MAX PLANCK INSTITUTE FOR HUMAN COGNITIVE AND BRAIN SCIENCES LEIPZIG

Haakon G. Engen: On the endogenous generation of emotion. Leipzig: Max Planck Institute for Human Cognitive and Brain Sciences, 2017 (MPI Series in Human Cognitive and Brain Sciences; 183)

On the Endogenous Generation of Emotion

Impressum

Max Planck Institute for Human Cognitive and Brain Sciences, 2017

Druck: Sächsisches Druck- und Verlagshaus Direct World, Dresden

Titelbild: © Edvard Munch: *Friedrich Nietzsche*, 1906, Detail

ISBN 978-3-941504-69-1

On the Endogenous Generation of Emotion

von der Fakultät für Biowissenschaften, Pharmazie und Psychologie

der Universität Leipzig

eingereichte

D I S S E R T A T I O N

zur Erlangung des akademischen Grades
doctor rerum naturalium
Dr. rer. nat

vorgelegt von
Cand. Psychol. Haakon G. Engen
Geboren am 4. August 1982 in Lørenskog, Norwegen

Leipzig, den 01.07.2016

Acknowledgements

Whatever merit this thesis might have, it is largely due to:

Professor Tania Singer, for her patience and support, and for giving me the opportunity and independence to develop myself as a researcher in my time as a graduate student.

Dr. Philipp Kanske, for his valuable feedback and steady voice of sanity in the development of paradigms, analyses, and manuscripts.

Dr. Jonny Smallwood, for inspiring me to think deeply about what makes self-generated mental contents special.

And, last, but absolutely not least, Sandra Zurborg, whose contributions to this thesis, to my sanity, and well-being are beyond words.

Contents

Preface

"Mit einer ungeheuren und stolzen Gelassenheit leben; immer jenseits—. Seine Affekte, sein Für und Wider willkürlich haben und nicht haben, sich auf sie herablassen, für Stunden; sich auf sie setzen, wie auf Pferde, oft wie auf Esel: — man muss nämlich ihre Dummheit so gut wie ihr Feuer zu nützen wissen."

Friedrich Nietzsche, *Jenseits von Gut und Böse*, 284

In the introduction to her excellent book exploring the place of emotion in contemporary moral philosophy, Martha Nussbaum (2008) describes emotions as forces that "[…] shape the landscape of our mental and social lives (p. I)", as "geological upheavals of thought (p. I)" that are "intelligent responses to the perception of value (p. II)". While Nussbaum's book is a great step forward in rehabilitating emotions as valuable contributors to rationality, it is notable that even she adopts a largely passive model of emotion. This view of emotions as states that come over us, that we are relatively passive "victims of", that shape our lives and inform us of the do's and don'ts of the world, has been the *leitmotif* for the intellectual exploration of emotion both historically and in modern times (Plamper, 2015). However, there is increasing evidence that this paradigm of passivity is, at least in some cases, mistaken. For one, we have a considerable degree of leeway in heeding the purported evolutionary wisdom of our emotions. Literally hundreds of studies have shown that the way we react to emotional events is, at least in part, dependent on how we choose to relate to them (see J. J. Gross, 2015a for a recent review). Moreover, evidence suggests that individuals not only *react* emotionally to events in the world, but seek out and use emotional reactions in an instrumental fashion in the service of goal achievement (Ford & Tamir, 2012; M. Y. Kim, Ford, Mauss, & Tamir, 2015; Tamir, Mitchell, & Gross, 2008). As such, it is increasingly apparent that we are not merely the recipients of the wisdom of emotions, but stand in an active dialectic relationship with them. Importantly, evidence is increasingly showing that flexibility and fluency in choosing what emotional states to experience might be a key factor

not only in mental health, but in flourishing (Bonanno & Burton, 2013; Fredrickson, 2013; Kashdan & Rottenberg, 2010).

The current thesis investigates one important means by which one can influence what emotional states one experiences, namely the self-generation of emotional states based on endogenous sources of information. Such states range from the melancholia of reminiscence to the anticipation of future joys, and constitute important parts of our emotional lives (Solomon, 2003). Importantly, while such endogenously generated emotion can in some cases be sources of anguish, as seen in some pathological cases (Brewin, Gregory, Lipton, & Burgess, 2010; Cooney, Joormann, Eugène, Dennis, & Gotlib, 2010), they can also be elicited in a volitional fashion with relative ease: By guiding one's thoughts to a cherished (or maligned) emotionally significant situation (past, future, or hypothetical), one can (under ideal circumstances) cause oneself to experience strong and vivid emotional experiences (Salas, Radovic, & Turnbull, 2012). Such endogenously generated emotions afford us a means to self-induce and experience emotional states independently of the external world, and, potentially actively utilise emotion in the service of self-regulation (Fredrickson, 2013).

Despite the ubiquity of endogenous emotions, comparatively little research has focused on how they come to be, much less how they can actively be used for self-regulation. The current thesis aims to rectify this by investigating 1) the neural and behavioural means by which endogenous emotion is generated, and 2) how the capacity to generate such emotion can be used in an active way to deal with external emotional stressors.

The investigation of how endogenous emotion generation occurs took two complimentary approaches: First, using functional magnetic resonance imaging (fMRI) in two large (N = 32/293) representative samples, the neural and behavioural foundations of endogenous emotion generation in the normal population was investigated. This was done using a newly developed paradigm that allowed naturalistic and nuanced measurement of optimal emotion generation. This was complemented by investigations of the structural and

functional neural signatures of endogenous emotion generation in a sample of long-term meditation practitioners. These practitioners had extensive experience in practices that centrally involve the active generation of the positive emotional states of loving-kindness and compassion. Thus, they constituted an expert population that enabled the investigation of structural and functional neural changes associated with extensive experience in the endogenous generation of emotion. Together, these approaches allowed the triangulation of the neural component process architecture supporting the volitional endogenous generation of emotion.

Similarly, the question of how endogenous emotion generation can be used for emotional self-regulation was also investigated in both normal and expert populations. It has been proposed that endogenous emotion generation has special utility for self-regulation by allowing the individual to buffer against negative stressors by self-inducing positive emotion. By investigating the neural correlates of using compassion meditation to regulate emotional reactions to negative stressors, the neural mechanisms and behavioural consequences of buffering against negative emotion could be investigated. Next, by investigating how individual differences in the ability to generate emotions was related to emotion regulation and coping styles in the normal population, it could be ascertained whether buffering, or other modes of emotion management, was associated with efficacy at self-generation of emotion. Thus, taken together, these findings provide an insight into the mechanisms by which endogenous emotion generation can be used for emotional self-regulation.

In the following an overview of the thesis is given. In Part I, the theoretical and empirical background for the dissertation is presented. In Chapter 1, the theoretical background for the current set of studies is described, discussing the historical conception of emotion as passive states of mind, and how a significant counter-current in the continental, existentialist philosophical tradition has argued for a more active conceptualisation of

emotion. Chapter 2 follows this up with a review of the extant behavioural and neuroscience literature on how endogenous emotion is generated, culminating in a working model of the component process architecture of endogenous emotion generation that guided the current work. The chapter also discusses empirical work suggesting a role for endogenous emotion generation in coping and emotion regulation, culminating in the theoretical account that guided the current investigations, identifying the potential mechanisms by which endogenous emotion generation can be used in the service of emotional self-regulation. This is followed by Part II, in which the empirical investigations that are the heart of the current thesis is presented. These studies are either published (H. G. Engen & Singer, 2015) or under review in peer-reviewed journals at the time of writing, and the current thesis reproduces these as submitted. As the paper format affords limited space for the discussion of methodological details, Chapter 3 prefaces the empirical studies with a detailed discussion of the experimental and analytical methods used, including an account of the development of a novel paradigm used to investigate endogenous emotion generation skills in a naturalistic fashion. Chapter 4 presents the results from this paradigm in a study that sought to establish the component process neural architecture of emotion generation in a large, representative, population. Additionally, the study sought to distinguish general neural mechanisms from those supporting specific implementations of emotion generation, such as generating specific states with specific valences, or containing specific information modalities. Chapter 5 sought to validate the candidate neural architecture described in Chapter 4 by investigating the functional neural correlates of loving-kindness meditation (a technique centrally involving the generation of positive emotion), and how expertise in this technique is reflected in morphological changes in cortical thickness. In the same sample of long-term meditators, Chapter 6 reports the behavioural and neural effects of using compassion meditation to regulate emotional reactions to negative stressors, and how this compares to reappraisal; a "gold-standard" emotion regulation strategy (Buhle et al., 2014; McRae, Ciesielski, & Gross,

2012a) that involves generating cognitive interpretations to change the emotional meaning of negative stressors. Thus, Chapter 6 investigates the neural mechanisms supporting active efforts to regulate responses to negative external stressors using endogenous emotion generation, and differentiates this from cognitive forms of emotion regulation. Finally, Chapter 7 investigates whether and how emotion generation skills relate to adaptive emotion management styles and trait tendency to experience positive or negative emotion in a subset (n = 288) of the sample investigated in Chapter 4. Part III summarises the current studies and discusses limitations, implications and future directions for this research. Chapter 8 provides an integrative discussion of the current findings relative to the working models proposed in Chapter 2, while Chapter 9 discusses the implications for our fundamental understanding of emotion and emotion regulation, as well as limitations of the current work and questions and directions for future work hinted at by the present findings.

Part I: Theoretical and empirical foundation

Chapter 1: Emotion as action: Philosophical background

1.1. Chapter overview

In this chapter, the philosophical and historical background for the current thesis is presented. Emotion has traditionally been thought of as being an adversary of rationality and consequently either being an impediment to self-regulation or something that itself needs to be regulated. There exists, however, a significant counter-current to this line of thought, rooted in the continental tradition of philosophy. This line of thought states that emotions are not mere reactions, but constitute strategies by which we adapt to the external world, suggesting they can play an important role in self-regulation. Importantly, it has been argued that emotion stemming from endogenous sources can be flexibly used in the service of goal achievement, and even counteract emotional reactions imparted on us by the external world.

1.2. The adversary of rationality: Emotions in the history of philosophy

"The heart has its reasons of which reason knows nothing"

— Blaise Pascal

As a subject of philosophy, emotions bear the dubious distinction of being both a perennial topic of inquiry and being almost universally dismissed as being essentially opposed to rational thinking. Frequently described as reactive and irrational, the emotions are traditionally thought of as harbingers of discord, perturbers of the soul and the enemies of harmony on both societal and individual levels (Nussbaum, 2008; Plamper, 2015). As such, it is perhaps not surprising that emotions have been a central topic of Western philosophy as far back as the pre-Socratics, perhaps most notably in the ethical teachings of the Stoics (Graver, 2009). In large part, these teachings focus on how to use rationality to reign in, or control

one's emotional reactions (D. M. Gross, 2007a). Typical to this intellectual tradition is the assumption that emotions are occurrences over which we have no influence. This understanding has been highly influential in forming the modern understanding of emotions as more or less adaptive, evolutionarily imparted reactions to significant stimuli in the environment. For instance, as this model has it, exposure to events that signify threat to the organism is likely to elicit an emotional reaction of fear, concomitant with behaviours (e.g. freezing), evaluations (e.g. "this is a horrible thing happening") and physiological and cognitive reactions resulting in the organism being mobilised to deal with a given threat (J. J. Gross, Sheppes, & Urry, 2011; Ledoux, 1998). Importantly, these reactions are taken to occur in a reflexive manner, such that exposure to threat automatically elicits emotional behaviours, irrespective of what the goals of the individual might be. Thus, as our opening quote indicates, emotions are often thought as having the paradigmatic quality of occurring due to a logic — such as evolutionarily inherited survival concerns — that is inscrutable, impenetrable, and essentially uncontrollable to and by one's rational mind. Aside from the potentially deleterious effects acting in affect can have, it was this reflexivity that made emotions inimical to ancient thinkers, on whose account emotional reactions are, in a very real way, partially losing mastery over oneself and becoming subject to rules not one's own.

Perhaps understandably then, systems raised in opposition to emotion have largely focused on devising means by which self-governance can be upheld or regained through the application of rationality. Thus, Marcus Aurelius, a noted Stoic, advises that by "[g]et[ing] rid of the judgment; you are rid of the 'I am hurt'; get rid of the 'I am hurt,' you are rid of the hurt itself" (Meditations, III, 16). Thus, by consciously, effortfully, altering one's judgements through rational thoughts, one can mend oneself of irrational emotional reactions. Moreover, by cultivating thought and rational judgement, it is held that one can diffuse emotional reactions altogether, for, as Epictetus (another famous Stoic) maintained "[m]en are disturbed, not by things, but by the principles and notions which they form concerning things"

(*Enchiridion*, 5). Thus, by training ourselves to see things as they truly are (which is to say acceding only to the properties they have in themselves) we can avoid emotional reactions wholly. Importantly, even though emotion is here posited to be the consequence of thought, it is seen as a side-effect of faulty thinking. This leads us to impart importance to things and events that are, in fact, emotionally neutral. Thus, an optimally rational actor should not incur the vagaries of emotions and, in effect, be wholly in control of oneself in perfect equanimity.

It is difficult to overstate how influential the notion of equanimity as an ideal for self-control has been. The notion that control of emotions involves achieving a neutral, non-emotional, rational state of mind was endorsed not only by the Stoics, but ran through most ancient Western systems proposing a means to achieve *eudaimonion* (e.g. Cynicism, Epicureanism, Peripateticism, Platonism, Pyrrhonism), and is arguably one of the central pillars of enlightenment in Buddhism (Lama & Ekman, 2008). While there is increasing acknowledgement of the potential intelligence and benefits of emotions in affective science (Barrett, 2011; Damasio, 1994; Kashdan & Rottenberg, 2010; Nussbaum, 2008), this adversarial account of emotion is still highly influential and can be thought of as the standard model organising both philosophy and empirical research into both emotions, and emotion management (J. J. Gross, 2015b; 2015a; J. J. Gross & Barrett, 2011).

1.3. Emotions as ways of interacting with the world

There is, however, a significant philosophical counter-current arguing for emotions being active means of engaging with the world. Per this model, emotions should not (exclusively) be thought of as reactions that should be avoided or suppressed, but occur in the context of, and, importantly, as an expression of our goals and intentions. On such an account, emotion is not something that needs to be controlled by our rational faculties of thought, but rather are extensions and expressions of rationality. Importantly, if we take this model seriously, emotion control involves more than merely suppressing or negating the occurrence

of emotional reactions, but rather selecting when and what emotional experiences one has at any given time – i.e. that emotions can be actively recruited and employed to further self-governance. In the following sections, three such accounts will be presented, as proposed by Descartes, Sartre, and Solomon, with the aim of demonstrating the means and mechanisms by which emotions are not something that (exclusively) happens to us, but entities we actively employ to self-regulate.

1.3.1. Cartesian emotion regulation

"To arouse boldness and suppress fear in ourselves, it's not enough to have a volition to do so. We have to set ourselves to think about the reasons, objects, or precedents which argue that the danger isn't great, that there's always more security in defence than in flight, that we'll gain glory and joy if we conquer, and nothing but regret and shame if we flee—things like that. Our passions can't be aroused or suppressed directly by the action of our will, but only indirectly by our representing to ourselves things that are usually joined with the passion we want to have or opposed to the one we want to fend off."

Rene Descartes, *Passions IV,* 45

In addition to being one of the great philosophers of mind and epistemology, Descartes formulated a comprehensive account of "the passions" or what we now recognise as emotions. Largely due to being perceived as espousing a strong dualism between emotion and cognition, Descartes has been the butt of much criticism in recent affective science. In particular, he has been read as stating that cognition can, and should, be in charge of emotional responses, and as such has been taken as being one of the strongest proponents of the dualistic adversarial account of emotion and cognition, as discussed above. Evaluating this charge is beyond the scope of the current treatment (see e.g. Damasio, 1994; D. M. Gross, 2007a), but it is interesting to note that the Cartesian account of how one can go about controlling ones emotions (as quoted above) appears not to follow an adversarial account, at least in as much as it refuses the possibility of volitionally influencing the course of one's emotions by mere cognition. Rather, in the above quote, Descartes proposes that emotions can

only be controlled by the counter-generation of emotions through creating internal representations, and that this can be done either in goal achievement or to counter ongoing emotions. While thoroughly supporting the notion that emotions are reflexive and following a specific trajectory, an implication of this view is that emotions can be used to achieve goals by using imagination to achieve what in modern parlance would be called an embodied representation of a given emotion. Following on this, Descartes makes apparent a distinction between exogenous emotions, that we can fend off, and endogenous emotions, that are, more or less, something we can control. This opens the intriguing possibility that a comprehensive account of emotion control needs to include the capacity to strategically self-generate emotional states both to motivate behaviour and to deal with emotional reactions to the external world.

1.3.2. No exits, no excuses: Sartre on our responsibility for emotion

"The existentialist does not believe in the power of passion. He will never regard a grand passion as a destructive torrent upon which a man is swept into uncertain actions as by fate, and which, therefore is an excuse for them. He thinks that man is responsible for his passion."

Jean-Paul Sartre, *Existentialism Is a Humanism*

Jean-Paul Sartre can be thought of as having followed up this Cartesian model by extending it to all emotion, including emotions caused by happenings in the external world (Sartre, 1939). Rather than thinking of emotion as something that happens to us, Sartre claimed that we are essentially responsible for our emotional reactions. Indeed, rather than calling them reactions, on Sartre's view, emotions are strategies we (consciously or non-consciously) use to deal with the world (see also Frijda, 2007). Essentially arguing against the view that emotions simply happen to us, Sartre fundamentally changes the status of the emoter (possibly exempting pathology) to someone that is responsible for their emotional state. The crux of Sartre's argument is that a view of emotions as passive is merely a vehicle

for the "bad faith" (*mauvaise foi*) of referring to our emotional reactions as post-hoc excuses for behaviours that at a later point in time turn out to be undesirable. As such, Sartre argues that all emotion occurs in the service of goal-achievement — in his view essentially ego-defence. Thus, while Sartre's emotions are endogenously caused, the precise strategic end might be camouflaged from our conscious recognition. As such, the Sartrean view argues strongly that all emotion has endogenous causes. This is similar to the view that judgments determine emotion, but is taken further by emphasising that emotions are actions, that they in effect stem from our goals and thus should not be thought of as mere reactions to the external world.

1.3.3. Robert Solomon on emotion as action

"Our emotions do not render us passive but the very opposite; they are sometimes the engine of our behaviour and the actively chosen motivation of meaningful action."
Robert Solomon*, On the Passivity of the Passions*

Following on Sartre, Robert Solomon expanded on the notion of responsibility to change the ontological status of emotions from happenings to actions, in as much as they are constituted by, and occur as consequence, of a series of volitional acts. Key to his argument is the observation that, at least in humans, emotions are extended processes rather than the circumscribed impulsive responses they are often portrayed as in philosophy and modern research. Indeed, in many cases emotions are in fact something that we actively pursue and cause to be in ourselves, and as such can only be thought of as volitional objects. Mirroring Descartes, he highlights that what he calls the "emergency paradigm of emotion" cannot account for the frequent occurrence of emotions that can only said to be products of our own cognitive processes. Thus we can, as Solomon suggests in striking verisimilitude to Descartes, "nurture" emotions by both rational argument (e.g. making oneself angry by "convincing" oneself that one has been slighted) and controlled and guided access of memories (e.g. at a funeral service, Solomon 2001, p. 202). Indeed, as he points out, the very distinction

frequently drawn between having an emotion and thinking about that emotion is problematic, in as much as thinking about the justifications for one's anger is part and parcel of that emotion and thus tends to elicit the very emotional reaction under deliberation. Supporting this point, a large part of both modern clinical practice and traditional philosophical remedies for emotion (e.g. Stoicism and certain Buddhist practices) aims at fostering this meta-cognitive distance, with the ultimate aim of enabling the individual to not only understand why they are experiencing emotions, but also to decide what emotions they can experience. Importantly, this line of reasoning suggests that emotions are not (just) reactions, but should also be thought of as states that we actively instil in ourselves to adapt to one`s context.

1.3.4. Summary: Endogenous emotion as a means of self-regulation

These three accounts together point to something that has largely been neglected in modern psychology: That emotions are not (exclusively) something that happen to us, but rather reactions that we bear responsibility for, that we possess some degree of control over, and that we actively use to regulate ourselves and our environments. Of special note are emotions stemming from endogenous sources, such as thoughts or memories, both because we are relatively better able to control their occurrence, but also because, as Descartes pointed out, we can actively use these to counter emotions stemming from external sources. Moreover, as Sartre and Solomon point out, such endogenous emotion can be used both to motivate oneself in pursuit of long-term goals, and can be strategies with which one adapts oneself to the world, and also a means of altering the external (particularly social) world. This raises the intriguing possibility that the capacity to self-generate emotional states based on endogenous sources of information might serve as tool for self-regulation.

1.4. Harnessing fire: Endogenous emotion generation as a means of emotional self-regulation

Following on this way of thinking about emotion, the core proposition investigated in this thesis is that emotions are not simply something that occurs to us, but something that we enact and use to govern ourselves. Following Descartes, I specifically propose that emotions can be actively used to control one's emotional milieu via the expedient of generating emotions based on endogenous sources of information, such as our deliberations or memories. Moreover, I propose that this ability is a central aspect of self-regulation enabling one to deal with emotion both through increased fluency with emotional situations through simulation and also through the active use of emotion generation to deal with emotional stressors.

The goal of the current thesis was to empirically investigate these hypotheses, using a combination of neuroimaging, psychophysiological and psychological methods. As endogenous emotion generation has received relatively little attention as a topic in its own right, the thesis has two main topics: First, it aims to develop and test a model of the neural and psychological component processes that support endogenous emotion generation. Second, based on this model, it seeks to explore how endogenous emotion generation skills can be utilised as a means of emotion regulation and coping. Preceding the empirical portion of the thesis (Chapters 4-7), the following chapter provides an overview of the current state of knowledge regarding the endogenous generation of emotion in psychology and affective neuroscience and develops the working behavioural and neural working model of endogenous emotion generation that guided the present research.

Chapter 2: Endogenous generation of emotion

2.1. Chapter overview

This chapter presents an overview of the current state of knowledge on the behavioural and neural bases of endogenous emotion generation. Starting with a discussion of how to define and differentiate endogenous emotion generation from other related affective phenomena, the chapter reviews extant behavioural and neuroimaging work investigating how endogenous emotions are generated and what subjective, behavioural and physiological effects this has. This review culminates in a working neural component process model of endogenous emotion generation that guided the current research. Based on this model, the chapter next discusses how the self-generation of emotional states can enable emotional self-management, proposing two distinct routes by which the endogenous generation of emotional states can be used for self-regulation. The chapter ends with a presentation of the hypotheses that guided the current work, and an overview of the empirical part of the thesis.

2.2. Conceptualising endogenous emotion generation

Unlike the generation of emotional reactions to exogenous cues, comparatively little work has focused on understanding endogenous emotion in their own right (Wilson-Mendenhall, Barrett, & Barsalou, 2013). To my knowledge, there has hitherto been no attempts at integrating knowledge of how emotion is generated from endogenous sources of information into a mechanistic framework. The current section reviews behavioural, psychophysiological and neuroimaging evidence on self-generated emotional states, with the aim of developing a neural component process model of endogenous generation of emotion (EnGE). Starting off, the following provides the working operationalisation of endogenous emotion that guided this model development and how EnGE can be differentiated from other, related, topics.

2.2.1. Operationalisation and definitions

To guide the following literature review and model development, I will here adopt an operationalisation of endogenously generated emotions as emotional states whose causes can be traced to an internal representation. I will refer to the process by which such emotional states come about as endogenous generation of emotion (EnGE; contrasted with exogenous generation of emotion (ExGE)). Examples of EnGE are when an emotion occurs due to a train of thought, memories, imagery or bodily interoception. An important aspect of endogenously generated emotions (and internal representations in general), is that these can be elicited both voluntarily and spontaneously. Thus, emotional states can be elicited by, for instance, goal-directed recall of a given emotion-eliciting event in the past or future (Benoit, Szpunar, & Schacter, 2014; Fitzgerald et al., 2004), or through intrusive thoughts and rumination (Brewin et al., 2010; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), or simply in the course of ordinary mind-wandering (Ruby, Smallwood, Engen, & Singer, 2013). Here I will focus on the voluntary component of EnGE in line with the goal of the current work of investigating its potential utility as a means of self-regulation, and because investigations of spontaneous EnGE is still in its infancy (but see Poerio, Totterdell, Emerson, & Miles, 2015b; Ruby et al., 2013). While this could be seen as a limitation of the current investigation it should be noted that evidence suggests considerable overlap between the psychological and neural mechanisms and effects of voluntary and involuntary generation of mental content (Andrews-Hanna, 2012; Smallwood & Schooler, 2015; Smallwood et al., 2013). Thus, it is likely that the conclusion drawn from the study of voluntary EnGE generalises to spontaneous EnGE, at least in terms of psychological and neural mechanisms.

Practically, the review is therefore limited to studies of self-induced emotion that relied either on non-emotional or minimally emotional stimuli, and therefore can be said to arise following processing of information endogenous to the individual. Moreover, given the objective of devising a general model of EnGE, the review focuses on investigations of non-

pathological populations. Before proceeding, the following section discusses the differentiation of EnGE as here conceptualised and the related topics of exogenous emotion generation, emotion regulation, mood generation, and constructive memory.

2.2.2. Differentiating endogenous emotion generation from related phenomena

2.2.2.1. Exogenous emotion generation

Based on the preceding operationalization, the relationship between exogenous (ExGE) and endogenous generation of emotion (EnGE) can now be discussed. On the surface, drawing this distinction is relatively simple, with endogenous emotions having causes internal to the individual, such as thoughts, memories, bodily sensations, or imagery. This contrasts with exogenously generated emotions, who in turn have a clear causal referent in the external world, such as an emotion-provoking situation or stimulus. Following on this, individual differences in ExGE tend to revolve around how one responds to external stimuli (Hamann, 2004). This is of some importance, because it is probable that this is at best partially overlapping with the likely sources of individual differences in EnGE, which also involves variation in the ability to recall emotional information (Gollnisch & Averill, 1993; G. A. Miller et al., 1987) or the capacity to generate mental imagery (G. A. Miller et al., 1987; Zeman, Dewar, & Sala, 2015). Moreover, at least in the volitional case, EnGE abilities are also likely to be affected by individual differences in the ability to control such as mnemonic or ideational processes (Banich et al., 2009). Thus, while endogenous emotions can occur with seeming spontaneity, they are (excepting pathology) in some way dependent on cognitive processes that involve the directed generation of emotionally relevant narratives leading to autonoetic simulations of emotional states of affairs (Niedenthal, Winkielman, Mondillon, & Vermeulen, 2009; Wilson-Mendenhall et al., 2013)

That said, the distinction between exogenously and endogenously generated emotions is fluid. For instance, in cases of severe anxiety, endogenous processes are arguably responsible for the generation and maintenance of the emotional state despite it having an external referent (Cha et al., 2016). On the other hand, external stimuli can sometimes entrain internal emotion generation processes, such as when reading a story with emotional qualities, where stimulus properties are not evocative of emotion outside of the narrative context it appears in. Moreover, recent meta-analyses of neuroimaging studies of emotion (Kober et al., 2008; K. A. Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012), suggest that even processing of external stimuli to a large degree involves neural mechanisms supporting the generation of such simulations (Hassabis & Maguire, 2007; Schacter & Addis, 2007). Thus, drawing a strict conceptual boundary between ExGE and EnGE might be impossible, requiring empirical delineation in terms of underlying processing.

2.2.2.2. Emotion regulation

Emotion regulation can be defined as a process in which on expends efforts to influence which emotions one has, the intensity of these emotions and/or how they are expressed (J. J. Gross, 2007b). Distinguishing emotion generation from emotion regulation is an important topic in affective science (J. J. Gross et al., 2011), that is heavily influenced by the fundamental theory of emotions one holds (J. J. Gross & Barrett, 2011). For instance, a basic emotion theorist can distinguish between the two with relative ease on account of having a stimulus-reaction model of emotion, meaning that emotion regulation simply involves managing the behavioural consequences of a given emotional reaction. Conversely, on a constructivist account, both emotion generation and emotion regulation involve changes in the situation-by-organism meaning-making interaction (Barrett, 2014). Thus, separating the two becomes conceptually difficult as any act of emotion regulation can be construed of as an act generating a new emotional state (J. J. Gross et al., 2011). However, in the case of

exogenous emotion, one can, at least in principle, make a distinction between a) processes that are initiated by the occurrence of a given event or stimulus and b) those that act to modulate the trajectory of these processes (J. J. Gross et al., 2011). For endogenous emotion, no similar appeal to ontogeny can be made to differentiate events of generation from events of regulation. Rather, generation and regulation likely depend on overlapping processes that are essentially internal to the emoter. Moreover, as will be discussed below, it is possible that EnGE can at times be used as a means to regulate emotional reactions. Providing a comprehensive resolution to this topic is beyond the scope of the current investigation, and I therefore take a functional approach to distinguish the two. Specifically, I propose that emotion generation occurs when an emotional feeling state is *de novo*, where previously there, subjectively, was none. Conversely, emotion regulation occurs when ongoing subjectively experienced emotional states are somehow modulated, intentionally or otherwise. Thus, distinguishing the two is on this, albeit simplified, account, a matter of causality and time. However, this should not be construed of as a strict ontological divide as they are likely reliant on similar neural and psychological mechanisms, especially those supporting the construction of mental representations (Kober et al., 2008; K. A. Lindquist et al., 2012; K. A. Lindquist & Barrett, 2012).

2.2.2.3: Mood generation

Endogenous emotion also needs to be differentiated from the closely related construct of mood states. The usual means of differentiating mood and emotion is by stating that emotions are phasic responses to events, whereas mood states represent the tonic level of affect. Moreover, moods are usually thought of as being more diffuse in terms of their expression and duration, usually resulting in lower intensity “background” affective experiences that can last for a protracted period of time (R. J. Larsen, 2000). Another important difference is that moods usually are thought of as having no clear eliciting cause in

the environment (Magen & Gross, 2010). Together, these criteria make it fairly straight forward to differentiate moods from (exogenous) emotional reactions.

In the case of endogenous emotion, however, this distinction is blurred. For one, while endogenous emotion can be thought of as phasic responses to internal processes, these processes themselves are likely extended in time, and embedded in the ongoing stream of consciousness (R. J. Larsen, 2000; Ruby et al., 2013). This means that the initiation and duration of endogenous emotional events are difficult to specify. Second, like moods, endogenous emotions are, by definition, not (directly) resultant on the perception of external events (R. J. Larsen, 2000). Thus, in one sense endogenous emotions are more similar moods than exogenous emotions. Unlike moods, however, endogenous emotions, as we here define them, are clearly consequent on a specific internal representation, and, crucially, on the maintenance of this representation at the focus of attention. Thus, it is possible to generate a positive emotional state by thinking about good times past, despite being, at the moment, dysphoric (e.g. Holmes, Lang, & Shah, 2009; Holmes, Mathews, Dalgleish, & Mackintosh, 2006; Pictet, Coughtrey, Mathews, & Holmes, 2011). Moreover, while such EnGE might provide alleviation of a mood (as suggested by Descartes in Chapter 1), this is effectively an act of regulation as it is here defined, such that endogenous emotion overrides the mood, possibly temporarily, with the mood state reinstating itself once active EnGE efforts cease. Summarising, endogenous emotion can be thought of as occupying a middle ground between exogenous emotional reactions and mood states that are clear reactions to endogenous events, but whose effects are not pervasive outside of the context in which the reaction was elicited.

2.2.2.4: Constructive memory

At the core, all psychological processes involving the internal generation of representations are in some way reliant on constructive memory processes (Hassabis & Maguire, 2007). Ranging from planning how to achieve one's future goals to reminiscing

about the distant past, similar psychological and neural mechanisms seem to be at the core of our abilities to generate simulations of the world (Addis, Pan, Vu, Laiser, & Schacter, 2009; Schacter & Addis, 2007; Schacter et al., 2012; Spreng & Grady, 2010). As mentioned above, EnGE is likely to rely heavily on the ability to create such lifelike, autonoetic, first-person simulations, and is therefore likely to rely on constructive memory processes. One crucial difference, however, is that there is no inherent requirement for constructive memory to be embodied, i.e. that the simulations should result in a manifest physiological equivalence between the simulated state and the current state of the body. As the term suggests, simulation involves a distinction between the internal model of the world and the actual constitution of the world. Thus, there is inherently an analogous, or "as if" quality to these simulations. This does not necessarily pertain to EnGE which, both by definition and in terms of subjective experience, results in genuine, experienced emotional states (Salas et al., 2012). This is not to say that EnGE cannot be used for similar purposes as constructive memory, such as planning or mentally working through problems (past or future) that are pertinent to one's goals, but that the product of the EnGE process is an experienced and embodied emotional state and not merely a cognitive or mnemonic representation of that state. As such, the notion of EnGE adopted here is related to the notion of embodied simulation frequently used in the literature on motor imagery (Gallese & Caruana, 2016).

2.3. Characterising EnGE as a psychological phenomenon

In this section, extant behavioural and psychophysiological investigations of EnGE are reviewed, with the aim of developing a characterisation of it as an empirical phenomenon and identifying the key features an account of EnGE must contain.

Much of our knowledge about EnGE comes from studies utilising endogenous emotion generation as a means to induce emotion. Typically, this type of experiment involves participants being asked to generate emotional states either purely endogenously, by

remembering past events or by imagining hypothetical scenarios (Damasio et al., 2000; Gemar, Kapur, Segal, Brown, & Houle, 1996; George, Ketter, Parekh, & Herscovitch, 1996; Holmes et al., 2006; Holmes & Mathews, 2005; Kimbrell et al., 1999; Liotti et al., 2000; Mayberg et al., 1999; Morina, Deeprose, Pusowski, Schmid, & Holmes, 2011; Pardo & Raichle, 1993; Salas et al., 2012; Wilson-Mendenhall et al., 2013), or by being asked to up-regulate emotional aspects of externally presented emotional stimuli (e.g. McRae, Misra, Prasad, Pereira, & Gross, 2012c; Ochsner et al., 2009b; Otto, Misra, Prasad, & McRae, 2014). As the latter approach is closer to the current definition of emotion regulation (cf. the distinction between generation and regulation drawn above), and because relatively few studies have used this methodology, the following will focus on studies where elicitation of emotional states relied minimally on stimulus properties.

Overall, this literature suggests that EnGE-based emotion induction is highly effective and result in strongly experienced emotional states, particularly for positive emotions (Riquelme, Radovic, Castro, & Turnbull, 2015; Salas et al., 2012). Using this self-induction approach, successful EnGE has been reported using mental imagery (Holmes & Mathews, 2005; 2010), narrative scripts of both impersonal (Wilson-Mendenhall et al., 2013) and personal varieties (Salas et al., 2012), semantic narrative analysis (Holmes et al., 2006; Vrana, Cuthbert, & Lang, 1986), and auditory imagery (Beaty et al., 2013; Williamson et al., 2012). Interestingly, it appears that both autobiographical recall and immersion in hypothetical emotional events result in emotional reactions. This suggests that EnGE should not be conceptualised as simple recall of a previously experienced emotional state, but rather a simulation of a situation in which a given target emotion might occur, akin to general constructive memory abilities like episodic recall and prospection (Schacter & Addis, 2007). However, evidence suggest that this type of simulation goes beyond mere subjective appraisal and involves the activation of physiological markers similar to exogenously generated emotions. Philippot, Chapelle, and Blairy (2002) demonstrated this in an experiment where

they investigated the relationship between respiration rate and subjective experience of emotion. Participants were instructed to put themselves into different emotional states (joy, sadness, anger, and fear) by altering their respiration patterns, but were given no instruction as to what pattern of respiration might be associated with each emotional state. In addition to having marked subjective effects, this resulted in participants adopting respiration patterns matching those seen in previous studies of emotional arousal, and that were differentiable by type of emotion in question. In a second experiment, participants were surreptitiously induced to engage in these patterns of breathing. This resulted in increased subjective experience of the emotions associated with a given breathing pattern. Thus, bodily signals of emotion are not only concomitants of self-induced emotional states based on endogenous information (see also Fawver, Hass, Park, & Janelle, 2014; Kleinke, Peterson, & Rutledge, 1998; G. A. Miller et al., 1987), but can in themselves cause emotional states to come into being. This further means that a comprehensive account of EnGE should include means by which physiological signals can be used to generate emotion, either alone or in combination with other modalities (Gallese & Caruana, 2016; Niedenthal et al., 2009; Seth, 2013). Finally, in addition to being embodied, EnGE appears to have a clear influence on behaviour, such that, for instance, self-generated emotional states can affect sport performance (Rathschlag & Memmert, 2014), or even impact on baseic motility, as is seen in the impact of EnGE on gait (Fawver et al., 2014).

Summarising, evidence demonstrates that EnGE should not be thought of as being a "cold" cognitive simulation of the facts and appraisals that surround a given emotion. Rather EnGE is an embodied phenomenon, resulting in emotional reactions with clear subjective and physiological outcomes. Thus, an account of EnGE needs to detail not only how emotional information is recalled, but also how this leads to emotional physiological reactions.

2.3.1. Individual differences in EnGE abilities

Another aspect of EnGE that has been revealed in the self-induction literature is that individuals differ in their ability to self-generate emotions. Miller and colleagues (1987) investigated trait and behavioural markers of the capacity to generate emotional imagery. Participants with good imagery abilities generated stronger and more differentiated physiological signatures of emotional arousal when requested to envisage different emotional situations using the emotional script procedure. Conversely, participants self-describing as bad imagers did not show this strong coupling between emotion scripts and physiological responses. Critically, there were no apparent differences between the good imagers and people self-described as poor imagers on several personality and cognitive variables. Thus, a concomitant of good emotional imagery ability appears to be the ability to generate full-fledged emotional reactions.

One caveat to this is that the Miller study, like most other self-induction studies, only investigated individual differences in the capacity to use mental imagery to generate emotion. While there is extensive evidence that mental imagery and emotion might have a preferential relationship (Holmes & Mathews, 2005; 2010), to date no evidence exists mapping whether EnGE abilities are predicated exclusively on the capacity to engage in mental imagery. Conceivably, in the normal population, people can differ both in efficacy and implementation of EnGE, with some individuals, for instance, relying on musical imagery, semantic analysis or bodily interoception to instigate EnGE. Indeed, at least in cases of pathology, semantic analysis in the form of verbal rumination appears to become the predominant mode of EnGE employed (Koster, De Lissnyder, Derakshan, & De Raedt, 2011), which could stem from decreased efficacy of EnGE based on visual imagery (Raune, MacLeod, & Holmes, 2005; Stöber, 1998). Similarly, while evidence exists to suggest that mental imagery outperforms verbal semantic analysis in terms of both short term and long term self-induction effects (Holmes et al., 2006; Holmes & Mathews, 2005; Pictet et al., 2011; Vrana et al., 1986), it is

not clear whether, for instance, combining multiple modalities of generation is more (or less) effective at generating different emotional states. These are important questions, because they can provide information about the core mechanisms of EnGE and because they would provide guidance for how to optimise and individually adapt interventions aimed at training this skill – the topic of our next section.

2.3.2. Training EnGE

An important aspect of the Miller (1987) study mentioned above is that even poor imagers got better with repeated testing, as indicated by both enhanced subjective emotional experience and better correspondence between subjective and physiological responses. Thus, it appears that the capacity to self-generate emotions can be trained, at least in the case of EnGE using mental imagery. More evidence for this comes from recent work by Emily Holmes and colleagues (Holmes et al., 2006; 2009; Pictet et al., 2011) showing that EnGE can be trained, and that such training has carry-over effects on both affective and cognitive measures. Importantly, Holmes and colleagues have recently shown that training positive imagery can provide lasting improvement for symptoms of depression (Blackwell & Holmes, 2010; Holmes et al., 2009; T. J. Lang, Blackwell, Harmer, Davison, & Holmes, 2011).

Another line of evidence pertaining to the trainability of EnGE stems from research into the effects of loving-kindness (LKM) and compassion (CM) meditation. These techniques centrally involve training through meditation in self-instillation of a positive emotional state of benevolence, warmth, concern and motivation to help others (Fredrickson, 2013; Goetz, Keltner, & Simon-Thomas, 2010; Salzberg, 2001; Singer & Klimecki, 2014). Recently, a number of training studies have shown that even short-term training of LKM and CM is efficacious at increasing experienced positive affect both in the lab and in daily life (Zeng, Chiu, Wang, Oei, & Leung, 2015), as well as being associated with improvements on psychophysiological (Kok et al., 2013) and neural (Klimecki, Leiberg, Lamm, & Singer,

2013; Klimecki, Leiberg, Ricard, & Singer, 2014) markers of resilience. Central to these meditation practices is training how to engage in vivid mental imagery, the recitation of verbal mantras and increasing interoceptive awareness of the psychosomatic sensations (particularly warmth) associated with the emotional states of loving-kindness and compassion (Fredrickson, Cohn, Coffey, Pek, & Finkel, 2008; Jazaieri et al., 2012; Klimecki et al., 2013; 2014; Leiberg, Klimecki, & Singer, 2011). Thus, LKM and CM training is a real-life example of a means by which EnGE can be trained, and also that such training can have marked benefits in terms of mental health (Fredrickson, 2013; Holmes et al., 2006; 2009).

2.3.3. Summary: Conceptualising EnGE

Summarising the extant behavioural investigations, it appears that EnGE is both something that individuals are capable of with relative ease, and that tends to elicit subjectively (Salas et al., 2012) and physiologically (G. A. Miller et al., 1987; Philippot et al., 2002) potent emotional states. This demonstrates the supposition that EnGE should be thought of as generating *de facto* embodied emotional states. Further, evidence suggests that EnGE can be implemented using a variety of different information modalities, ranging from episodic imagery to interoception. While it appears that episodic imagery of emotion might be most effective as a generation technique (Bergman & Craske, 2000; Holmes et al., 2006), evidence also supports the efficacy of other modalities. Moreover, there appears to be considerable individual differences in EnGE, suggesting that individuals might vary in how they tend to implement EnGE, and how efficacious this implementation is.

As such, the extant behavioural and psychophysiological evidence suggests EnGE results in emotional states with clear physiological and subjective concomitants. Therefore, a complete account of EnGE as a phenomenon needs to include explanations of how both these aspects are generated. Moreover, evidence that EnGE can be implemented based on a variety

of different information modalities, means that it needs to account for both modal and supramodal mechanisms of implementation.

2.4. The neural architecture of EnGE

In this section, we make the shift from discussing the psychological mechanisms of EnGE to reviewing extant work on how these mechanisms are neurally implemented. In the main, research on the neural basis of emotion have followed behavioural and psychophysiological work relying heavily on (primarily visual) stimuli to elicit emotions. Indeed, it has been estimated that only 6% of previous neuroimaging studies have provided information on EnGE, primarily in the form of mental imagery (Wilson-Mendenhall et al., 2013), meaning our understanding of EnGE is in part limited by a lack of research. This problem is aggravated by the many different protocols used to elicit emotions in these few

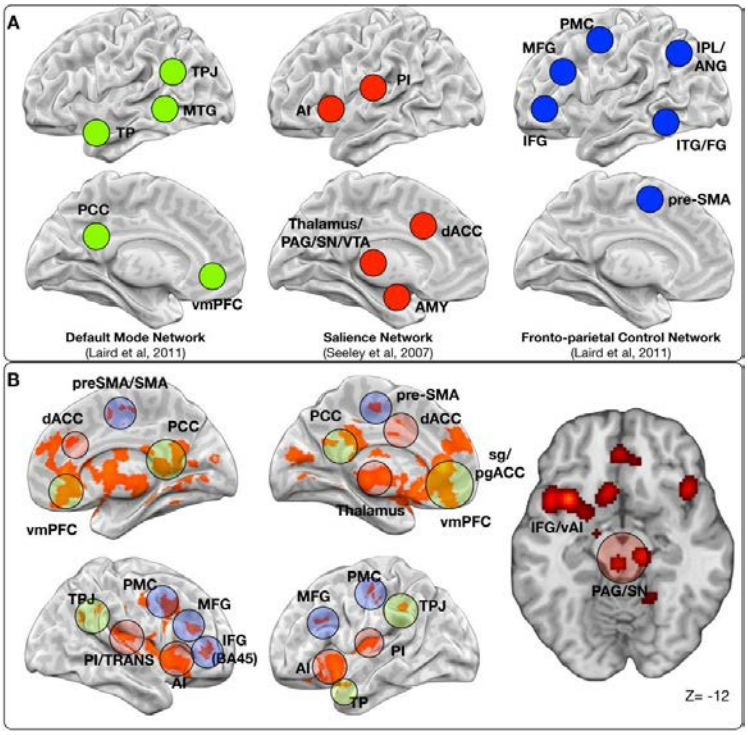


Figure 2.1: Neural networks of EnGE. A) Schematic of three large scale intrinsic connectivity networks (ICNs) (adapted from Laird et al., 2011; Seeley et al., 2007). **B)** Illustration of activations in the early PET studies made using GingerALE meta-analysis toolbox. NB: For illustration only as $p < .05$ uncorrected. Colour-coded circles denote overlaps between ICNs and meta-analytic findings.

studies, as the proximate goal of ensuring the uniform elicitation of strong emotional states has lead researchers to use different stimuli, such as visual or auditory cues, to guide the generation process. Aside from epistemic concerns of whether such paradigms can be said to rely on endogenous sources of information, the use of different information and instruction modalities makes it difficult to differentiate neural mechanisms supporting modality-specific implementation of EnGE from supramodal mechanisms supporting EnGE in general. This is a matter of some concern, since the majority of studies were performed on relatively small samples. Similarly, the goals of these studies have mainly been to investigate different neural effects of the outcomes of the generation process (i.e. of different emotional states), and have therefore not focused on identifying the neural mechanisms of emotion generation *per se*.

One notable exception to this heterogeneity is a series of early positron emission tomography (PET) studies (Damasio et al., 2000; Gemar et al., 1996; George et al., 1996; Kimbrell et al., 1999; Liotti et al., 2000; Mayberg et al., 1999; Pardo & Raichle, 1993; Reiman et al., 1997) that investigated the neural signatures of volitional autobiographical recall of significant emotional experiences. As these studies were all performed using the same imaging modality, with comparable experimental protocols and sample sizes, these studies are amenable to aggregation via meta-analysis. (However, the low cumulative sample sizes of these studies (N = 122) means that findings should be taken as illustration more than statistical fact.) When combined, these studies suggest EnGE is supported by at least three large scale neural networks (Figure 2.1):

1) The Default Mode Network (DMN; Buckner, Andrews-Hanna, & Schacter, 2008; Raichle & Snyder, 2007; green in Figure 2.1A) including medial prefrontal, posterior cingulate/precuneus, and left temporoparietal regions. This network is known to play an important task in different modes of constructive memory, requiring the internal generation of representations or simulations (Spreng, Mar, & Kim, 2009).

2) The extended Salience Network[1] (SN; Seeley et al., 2007; red in Figure 2.1A) including most prominently insula and dorsal cingulate regions, in addition to subcortical (amygdala and basal ganglia) and brain-stem regions like the substantia nigra, ventral tegmental area and periaqueductal grey, known to support affective processing of both reward and punishment (Berridge & Kringelbach, 2013; Buhle et al., 2013).

3) The Frontoparietal Control Network (FPCN; Brass, 2002; Cole et al., 2013; Laird et al., 2011; blue in Figure 2.1 A), including inferior and middle frontal gyri, and pre-supplemental motor area, as well as posterior parietal regions not seen here. This network is involved in a wide range of cognitive control tasks, and it has been suggested that it serves a domain-general task implementation network based on work showing that the FPCN flexibly couples to other networks to facilitate goal achievement (Cole et al., 2013; Spreng, Stevens, Chamberlain, Gilmore, & Schacter, 2010).

Consistent with this three-network structure, Harrison and colleagues (Harrison et al., 2008) reported an fMRI study investigating the difference in brain network connectivity during self-induction of sadness via autobiographical recall. Importantly, Harrison and colleagues used independent-component analysis of fMRI time-series, meaning that they could investigate modulations of connectivity within actual intrinsic connectivity networks. Of the five components thus identified, DMN, SN, and right and left FPCN all showed significantly increased functional connectivity during sad recall, suggestive of them being core functional networks in EnGE. Finally, recent meta-analyses of the neuroimaging

[1] The cortical extent of this network is also variably known as, or overlaps with, the ventral attention network (Vossel, Geng, & Fink, 2014), the paralimbic network (Harrison et al., 2008; Kober et al., 2008), or the cingulo-opercular network (Dosenbach, Fair, Cohen, Schlaggar, & Petersen, 2008), and has in different ways been associated with the adaptation of behaviour to external demands. We here use the formulation of the network proposed by Seeley et al. (2007) as this explicitly includes subcortical and brain-stem regions and is in this configuration thought not only to detect, but also to support allostatic adaptation of the organism to emotional demands (Touroutoglou et al., 2016; Touroutoglou, Hollenbeck, Dickerson, & Barrett, 2012).

literature on (primarily exogenously elicited) emotion indicate that the neural basis of emotion clusters in distinct functional components (Kober et al., 2008; K. A. Lindquist et al., 2012). As is evident from comparing Figures 2.1 A and 2.2 there is considerable overlap between the activations reported in these early PET studies and those reported in these meta-analyses, one interesting exception being the lack of activation in regions corresponding to the visual association component (yellow in Figure 2.2) in the PET studies consistent with there being minimal exogenous stimulation in these experiments.

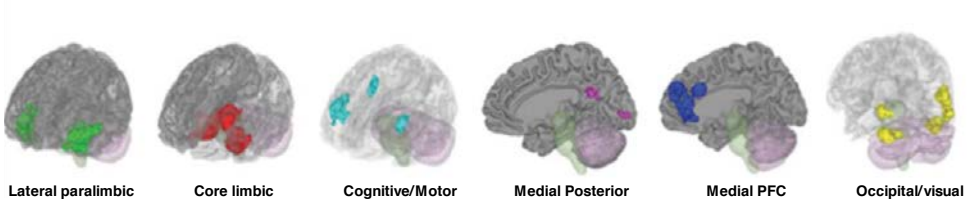


Figure 2.2: **Functional components of emotion generation.** Groupings of neural regions shown to co-activate in a recent meta-analysis of neuroimaging studies of emotion. Adapted with permission from Kober et al. (2008).

Before proceeding, the possibility should be mentioned that this account is inherently biased towards mnemonic forms of EnGE, as the early PET studies all involved forms of autobiographical recall as the basis for self-induction. Moreover, as most of the studies discussed investigated negative emotional states, it is unknown whether positive EnGE is supported by the same networks. However, given the lack of neuroimaging studies investigating EnGE of other emotional states or induction modalities, the relative independence of the three networks in question (Laird et al., 2011; Yeo et al., 2011), and their role in the general neural reference space of emotion (Kober et al., 2008; K. A. Lindquist et al., 2012), the remainder of this chapter takes this three-network structure to guide the development of a working model of EnGE.

2.4.1. State characteristics determine neural implementation of EnGE

A core lesson from the behavioural literature review above is that EnGE should be thought of as a constructive phenomenon, such that different components of emotion (e.g. imagery of emotional situations, semantic knowledge, bodily states) can all be used to self-elicit emotional states. As most current research has focused on the elicitation of single emotional archetypes of e.g. sadness, fear, or joy, using single implementation modalities, little is known about how such construction is neurally implemented. One exception to this is a recent study by Wilson-Mendenhall and colleagues (2013), in which participants generated emotions by immersing themselves in affective scenarios that varied such that they either emphasised physical or social threat. Supporting the notion of EnGE as a process by which embodied emotional experiences are self-elicited, they found that the generation of both kinds of threat activated neural systems associated with action planning, consistent with the role of threat perception in facilitating behavioural resolution (Whalen, 1998). Moreover, they found that social threat primarily elicited activation of DMN regions, particularly mOFC and TPJ, consistent with the known role of these regions in social processing (Decety & Lamm, 2007; J. P. Mitchell, 2009; Saxe & Powell, 2006; Spreng et al., 2009). Conversely, physical threat elicited activation of attention networks, including both FPCN and dorsomedial portions of the SN. Activation of these attention networks is a known consequence of exposure to (particularly negative) emotional stimuli, and is thought to underlie the influences of emotion on goal-directed behaviour/processing. Similarly, Damasio and colleagues (2000; see also Fitzgerald et al., 2004) found that autobiographical recall of situations in which individuals experienced discrete emotional states (e.g. sadness or joy), differentially activated neural regions involved in somatic representation and modulation of homeostasis. This is consistent with EnGE eliciting bodily states similar to those experienced in the original emotional situations.

Thus, dependent on the emotional state generated, EnGE appears to involve the recruitment of appropriate neural systems to construct and prepare the organism for dealing with the generated emotional state. While speculative at present, it is possible that the FPCN functions to support this coupling, given its role as a system of supramodal "flexible hubs" (Cole et al., 2013) that organises and coordinates activation in other networks in a task-dependent manner. In addition to supporting a general embodied simulation account of EnGE (Niedenthal et al., 2009; Seth, 2013), this opens for the intriguing possibility that EnGE can be used to actively prepare the organism for events, emotional or otherwise, as we will discuss in more detail below when considering the potential role of EnGE as a means of emotion management.

2.4.2. Temporal dynamics of EnGE networks

If comparatively little data exists on the structural basis of EnGE, next to nothing is known about its dynamics. One exception to this is an fMRI study by Daselaar and colleagues (Daselaar et al., 2008), investigating the neural time course of autobiographical memory retrieval, including memories with emotional qualities. Of particular interest for Daselaar et al. was the neural regions involved in the retrieval and (re)construction of episodic memories, and their subsequent maintenance, elaboration and reliving as an episodic simulation of an autobiographical event. Daselaar et al. found that the initial recall and construction of autobiographical events was supported by activation of different regions from those supporting their subsequent elaboration and experience: Initial recall elicited activation in dorsal frontal regions, retrosplenial cortex and hippocampus, whereas elaboration of these memories activated ventrolateral prefrontal regions. These findings are largely consistent with research on constructive memory in general (Addis et al., 2009; Addis, Wong, & Schacter, 2007). Critically, Daselaar et al. found that the emotional intensity of recalled memories was only predicted by activation in the initial phase, centred on aspects of the extended SN,

including limbic regions and basal ganglia. Conversely, the vividness of these memories correlated with activation in the later elaboration period, centred on left VLPFC and PCC, both regions thought to be part of the DMN (Yeo et al., 2011). As such, this finding suggests that core affective qualities are imparted on endogenously generated experiences relatively early in the generation process, whereas their phenomenal qualities rely on extended processing. This could suggest that the core affective state serves to anchor the generation process, but it is still an open question whether this is the case outside of EnGE via episodic memory.

Supporting the notion that EnGE in general is characterised by early generation of core affective properties, Suess and Rahman (2015) recently found that early stages of mental imagery of emotional facial expressions is highly similar to perceptual processing. Seuss and Rahman specifically investigated two event-related potential (ERPs) components known to be modulated by the emotional qualities of visual stimuli, the early posterior negativity (EPN) that occurs after 200-300 ms, and the late positive potential (LPP; Hajcak & Olvet, 2008) that has a posterior distribution and occurs after ~600 ms. Importantly, these components have been suggested to reflect different processes: The EPN is thought to reflect initial attentional orientation and discernment of core affective properties of stimuli (Citron, Weekes, & Ferstl, 2013). Conversely, the LPP is thought to reflect extended processing of stimuli, ostensibly related to "fleshing out" the initial percept with information from long term memory (Hajcak & Olvet, 2008). When comparing perception with mental imagery of emotional faces, Suess and Rahman found no discernible differences in the EPN, but marked differences in distribution patterns for the LPP. While differences in temporal resolution between electroencephalography (EEG) and fMRI (milliseconds vs. seconds) and the spatial coarseness of ERP components means that care must be taken in interpreting these results, it is interesting to note that the early attentional orienting thought to be measured by the EPN corresponds closely to the sort of stimulus-driven attention that is thought to be instantiated

by the Salience Network (Seeley et al., 2007; Vossel et al., 2014). Conversely, extended construction of representations of the form LPP is thought to be associated with is most frequently attributed to Default Mode Network (for partially confirming evidence from combined EEG-fMRI see Liu, Huang, McGinnis-Deweese, Keil, & Ding, 2012; Schacter & Addis, 2007). Interestingly, Seuss and Rahman found that the LPP component during imagery had a clear midline distribution, with apparently distinct generators in the frontal and posterior aspects of the brain, consistent with the topography of the DMN.

While again biased by the fact that these studies focused on forms of episodic imagery, this suggest that EnGE should be construed of as an extended process with at least two distinct stages related to the recall and subsequent maintenance and elaboration of the emotional state into a veridical internal experience, or simulation. Importantly, it appears that the coarse core affective qualities of the experience are imparted early in the EnGE process with subsequent processing elaborating on these (see Barrett, Mesquita, Ochsner, & Gross, 2007a for an argument for this relationship holding for emotional experiences in general.).

2.4.3. The role of amygdala in EnGE

As a final aside it should be mentioned that the early PET studies discussed above did not report activation of the amygdala during EnGE. The amygdala is traditionally thought to be an essential part of the neural machinery of emotion processing (Canli et al., 2005; Ghashghaei & Barbas, 2002; Murray, 2007; Seymour & Dolan, 2008; Whalen, 1998), thought to be especially central in generating behavioural and physiological emotional reactions (Ochsner et al., 2009b). Moreover, it is well-established that the amygdala plays an important role in the formation of emotional memories, and it is thought to play a role in enabling the resilience of emotional memory to forgetting (Yonelinas & Ritchey, 2015). Indeed, it has been suggested that the amygdala plays a key role in autobiographical memory by "infusing" recall with emotion and causing physiological reactions congruent with the originally

experienced state (Ally, Hussey, & Donahue, 2013; Svoboda, McKinnon, & Levine, 2006) [2]. It is possible that practical issues like low sample size, the precise emotional states investigated and/or the use of PET might have occluded amygdala activation in these early studies. A more interesting possibility is that the amygdala might not play a similar core processing role in the elicitation of endogenous emotional states. Supporting this, extant evidence for the role of amygdala in emotion is primarily based on visual processing of stimuli (Dolan & Vuilleuimier, 2006), where it is thought to support the rapid allocation of attention to biologically relevant stimuli (Ohman, 2002; Ohman, Carlsson, Lundqvist, & Ingvar, 2007; Ohman, Soares, Juth, Lindström, & Esteves, 2012). Leaving aside the discussion of whether such amygdala-based modulation is automatic or not, it is notable that the amygdala is most strongly implicated in perceptual, most notably visual (Koelsch et al., 2013; Whalen et al., 2004), modes of processing (Pessoa & Adolphs, 2010). Thus, the absence of amygdala activation reported in these studies might reflect the relative lack of importance of the amygdala in generating emotional reactions based on endogenous information due to the lack of incoming stimuli. However, evidence also suggests that the amygdala might be important in attributing such endogenously generated information to the external world (McRae, Misra, Prasad, Pereira, & Gross, 2012c; Ochsner et al., 2009b). It should also be noted that more recent studies utilising fMRI have reported amygdala activation during both hypothetical (Wilson-Mendenhall et al., 2013) and autobiographical EnGE (Daselaar et al., 2008), though both protocols involved presenting participants with stimulus-cues meaning it is unclear whether amygdala activation was caused by stimulus processing or EnGE. Thus, the precise role of amygdala in EnGE is still an open question.

[2] However, recent meta-analyses did not find support for amygdala being involved in either general, or specifically emotional kinds of autobiographical memory (Martinelli, Sperduti, & Piolino, 2013).

2.5. Towards a working model of EnGE

The core proposition of the current thesis is that EnGE should be construed of as a process in which constructive cognitive mechanisms are employed to generate representations of emotional qualia that result in embodied emotional experiences or simulations. These qualia can take many different forms and include information from a multiplicity of information processing modalities, ranging from full-fledged visual episodic simulations of experienced or hypothetical events (e.g. imagining an upcoming event as going good or bad), semantic representations (e.g. ruminating about one's shortcomings), or bodily states (e.g. taking slow, deep breaths to put oneself at ease). As research into the effects of different modalities has been focused on finding the optimal means of self-inducing emotion, usage of these modalities have largely been thought of as being mutually exclusive (e.g. Holmes et al., 2006). However, in as much as emotional experiences are rich and multi-modal phenomena (Barrett et al., 2007a), it is likely that a natural implementation of EnGE involves using multiple modalities. Moreover, as there is significant variation in the ability to generate emotion, it is likely that individuals differ in the degree to which they show affinity for – and efficacy with – different modalities.

This section aims to synthesise the available evidence into a formal working model of EnGE as a psychological process, with special emphasis on specifying the constituent component processes supporting the generation of endogenous emotion and how these map onto the neural reference space (K. A. Lindquist et al., 2012) indicated by earlier studies.

2.5.1. Psychological component processes of EnGE

For it to be meaningful to speak of endogenous emotions, they need to have a core similarity to exogenous emotion in resulting in as much as they should be associated with subjective feeling states, with the primary difference between the two being how they are generated. To the degree that this assumption holds (cf. section 2.2), it should be possible to

adapt extant models of exogenous emotion generation to the endogenous case. Gross (J. J. Gross, 1998a; 2007b; 2015a) has proposed that research on the generation of emotion could be summarised in what he calls the *modal model of emotion* (Figure 2.3A). In this model, emotions are essentially reflex-like, short-term, adaptive reactions to external events with distinct coordinated effects on behaviour, cognition and physiology. Thus, an emotional reaction results on an individual 1) being in an emotional situation (e.g. seeing a snake) that is 2) attended to and 3) appraised as being relevant to the ongoing goals of the individual. If these criteria are fulfilled, an emotional response is elicited that aims at altering the situation so as to bring it closer in line with one's ongoing goals (e.g. in the case of the snake, simple avoidance of bodily harm; Carver & Scheier, 1998; Magen & Gross, 2010). For exogenous stimuli, this typically involves factors in the world such as being in an uncontrollable environment in the presence of a potential threat (e.g. being in the wild in the presence of a venomous snake). Conversely, being in a safe environment (e.g. being in a zoo and viewing a snake in a terrarium) would stop (or not initiate) the emotion generation process, on account of it not being relevant to goal achievement at that point in time.

A straight forward adaptation of this model to the exogenous case would be to replace the eliciting situation with an internal representation of a situation (Figure 2.1B). Thus, endogenous emotion generation would then be initiated by an internal model of the world, requiring the generation of a psychologically relevant simulation, such as reminiscing about a previous situation in which an emotion occurred, or the generation of a narrative context in which to interpret a hypothetical event. This, in turn, is likely to require the engagement of mechanisms related to directed search of memory for appropriate parameters and ultimately combining sources of information into a unified concept or simulation equivalent to the external situation (Schacter & Addis, 2007). As such, it appears plausible that EnGE requires the involvement of mechanisms of psychological construction of situated representations (Addis et al., 2009; K. A. Lindquist & Barrett, 2012; Schacter & Addis, 2007; Wilson-

Mendenhall et al., 2013; Wilson-Mendenhall, Barrett, Simmons, & Barsalou, 2011). Thus, further modification to the emotion generation loop is required by specifying how an internal

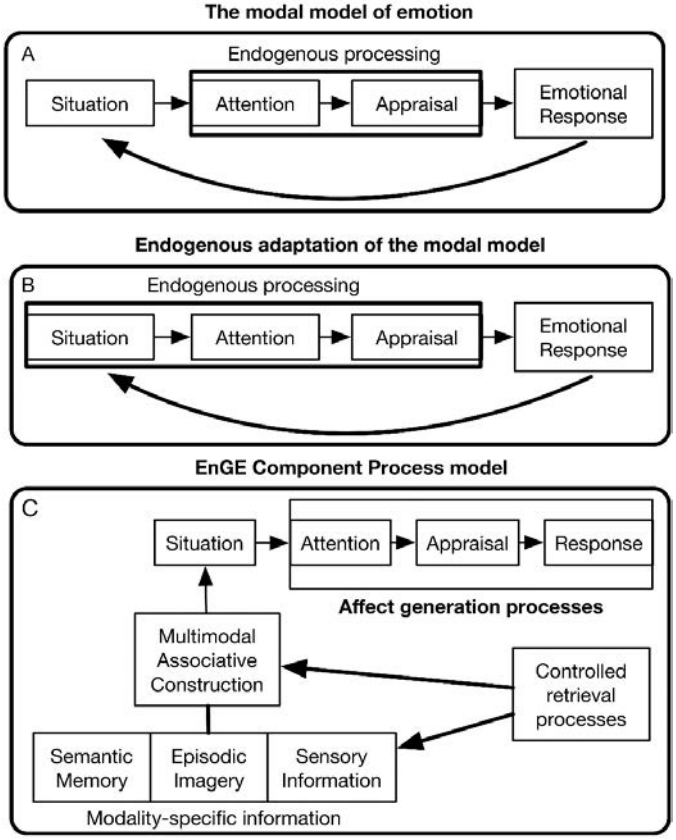


Figure 2.3: Models of emotion generation. A) The modal model of emotion generation as proposed by Gross (J. J. Gross, 2007b). B) The endogenous adaptation of the modal model, such that eliciting situations are contingent on internal processes. C) The elaborated component process model of endogenous emotion generation that guided the current research.

emotionally-relevant representation is generated. Likely, this process occurs through a form of multimodal associative construction (Hassabis & Maguire, 2009), by which semantic, contextual and sensory (including interoceptive) information relevant to a given emotion representation is reactivated, retrieved into awareness, and ultimately integrated into a complete emotional experience (see Figure 2.3C). This construction process can be triggered either incidentally in the course of one's stream of thought, or volitionally, through active,

guided retrieval (Hassabis & Maguire, 2007). In the latter case, a top-down control system is likely involved guiding retrieval of modality-specific information (Wheeler, Petersen, & Buckner, 2000), as well as entrainment of the representation formation process in the service of goal-achievement (Gerlach, Spreng, Madore, & Schacter, 2014; Spreng et al., 2010).

As for the subsequent steps of the emotion generation cascade, evidence provides some support for an analogy between the modal model and EnGE, with recent work showing that individuals are flexible in terms of what aspects of recalled emotional states they pay attention to and the appraisals they make about their own endogenously generated emotions. For instance, it has been shown repeatedly that the emotional consequence of recalling a past emotional incident depends on the objectives and mental stance of the individual to either go into the emotional state or attempt to abolish it (Denkova, Dolcos, & Dolcos, 2015; Kross & Ayduk, 2008; Kross, Ayduk, & Mischel, 2005; Kross, Davidson, Weber, & Ochsner, 2009). Thus, at least for EnGE via autobiographical recall, it appears that it is possible to draw a distinction between mechanisms supporting recall and episodic representation formation, and those enacting the affective consequences of those representations. An alternative could be that EnGE involves first modulating ones core affective bodily states (Russell, 2003; 2009), that then serves as an "anchor" for subsequent representation formation efforts, consistent with the previously discussed work by Daselaar and colleagues (2008), who found amygdala activation during initial recall of the core of emotional autobiographical memories but not during later elaboration of such memories. As of yet, evidence is not available to arbitrate between these two possibilities.

Summarising, I propose that EnGE should be conceptualised as consisting of three core processes: 1) A multimodal associative representation process, 2) a process involved in the generation of physiological emotional responses, and in the case of volitional EnGE, 3) a control process supporting active retrieval of information from memory. In the following the

possible neural mapping of this component process architecture is discussed, with reference to the three major intrinsic connectivity networks implicated in previous studies of EnGE.

2.5.2. The role of the Default Mode Network in internally generated cognition

As previously discussed, a closely related topic to endogenously generated emotion is that of self-generated thought (SGT), consisting of more or less spontaneously occurring episodic memories, streams of thought, and other qualia depending on internal sources of information (Smallwood & Schooler, 2015). Indeed, as we have shown previously, self-generated thought is often the precursor of emotional reactions (Ruby et al., 2013), meaning that drawing a clear distinction between the two is not trivial. As such, one would expect that EnGE and SGT share important features in terms of their neural implementation and, by extension, their psychological component processes. Consistent with this, there is a large degree of overlap between the reference space for EnGE presented previously (Figure 2.1B) and that thought to support SGT, with the most notable overlap being in the DMN and FPCN. Reviewing the literature on the neural bases of SGT, Andrews-Hanna, Smallwood and Spreng (2014) discussed the plausible component processes instantiated by these networks, pointing out that DMN activation is reported across a wide range of tasks including theory of mind, reasoning, scene construction, mental imagery, episodic recall, prospection and perspective taking, and other psychological phenomena requiring the generation of internal representations (Addis et al., 2007; 2009; Gerlach et al., 2014; Schacter et al., 2012; Schacter & Addis, 2007; Spreng et al., 2009; 2010; Spreng & Grady, 2010). Interestingly, based on intrinsic connectivity and coactivation characteristics, the DMN can be parcellated into distinct subcomponents (Andrews-Hanna, Reidler, Sepulcre, Poulin, & Buckner, 2010): A dorsal-medial system (blue in Figure 2.4, consisting of dorsomedial and lateral PFC, temporal lobes and temporoparietal junction) that is relatively independent from the medial-temporal subsystem (green in Figure 2.4, consisting of retrosplenial cortex, inferior temporal cortex and

ventromedial prefrontal cortex). These subsystems both interact with a core system centred around the anterior medial PFC, PCC and angular gyrus (yellow in Figure 2.4). When Andrews-Hanna et al. (2012; see also Andrews-Hanna et al., 2010) investigated the associations of these subsystems with different psychological terms using the NeuroSynth database (Yarkoni, Poldrack, Nichols, Van Essen, & Wager, 2011), they found evidence for

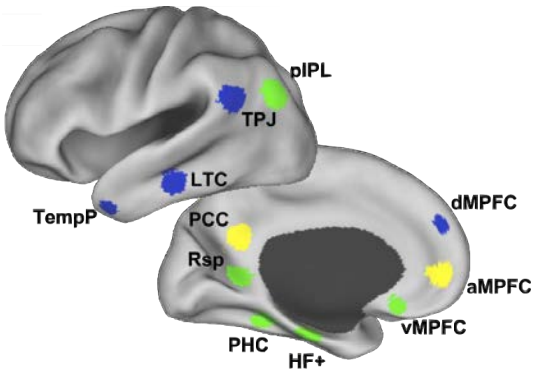


Figure 2.4: Core- and subsystems of the DMN. Adapted with permission from (Andrews-Hanna et al., 2010). Yellow = core DMN system to which both subsystems are coupled. Blue = dorsal-medial subsystem. Green = medial-temporal subsystem. Subsystems are coupled to the core, but not each other.

functional dissociation between these component: The dorsal medial subsystem was most closely associated with processes requiring conceptual processing and simulation formation. Conversely, the medial-temporal subsystem was associated with mnemonic processes, particularly those requiring scene formation or episodic simulations. Finally, the core system was found to be most closely associated with self-referential and affective processes, consistent with the strong connectivity of anterior medial PFC to both limbic and subcortical regions involved in core affective processes and somatic regulation (Roy, Shohamy, & Wager, 2012), and the association of PCC with supramodal awareness (Leech & Sharp, 2014). Summarising, the DMN consists of at least three systems associated a) accessing memory, b) creating inner mental models based on these memories, and c) evaluating the self-relevance of these models. These are all essential processes for the generation of internal

simulations and evaluation of past and future events, thought to be one of the major adaptive functions of self-generated thought (Smallwood & Schooler, 2015). As such, the DMN is a likely neural substrate for the multimodal representation formation process proposed to be involved in EnGE above.

2.5.3. The Salience Network as a unique signature of EnGE

If DMN supports the generation of representations supporting the experiential aspect of emotions, it is relatively far removed from neural regions supporting the perception and generation of the somatic aspects of emotions, which is rather thought to be supported by the extended Salience Network (Seeley et al., 2007). The SN is composed of structures in the brainstem, limbic system and paralimbic regions, like the insula, amygdala, periaqueductal grey (PAG), substantia nigra, and hypothalamus. In addition to there being extensive evidence connecting these structures to the experience of emotion (Kober et al., 2008; K. A. Lindquist et al., 2012; Vytal & Hamann, 2010), these structures are known to support interoception, pain processing, and motivational processes, as well as general hedonic processing (particularly amygdala and ventral striatum) and homeostatic regulation (particularly hypothalamus) (Berridge & Kringelbach, 2013; Buhle et al., 2013; Corradi-Dell'Acqua, Tusche, Vuilleumier, & Singer, 2016; Craig, 2011; Damasio et al., 2000; Jabbi, Bastiaansen, & Keysers, 2008). In concert, these regions are well suited for the generation of the physiological concomitants (i.e. bodily states) associated with emotional reactions. As such, it is possible that the SN supports the generation of core affective, somatic properties of endogenous emotional experiences, providing the embodiment of the simulation generated by the DMN. Thus, SN activation is potentially a signature of EnGE differentiating it from SGT, supporting the generation of embodied emotional reactions to endogenous information.

2.5.4. The Frontoparietal Control Network and goal-directed control of memory

Finally, FPCN activation as seen in the early PET studies could reflect active control of mnemonic processes. While the processes supported by the DMN are arguably sufficient for the generation of internal representations, co-activation of DMN and FPCN is frequently reported (e.g. Gerlach et al., 2014; Spreng et al., 2010; Spreng & Grady, 2010; Spreng, Sepulcre, Turner, Stevens, & Schacter, 2013; Whitman, Metzak, Lavigne, & Woodward, 2013), in tasks where participants are required to generate internal simulated representations in the service of attaining a specific goal, such as planning how to do something (Gerlach et al., 2014; Spreng et al., 2010; Spreng & Grady, 2010). Interestingly, FPCN and DMN coupling does not appear to occur when imagining the outcomes of such plans, suggesting that the FPCN is involved in structuring the generation process in terms of particular set-points (Gerlach et al., 2014).

Another, complementary, possibility is that FPCN serves to guide the retrieval of modality-specific information in line with its proposed role as a system of supramodal "flexible hubs" (Cole et al., 2013). Evidence suggests that modality-specific information is stored in primary sensory association cortices, and that vivid reconstruction of memories involves reactivation of similar neural systems involved in their initial processing (Wheeler et al., 2000). This process of reactivation is thought to be supported by the ventrolateral portion of the prefrontal cortex corresponding to the anterior IFG, particularly BA 45 and 47. The left IFG is variably reported to be part of the DMN (e.g. Yeo et al., 2011) and a part of the FPCN (e.g. Harrison et al., 2008; Laird et al., 2011), with evidence suggesting that IFG is not a part of the core functional modules of the DMN (Andrews-Hanna et al., 2010). Moreover, the IFG is a central nexus with intrinsic connectivity to both DMN and more central nodes of FPCN (Spreng et al., 2013). As such, the left IFG might serve as a junction through which DMN can be recruited in a goal directed representation generation (Spreng et al., 2010), a supposition supported by findings that the coupling between the FPCN, and particularly left IFG, and

DMN is a predictor of creativity (Beaty, Benedek, Silvia, & Schacter, 2016; Beaty et al., 2014). The role of IFG in the cognitive control of memory is well documented, and in particular the orbital part of the IFG that is implicated in the early PET studies appears to support strategic mnemonic access to conceptual representations in service of goal achievement (Badre & Wagner, 2007). The left IFG is also heavily implicated in language function, encompassing in its posterior aspects Broca's area (Eickhoff et al., 2011), known to be central for language production. Interestingly, the anterior aspects of the IFG, including the orbital part appears to be particularly involved in semantic fluency tasks (S. Wagner, Sebastian, Lieb, Tüscher, & Tadić, 2014) that require the generation of words based on category membership. Thus, it could be that its involvement in EnGE as reported in these early studies reflects retrieval of memories based on valence category. Consistent with the flexible hub account of the FPCN (Cole et al., 2013), this could suggest that the role of FPCN in volitional EnGE is to facilitate activation of DMN in the service of goal achievement, combined with supporting the retrieval of information from modality-specific sensory cortices.

2.5.5. A neural component process architecture of EnGE

Summarising, the neural component process architecture proposed involves three major intrinsic connectivity networks, the DMN, SN and FPCN, working in concert to generate emotional states based on endogenous sources of information (see Figure 2.5). In this, it is proposed that the DMN operates primarily in the service of retrieving and integrating information from long-term memory and generating simulations or representations corresponding to hypothetical or previously experienced emotional situations. The SN is hypothesised to be involved in the generation of core affective states rooted in physiological sensations. Finally, the FPCN coordinates activation of DMN and SN in the service of achieving the goal of self-inducing an emotional state, and potentially guiding memory search

by coupling with and activating networks supporting of representations of modality-specific information. To the degree that this model holds, one would expect distinct aspects of emotion generation to correlate with each of these networks. Specifically, one would expect the affective intensity of the experienced emotion to be associated with SN activation. Conversely, one would expect DMN activation to primarily reflect the type of retrieved information that constitutes the generated emotional experience. Finally, FPCN, subserving cognitive control of the representation construction process, should show consistent activation during effortful generation, as well as coupling to modality-specific sensory processing networks as a function of the specific representation being generated.

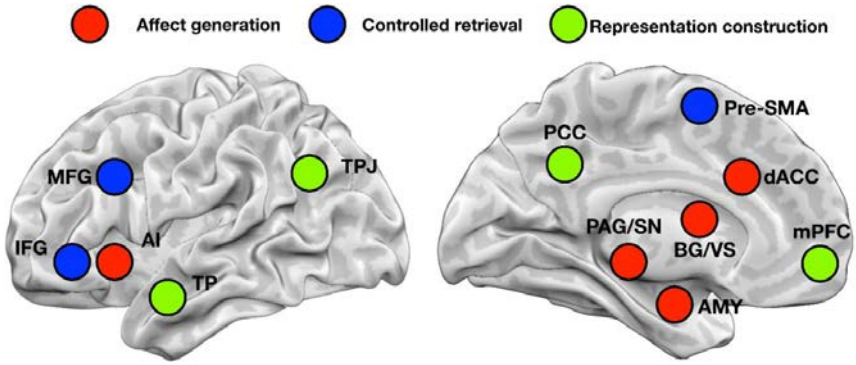


Figure 2.5: Neural component processes of EnGE. Hypothesised mapping of component processes to the three networks implicated in earlier PET studies of EnGE.

2.6. EnGE as a means of emotion management

In this section we move from the primarily theoretical question of how EnGE can occur in terms of psychological and neural mechanisms to the question of what utility EnGE might have. As we discussed in Chapter 1.3, one of the most intriguing aspects of EnGE is that it can allow us a measure of control over our emotional reactions. There are many ways by which one can manage one's emotions. For instance, a number of means exist by which cognitive processes can be used to alter *how* or even *if* we process emotional information (J. J. Gross, 1998b; 2007b). There is also a large literature on the efficacy of different means of coping with emotional stressors (Lazarus & Folkman, 1984). While the literature on emotion

regulation tends to emphasise the momentary regulation of emotional reactions, the coping literature has a more long-term perspective, such as dealing with bereavement. This aside, there is considerable overlap between these two fields, such that, for instance, the use of cognitive strategies to reinterpret events can both be used in a reactive, regulatory way and as a means of long-term coping (J. J. Gross, 2015a). In the following I will refer to the confluence of these two fields as *emotion management*, by which I mean the range of strategic (i.e. volitional) means that an individual can use to influence their present or future emotional states.

It is increasingly becoming apparent that whether or not one reacts to external emotional stimuli can be regulated by a wide range of cognitive and behavioural strategies (Webb, Miles, & Sheeran, 2012), some of which are remarkably simple. For instance, we recently showed that the preferential processing of affective stimuli (fearful and angry faces) can be eliminated by the simple expedient of focusing on the gender of the individual expressing the emotion rather than the emotional expression itself (H. G. Engen, Smallwood, & Singer, 2015). Roughly speaking such regulatory efforts can be grouped into efforts aimed at regulating emotion as it occurs and those aimed at predicting and preparing one to deal with potential future stressors (J. J. Gross, 1998a; 2007b; S. E. Taylor & Schneider, 1989). In the following, I will discuss evidence for how EnGE can be used in either of these capacities, and discuss how they can be conceptualised within arguably the dominant and most developed mechanistic framework of emotion management: the process model of emotion regulation (J. J. Gross, 1998b; 2015b).

2.6.1. The process model of emotion regulation

Emotion regulation and coping research has traditionally been focused on investigating the effects of different cognitive or behavioural strategies to deal with emotional stress as it occurs. One of the most influential taxonomies of such strategies is the process model of

emotion regulation proposed by James Gross (J. J. Gross, 1998b Figure 2.5). This model is based on the modal model of emotion discussed in Chapter 2.4.1 (see Figure 2.3), classifying different approaches to influencing the emotional reactions one has into different families of strategies based on where in the generation process they intervene (J. J. Gross, 2015a).

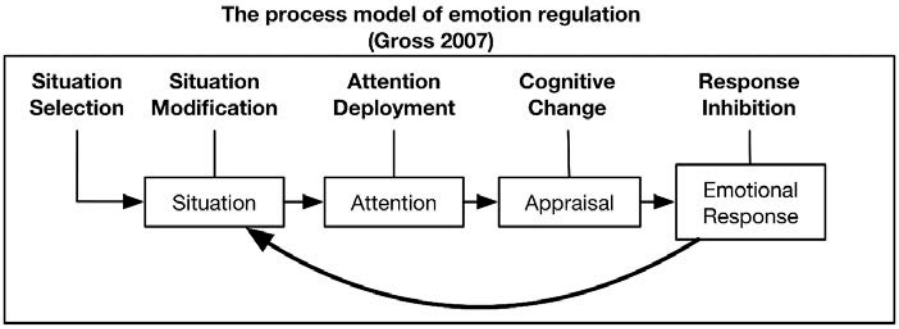


Figure 2.6. The process model of emotion regulation, adapted from writings by James Gross (J. J. Gross, 2007b; 2015a).

Of these, strategies involving attentional deployment and cognitive change have received considerable attention in the literature, both because they have been shown to be highly effective, and because they have the potential to be translated into clinical interventions for individuals with emotion regulation difficulties (Aldao, Jazaieri, Goldin, & Gross, 2014; Blechert et al., 2015). Such strategies involve actively adopting a different cognitive stance to the eliciting event, either by thinking differently about it (as in the strategy of reappraisal; Goldin, McRae, Ramel, & Gross, 2008; McRae, Ciesielski, & Gross, 2012a) or somehow reducing their importance by paying them less attention and distracting oneself (Dörfel et al., 2014; Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2011). Extensive research shows that such cognitive strategies can be effective means to control emotional reactions in many contexts (Webb et al., 2012), and are thought to gain their efficacy in large part from altering the preconditions for emotional reactions, particularly by blocking or altering the affective appraisals made about stimuli (J. J. Gross, 2007b). As such, these strategies are examples of *high leverage* strategies (Magen & Gross, 2010) that impact on early and highly influential

components of the emotion generation cascade. Thus, if EnGE is an effective means of emotion management, it is likely to impact on situational, attentional or cognitive processing components of the ExGE process.

2.6.2. Positive EnGE as a reactive emotion management strategy

A hallmark of EnGE is that it allows the individual to experience emotional states independently of the external environment. Recently, Barbara Fredrickson (Fredrickson, 2013) suggested that the ability to self-generate positive emotions might be particularly important part of EnGE, with the potential to increase resilience and improve well-being. This suggestion was based on substantial evidence showing that experiencing positive emotion is associated with a wide range of mental and physical health benefits (Catalino & Fredrickson, 2011; Fredrickson, 2001; Fredrickson et al., 2008; Kok et al., 2013; Tugade & Fredrickson, 2004), in the large related to enhancing physiological and mental self-regulation. Of particular interest for present purposes, it has been found that emotionally resilient individuals actively seek out positive emotional experiences and show enhanced proficiency in generating positive interpretations of negative situations (Tugade & Fredrickson, 2004) consistent with them actively generating positive emotional states to "bounce back" when confronted with difficulties. Similarly, it has been found that individual differences in the accessibility of positive emotional memories, especially in times of distress, is associated with trait resilience, even when controlling for average trait positivity (Philippe, Lecours, & Beaulieu-Pelletier, 2009). Another line of evidence for the importance of positive emotion generation in mental health comes from the work of Emily Holmes and colleagues (Holmes et al., 2006; 2009; Holmes & Mathews, 2005; Holmes, Blackwell, Burnett Heyes, Renner, & Raes, 2016; Morina et al., 2011). Holmes and colleagues have investigated the regulatory effects of positive mental imagery in both normal and clinical populations. Interestingly, decreased mental imagery and increased reliance on verbal descriptions when processing emotional

material appears to be common sequela of depression (Bergman & Craske, 2000). It has been argued that this shift might be causal in the ætiology of depression as it diminishes the capacity of the individual to deal with negative emotion (Stöber, 1998). Summarising the work by Holmes and colleagues, they have found 1) that individuals readily generate emotional reactions based on mental imagery 2) that the capacity to do so can be trained and 3) that such training can work as a "cognitive vaccine" against affective maladies, even in ongoing depression. Similarly, Gulia Poerio and colleagues (Poerio, Totterdell, Emerson, & Miles, 2015a; 2015b) have shown that positive mind-wandering or day-dreaming can have beneficial effects, undoing the effects of externally induced negative affect, and being associated with increased positive affect and feelings of social connectedness. Finally, recent investigations suggest that a key beneficial effect of compassion and loving-kindness meditation (both centrally involving the generation of positive affective states) is enhanced resilience in the face of negative stressors (Fredrickson et al., 2008; Klimecki et al., 2013; Kok et al., 2013; Singer & Klimecki, 2014).

Summarising these findings, it appears that positive EnGE is closely associated with emotional resilience and emotional well-being and that both directed and spontaneous modes of EnGE can have these effects. However, as of yet it is unclear what mechanisms might be underlying this effect. Returning to the process model of emotion generation, one possibility is that having access to self-generated positive emotions might enable one to distract oneself from negative situations. Distraction is known to be a highly effective emotion regulation strategy (Kanske et al., 2011; McRae et al., 2010; Sheppes & Gross, 2011) and is thought to enable emotion regulation by minimising attention allocated to processing emotional materials early in the emotion generation process (Sheppes & Gross, 2011; Thiruchselvam, Blechert, Sheppes, Rydstrom, & Gross, 2011). Possibly, positive EnGE abilities could enable one to self-generate alternate, more appetitive, objects of attention (see Feindler, Marriott, & Iwata, 1984; Smallwood, Brown, Baird, & Schooler, 2012). If such a *distraction hypothesis*

was true one would expect that positive EnGE should result in diminished processing of negative stimuli (c.f. Thiruchselvam et al., 2011). Moreover, to the degree that EnGE is an effective means of distraction, one could expect to see a correlation between EnGE abilities and trait use of distraction as a means to regulate emotion.

Another possibility is that positive EnGE can cause changes in the interpretation of a stimulus by facilitating other emotion regulation processes. For instance, by self-generating a positive emotional state, it is possible that one can draw on the documented effects of positive emotional states to widen the scope of attention and flexibility of thought (Fredrickson & Branigan, 2005). This could potentially enable one to devise better adapted and more effective reappraisals of emotional stressors one is confronted with (Diedrich, Hofmann, Cuijpers, & Berking, 2016). Such reappraisal has been found to be a highly effective means of emotion regulation with extensive benefits both in terms of altering affect, and long term health benefits (Blechert et al., 2015; Buhle et al., 2014; Folkman & Moskowitz, 2000; McRae, Ciesielski, & Gross, 2012a). Facilitating the efficacy and occurrence of reappraisal would therefore be expected to enable the individual better to regulate their emotional states. If this *facilitation hypothesis* was true, one would expect that positive EnGE would lead to similar neural and behavioural consequences as reappraisal, and that EnGE abilities should be associated with trait usage of strategies that involve internally changing how one thinks about different situations.

Finally, an intriguing possibility is that positive EnGE can work even earlier in the emotion generation process and constitute a means of situation modification, directly altering the emotional significance of a given event. While situation modification most commonly refers to behavioural means of altering the physical external situation, it should be noted that the emotional significance of a situation is a function not only of the state of the external world, but also of the state of the individual (J. J. Gross, 2015a). This, for instance, is seen when people chemically alter their mental states, where a common concomitant is altered

perception of the emotional significance of situations. Another example of this can be seen in affective congruence effects in which being in a certain mood state makes you more likely to notice and process stimuli of the same valence. Research shows that such affective congruency effects are highly prevalent and elicited by largely automatic means (Russell, 2014). Unlike the previous hypotheses discussed, such regulatory effects might be better characterised as enhancing the ability to *endure* negative emotional by buffering with positive emotion than outright alteration of negative emotional states into neutrality or positivity. This might be particularly helpful in cases where the situation makes the down-regulation of negative affect difficult or suboptimal, such as in a caretaker setting where an empathic link is needed for optimal care (Manzano García & Ayala Calvo, 2012; Singer & Klimecki, 2014). Importantly, unlike *distraction*, if this was the mechanism of action one would not expect diminished processing of negative stimuli. Moreover, unlike the *facilitation hypothesis*, the *buffering hypothesis* would suggest that regulatory effects would be neurally and behaviourally supported primarily by increased positive emotion, and not improved ability to down-regulate negative emotions.

Presently, insufficient evidence is available to arbitrate between these three hypotheses, though it should be noted that recent functional MRI evidence shows that engaging in positive emotion generation via compassion meditation does not appear to result in reduction of either neural correlates nor subjective experience of negative affect when confronted with adverse stimuli (Klimecki et al., 2013), suggesting that a distraction account might not be applicable.

2.6.3. Emotion simulation as pre-emptive emotion management

Hitherto, we have followed the literature, in describing emotion regulation and coping efforts as occurring as a consequence of an emotional stressor being present in the environment. However, as Taylor and Schneider (1989) pointed out, such a reactive account

of emotion management ignores the panoply of behaviours and cognitions people engage in pre-empting and preparing themselves for potential future stressors, where success involves the stressor not occurring. Thus, for instance, a person tasked with giving a talk can motivate themselves to extra preparation and rehearsal by envisaging the emotional consequences of *not* preparing, thereby avoiding the stressor altogether by making extra efforts to prepare. Conversely, by imagining the positive outcome of a given action it is possible to motivate oneself despite the mundaneness or aversiveness of that action (Oettingen & Mayer, 2002). As such, this notion of emotion management as reactive to external events overlooks one of the most distinctive features of human cognition: The capacity to simulate potential events in the service of problem solving and goal achievement (Schacter & Addis, 2007).

In the context of emotion management, this capacity can enable both prediction of upcoming stressors and preparation for them by, for instance, adopting appropriate emotional states and mustering appropriate levels of arousal to deal with a situation (M. Y. Kim et al., 2015; Tamir et al., 2008; S. E. Taylor & Schneider, 1989). The occurrence of such instrumental motives in emotion has recently been documented, and appears to be a highly effective and utilised means of self-regulation (Ford & Tamir, 2012; M. Y. Kim et al., 2015; Millgram, Joormann, Huppert, & Tamir, 2015; Tamir et al., 2008). EnGE might be particularly useful for this as it allows the individual to experience (through embodiment) the likely emotional outcomes of future events, thereby allowing the mobilisation of appropriate coping resources. Similarly, EnGE can be utilised to simulate possible alternate outcomes to future events, enabling one to select appropriate goals for emotion management efforts. Optimal goal-selection in a given the situation could be afforded by a simple simulation heuristic (Kahneman & Tversky, 1982), by which goals are selected based on the ease by which they can be imagined. Thus, the ease with which an emotional state can be generated could serve as an indicator that emotion management of this kind can be achieved. Referring again to the process model of emotion regulation, this hypothetically means that EnGE can be

used to enable situation selection, by predicting and diffusing stressors, as well as situation modification, by altering the internal component of the situation to fit potential upcoming challenges, thereby enabling increased fluency in dealing with emotional stressors.

2.6.4. Two routes of EnGE-based emotion management

Summarising, I suggest that there exist at least two distinguishable routes by which EnGE can be used for emotion management: 1) By enabling the individual to generate, in particular positive, emotional experiences, in effect buffering them from negative vagaries of the environment, or 2) by allowing them to simulate scenarios including potential emotional states based on past experience, thereby enabling pre-emptive action to be taken and plans to be made that either enhance or decrease the likelihood of that state arising. Critically, while I propose that these two routes are dissociable, they are likely to work in concert, such that EnGE abilities would be associated with both reactive and prospective emotion management.

2.7. Hypotheses and questions

The current thesis has two overarching goals. First, it seeks to test the validity of the neural component process model of endogenous emotion generation proposed above. This was done using a combination of behavioural and neuroimaging methods were utilised to investigate different ways in which goal directed emotion generation can be implemented. By triangulating across different modes and methods of emotion generation the thesis aims to propose to establish a general neural architecture of EnGE. Informed by the enhanced understanding of the underlying mechanisms of EnGE the second focus of the thesis to explore the question of whether EnGE can be used as a means of emotion management. Specifically, potential benefits of emotion generation ability on trait markers of resilience and coping, and the relation of emotion generation to emotion regulation were investigated. In the

following the specific hypotheses guiding the current research are presented, before an overview of the following empirical investigations is provided.

2.7.1. Hypothesis 1: A neural component process architecture of EnGE

The first part of this thesis provides test the neural component process architecture proposed above in Chapter 2.4 (see also Figure 2.5). Specifically, it is hypothesised that EnGE should be neurally implemented by the cooperation of three major intrinsic connectivity networks: The Default Mode Network (DMN), the Salience Network (SN), and the Frontoparietal Control Network (FPCN). These are hypothesised to implement dissociable psychological component processes supporting the endogenous generation of emotion. Specifically, DMN is hypothesised to implement the generation of representations of affective situations while the SN is hypothesised to coordinate the emotional responses to these. Finally, the FPCN is proposed to support the cognitive control of the representation generation process. Importantly, the FPCN is proposed both to support the representation generation process in general, and to support the retrieval of emotion-related information in modal association regions.

2.7.2. Hypothesis 2: Neural plasticity of EnGE-architecture

To the degree that the above hypothesis holds, one would expect to see morphological changes in this network as a function of expertise in EnGE relative to a normal population. Moreover, to the degree that EnGE-expertise is an emergent skill distinguishable from its processual subcomponents, one would expect that such changes should centre around the components of the network that are most important for the coordination of component processes, here hypothesised to be supported by the FPCN.

2.7.3: Hypothesis 3: Neural mechanisms of EnGE-based regulation

In Chapter 2.5 two distinct means by which EnGE can be used for emotion management was proposed: 1) A reactive mode, by which positive emotional states can be generated in response to external stressors and 2) a pre-emptive mode, allowing the individual predict and prepare for upcoming stressors. In the current thesis, the neural bases of the reactive form of emotion management is investigated. Based on the current review, one would expect that efforts to use EnGE to regulate one's emotional reactions should rely on a similar neural architecture to that proposed in hypotheses 1 and 2. Moreover, by comparing the neural bases of EnGE-based emotion regulation efforts with other emotion regulation techniques it should be possible to determine the mechanism by which any observed regulatory effects occur. Chapter 2.5.2 further proposed three potential mechanisms for reactive EnGE-based emotion regulation: *distraction, facilitation,* and *buffering*. In the context of a negative stressor, the *distraction* and *facilitation* accounts would both predict increased positive affect with a concomitant decrease in negative affect. However, the accounts diverge in terms of neural effects with the *distraction* account predicting the involvement of EnGE circuits alone, and the *facilitation* hypothesis predicting involvement of EnGE circuits in conjunction with those supporting other forms of emotion regulation. Finally, the *buffering* account diverges from the two preceding by proposing that EnGE-based regulatory efforts should primarily enhance the ability to endure negative stressors, meaning that it should result in increased positive emotion without, necessarily, a concomitant drop in negative emotion.

2.7.4: Hypothesis 4: EnGE influences trait emotion and emotion management

To the degree that EnGE is an efficacious emotion regulation technique one would expect to see evidence of this in its relationship with indices of adaptive emotion management and trait affect. Specifically, if the reactive mode is efficacious, one would expect EnGE abilities to be associated with usage of emotion-focused emotion management. Conversely, if

the pre-emptive mode is efficacious, one would expect a relationship between EnGE abilities and instrumental emotion management styles, that emphasise taking pre-emptive action to diffuse the impact of emotional events. Additionally, if EnGE facilitates adaptive emotion management as is here proposed, one would expect to that individual differences in EnGE abilities mediate the relationship between adaptive emotion management and trait affect.

2.8: Overview of dissertation

The empirical body of work (Chapters 4-7) in this thesis is published or is under review in peer-reviewed journals. As the article format does not allow sufficient space for in-depth discussion of the methods used, Chapter 3 provides an overview of the different experimental methods employed, and discusses how they can be utilised to answer the questions posed above.

The focus of Chapters 4 and 5 was to investigate what neural processes support the generation of endogenous emotion. In Chapter 4, a large scale study investigating the neural component structure of EnGE is reported. This study focused on establishing the neural architecture of EnGE, differentiating implementation-specific mechanisms from those supporting EnGE in general. Chapter 5 presents a study in which the effects of long-term practice of volitional emotion generation via loving-kindness and compassion meditation on brain structure was investigated. To the degree that our neural component process model holds, one would expect that such experts show structural changes in these networks. Together, these chapters enabled testing of the proposed component process architecture of EnGE both in terms of functional activation and long-term brain change.

In Chapters 6 and 7 the thesis shifts to the question of the utility of EnGE. Chapter 6 investigates the feasibility of using EnGE as an emotion regulation strategy when confronted with an external stressor and the neural mechanisms supporting such regulatory effects, by investigating the behavioural and neural consequences of using compassion-meditation to

down-regulate emotional reactions to negative film clips. Further, this was compared with emotion regulation implemented using reappraisal, allowing a differential test how EnGE-based regulation occurs in light of the process model of emotion regulation. Finally, Chapter 7 investigates whether and how trait abilities to generate positive and negative emotion predicts trait affectivity and emotion management styles, as measured by self-report. This allowed investigation of the relative importance of general or valence-specific EnGE abilities for different forms of emotion management, and also to directly test whether EnGE causally mediated the relationship between emotion management styles and trait affect.

Part II: Experiments

Chapter 3: Methods

3.1. Chapter overview

This Chapter provides an overview of the methods employed in the studies reported in Chapters 4-7 and how they relate to the central research questions. The first goal of the thesis was to establish the neural mechanisms supporting endogenous emotion generation. This was done in two experiments investigating 1) how endogenous emotion generation is implemented in the normal population (Chapter 4) and 2) how expertise in emotion generation has long term effects on brain architecture (Chapter 5). The second goal was to investigate how EnGE can be used as a mechanism for emotion management. This was done by 1) the neural mechanism of emotion regulation using compassion meditation in a population of expert meditators (Chapter 6) and 2) establishing how emotion generation abilities correlate with trait affect and emotion management style in the normal population (Chapter 7). In this Chapter an overview of the methods used in these investigations is given, starting with a description of the two samples, followed by presentations of the experiments utilised and the measures and analytical approach taken.

3.2. Overview of the empirical body of work

The empirical body of work in this thesis is either published (Chapter 7; H. G. Engen & Singer, 2015) or is currently under review (Chapters 4-6). Chapters 4-7 consist of the article manuscripts as they have been submitted. As the article format does not allow sufficient space for in-depth discussion of the methods used, the present chapter provides an overview of the different experimental methods employed, and discusses how they can be utilised to answer the questions posed above.

In the current thesis two complimentary approaches were taken to map the neural and behavioural mechanisms of EnGE: First, Chapter 4 investigated how volitional EnGE was

implemented in the normal population, using a newly developed, naturalistic fMRI paradigm (known as the RAGE, described in more detail below) in two independent representative samples (*N*=32 and 293). This paradigm enabled the investigation of how EnGE is neurally implemented in the normal population and provided ample power to test our component process model in Chapter 4. The larger of these two samples stemmed from a large-scale longitudinal mental training study (the *ReSource Project*). As part of this project participants completed an extensive battery of trait measures of socio-affective skills. In Chapter 7, this was used to investigate whether EnGE abilities predicted trait affectivity, emotion regulation and coping styles in the normal population.

Second, the neural and behavioural correlates of EnGE expertise were investigated. This was accomplished by investigating the morphological differences in brain structure in a sample of meditators with extensive experience in loving-kindness and compassion meditation ($N = 17$). As discussed in Chapter 2.3.2, a central aspect of these meditation practices is the active self-generation of positive emotional states. Thus, by comparing practitioners to a matched sample of normal controls, we could in Chapter 5 investigate the structural brain bases of EnGE expertise. The aim of this study was to verify our results from the normal population, the reasoning being that long-term practice in emotion generation should lead to durable changes in our putative emotion generation networks. Finally, as compassion meditation centrally involves dealing with the suffering of others, we could in Chapter 6 directly test whether the generation of positive, compassionate affect could be used as a reactive emotion management strategy to deal with exogenous stressors as discussed in Chapter 2. Moreover, by comparing the neural and behavioural outcomes of compassion relative to the well-described emotion regulation strategy of reappraisal we could directly test whether any observed regulatory effects were consistent with the distraction, facilitation or buffering hypotheses of EnGE based emotion regulation proposed in Chapter 2. To begin, the following section provides a detailed presentation of the two samples under investigation.

3.3. Samples

3.3.1. The ReSource Project

Chapters 4 and 7 are based on data from a newly developed functional magnetic resonance imaging paradigm (the Regulation and Generation of Emotion; the RAGE, see below), as it was it was implemented in the *ReSource Project* (Singer et al., 2016)— a large scale longitudinal mental training project, the baseline data of which are reported in the current thesis. Utilising the large, representative sample gathered in the *ReSource Project* allowed for the investigation of endogenous generation of emotion as it naturalistically occurs in terms of behavioural and neural implementation. Out of a total sample of 332 recruited for the *ReSource Project*, 305 participants completed the RAGE. In Chapter 4, a further 12 participants were excluded due to data quality issues leaving a final sample of 293 (170 female, mean age = 40.4, range: 20-55, SD = 9.3). Furthermore, as a step in the development of the paradigm used, a demographically matching sample of 32 (15 female, mean age = 30.3, range 21-51, SD = 9) underwent the procedure, allowing us to provide within-study replication of our findings. Finally, as the *ReSource* study included extensive psychometric assessment, we were additionally able to investigate the degree to which endogenous emotion generation abilities are associated with beneficial — or pernicious — affect and coping styles, A further 5 participants had incomplete questionnaire data, leading to a sample of 288 in Chapter 7 (168 female, mean age = 40.42, range: 20-55, SD = 9.29).

3.3.2: Long-term meditation practitioner study

Chapters 5 and 6 are based on data acquired in the context of a project investigating expert, long-term practitioners (LTMs; $N = 17$; 5 women; 45-62 years, mean ± SD age = 56 ± 5 years) of loving-kindness (LKM) and compassion meditation (CM), together with a control group matched on age and sex (5 women; 46-63 years, mean ± SD age = 54 ± 6 years), education level (LTM: mean ± SD years of education = 14 ± 3 years; Controls: 14 ± 3 years)

and IQ (103.6 ± 20.0 vs. 102.4 ± 28.5; assessed using Raven's Progressive Matrices). Moreover, participants and controls had equivalent affective status, reporting similar, subclinical symptoms of depression and anxiety (Beck's Depression Inventory (Beck et al., 1996); mean ± SD in LTM: 5.9 ± 3.9; controls: 6.6 ± 7.0; $p > 0.7$; Spielberger Trait Anxiety Scale, (Spielberger et al., 1983) mean ± SD in LTM: 37.3 ± 9.9; controls: 37.0 ± 9.1; $p > 0.7$). As reviewed in Chapter 2.3.2, this family of meditation techniques is characterised by the active generation of positive emotions of caring and compassion in a meditative setting, making them an "in-the-wild" example of endogenous emotion generation. A strong test of our component process model of EnGE would be to find evidence of structural plasticity within the relevant networks in people who have trained EnGE. Given the, on average ~40,000 hours of practice our sample reported in these techniques, this allowed us to investigate the effects of emotion generation expertise in terms of long-term brain plasticity, investigating whether extensive practice in EnGE resulted in structural changes in our putative generation network. Chapter 5 investigates whether there is evidence of brain plasticity as a function of emotion generation expertise, by investigating cortical thickness changes in practitioners compared to the group of matched controls. Chapter 6 is focused on a subsample (n=15) of practitioners (5 women; age range=45-62 years, age mean = 56.1 SD=4.6 years) who further completed a task designed to investigate how EnGE abilities in the form of compassion meditation can be used to regulate emotional reactions to exogenous negative stimuli.

3.4. Experimental methods

As the study of EnGE as a process in its own right is a relatively underdeveloped topic, its investigation required the adaptation or development of new experimental methods. The rationale guiding these experiments and their development is provided in this section, as well as an account of how their design allowed us to address our core hypotheses.

3.4.1. Regulation and Generation of Emotion: The RAGE paradigm

Based on our considerations in Chapter 2, we developed the Regulation And Generation of Emotion (RAGE) task (see Figure 3.1) as a means to sample EnGE abilities as they naturalistically occur in a normal population. Based on pilot data where participants were allowed participants full freedom in how to generate positive and negative emotional states, it was found that participants tended to gravitate towards using multiple modalities and that these primarily included variants of episodic imagery (i.e. mentally envisaging a narrative), semantic analysis (i.e. thinking about a situation in terms of words), bodily interoception (i.e. focusing on different bodily sensations associated with emotions) and auditory imagery (i.e. mentally recalling emotion-evoking music or the timbre of voices). Replicating previous research, participants reported both that they could generate emotions with relative ease (Wilson-Mendenhall et al., 2013), and that the emotional states thus generated were subjectively experienced as real emotional states (Riquelme et al., 2015; Salas et al., 2012). The aim of the main experiment was to retain as naturalistic a measure as possible, such that we could measure EnGE abilities as they occur in the normal population in terms of evoked subjective affect, while at the same time retaining experimental control such that meaningful neural and psychophysiological measurements could be performed. As such, participants were permitted to generate emotions in whichever way they themselves felt would elicit the most robust emotional states. Further, they were allowed to generate either high or low arousal exemplars of positive and negative emotion. We opted for a short-epoch design inspired by extant studies of extended emotion regulation and emotion generation processes (Goldin et al., 2008; 2005). This short epoch design was chosen as it would allow us to differentiate processes based on their temporal activation patterns, in accord with our neural component-process model. The epoch was split into a Generation phase, in which participants generated positive or negative emotions, and a Modulation phase, where participants either maintained

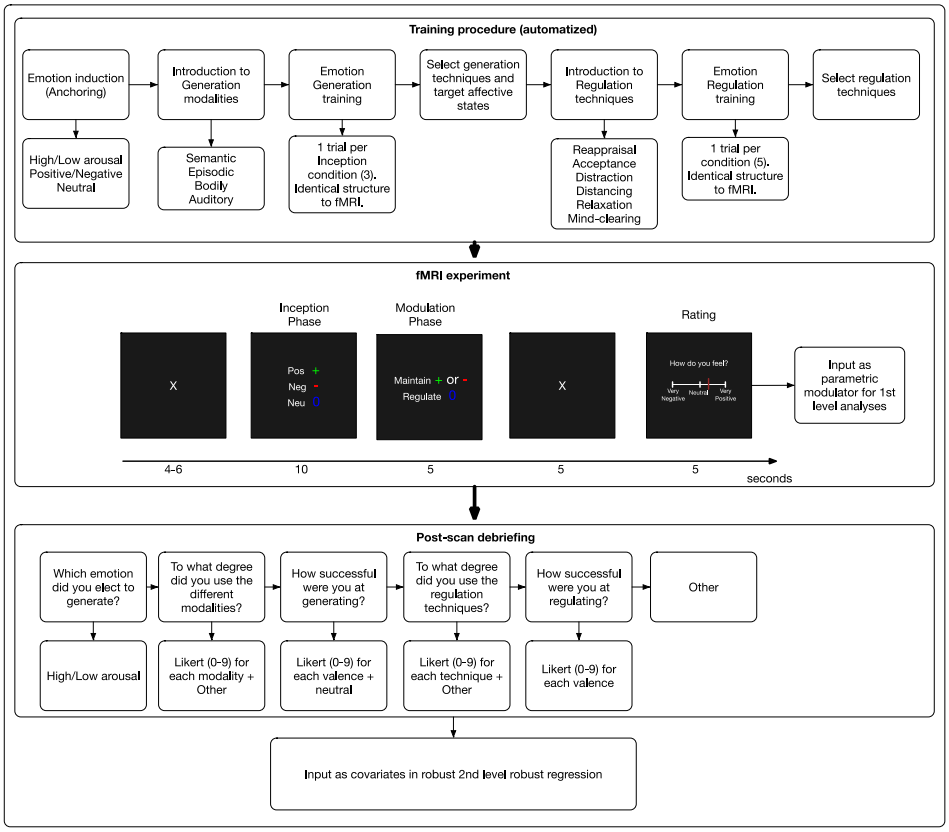


Figure 3.1. The RAGE paradigm. Schematic overview of the complete experimental procedure for the RAGE paradigm.

their generated state or down-regulated it, again using whichever method they best saw fit. Importantly, this allowed us to differentiate systems involved in the initial generation of the emotional state from those involved in the elaborated representation and maintenance of these states over the course of time.

Previous work on EnGE has reported response to depictions of hypothetical scenarios (Oosterwijk et al., 2012; Wilson-Mendenhall et al., 2011; 2013) or autobiographical memories (George et al., 1996; Liotti et al., 2000; e.g. Mayberg et al., 1999). As the goal of these studies were primarily to investigate the neural correlates of the emotional states thus elicited, they depended to different degrees on the presentation of a short-form version of the

target scenarios via verbal cues or auditory short-form versions of the scenarios. This means that the results from these studies potentially conflate processing of external information with processes central to EnGE as we conceptualise it here, particularly pertaining to goal-directed retrieval of information from long term memory. To avoid these problems, we opted for a purely cue-based approach in EnGE abilities. In different contexts, such cue-based paradigms have been used in the past to investigate the endogenous processing of emotional information (Singer et al., 2004; e.g. 2006) and tends to provide robust and circumscribed estimates of the neural mechanisms supporting internal mental processes (see e.g. Lamm, Decety, & Singer, 2011).

A problem with this cue-based approach is that one runs the risk of losing experimental control by not having an external referent (e.g. a visual stimulus) entraining processing between individuals. To counteract this, participants completed a supervised computerised training session prior to entering the scanner. First, to ensure that participants all generated similar emotional states, they underwent a multimodal emotion induction in which high and low arousal, positive and negative emotional states (as well as a neutral, baseline state) were induced using normed affective material, including musical clips, images and verbal descriptions of the target emotional states and their bodily concomitants. In addition to ensuring similar representations of the goal emotional states, this induction was designed to match the primary four modalities reported used in our piloting (i.e. auditory imagery, episodic imagery, semantic analysis, and bodily interoception). Following induction, participants selected whether they generated high or low arousal exemplars of positive and negative emotion, in addition to *how* they generated them by selecting one or more of the four modalities to use in combination, or self-define a means, based on how they themselves were best able to generate their elected target states, as determined by three practice trials. Thus, the approach taken was to keep the target emotional states as constant as possible between participants, but allow them to elect the methods by which to achieve them, ensuring optimal

implementation across individuals. Moreover, by having individuals generate both positive and negative states we could for the first time distinguish individual differences in EnGE abilities *per se* from the ability to generate specific emotional states — of importance for distinguishing between the buffering and fluency hypotheses of EnGE-based emotion regulation in Chapter 7.

The self-selection of generation methods also served two important functions by of allowing the answering of important questions not addressed in previous literature: First, it allowed the differentiation of neural systems supporting the implementation of EnGE reliant on modality-specific information (e.g. using episodic imagery or semantic analysis) from those supporting the elaboration of subjective experience of EnGE itself, i.e. in a modality-independent manner. This is of particular importance as it allows a means of differentiating component processes supporting the generation of core affect from those supporting their conceptualisation (i.e. their interpretation). Second, and in accordance with the goal of naturalistic measurement of EnGE abilities, allowing individuals to utilise their optimal EnGE approaches enabled us to investigate trait EnGE abilities in an objective fashion. This is important because previous studies have been limited to investigating either EnGE via a single modality (most often episodic imagery), or have investigated the efficacies of different modalities (e.g. episodic imagery vs semantic analysis as in Bergman & Craske, 2000; Holmes et al., 2006). However, EnGE as it naturalistically occurs is likely to be multi modally implemented, much like imagination or general cognition (Barsalou, 2008). As such, individual differences in usage of a given modality (e.g. semantic analysis) might not adequately capture EnGE abilities in general, or conceal non-linear effects of combining modalities. By allowing participants to freely combine modalities to whichever degree they felt most efficacious this question could be addressed.

Finally, the RAGE allowed the differentiation between circuits supporting the *de novo* generation of emotional states and those supporting their subsequent modulation or down-

regulation. Participants trained down-regulation using one or more of six different emotion regulation tactics derived from the literature (Reappraisal, Distancing, Distraction, Relaxation, Clearing, Acceptance) or a self-defined alternative. Participants were instructed to use the technique or combination of techniques to whichever degree they found was the most efficacious for both valences, as determined by a set of the trials in which participants both generated and regulated their emotional states. Moreover, by including partial trials (Ollinger, Shulman, & Corbetta, 2001) where the Modulation phase was omitted, the neural signatures of active maintenance of emotional states could be differentiated from those associated with either volitional down-regulation of those states or natural decay (see Figure 3.2). Together, this design allowed testing of the neural component process architecture proposed in Chapter 2, by investigating the activation dynamics of networks using a similar procedure to that successful used in the study of the neural processes supporting constructive memory (Addis et al., 2007; 2009; Daselaar et al., 2008).

Summarising, the RAGE affords the investigation of individual differences in EnGE abilities by allowing individuals optimal implementation. Additionally, the paradigm allows the investigation of implementation methods in terms of both absolute efficacy and potential combinatorial effects, as well as allowing the differentiation of modality-specific from core emotion generation systems in the brain. Finally, by having participants both generate and regulate endogenous emotion it was possible to differentiate neural systems specifically related to the generation of emotion from those supporting general emotion control. In the current thesis data from RAGE was used to 1) investigate the neural mechanisms supporting EnGE (Chapter 4) and 2) investigate whether EnGE abilities were predictive of emotion management styles and trait affect (Chapter 7). The latter allowed the testing of the fourth hypothesis proposed in Chapter 2, that EnGE should be related with trait differences in emotion management skills and, importantly, mediate the relationship between these skills and trait affect.

3.4.2. "State-fMRI" of loving-kindness meditation

The second hypothesis of this thesis is that there should be discernible structural changes of the proposed EnGE-network in individuals with expertise in the self-generation of emotion. In Chapter 5, the neural correlates of EnGE-expertise were explored, by investigating the overlaps between cortical thickness in the sample of LKM and CM (see Chapter 2.3.2) experts relative to a control group. Importantly, previous work has shown that LKM training reliably improves experienced positive affect, but that this is increase is reliant on persistent engagement in LKM (Cohn & Fredrickson, 2010; Fredrickson et al., 2008). As such, LKM appears to directly result in the generation of positive affective states. Moreover, LKM is a highly standardised technique such that LKM-based emotion elicitation is likely to progress in a similar manner across individuals. This means that it is a good candidate for elucidating the neural mechanisms supporting expertise in EnGE. To ensure that any findings were specific to LKM, a multi-modal approach was adopted, comparing the cortical thickness changes with results from a functional MRI investigation based on resting state methodology. In a typical resting state fMRI experiment (rs-fMRI) participants are scanned without any particular task and are simply requested to either rest while fixating on a cross or with eyes closed. By investigating either the time-series or amplitude of low frequency fluctuations of voxels in the brain, an extensive literature (Buckner et al., 2008; Raichle & Snyder, 2007) shows that while doing this, the brain tends to enter a discrete state characterised by engagement of the Default Mode Network mentioned in Chapter 2 (Buckner, Krienen, & Yeo, 2013). While traditionally conceived of as a "task-negative" or "off" mode of the brain, recent evidence strongly suggests that this activation pattern is characteristic of the state of mind-wandering or self-generated thought (Andrews-Hanna, 2012; Andrews-Hanna et al., 2014; Smallwood & Schooler, 2015). As such, the resting state of the brain is more properly thought of as an active, generative cognitive state that, importantly, appears to be distinguishable from mental states imparted by meditation (Mrazek, Franklin, Phillips, Baird, & Schooler, 2013).

At the same time there is increased interest in investigating the neural bases of discrete brain states, with evidence suggesting that such states are supported by the large scale coupling and co-activation of networks supporting the function of said brain state (C. D. Gilbert & Sigman, 2007; Milazzo et al., 2016). Thus, in Chapter 5 activation in the resting state was contrasted with that occurring when our sample of LTMs engaged in LKM, reasoning that this should reveal neural activation specifically related to the generation of positive affect, rendering novel insight into the neural bases of the state of EnGE. Specifically, as we contrasted the generation of LKM with the resting state—which itself involves generation of cognitive material—findings should engage primarily the neural substrates of generating an emotional state of loving kindness. Moreover, as the main topic of Chapter 5 was to investigate how expertise in emotion state generation using meditation influenced brain morphology, this allowed us to differentiate effects structural alterations specific to LKM from those reflecting general long-term meditation practice.

3.4.3. EnGE as a means to counter with exogenous stressors

The third hypothesis of the thesis is twofold, stating 1) that using EnGE in response to external stressors is a viable means of emotion regulation, and 2) that such emotion generation should rely on largely the same neural network as described above. In chapter 6, this is tested by investigating the neural and behavioural correlates of using compassion meditation (CM) to regulate emotional reactions in response to negative external stimuli. A major difference between LKM and CM is that CM involves both the generation of a state of loving-kindness and the application of that state to a specific person in the world that is in a state of suffering (Goetz et al., 2010). As such, compassion meditation can be thought of as a means by which emotions can be regulated in response to negative events in the world, particularly events social in nature. This is of some importance as recent evidence suggests that standard emotion regulation techniques can result in emotional bluntness or callousness (Cameron & Payne,

2011), suggesting that they might not be particularly well suited to dealing with emotional situations where empathy is required (Singer & Klimecki, 2014). Critically, CM involves the active generation of positive affect meaning that it is an apt model technique for testing the buffering hypothesis of EnGE based emotion management.

Chapter 6 investigates the efficacy of CM to regulate emotional reactions to negative socio-affective film stimuli depicting people in distress was tested. The paradigm employed was modelled on a previously published emotion regulation paradigm optimised for investigating the temporal dynamics of neural processes supporting different emotion regulation strategies (Goldin et al., 2008; see Figure 3.3). Using a slow event-related design, participants were presented with negative film stimuli previously shown to elicit strong negative affect (Klimecki et al., 2014), with matched neutral film clips serving as an induction check. Participants were instructed to either passively view the film clips or use CM to regulate their emotional reactions, allowing a contrast between the conditions to reveal the neural signatures of using EnGE as a means of dealing with negative environmental stressors. Additionally, participants regulated their emotional reactions using positive Reappraisal, an emotion regulation strategy that involves the active generation of alternate interpretations (appraisals) of emotional stimuli, thereby altering their emotional meaning (Blechert et al., 2015; Buhle et al., 2014; J. J. Gross, 2015b; McRae, Ciesielski, & Gross, 2012a) . In addition to being a high leverage strategy, Reappraisal is the most thoroughly investigated strategy of the emotion-regulation literature and can be thought of as a "gold-standard" against which the efficacy of other strategies can be gauged (Blechert et al., 2015; Buhle et al., 2014; Goldin et al., 2008; J. J. Gross, 2007b). Critically, positive reappraisal and CM involve engaging with negative stimuli in markedly different ways: Reappraisal involves inhibiting a prepotent and immediately given negative interpretation and replacing it with a positive interpretation (Blechert et al., 2015; Buhle et al., 2014; Goldin et al., 2008; J. J. Gross, 1998a; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). CM, conversely, has no inhibitory

component (at least conceptually), but rather the pivots on the *de novo* generation of a positive emotional state that is then applied to the object of negative affect (Singer & Klimecki, 2014). Thus, reappraisal and compassion should be similar in their reliance on generation materials, but differ in terms of both experienced affect, with CM resulting in more mixed emotional states, and concomitant neural consequences. Importantly, evidence already exists on how reappraisal differs from different types of emotion regulation using distraction (Dörfel et al., 2014; Kanske et al., 2011; McRae et al., 2010) in terms of both behavioural outcomes and neural bases, allowing one to infer to what degree CM-based meditation is similar to either of these techniques. Overall, this paradigm allowed us to identify which of the three possible mechanisms of reactive EnGE-based emotion management (discussed in Chapter 2, 2.7.2) best characterised CM.

3.5. Methods and analyses

To test the hypotheses proposed in Chapter 2, a variety of measures were used, spanning from questionnaire measurements of trait affective styles to measures of the thickness of the cerebral cortex. Here I provide an overview of the methods used, the analytical approaches taken and the rationale guiding their use vis-a-vis testing the core hypotheses of the thesis.

3.5.1. Assessing the neural architecture of EnGE

In the experiments described herein three main approaches were taken to investigate the neural mechanisms supporting EnGE: 1) Measurements were performed of what neural regions are activated during EnGE relative to neutral baselines suggesting that they encode different processes, 2) network-focused methods were used to investigate how different neural regions correlate during EnGE, and 3) how expertise and long-term practice of EnGE is associated with changes in the structure of the brain. Of these, the first two rely on functional

magnetic resonance imaging (fMRI) which measures neural activity via the proxy of hemodynamics, while the latter relies on measuring the relative quantities of protons in different tissue types. In the following I will provide an overview of the methods employed in each of the three domains.

3.5.1.1. Activation-based fMRI

The measurement of neural activation via the proxy of hemodynamics using fMRI is a cornerstone technique in modern cognitive and affective neuroscience, allowing the measurement of fluctuations of neural activation by proxy of measuring changes in blood-oxygen level dependent (BOLD) signals in the brain (Logothetis, Pauls, Augath, Trinath, & Oeltermann, 2001), with millimetric accuracy. As such, the BOLD signal is a cumulative signal, measuring the mass activity of neurons, with the guiding assumption being that increased neural activation results in increased metabolic demands, and consequently larger BOLD signals. Importantly, BOLD is not an absolute measure of neural activity, since even inactive neurons utilise oxygen for their maintenance. Temporally, the resolution of the fMRI signal is limited by two sources: First, it is an indirect measure of cardiovascular effects resultant on neural activity signals are temporally shifted by ~4-6 seconds in normal individuals and extend over a period of about 20 seconds following the onset of a stimulus (Handwerker, Ollinger, & D'Esposito, 2004). Second, a brain-wide acquisition of this signal usually takes around 2 seconds, which limits the sampling rate of this signal at any given point in the brain. As such, fMRI as a method is best used to investigate neural processes that extend over time. Similarly, by investigating how signal in voxels co-varied with usage of different generation modalities, we could identify regions whose activation likely supported implementation of modality usage.

In the current thesis activation patterns were investigated in three different ways. First, differential BOLD signals in brain regions as a function of experimental conditions were

investigated. This is the mainstay approach for fMRI-based cognitive neuroscience, and involves convolving a design matrix with a canonical hemodynamic response function (Friston, 1995). This allows the parcellation of an fMRI time-series into events, allowing the differentiation of discrete phases of an experiment in terms of the BOLD signal at each voxel. Importantly, this allows estimation parameters of signal strength in different conditions. Next, basic cognitive subtraction logic can be applied, in which brain-wide signal in a baseline measurement is subtracted from the signal apparent in an experimental condition of interest to reveal brain regions that encode, or are sensitive to, the process in question. For instance, in Chapter 7, participants were presented with negative and neutral videos. By subtracting the BOLD signal in the negative condition from the neutral condition, brain regions that responded stronger to specifically emotional content in videos could be identified. Moreover, by investigating modulations of the activation strength by subjective report, we could identify brain regions that parametrically varied as a function of the intensity of experienced emotional states, consistent with them supporting the generation core affective aspects of emotional experiences (see e.g. Heinzel et al., 2005).

One limitation of this approach is that it is inherently tied to an experimental design and thus cannot be used to address questions of brain activity during different cognitive states, that typically have a longer extent than e.g. a few seconds. Moreover, the reliance on a design matrix to parcellate fMRI time series in the above approach means that it is not well-suited to explore the neural bases of states that are not particularly amenable to translation into an experimental task, such as self-induction of meditative states. In Chapter 5, this was circumvented by investigation of the amplitude of low frequency fluctuations (ALFF; Yang et al., 2007; Zou et al., 2008) of fMRI signal during the meditation state of loving-kindness. ALFF is a spectral method that quantifies the degree to which a given brain region shows evidence of slow fluctuations (0.01-0.08 Hz) of BOLD signal. While traditionally applied as an individual difference measure of resting state activation, ALFF has been shown to vary

within-person as a function of stimulation (Jao et al., 2013; Yan et al., 2009) and the content of thoughts during task-free scanning (Gorgolewski et al., 2014; Kühn, Vanderhasselt, De Raedt, & Gallinat, 2012), suggesting that it is also sensitive to changes in activation corresponding to cognitive states. By contrasting ALFF during LKM with that of a normal resting state, the neural basis of a sustained state of EnGE could be investigated. Moreover, as the resting state is itself generative (as discussed in Chapter 2) and is frequently loaded with affective material (Gorgolewski et al., 2014) this contrast should allow us to identify neural systems specifically involved in the controlled generation of emotional states.

Finally, in Chapter 4, masking logic was used to provide a nuanced test of dynamic hypotheses regarding the component processes supporting EnGE. Masking is, in effect, a simple logical procedure, enabling one to identify conjunctions and disjunctions of neural activations as a function of condition. As previously mentioned, the standard approach to mapping activations involves convolving a design matrix with an HRF. This means that any reported activation is, in effect, a measure of the relative fit of the empirical fluctuations in the signal to the predicted HRF given processing occurring at a given point in time. While this is unproblematic in the context of simple stimulus-response type processes, this means that extended, multi-component processes like the one proposed for EnGE will be blurred into one activation map. One way of getting around this is by having the design matrix include multiple regressors at different points of the process. We specifically propose that there is a differentiation between processes involved in the initial generation and subsequent elaboration of emotional experiences. Based on previous work (Addis et al., 2007; 2009; Daselaar et al., 2008) we therefore modelled the first 10 seconds and the subsequent 5 seconds of our 15 second trial separately. Second, our design included several conditions that we expected to differentially engage component processes, allowing us to parcel out activation associated with different processes (see Chapter 4 for more detail).

3.5.1.2. Network-based fMRI

A different way of approaching the question of how function is reflected in brain dynamics is to investigate modulations of correlations between different regions as a function of conditions. This is of particular relevance for the current work as we specifically propose that different component processes are instantiated by different functional networks. We investigated this in Chapter 4 using complementary data-driven and model-based approaches.

Constrained principal components analysis (Woodward et al., 2006) is multivariate method that draws on the fact that any given fMRI measurement has several thousands (in our case ~30000) time series measurements, one for each voxel. By using PCA, it is possible to lump these voxels together into components that are highly intercorrelated (Friston, Frith, Liddle, & Frackowiak, 1993) and thus are formally known as functionally connected networks (Friston, 2004), that tend to activate in a coordinated fashion. By constraining the investigated time series to e.g. the period following a task-instruction, it is thus possible to identify networks that are of special relevance to a given task. Importantly, the resultant networks are formed by their different dynamics during different conditions and not intrinsic connectivity, meaning that spatially overlapping, task-relevant functional units composed of multiple networks can be identified (Woodward et al., 2006; Woodward, Feredoes, Metzak, Takane, & Manoach, 2013). This is of special importance here, given our hypothesis that FPCN coordinates activation of both DMN and SN.

While CPCA can be used to provide insight the dynamic functional organization of a given task based on its condition structure, it is silent about how between-subject variance in implementation of a task is reflected in brain networks. To get at this, we used whole-brain mediation analyses (Wager et al., 2008) to identify brain networks supporting usage of a given modality to generate emotion, by performing a whole-brain search for voxels that mediated the relationship between activation of a region and self-reported modality usage (i.e. a mediation of a correlation between brain activity and behaviour). This analysis had two

goals: First, we wanted to investigate the neural networks supporting implementation of each modality, seeking support for our notion of the embodiment of EnGE. Second, we sought to identify networks shared by all modalities, as these most likely support general EnGE processes such as representation or controlled retrieval, in line with our hypothesised component process structure.

3.5.1.3. Cortical thickness

Finally, Chapter 5 investigated how EnGE expertise was related to structural plasticity of cortical grey matter. This was done by measuring the thickness of the grey matter sheath of the previously described sample of LKM and CM experts and how it differed from that of matched controls. Cortical thickness is a sensitive proxy for total grey matter in a given region (Hutton, Draganski, Ashburner, & Weiskopf, 2009), with grey matter concentrations consistently showing expertise and training effects (Draganski & May, 2008). Thus, the involvement of a region in a given process can be inferred from changes in cortical thickness of that region with training. In lieu of a proper longitudinal design training EnGE abilities, we opted for an expert approach and dissociated meditation-general effects from LKM-specific changes using the "state"-fMRI method described above. Given that the meditators tested were specifically experts in meditation methods requiring EnGE, we therefore expected to find structural differences in the neural reference space for EnGE described in Chapter 2, possibly most centrally involving regions involved in the volitional initiation and shielding of mental states, i.e. the FPCN (Andrews-Hanna et al., 2014).

3.5.2. Psychophysiological measurement of embodied emotion

As discussed, the component process model proposed in Chapter 2 presupposes that EnGE results in embodied simulations of emotion states that are largely similar in terms of physiological reactions to emotional states elicited by external events. To verify this, we

investigated the relationship between EnGE and galvanic skin responses (GSR) concurrent with the RAGE scanner session, acquired using two Ag-Cl electrodes attached to the left index and middle fingers. Using Continuous Decomposition Analyses as implemented in the LedaLab toolbox (Benedek & Kaernbach, 2010a), we could distinguish phasic changes in GSR reflective of e.g. stimulus processing, from longer-duration trial-wise fluctuations in skin conductance levels (SCL). SCL is a commonly used (Kreibig, 2010) and highly reliable (El-Sheikh, 2007) measure of autonomic arousal, with evidence suggesting that SCLs are elevated as a function of increasing experienced emotional intensity (J. J. Gross, 1998a; Levenson, 2014; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005), at least in exogenously elicited emotional states. Observing a relationship between subjective ratings and SCL would therefore suggest that EnGE as implemented by our design had both subjective and physiological consequences, consistent with EnGE producing embodied simulations of emotional states and not merely cognitive representations of emotional situations.

3.5.3. Assessing the role of EnGE in trait affect and emotion management

As part of the *ReSource* study, participants completed a comprehensive package of questionnaires measuring a range of social and affective traits, including self-report measures of self-regulation capacities and trait tendency to use different emotion regulation and coping strategies, as well as several measures of affective styles and tendency to experience positive and negative emotional states in daily life. In Chapter 7 we utilised these measurements to investigate how individual differences in the capacity to generate emotion in the RAGE paradigm correlated with individual differences in trait affectivity and emotion management styles. This allowed a test of the hypothesis that EnGE can be actively used as a means of emotion management. Drawing on the multivariate nature of our data, we took a latent variable approach, and combined all relevant scales for trait affect and, separately, emotion regulation, self-regulation and coping technique usage using principal components analysis

(PCA). The resultant components (see Chapter 7 for more detail) can thus be taken as reflecting overall trait affect and tendency to utilise different families of emotion management. Taking the degree to which each participant loaded on each of these dimensions, we thus devised composite individual difference scores that were not biased by any particular formulation in any particular questionnaire, and thus likely constitute a measure of actual, self-experienced, affective style. Importantly, by having separate factors for trait affect and emotion management, we could validate our factors by establishing that loadings on emotion management style correlated in the expected fashion with trait affect.

A major problem to investigating the relationship between EnGE and emotion management styles is that the capacity to generate emotions are construably associated overall trait affect. Thus, a happy person might be more adept at generation of positive emotional states just because they are happy. Moreover, it is a known fact that trait affect is associated with usage of specific adaptive emotion management styles, meaning that any correlation observed could be simply because emotion management improves affect that in turn improves EnGE. We tackled this problem in two ways: First, we investigated both average and relative ability to generate both positive and negative emotion. Importantly, while investigating one of these parameters, we controlled for the other meaning that any relationships observed were specific to general or valence-specific EnGE abilities. Second, we adopted a causal mediation modelling approach, directly testing whether EnGE-abilities mediated the observed relationship between different kinds of adaptive (and maladaptive) emotion management styles and trait affect. This allowed us to investigate whether EnGE abilities showed evidence of being part of the mechanism supporting the beneficial (or detrimental) impact of emotion management style on trait affect.

3.5. Conclusion

The approach taken in this thesis is inherently multi-modal and multi-method in line with the stated goal of testing a comprehensive neural component process model of EnGE, as well as investigating the consequences of EnGE abilities for emotion management and well-being. the following four chapters, the manuscripts for the empirical investigations as they are published (Chapter 7; H. G. Engen & Singer, 2015) or currently under review in peer-reviewed journals are presented, whereupon Chapter 8 provides a summary and discussion of this work in terms of our hypotheses and Chapter 9 discusses limitations, implications and future directions for the current work.

Chapter 4: The neural component-process architecture of endogenously generated emotion[3]

Haakon G. Engen, Philipp Kanske, & Tania Singer

4.1. Abstract

Despite the ubiquity of endogenous emotions and their role in both resilience and pathology, the processes supporting their generation are largely unknown. We propose a neural component process model of endogenous generation of emotion (EnGE) and test it in two fMRI experiments (N = 32/293) where participants generated and regulated positive and negative emotions based on internal representations, using self-chosen generation methods. EnGE activated nodes of Salience (SN), Default Mode (DMN), and Frontoparietal Control (FPCN) Networks. Component processes implemented by these networks were established by investigating their functional associations, activation dynamics and integration. SN activation correlated with subjective affect, with midbrain nodes exclusively distinguishing between positive and negative affect intensity, and dynamics consistent with it supporting the initial generation of core affect. Dorsomedial DMN, together with ventral anterior insula, formed a pathway supporting multiple generation methods, with dynamics suggesting it is involved in the experiential representation of emotion. Finally, both SN and DMN coupled to left frontal portions of the FPCN which correlated with both subjective affect and representation formation, consistent with FPCN supporting the executive coordination of the generation process. These results support a component process mapping of EnGE onto major neural networks, providing a foundation for research into endogenous emotion in normal, pathological, and optimal function.

[3] [3] The current chapter is presently under review, and the final, published version, might show discrepancies from the present version. Presented here is the revision of the manuscript as of 24.06.2016.

4.2. Introduction

From melancholic reminiscence to joyful anticipation, we frequently experience emotions caused by internal mental processes, such as thoughts and memories (Killingsworth & Gilbert, 2010). Such endogenous emotion is described as richer and more intense than emotion elicited by external events (Salas et al., 2012), and is known to play an important role in affective psychopathology, such as depression (Nolen-Hoeksema et al., 2008)and anxiety (Freeston, Dugas, & Ladouceur, 1996). There is also evidence that the endogenous generation of positive emotional states can used as an effective means to regulate emotional reactions to external events (Engen and Singer 2015). Moreover, the trait tendency to do this is a predictor of psychological resilience (Tugade & Fredrickson, 2004). Thus, understanding the psychological and neural mechanisms of endogenous generation of emotion (EnGE) can yield important insight into normal, pathological, and even optimal emotional function.

Despite this, research into EnGE has been limited, stemming mainly from behavioural studies using EnGE as a method to induce emotional states. This research shows that EnGE can be occasioned by multiple information-processing modalities, including mental imagery and semantic analysis of emotional information (Vrana et al., 1986), interoception of bodily signals (Philippot et al., 2002)or recall of episodic autobiographical memories (Mayberg et al. 1999). It has also been shown that EnGE can effectively occur when individuals immerse themselves in hypothetical scenarios (Wilson-Mendenhall et al., 2013).This latter finding demonstrates the theoretically important point that EnGE is not limited to reinstantiation of previously experienced emotional situations, but can also simulate states appropriate for novel contexts. Indeed, emotions are frequently elicited by spontaneous cognition about future events (Ruby et al., 2013), suggesting that an important use of EnGE is predicting the affective relevance of hypothetical future scenarios (Baumgartner, Pieters, & Bagozzi, 2008). While these studies were not focused at exploring EnGE as a process in its own right, they show that multiple means (e.g. different strategies or different information modalities) can be

used to generate emotional states, dependent on the representational content of the target emotional experience. Mirroring recent constructivist theories of emotion (Barrett & Wilson-Mendenhall, 2014; Russell, 2014), this suggests that a comprehensive account of EnGE needs to distinguish between 1) processes supporting the generation of the hedonic or core affective quality of an endogenous emotional experience from 2) processes supporting the formation of representations of the context to which this affective state applies (or stems from). Importantly, this opens for the possibility that the two are mechanistically distinct, with neural systems supporting the generation of affective qualities being differentiable from those supporting the generation of elaborated experiential representations.

Presently, neuroimaging studies of EnGE using comparable protocols are limited, making evaluation of this hypothesis difficult. One exception is a series of early PET experiments in which participants generated emotional states by volitionally recalling significant emotional experiences (Damasio et al., 2000; Gemar et al., 1996; George et al., 1996; Kimbrell et al., 1999; Liotti et al., 2000; Mayberg et al., 1999; Pardo & Raichle, 1993; Reiman et al., 1997). Considered in aggregate (see *Appendix:* Figure A2.1.1), these studies implicate three large scale functional networks in EnGE: 1) The Default Mode Network (DMN; Raichle & Snyder, 2007), including ventromedial prefrontal cortex (vmPFC), posterior cingulate cortex (PCC), left temporoparietal junction (TPJ) and hippocampus (HC), 2) the extended Salience Network (SN; Seeley et al., 2007)including anterior insula (AI), dorsomedial PFC (dmPFC), and structures in basal ganglia and midbrain and 3) The Frontoparietal Control Network (Laird et al., 2011; FPCN; Spreng et al., 2010; 2013) centred on lateral and dorsomedial prefrontal, and inferior parietal cortices. There is a notable overlap between this putative neural architecture and that known to support the construction of mental representations in general: DMN is associated with numerous forms of psychological processes involving simulation based on endogenous information (Spreng et al., 2009), and appears to be involved in the integration of information about a given topic into detailed

episodic representations/simulations. Interestingly, DMN does not appear to support the initial generation of the representational core that these details pertain to (Addis et al., 2007). Rather, this initial generation is thought to involve the direct activation of domain-specific and task-relevant networks (Hassabis & Maguire, 2007)In the context of emotion, the SN is a likely candidate such a network. Composed of cortical (AI, dorsal ACC), limbic (amygdala (AMY), ventral striatum (VS)), and midbrain structures (periaqueductal grey (PAG), substantia nigra/ventral tegmentum (SN/VTA)), the SN is closely associated with the generation of core affect and homeostatic regulation (K. A. Lindquist & Barrett, 2012; Seeley et al., 2007). Interestingly, DMN and SN appear to be intrinsically anti-correlated (Buckner et al., 2013; Spreng et al., 2013), strengthening the claim that they support dissociable component processes in EnGE. This anti-correlation also suggests the need for an intermediary network coordinates and maintains activation of the SN and DMN, pointing to the need for executive processes to coordinate and maintain the generation process. Possibly, the FPCN supports this role as it is known to support adaptive cognitive control in general (Cole et al., 2013). FPCN is known to interface with SN (Dosenbach et al., 2008), affording a pathway by which core affective states can be generated in a goal-directed fashion. Similarly, FPCN and DMN are known to couple during goal-directed internal mentation (Spreng et al., 2010) and to be implicated in the domain-general control of retrieval processes important for representation formation (Badre & Wagner, 2007).

To the degree that this functional process architecture holds, an interesting question is how these processes interact over the course of a given EnGE event. Addis and colleagues (Addis et al., 2007; 2009; see also Daselaar et al., 2008) have shown that the construction of endogenous simulations of events involves distinct generation and elaboration phases, with the initial phase involving retrieval of the core semantic features of the representation and the subsequent elaboration phase involving the elaboration of the core information in question with details about the specific event, supported by the DMN. If this model holds for EnGE,

one would expect involvement of SN primarily in the early stages of process, corresponding to core affect serving as a semantic anchor for later elaboration efforts. Plausibly, however, the opposite could be true, such that generation involves setting up representations of emotional situations, which in turn elicit core affective states (Kross et al., 2009).

The objective of the current study was to investigate this and to establish a comprehensive neural component process architecture for EnGE. Based on the above considerations we expected EnGE to be neurally implemented by DMN, SN, and FPCN. Each of these networks were hypothesised to support separable component processes of EnGE. Specifically, we suggest that SN supports the generation of core affective states that serve as a guide for the formation of detailed representations via processes instantiated in the DMN, resulting in an emotional experience. Finally, we propose that FPCN supports the executive maintenance and coordination of the generation process, coupling with both SN and DMN. Importantly, as we propose they form the functional core of EnGE, we expect that these networks should partake in EnGE irrespective of the hedonic quality of the emotional state or the precise means or modality used to generate it. We tested this model in two experiments (N = 32 and N = 293) with a newly developed paradigm aimed at maximizing ecological validity and generalizability of EnGE. To ensure that participants generated comparable emotional states, they were anchored using a multimodal emotion induction procedure prior to scanning. This procedure elicited multiple markers of emotional states (semantic, visual, auditory, and bodily) prior to scanning, avoiding artificially biasing participants' implementation towards particular information modalities. In order to maximise ecological validity and task compliance, participants were instructed to implement EnGE as they experienced most efficacious. Thus, in Experiment 1 participants were given complete freedom in how they generated emotions, while in Experiment 2 they were allowed to combine four generation modalities (Semantic Analysis, Episodic and Auditory Imagery, and Bodily Interoception; i.e.

the endogenous analogues to the modalities used in the induction procedure), in whichever way they found most effective.

Participants then completed a cue-based fMRI paradigm (Figure 4.1 A). Trials consisted of a Generation phase and a Modulation phase. In the Generation phase, participants used their self-selected techniques to generate positive and negative emotional states, or actively attempted to remain neutral. Thus, we could distinguish the neural correlates of general emotion generation from those supporting generation of particular implementations of generation. In the subsequent Modulation phase, participants maintained this state (Maintain condition), actively suppressed it (Regulate condition), or simply ceased their generation efforts (Cease condition; Experiment 1 only). This approach enabled us to dissociate neural systems supporting different component processes based on their activation profiles. Finally, participants provided ratings of their affective states following each trial, allowing identification of the neural correlates of generation success.

4.3. Methods

4.3.1. Participants

For Experiment 1, 32 participants were recruited from an in-house participant database (15 female, mean age = 30.3, range 21-51, SD = 9). For Experiment 2, participants were recruited in the context of the large-scale longitudinal *ReSource Project* (see *Appendix:* A1.1.1 *ReSource eligibility criteria* for details on recruitment). Baseline data from this study were used. 332 participants were recruited for the *ReSource Project*, with 305 participants completing the current paradigm. Of these, 5 participants were excluded on account of missing auxiliary data (post-scan questionnaire, structural MRI) and technical difficulties. 4 participants reported difficulties (e.g. nausea or sleepiness) during the scanning session and were dropped from analysis. From the sample of 296 with complete data, a further 3 participants were removed due to aberrant behavioural report and/or unacceptable data quality

after preprocessing (> 1 voxel movement, > 5% corrupted time points, design VIF > 2), leaving a final sample of 293 (170 female, mean age = 40.4, range: 20-55, SD = 9.3). All participants had normal or corrected to normal vision. The study was approved by the Research Ethics Committees of the University of Leipzig (number 376/12-ff) and the Humboldt University in Berlin (numbers 2013-02, 2013-29, and 2014-10). All participants gave written informed consent, were paid for their participation, and were debriefed after the study was completed.

4.3.2. Experimental procedure

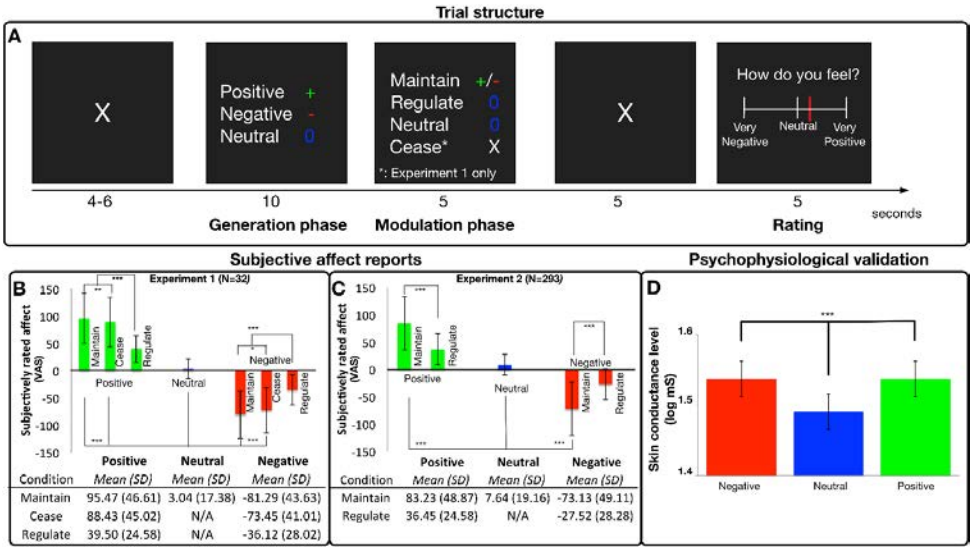


Figure 4.1: Experimental task, behaviour and psychophysiological validation. A) Schematic of a single trial. **B)** Subjective ratings of affect in Experiment 1. **C)** Subjective ratings of affect for Experiment 2. **D)** Main effect of Condition on skin conductance levels (SCL) in a subset ($N = 244$) of participants in Experiment 2. Error-bars = within-subject standard errors (Loftus & Masson, 1994). *: $p < .05$, **$p < .01$, ***$p < .001$.

Before scanning, participants underwent an automated training procedure (see *Appendix:* A1.1.2 *Training procedure*for details), including a multimodal emotion induction aimed at minimising between-participant variance in implemented emotional states. In Experiment 2, participants were also instructed in the use of four generation modalities

(Semantic, Episodic, Auditory, Bodily), and instructed to select to which degree they to use each in the following experiment according to their own preferences. Additionally, participants were shown a number of neutral stimuli (e.g. pictures of scenery) and instructed to actively attain the sort of neutral emotional state depicted in the Neutral and Regulation conditions (see below). After the scanning session, participants were debriefed. In Experiment 1 verbal debriefing was done with an experimenter. In Experiment 2 participants reported the degree to which they used each of the generation modalities using a 9 point Likert scale.

Each trial (Figure 4.1 A) started with a 4-6s white fixation cross indicating the start of trial. Then a 10s Generation phase was entered, in which subjects were shown a colored symbol indicating which emotional state to generate (Red minus = Negative, Green plus=Positive, Blue 0=Neutral). This was followed by a 5s Modulation phase where participants either maintained the generation of the emotional state or down-regulated it so as to attain a neutral emotional state. In the Maintain condition the instruction symbol remained the same as in the Generation phase. In the Regulation condition, the symbol changed to a blue 0. Finally, in Experiment 1 we included a partial-trial condition where the instruction cue changed to a fixation cross (Cease condition; Experiment 1 only). For the Neutral condition the symbol did not change, but remained a blue 0. Thus, Experiment 1 consisted of a total of 7 different conditions (Maintain Positive/Negative, Regulate Positive/Negative, Cease Positive/Negative and Neutral). Experiment 2 omitted the Cease condition and thus had a total of 5 conditions. Experiment 1 had two runs of 5 trials per condition (35 per run), while Experiment 2 had a single run of 10 trials per condition (50 total). Condition sequence was pseudo-randomised, ensuring no direct repetitions of conditions occurred. Finally, a 5s fixation cross was presented followed by a 5s presentation of a continuous Visual Analogue rating Scale ranging from “Extremely negative” via “Neutral” to “Extremely positive” (range

+/- 251 from the neutral point (0)). Initial cursor position was jittered randomly around the Neutral point. Participants responded using a button box and the right hand index and middle finger. Participants were instructed to report their affective state as it was at the moment of report. Stimuli were back-projected using a mirror setup. Task setup was identical in both experiments, except for the omission of the Cease condition in Experiment 2 due to time constraints.

4.3.3. MRI acquisition

For both experiments, MRI data was acquired on a 3T Siemens Verio Scanner (Siemens Medical Systems, Erlangen, Germany) using a 32-channel head-coil. High-resolution structural images were acquired using a T1-weighted 3D-MPRAGE sequence (TR = 2300ms, TE = 2.98ms, TI = 900ms, flip angle = 7°, iPat = 2; 176 sagittal slices, FOV = 256mm, matrix size = 240×256, 1^3 mm voxels; total acquisition time = 5.10 minutes). For the functional imaging, we employed a T2*-weighted gradient EPI sequence that minimised distortions in medial orbital and anterior temporal regions (TR = 2000ms, TE = 27ms, flip angle = 90°, iPat = 2; 37 slices tilted ~30° from the AC/PC axial plane, FOV = 210 mm, matrix size = 70×70, 3^3 mm voxels, 1mm gap). For Experiment 2 we acquired B0 field maps using a double-echo gradient-recalled sequence with matching dimensions to the EPI images (TR = 517 ms, TE = 4.92 and 7.38 ms).

4.3.4. fMRI preprocessing

Preprocessing was performed using a combination of SPM12 (r6225) functions and the ArtRepair toolbox (Mazaika, Whitfield, & Cooper, 2005) running on Matlab 2013b. Functional images were realigned (Experiment 1) or realigned and unwarped to additionally correct for distortion using B0 field maps (Experiment 2). ArtRepair procedures were then

employed, including slice wise artefact detection and repair using interpolation (art_slice; 5% cutoff), time series diagnostics (art_global) identifying and repairing via interpolation volumes showing large global intensity fluctuation (> 1.3 %), volume-by-volume movement exceeding 0.5 mm and overall movement (> 3mm), and despiking with a 5% signal change cut-off (art_despike). T1 structural images were registered to the mean realigned volume and segmented. Using DARTEL (Ashburner, 2007) procedures, functional images were normalised and smoothed with an isotropic kernel of 8mm FWHM.

4.3.5. 1st level fMRI analyses

Individual-level models included separate sets of regressors for the Generation and Modulation phase. For the Generation phase, 3 regressors were specified corresponding to the emotional target (Positive, Negative, Neutral) of the trial. For the Modulation phase, separate regressors were specified for each condition. Thus, the model in Experiment 1 included an additional 7 regressors (Valence (Positive and Negative) * Modulation (Maintain, Cease and Regulate) + Neutral), while the model in Experiment 2, where the Cease condition was omitted included an additional 5 regressors. Regressors were convolved with canonical HRF functions with a 10s (Generation) or 5s (Modulation) duration, as well as regressors specifying parametric modulations by trial-wise subjective affect ratings. An additional regressor was specified for the Rating period. Movement parameters derived from the realignment step (6 regressors), their derivatives and squared values were added (24 regressors). Potential physiological confounds were controlled for by adding four additional regressors reflecting volume-wise mean signal from white matter and CSF, global signal, and highest-variance voxel time-course. Finally, the models included parametric modulation regressors coding for trial-wise ratings for all regressors of interest.

4.3.6. 2nd level analyses fMRI analyses

All 2nd level GLM analyses were conducted using robust regression (Wager et al. 2005), with covariates of no interest coding elected arousal level, age and gender. 2nd level models for Experiment 2 additionally included regressors coding self-reported generation modality usage (4 regressors) as continuous covariates.

All results were corrected for multiple comparisons using cluster extent Family Wise Error Rate (FWEc) correction at an alpha of $p<.05$, unless otherwise indicated. Cluster extents were estimated using Monte Carlo simulation and estimated intrinsic smoothness (3DClustSim and 3DFWHMx from the AFNI package (Forman et al. 1995)), as implemented in the NeuroElf package. Note that peak-forming thresholds were adapted for Experiment 1 ($p<.001$) and 2 ($p<.00005$) to account for differences in sample size. Correlational and mediation results also used a less strict peak threshold of $p<.0005$.

All analyses were masked with a grey matter template derived from the DARTEL created template, thresholded at 95% grey matter probability, supplemented by a hand-drawn masks of brainstem nuclei due to poor differentiation of white from grey matter in these regions.

4.3.7. Constrained principal component analysis

In Experiment 2, we adopted a data-driven approach using constrained principal components analysis (CPCA; see Woodward et al., 2013 for details) of fMRI time series using the CPCA-fMRI package (www.nitrc.org/projects/fmricpca). CPCA analysis of fMRI data is a multivariate method that involves a singular value decomposition of BOLD time series to identify functional networks followed by an estimation of BOLD change in each network over peristimulus time as a function of experimental condition. Here we used finite impulse response (FIR) modelling to identify task-specific functional connectivity networks based on the 15 bins (i.e. 30 seconds, allowing for hemodynamic lag) following the onset of

the generation cue. Importantly, using a FIR model allows hemodynamic response (HDR) profiles to be identified for each component separately, allowing the identification of task-relevant functional connectivity networks with dissociable temporal profiles. Finally, CPCA provides HDR estimates at the individual level, allowing the resultant predictor weights to be used to explore the correlates of individual differences in component activation.

4.3.8. Mediation analyses

To differentiate components of the generation network involved in generation using a specific modality from components involved in generation in general, we followed previous work aimed at identifying the large scale networks supporting emotion regulation performance via mediation modelling (Denny, Ochsner, Weber, & Wager, 2014). First, regions whose activation during generation of emotion (relative to neutral) was identified using robust regression. Mediation Effect Parametric Mapping (MEPM) as implemented in the M3 mediation toolbox (Wager et al., 2008) was used to investigate modality specific and modality general pathways of emotion generation. We performed a whole-brain search for voxels whose activity during emotion generation (relative to the neutral baseline) showing a relationship with reported use of a given modality that was mediated by the activity in regions independently correlated with usage of that modality in a robust regression model. Statistics were assessed using the bootstrapping approach implemented in the M3 toolbox (10,000 samples).

4.3.9. Analysis overview

The first objective of our analyses was to establish the overall neural architecture of EnGE. To achieve this, we first sought to establish the validity of our experiment by investigating subjective and physiological indices of emotional states. Next, we contrasted combined positive and negative EnGE with the neutral baseline, thereby identifying the overall neural basis of EnGE. We next sought to test the component process mapping

proposed in the introduction in two ways: First, based on the data from Experiment 1, we enacted a contrast-based decomposition, based on a model of the activation dynamics expected for each of the component processes. To complement this, we next performed a data-driven decomposition of the data from Experiment 2 using CPCA, to identify the functional networks central in EnGE. Together, the results from these three analyses allowed a description of the overall network and functional subcomponents supporting EnGE in general. Following on this, the second objective of the analyses was to differentiate general EnGE networks from those supporting specific implementations of EnGE, such as the generation of a particular valence, or using a specific modality. By investigating how subjective ratings for positive and negative generation parametrically modulated signal, we could differentiate regions activated in a valence-specific manner from those supporting specifically the generation of positive and negative emotional states. Finally, by investigating the correlation of activation with reported usage of different modalities, we could identify specific regions supporting modality-specific implementation, and, using mediation analysis, identify the networks supporting EnGE modality usage. Moreover, by comparing these networks we could differentiate parts of these networks supporting specific modalities from those supporting EnGE in general.

4.4. Results

4.4.1. Behavioural and psychophysiological validation

Our first objective was to validate our experimental design, using a combination of behavioural and psychophysiological measures to ascertain that participants were able to generate and regulate emotional states as measured by subjective and objective markers of emotional arousal.

Experiment 1			*t*-value (df = 31)	
Comparison			*Positive*	*Negative*
Maintain	vs	Neutral	11.25***	-10.50***
Cease	vs	Neutral	10.94***	-9.50***
Cease	vs	Maintain	-3.27**	2.43*
Regulate	vs	Maintain	-7.79***	6.88***
Cease	vs	Regulate	6.96***	-6.08***

Experiment 2			*t*-value (df = 292)	
Comparison			*Positive*	*Negative*
Maintain	vs	Neutral	27.24***	-27.94***
Regulate	vs	Maintain	-19.44***	20.69***

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 4.1: Comparison of self-reported experienced affect. Descriptives are reported in Figure 4.1

Post-trial ratings were analysed using paired t tests, reported in Table 4.1. Fig 4.1 B shows subjective ratings in each condition for Experiment 1. Relative to the Neutral baseline condition, increased reports of corresponding affect were observed for both Maintain and Cease conditions. The Cease condition also showed significantly higher ratings for both positive and negative affect compared to their respective Maintain conditions. Finally, regulation resulted in decreased ratings for both positive and negative emotion relative to the respective Maintain conditions. Figure 4.1 C shows subjective ratings as function of condition for Experiment 2. Relative to the neutral baseline condition, increased reports of corresponding affect were observed for both positive and negative Maintain conditions. Regulation conditions also showed decreased ratings for both positive and negative affect, relative to their respective Maintain conditions. These results demonstrate that participants were subjectively able to generate and regulate endogenous emotional states of both positive and negative valence in both experiments. Importantly, they also show that, while a generated emotional states decay without active maintenance, they remain subjectively significant for at

least a short time following generation, consistent with the representation of the emotional state persisting even without active generation efforts.

We next sought to establish whether participants' generation efforts also elicited objective emotional arousal responses. To this end, we concurrently assessed elicited skin conductance levels (SCL) in Experiment 2 (see *Appendix:* A.1.1.3. *Psychophysiological Data Acquisition and Preprocessing*). 225 recordings had acceptable data quality, and were used to investigate the impact of generation instructions on objective measures of emotional arousal, as well as their interaction with subjective ratings. As SCL is the most frequently reported measure in investigations of exogenously induced emotional states (Kreibig, 2010), an interaction would suggest that the elicited states can be construed of as bona fide emotional states and that behavioural ratings can be taken as proxy for emotional arousal. Using linear mixed modeling of trial-wise SCL responses during the Generation period, we predicted the trial-wise log-transformed estimates of skin conductance level measured in microsiemens (μS) using a subject-level random intercept model. The model further included a factorial fixed effect for condition (Generate Positive, Generate Negative, Neutral) and a continuous fixed covariate for scaled trial-wise ratings of subjective affect. To control for potential learning/fatigue effects, trial number was entered as a nuisance covariate (for more detail on the effect of fatigue in the current experiment, please see *Appendix:* A1.1.5. *Assessing the effect of fatigue on emotion generation*). This analysis revealed a main effect of Condition [$F(2, 11012.639)= 3.155$, $P < 0.05$], Rating [$F(1, 11013.700)= 4.625$, $p < 0.05$], as well as a Condition*Rating interaction [$F(2, 11014.119)= 17.815$, $p < 0.001$]. Bonferroni corrected t tests (Figure 4.1 D) were performed to clarify the main effect of Condition, showing that, relative to the neutral [mean = 1.205, SE =.0.32] baseline condition, higher SCL levels were observed for both negative [mean = 1.267, SD =.032; paired t test: $t(224) =4.44$, $p < 0.001$] and positive [mean = 1.268, SD= .032; paired t test: $t(224) =5$, $p < 0.001$] emotion generation conditions. Closer investigation of the Condition*Rating interaction showed that it consisted

of a significant difference in the slopes of the rating effect between negative and positive generation [$t(8770.515) = 5.63$, $p < .0001$]. Specifically, SCL had a negative relationship with ratings [$t(4278.64) = -3.71$, $p < .001$] during negative generation, and a positive relationship [$t(4278.64) = 4.16$, $p < .001$] during positive generation. Corresponding to the bipolar scale used (see Figure 4.1A), this shows that SCL levels increased with stronger affect ratings for both positive and negative emotion (Figure 4.1E). These results show that participants were capable of generating both positive and negative emotional states, as measured by both subjective and objective indices of emotional arousal, and these indices were correlated, such that behavioural report corresponded to objective physiological arousal.

Finally, we sought to explore what kind of emotional states participants elected to generate. Thus, during debriefing, participants in Experiment 2 were asked whether they generated high or low arousal exemplars of positive and negative emotional states after the experiment. 39% of participants reported generating high arousal positive emotional states, like joy or happiness, with the complementary 61% generating low arousal positive emotion like calmness or caring. Similarly, 29% of participants reported generating high arousal negative emotions like fear or anger, while 71% reported generating low arousal states like sadness or melancholia. All subsequent analyses in Experiment 2 control for this between-subject variance.

4.4.2. Exploring the neural architecture of EnGE

Our next objective was to establish whether our hypothesised three-network architecture of EnGE was in evidence. To identify the neural correlates of emotion generation we contrasted the combined Generation and Maintenance periods for both positive and negative affect generation with the *Neutral* baseline condition, with one sample *t* tests performed using robust regression (Wager, Keller, Lacey, & Jonides, 2005). For Experiment 1, a primary cluster-forming threshold of $p < .001$, $T > 3.38$ was used. In Experiment 2, a

more stringent threshold of $p < .00005$, $T > 3.95$ was used for the primary contrasts to balance increased power. Using Monte-Carlo simulation (Forman et al., 1995), cluster thresholds were determined to be $k > 40$ and $k > 10$, respectively, for FWEc $\alpha < .05$.

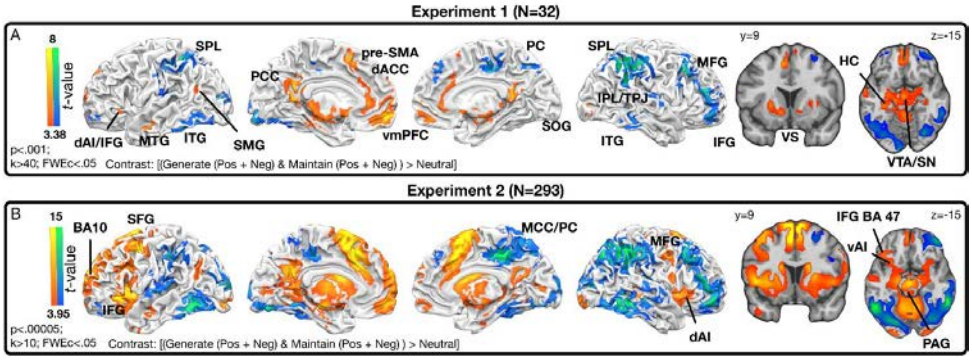


Figure 4.2: Core networks of endogenous emotion generation A) Regions activated in Experiment 1 for the Generation conditions (Positive and Negative Generate & Maintain) relative to the Neutral condition. **B)** Equivalent contrast for Experiment 2.

In Experiment 1 (Figure 4.2A, *Appendix*: Table A3.1.1) we observed activation in core nodes of the DMN (vmPFC, PCC, left TPJ, left middle temporal gyrus (MTG) and hippocampus), and SN (AI, dmPFC, including presupplemental motor area (pre-SMA) and dorsal anterior cingulate cortex (dACC)). Activation was also observed in nodes of the SN most closely associated with hedonic processing (VS, SN/VTA), as well as cerebellar regions. Deactivations were observed in right FPCN, in addition to inferior temporal gyrus (ITG) and superior occipital gyrus. In Experiment 2 (Figure 4.2B, *Appendix*: Table A3.1.1) we observed activation and deactivation patterns substantially similar to Experiment 1, albeit markedly stronger, consistent with the increased power in Experiment 2 ($N = 293$). Additional activation was observed in the frontal portions of the left FPCN (bilateral inferior (IFG) and middle (MFG) frontal gyri). Stronger activations were observed in midbrain, including both SN/VTA and PAG, as well as hypothalamus, thalamus, basal ganglia, and ventral AI. Again, deactivations centred on right FPCN and occipital regions.

These results replicate the PET studies discussed in the introduction, and support our contention that the DMN, SN, and FPCN key are components in the neural architecture supporting EnGE. They also expand on them demonstrating that this relationship holds for EnGE as it is freely implemented in the population. Finally, they suggest that EnGE additionally involves the active suppression of right frontoparietal and occipital regions, explainable by the known deactivation of these regions in internally focused processing (Andrews-Hanna et al., 2014).

4.4.3. Model-based component process mapping

Differentiating the generation from the elaboration of endogenous states into detailed representations is commonly done by having participants report the moment they subjectively experience to have completed the generation process (i.e. retrieval of core semantic information about an event), and begin the process of elaborated mental simulation by adding details about the context (e.g. Addis et al., 2007; 2009)In the context of EnGE, achieving a similar subjective differentiation is difficult, since emotional experiences are inherently situated in a context (Wilson-Mendenhall et al., 2011; 2013). We therefore took a model-based decomposition approach to test our component process structure. We reasoned that the 10 second Generation period of each trial should include activation of all constituent component processes (i.e. generation, elaboration, and maintenance), and that regions supporting these processes should be distinguishable by their activation dynamics in different conditions during the Modulation phase. By masking out activation attributable to either maintenance or elaboration from the 10 second Generation period, one should therefore be left with regions specifically involved in generation.

Specifically, we hypothesised that regions supporting the initial generation of the affective state should show early and phasic activation corresponding to their involvement in the generation of the affective core of the experiences. Importantly, they should also be

largely unaffected by modulation efforts, as such efforts should target the neural substrates of representation of the emotional experience rather than those involved in generation. Conversely, regions supporting the elaborated representation of emotional experiences should be affected by modulation efforts, and be mainly activated in the later part of the trial. Moreover, given that emotional experiences tend to persist over time (Buchanan, 2007; Verduyn, Delvaux, Van Coillie, Tuerlinckx, & Van Mechelen, 2009), it should be possible to dissociate the neural substrates of elaborated mental simulation, from those supporting the active maintenance of the generation process. We did this by contrasting activation in the Cease condition with the Maintenance condition (in the Modulation phase only). Figure 4.3 A schematically illustrates the hypothesised activation dynamics for each component as a

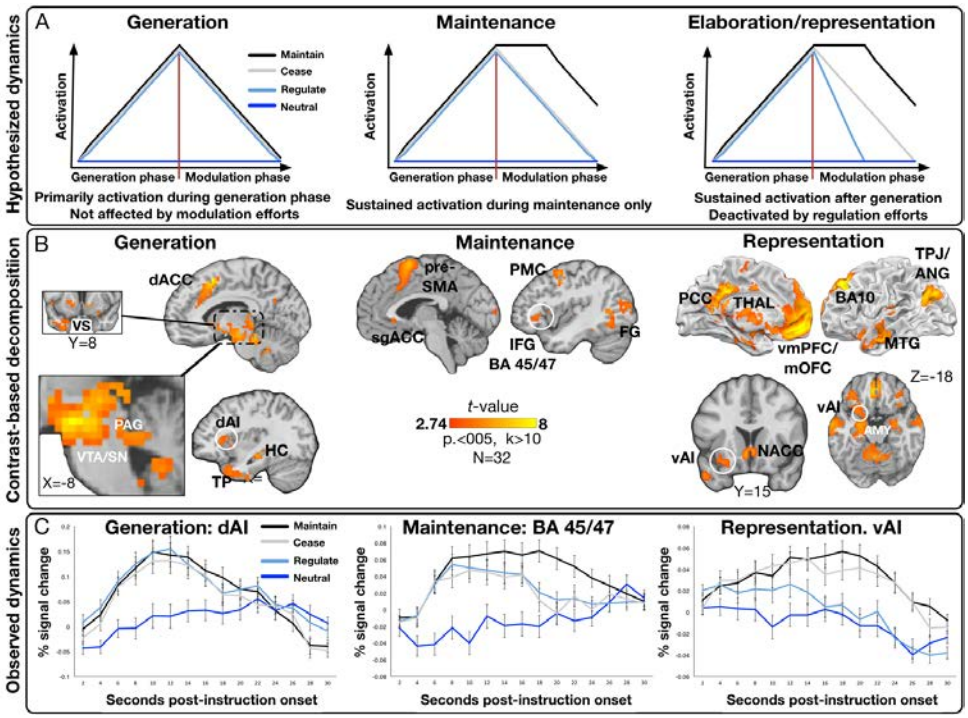


Figure 4.3: Model-based differentiation of EnGE networks. Results from the CPCA analysis showing the two components that predicted endogenous emotion generation ability, their dynamics and correlation with generation efficacy. **A)** The primary component, composed mainly of FPCN and DMN regions. **B)** The secondary component, composed mainly of FPCN and SN nodes. **C)** Conjunction of A and B, showing regions partaking in both task-relevant components.

function of condition. For these analyses alone we used a more lenient threshold of $p<.005$ (uncorrected), $k>10$, due to the lower power of the component contrasts.

To begin, component contrasts were calculated according to our process dynamic logic (see *Appendix:* Figure A2.1.2; Table A3.1.2). The Generate>Neutral contrast for the Generation period defined the neural reference space for EnGE overall (*Appendix:* Figure A2.1.2 A). Regions involved in the elaborated representation of emotional experiences were identified by contrasting the average of the Cease and Maintain conditions with the Regulate condition (*Appendix:* Figure A2.1.2 B). This was done because both Cease and Maintain conditions were associated with elevated subjective emotional experiences relative to both Neutral and Regulate conditions (Figure 4.1 B). We opted to use the Regulate rather than the Neutral condition since the Regulate condition actively suppresses emotional representations. Finally, regions involved in effortful maintenance of EnGE was identified by the Maintain>Cease contrast (*Appendix:* Figure A2.1.2 C). Next, the maintenance and representation contrasts were inclusively masked with the Generate>Neutral contrast to ensure that all activations were associated with EnGE. To ensure orthogonality of maintenance and representation maps, these were mutually masked (overlapping regions are reported in *Appendix:* Figure A2.1.2 D). Maintenance activation was primarily seen in pre-SMA, left IFG and premotor cortex – all known nodes of FCPN – in addition to fusiform gyrus and subgenual ACC. Representation processes were centred on nodes of DMN, including mOFC, PCC, left TPJ and MTG, in addition to bilateral AMY, NACC and left ventral AI. Finally, the Generate>Neutral contrast was exclusively masked with both maintenance and representation process contrasts, leaving exclusively activation not attributable to either of the two. This centred on dorsal AI and dACC, midbrain, and parts of ventral striatum, all known nodes of SN, in addition to hippocampus and temporal pole.

As a final step, we verified that our approach indeed identified regions with appropriate temporal dynamics by extracting FIR-fitted time courses using MarsBar for select

regions in each contrast. This revealed strong correspondence between observed and hypothesised dynamics (Figure 4.3 C).

These results conform to our hypotheses, showing that EnGE is supported by at least three separable component processes, and that these roughly overlap with each of the three core intrinsic networks observed in our main contrasts. Specifically, FPCN, appears primarily to support the active maintenance of generation efforts, while DMN primarily supports the representation of the generated states as evidenced by it being the primary target for down-regulation, as well as it remaining active even in the absence of generation efforts. Finally, the cortical and midbrain aspects of SN selectively responded in manner consistent with being involved initial generation of emotional states. Interestingly, we found that several regions in the limbic subcomponent of the SN (vAI, AMY and NACC) responded in a manner consistent with them supporting elaborated representations, suggesting that midbrain and limbic components of SN differ in their functional contribution to EnGE.

4.4.4. Functional significance and dynamic integration of EnGE networks

Having found evidence consistent with our hypothesised component process mapping, our next step was to a) establish the functional significance of these networks and b) investigate their dynamic interaction during EnGE. To address this we used constrained principal components analysis (CPCA; Lavigne, Metzak, & Woodward, 2015; Woodward et al., 2013) in our larger sample in Experiment 2. Briefly, CPCA is a data-driven multivariate method that combines multiple regression with PCA analysis to identify component of mutually correlating voxels, i.e. functional networks, involved in a task based on their specific task-related activation dynamics (see *Experimental Procedure* for detail). As CPCA provides individual-level estimates of activation of each network component, we could identify networks specifically predicting individual-differences in EnGE efficacy, thereby establishing both their involvement, and their functional significance. Finally, as CPCA does not enforce

spatial orthogonality on components it allows the identification of regions partaking in multiple network components with differing temporal dynamics, as would be expected if, as hypothesised, FPCN coupled to both SN and DMN.

Eigenvalue plots indicated that 6 components should be extracted using the scree criterion. To differentiate components supporting general task processes (e.g. sensory processing, motor responses) from those specifically supporting EnGE, we calculated the component-specific AUC of loadings in the task window (4-22 s post stimulus; allowing for 4-6 s hemodynamic lag) for the Maintain condition, subtracting the Neutral baseline. This yielded individual-level estimates of overall component activation during EnGE, that were orthogonalised and entered into a multiple regression model predicting individual differences in self-reported generation success (i.e. average affect ratings in the Maintain condition only).

Individual differences in component activation explained a significant amount of variance in generation success [$F(6,286) = 3.124$, $p < .01$, $R^2 = .062$], with two components directly predicting generation success. To interpret these, loading maps were thresholded at the dominant 10% of component loadings with $k > 30$ (Lavigne et al., 2015). The first component [$\beta = .153$, $t(292) = 2.675$, $p < .01$; Figure 4.4A] included central nodes of the DMN (vmPFC, PCC, left TPJ) and FPCN (bilateral BA47, BA45, MFG, and PMC), as well as VS (NACC, caudate) dorsal ACC, SMA/pre-SMA, mid cingulate cortex (MCC), bilateral STG/TRANS, left MTG and right somatosensory cortex, similar to the *Representation* network identified above. This similarity extended to activation dynamics, showing both early and sustained activation in the Modulation phase for the Maintain condition, and evidence of suppression in the Regulate condition. The second component [$\beta = .146$, $t(292) = 2.574$, $p = .01$; Figure 4.4B] included cortical nodes of SN (bilateral AI, pre-SMA) as well as portions of the FPCN (bilateral BA 47, left BA45, angular gyrus, and MFG), plus thalamus, occipital cortex and superior cerebellum. Dynamics closely resembled the *Generation* network identified above, with no observed difference between Maintain and Regulate conditions.

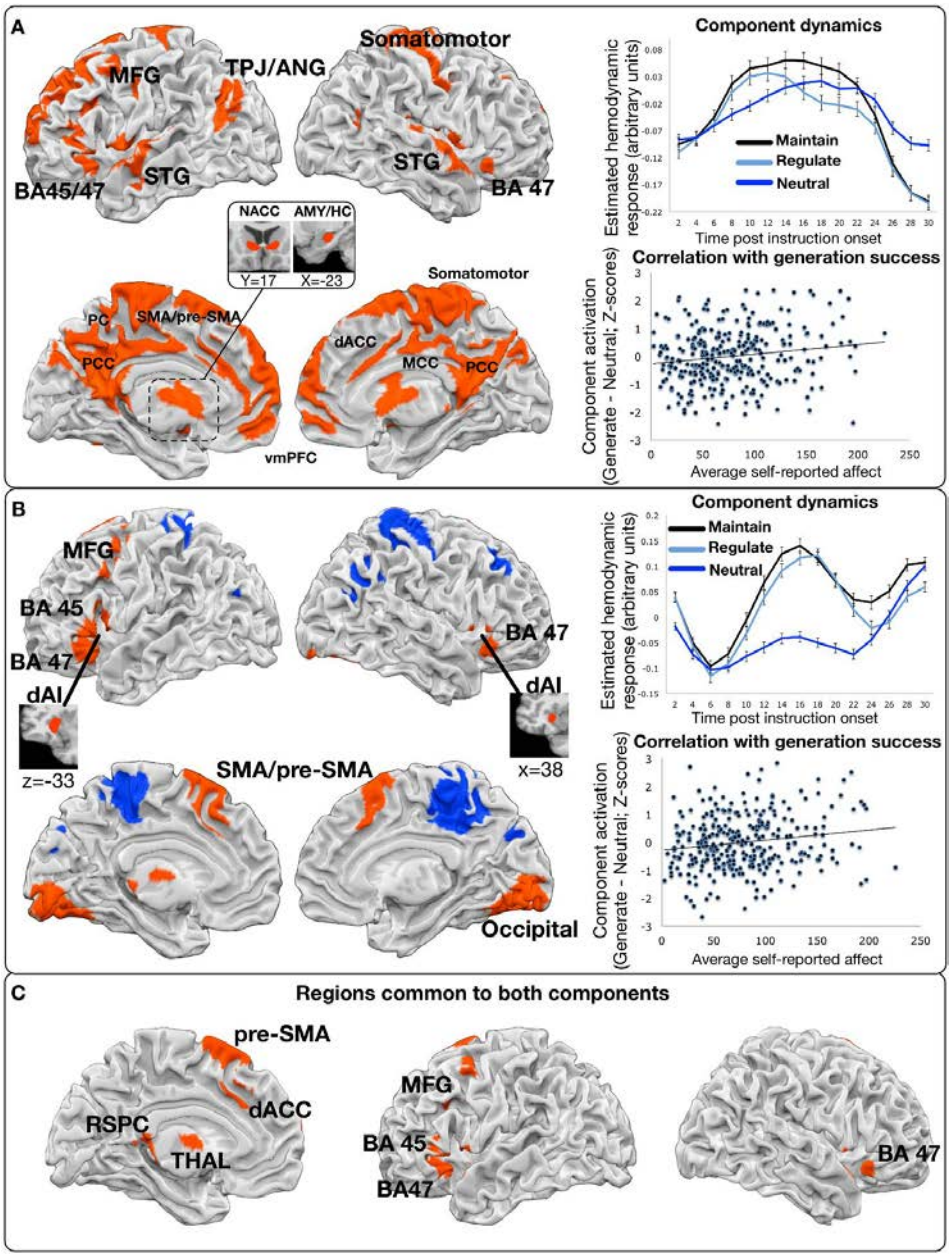


Figure 4.4: Dynamic differentiation and integration of EnGE networks. Results from the CPCA analysis showing the two components that predicted endogenous emotion generation ability, their dynamics and correlation with generation efficacy. **A)** The primary component, composed mainly of FPCN and DMN regions. **B)** The secondary component, composed mainly of FPCN and SN nodes. **C)** Conjunction of A and B, showing regions partaking in both task-relevant components.

Conjoining the individually thresholded component maps (Figure 4.4C; *Appendix*: Table A3.1.4), we found that left lateral FPCN regions (IFG, BA 45/45, MFG), as well as pre-SMA and dACC were part of both components, in addition to thalamus and retrosplenial cortex. Notably, this closely overlaps with the *Maintenance* network identified in our model-based analyses. These results expand on our model-based approach above, establishing the unique functional significance of our three candidate networks in EnGE. Further, they demonstrate that FPCN coupled with both SN and DMN, while these did not show evidence of coupling, supporting the hypothesis that FPCN coordinates activation of these two networks during EnGE.

4.4.5. Identifying the neural basis of core affect generation

Having found evidence for both the functional significance and dissociability of our putative EnGE networks, we next sought to establish neural implementation of core affect generation and representation formation. In post-scan debriefing, the participants in Experiment 2 reported higher success at generating positive than negative emotions. To avoid biasing results by potential effort or success effects, we therefore combined positive and negative valence conditions, and instead focused on trial-wise parametric modulation of contrast signal as a function of ratings. Using robust regression, we performed one sample *t*-tests on parametric modulation maps separately for positive and negative trials, averaging across all conditions involving emotion generation for both Generation and Modulation periods (*Appendix:* Table A3.1.5). To differentiate regions supporting emotion generation success in general from valence-specific regions, we conjoined the resulting FWEc thresholded maps (Figure 4.5).

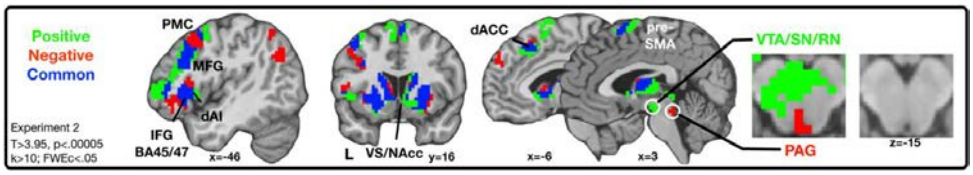


Figure 4.5: **Neural correlates of core affect**. Regions parametrically modulated by subjective ratings of affect in Experiment 2. Green and blue regions respectively show modulation in positive and negative conditions exclusively. Red coloured showed parametric modulation in both positive and negative conditions. Results are averaged across all emotional conditions for both Generation and Modulation phases.

In Experiment 1, this revealed valence-general modulation in the basal ganglia, including putamen and caudate body, while positive ratings uniquely modulated caudate head/nucleus accumbens (NACC), and negative ratings uniquely modulated left dorsal anterior insula and pre-SMA. In Experiment 2, valence-general modulation was more extensive, including left frontal portions of the FPCN, particularly inferior (IFG) and middle frontal gyri (MFG), as well as key nodes of the SN (dorsal AI, dorsal ACC, and pre-SMA) in addition to thalamus. Valence specific modulation of activation was observed in caudate head/NACC and SN/VTA for positive affect ratings, while modulation of deactivation was observed in occipital and right lateralised frontal regions overlapping with the deactivated regions reported in the main contrast. Negative affect ratings modulated activation in right dorsal anterior insula and periaqueductal grey. These results show that activation levels of FPCN and SN support successful emotion generation. Moreover, midbrain portions of the SN were shown to be recruited in a valence-specific fashion, consistent with the known association of these regions with domain general hedonic processing (Buhle et al., 2013; Kringelbach & Berridge, 2009). This supports our hypothesis that SN is particularly important for the generation of the affective core of EnGE. Further, in line with our CPCA analysis, we find that frontal FPCN supports a general role in the generation of core affective states, possibly associated with the initiation of the generation process.

4.4.6. Pathways of representation formation

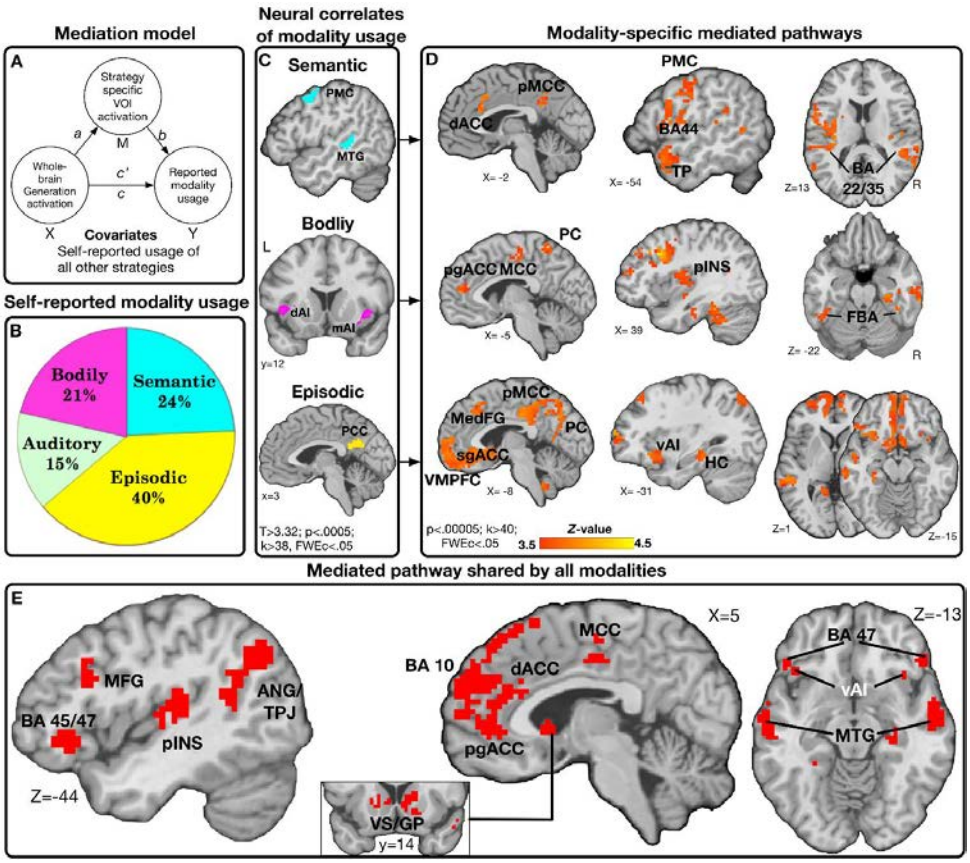


Figure 4.6: Pathways of modality-specific and general representation usage. Results from robust regression and mediation analyses of Experiment 2, differentiating general neural networks supporting emotion generation from modality specific instances. **A)** The mediation model used to perform a whole brain search for predictors (*X*) of reported modality usage (*Y*), that were mediated by activation in regions correlated with reported modality usage (*M*; reported in panel C and *Appendix*: Table A3.1.6). **B)** Reported usage of the four modalities in post-scan questionnaires. **C)** Regions correlated with modality usage. **D)** Mutually exclusively masked mediation maps, showing pathways uniquely supporting usage of each emotion generation modality. **E)** Conjunction of the three modality-specific pathways, showing the regions common to all three modalities.

Finally, we sought to identify the neural bases of representation formation. To this end, participants in Experiment 2 were constrained to use four specific generation modalities: 1) Semantic Analysis, involving the use of verbalised thoughts affective thoughts 2) Episodic Imagery, involving the generation of visual emotional imagery, 3) Auditory Imagery, involving the generation of affective soundscapes and 4) Bodily Interoception, involving focus on and interpretation of bodily signatures of emotional states (for precise instructions,

see *Appendix*: A1.1.4. *Generation strategy descriptions*) These modalities corresponded to our multimodal induction procedure, ensuring participants were equally primed to using each of them. Moreover, these modalities have clear analogues in daily life (e.g. thinking self-deprecating thoughts (semantic), remembering or anticipating an emotional event (episodic), humming a sad song (auditory), or noticing a dry mouth and racing heart when making a presentation (bodily)). Finally, participants were allowed to freely combine these modalities in whichever way they found best enabled them to generate emotional states. These four modalities can be combined to different degrees in an intuitive fashion (e.g. internal affective monologue combined with a concrete emotional episode where a specific song was playing) ensuring variance in the combinations utilised by participants.After scanning participants were requested to report to what degree they used each of these modalities on a 1-9 Likert scale. Overall usage (irrespective of combination) was determined by taking the sum total of these reports and proportionally comparing them (Figure 4.6B). This showed that the Episodic modality was the most used (40%), followed by Semantic (24%), Bodily (21%), and Auditory (15%). Entering the degree to which each participant reported using each modality as covariates in our main robust regression analysis (Generate & Maintain > Neutral), we could identify the neural correlates of modality usage (Figure 4.6C). Due to the noisy nature of self-report we used a more lenient cluster-forming threshold of $p < .0005$, $T > 3.32$ ($k > 42$, FWEc $p < .05$, reported in *Appendix*: Table A3.1.6). This revealed that use of the Semantic modality was correlated with activation of the left MTG, corresponding to the inferior border of Wernicke's area, as well as a region in the left DLPFC. Use of the Episodic modality was correlated with signal in the anterior superior PCC, a region known to be a part of the mnemonic subsystem of the DMN (Andrews-Hanna et al., 2014). Use of Bodily modality was correlated with signal in bilateral dorsal and mid-AI, a region known as interoceptive cortex representing bodily-signals (Craig, 2011). As no significant correlations were found for the Auditory modality, we did not explore this further.

We next identified the extended neural pathways by which these regions influenced the generation network as a whole using mediation analysis (Denny et al., 2014; Wager et al., 2008). Specifically, we implemented a mediation model (Figure 4.6A) performing a whole-brain search for voxels where the relationship between their Generate>Neutral contrast value and reported modality usage was mediated by the contrast values in the modality-specific regions identified above. Thus, these analyses identify voxels whose relationship with modality usage is mediated by activation of the modality specific regions, suggesting that they are part of the functional pathway by which that modality is implemented. For each analysis, reported usage of all other modalities were entered as covariates. To identify unique pathways for each modality mediation maps were thresholded at $Z > 3.25$, $p < .005$, $k > 30$, and masked exclusively with the maps for the remaining two modalities, revealing exclusive modality-specific pathways (Figure 4.6D; *Appendix*: Table A3.1.7). The Semantic pathway included left BA45 and BA22/35, approximating Broca's and Wernicke's areas respectively, left temporal pole and premotor regions, and dorsal ACC and anterior PCC, closely corresponding to the extended semantic system described in a recent meta-analysis (Binder, Desai, Graves, & Conant, 2009). The Episodic pathway included the majority of the DMN, including VMPFC, PCC, left MTG and HC, bilateral angular gyrus, ventral AI and left IFG, as well as subgenual ACC extending into ventral striatum, caudate and pallidum. Finally, the Bodily pathway included regions involved in body representation, including right posterior insula (Craig, 2011), bilateral fusiform body area and left extrastriate body area (J. C. Taylor, Wiggett, & Downing, 2007), in addition to MCC and PC, perigenual ACC and bilateral dorsolateral PFC, including premotor cortices.

To identify modality-independent pathways, we conjoined the thresholded maps, revealing a shared pathway (Figure 4.6E; *Appendix*: Table A3.1.7) overlapping with the *Representation* network identified above including the ventral AI and portions of the dorsomedial subsystem of the DMN (Andrews-Hanna et al., 2010), and a substantial portion

of the FPCN (bilateral IFG and MTG, left angular gyrus, MCC), as well as posterior insula. Thus, our findings support our hypothesis that DMN, together with vAI, support representation formation in cooperation with FPCN. Importantly, the *Generation* network was not involved in either general nor modality-specific pathways, supporting the hypothesised distinction between representation formation and core affect generation.

4.5. Discussion

We hypothesised that the endogenous generation of emotion (EnGE) involves the cooperation of three core functional networks: the saliency network (SN), the Default Mode Network (DMN) and the fronto-parietal control network (FPCN), respectively supporting core affect generation, episodic representation, and executive maintenance of emotional states. In two independent samples we found that EnGE activations centred on our three candidate networks. Decomposing these networks based on hypothesised activation profiles of component processes, we found support for our process-network mapping, showing that cortical and midbrain SN primarily contributed in the initial stages of the generation process and were unaffected by subsequent modulation efforts. Activation of cortical (dorsal AI, pre-SMA) and limbic (basal ganglia) nodes of SN were modulated by subjective experience of both positive and negative emotion, while midbrain nodes of SN showed valence-specific modulation, with PAG tracking with negative affect and SN/VTA tracking with positive affect. Overall, this is strongly supportive of the SN primarily supporting the initial generation of core affect in EnGE.

Conversely, activation of DMN was observed in all conditions where participants reported elevated affect, also after active generation had ceased, and was deactivated when participants suppressed their emotional states. Notably, this pattern was not exclusive to DMN, but was also observed for limbic regions (AMY, NACC) heavily implicated in affective processing (K. A. Lindquist et al., 2012), and ventral AI, known to be associated

with the intensity of emotional experience (Touroutoglou et al., 2012). Furthermore, dorsomedial DMN, together with ventral AI was found to be part of a general network supporting elaboration of core affect into detailed emotional experiences. Overall, this supports our hypothesis that the DMN plays a central role in the representational component of EnGE, expanding on it by showing that key affective regions partake in this process, which is a likely signature of the emotional nature of the representations in question.

We also found that left lateral and dorsomedial portions of FPCN together with inferior and posterior middle temporal gyri, uniquely activated during extended generation efforts. The FPCN was also found to be unique in being part of both components found to predict generation success in our data-driven decomposition analysis, coupling with both DMN and SN, consistent with it coordinating activation of these networks. Supporting this, left lateral frontal FPCN activation was found to predict trial-wise generation success and also to partake in the core, modality-independent pathway supporting representation formation. Thus, left lateral FPCN appears important for both the initiation and maintenance of EnGE, coherent with the known role of this region in the cognitive control of other internal processes, like memory retrieval and working memory (Badre & Wagner, 2007). In summary, our findings suggest that EnGE is a dynamic process in which left FPCN engages cortical and midbrain portions of the SN to establish a hedonic core affective state. Concurrently, FPCN couples to DMN, key limbic and insular regions, and regions supporting specific representational content, enabling the elaboration of the core affective state into an emotional experience.

It is notable that our findings show a large degree of overlap of with recent meta-analytical models of the neural bases of emotion derived from experiments using mainly exogenous (typically pictorial) stimuli (Kober et al., 2008; Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012). Based on clustering of co-activation patterns, Kober et al. (2008) described 6 functional networks supporting emotion processing. Interestingly, though the

precise functional clustering differs somewhat, the current data suggests the involvement of at least 5 of these clusters, suggesting a large degree of overlap between the neural bases of exogenous and endogenous emotion. Indeed, the one component primarily distinguishing the Kober et al. findings from the current results primarily include occipital regions known to be involved primarily in visual processing – as one would expect given the lack of emotionally relevant stimuli in the current experiment. As such, our findings suggest that the neural architecture of emotional processing is largely similar across induction modalities.

However, despite the similarity in overall architecture, the current results do suggest that EnGE might involve different functional roles for specific structures. This is most notable in the case of amygdala and basal ganglia. It is commonly assumed that these support generation of core affect, i.e. the qualities of valence and arousal that form the emotional foundation of experiences (Lindquist & Barrett, 2012). While the observation in the current data that basal ganglia activity is correlated with the intensity of both positive and negative subjective affect could be taken in support of such a relationship for emotional arousal, valence generation appears to be centered in midbrain regions, consistent with their known role in reward (SN/VTA; Berridge & Kringelbach, 2013) and aversion (PAG; Buhle et al., 2013). In our data, limbic structures like amygdala and nucleus accumbens appear instead to support the extended representation of emotional states, and thus are more closely linked to the experience than the generation of emotion. While this could be specific to endogenous emotions, we note that a recent meta-analysis of mainly exogenous emotion generation experiments did not find evidence for valence-specific processing in limbic regions (Lindquist, Satpute, Wager, Weber, & Barrett, 2016).

A natural question our findings raise is whether they are applicable to endogenous emotions that are not actively generated, but occur spontaneously. While ultimately an empirical question, extant evidence from the study of spontaneous mental activity in general suggests that this also is supported by the coupling of FPCN, DMN and mnemonic regions

(Christoff, Gordon, Smallwood, Smith, & Schooler, 2009), in a manner similar to what we observe. However, while the general architecture is similar, one could expect that spontaneous EnGE might show different dynamics. For one, while we find evidence that core affective states are elicited concurrently with representations when EnGE is volitionally initiated, in spontaneous EnGE the representation is likely to be a dynamic process in which occasionally affectively salient constellations appear. One possibility is that SN is then triggered via its dmPFC node, known to be associated with monitoring ongoing cognitive processes (Dosenbach et al., 2008).

Another interesting aspect of our findings is the similarity of the neural networks supporting EnGE to those supporting emotion regulation. Reappraisal, one of the most closely investigated and efficacious regulation strategies in the current literature, consistently shows activation in FPCN and, in particular, the same regions of the left lateral frontal cortex we found to be at the functional core of EnGE (Buhle et al., 2014). This region has also been implicated in a prefrontal-subcortical pathway predicting the capacity to regulate emotion using reappraisal (Wager et al., 2008), and is thought to play a key role in the cognitive control of memory (Badre & Wagner, 2007). Interestingly, a study comparing a variety of cognitive emotion regulation strategies showed that Reappraisal uniquely activated the left FPCN (Dörfel et al., 2014). This could relate to Reappraisal requiring the active generation of new emotional meaning, and thus indicate that Reappraisal partly depends on the capacity to endogenously generate emotion. Dörfel and colleagues also showed that right FPCN appears to form a core regulation network utilised across strategies, thought to implement inhibitory processes. We found that these regions were strongly *deactivated* during EnGE. This could point to a neural basis for distinguishing between the regulation and generation of emotion, an important issue in current emotion theory (J. J. Gross & Barrett, 2011). A corollary to this is whether emotion generation can itself be used as an emotion regulation technique. Recent work in our lab (H. G. Engen & Singer, 2015) suggests so, showing that meditation-based

generation of compassion can be used to actively regulate emotional responses to external stimuli, and is associated with activation of largely the same network we describe here. Importantly, no activation was observable in the right FPCN in that study, suggesting that this constituted a non-inhibitory type of regulation. Though it is unknown whether this generalises to other generation-techniques, this suggests that regulation based on counter-generation of emotion should be considered distinct from inhibitory strategies. The current findings appear to support this, showing that EnGE in general appears to, if anything, involve the deactivation of right FPCN. This is of potential practical importance, as it points to the possibility of developing interventions aimed at enhancing emotion generation ability, which could facilitate coping in individuals who are unable to utilise inhibitory strategies due to either circumstance or pathology.

4.5.1. Limitations

The large scale of the current data required the use of a relatively compressed paradigm, that could have skewed the results. For one, as we used a fixed length for the Generation phase, it is possible that some of the effects seen are attributable to anticipation of the next phase of the experiment. Future studies could avoid this by including a variable length generation phase akin to that used in previous studies of constructive memory (Addis et al. 2009). A potential limitation of our design is that we only used a single symbol (a blue 0) to denote that participants should aim to achieve a neutral emotional state. While piloting indicated that using a single cue was less confusing for participants than having separate regulation cues, it is possible that this could have lead to conflation between the neutral and regulation conditions. Another limitation of the paradigm was that we had no clear indications of the precise discrete emotions the participants generated or how these fluctuated over the course of task-implementation, as a function of e.g. fatigue or habituation. Future studies could investigate this by performing more detailed analysis of trial-wise contents of generated

states, which could provide more information about the differences between emotional states in terms of their neural underpinnings. Similarly, a more fine-grained analysis of trial-wise variation could provide valuable insight into potential fluctuations of generation strategies during repeated emotion generation, which could provide valuable insight into the relative efficacies of generation modalities. This would also be a possible way of minimizing potential confounds stemming from fatigue/habituation like we observed here. Moreover, acquiring more details on the specific scenarios used during EnGE (i.e. whether they are past or future related, or whether they involved specific emotional states), would allow for more nuanced modelling of how information about emotion is stored, retrieved, and combined in the construction of emotional experiences.

Chapter 5: Structural brain changes associated with expertise in loving-kindness meditation[4]

Boris Bernhardt, Haakon G. Engen*, Leon Skottnik, Matthieu Ricard, & Tania Singer*

5.1. Abstract

Previous work has established that expertise in attention and interoception-based meditation is associated with morphological changes in fronto-polar, insular, and cingulate regions. However, it is unclear if these effects generalise to forms of meditation involving the active generation of affective states, such as loving-kindness meditation (LKM). We investigated this in a group of long-term meditation practitioners (LTMs, mean ± SD 40 ± 9k hours of practice, primarily in loving-kindness and compassion meditation). Compared to meditation-naïve controls, LTMs showed increased thickness of fronto-polar and fronto-insular cortices, partially conforming with previous findings. To identify LKM-specific changes, findings were complemented by fMRI analysis, showing amplitude increases in low-frequency fluctuations during a LKM session relative to non-meditative rest in our LTMs in multiple prefrontal and insular regions bilaterally. Importantly, functional and structural findings overlapped, particularly in the left ventrolateral prefrontal cortex and anterior insula, suggesting that these regions play a central role in the generation of emotional states. Interestingly, these regions are not usually implicated in meditation, but previously associated with both general generative cognition and intensity of emotional experiences. Our results suggest that brain plasticity of meditation expertise may be specific to practice, motivating future, more nuanced investigations into the effects of specific meditation practices.

[4] The current chapter is presently under review, and the final, published version, might show discrepancies from the present version. Presented here is the submitted manuscript of 21.04.2016. * = authors contributed equally

5.2. Introduction

There has been a long-standing interest in the neurosciences to study brain plasticity (Cotman & Berchtold, 2002; Kolb, Gibb, & Robinson, 2003; Poldrack, 2000). Plasticity can occur across multiple time scales, from short-term to long-term, and at the level of structural networks. In humans, most previous studies addressing structural plasticity have focused on training or expertise effects on motor, sensory, or memory functions(Draganski et al., 2004; Taubert, Lohmann, Margulies, Villringer, & Ragert, 2011). In addition to validating training protocols, evidence of morphological plasticity allows nuanced insight into the central brain regions supporting different psychological processes and groups of individuals. Recently, there has been an increasing interest in the study of plasticity effects of mental training via meditation. Meta-analysis suggests that meditation training indeed has measurable effects on brain structure: In a recent meta-analysis(Fox et al., 2014), Fox and colleagues overviewed morphometric findings in 21 studies, collectively assessing more than 300 practitioners across different traditions, finding evidence for consistent grey matter changes in brain regions associated with executive attention, planning and interoception, such as the dorsal anterior cingulate (dACC), frontopolar (FP) cortex, and insula(Fox et al., 2014). As the studies reviewed in the Fox analysis includes both longitudinal investigations into plasticity as a function of training and cross-sectional studies this could suggest that ACC, FP, and insula are central in engendering meditative states.

It has been argued that there are several different families of meditation practice (Dahl, Lutz, & Davidson, 2015), each emphasising and relying on different cognitive processes. Importantly, the majority of the studies of brain plasticity and meditation have focused on an *attentional* family of practices (*e.g.* mindfulness meditation or Vipassana with their focus on paying attention breath). Thus, it is unclear whether these regions also support other meditation practices. For instance, a so-called *constructive* meditation practice is characterised by relying less on attention control and interoception and more on the active

construction of cognitive and affective states. One example of such a practice is loving-kindness (LKM) and compassion meditation (CM), that centrally involves the generation of positive affective states. Behavioural and autonomic research has suggested that the training of LKM and CM may be associated with increases in prosocial behaviour (Leiberg et al., 2011; Weng et al., 2013) and vagal tone as a proxy of physical health (Kok et al., 2013). It has been suggested that LKM training may increase positive affect when an individual is confronted with another's suffering, associated with a wish to alleviate this suffering (Singer & Klimecki, 2014). Using longitudinal fMRI, our group recently assessed short-term functional alterations after the training of loving-kindness and compassion in meditation-naïve participants (Klimecki et al., 2013; 2014). Individuals undergoing compassion training showed increased activations in subcortical networks, together with the medial orbitofrontal cortex (mOFC) relative to a group that trained spatial memory (Klimecki et al., 2013). In a follow-up study, the authors observed increased activations in perigenual and subgenual cingulate cortex (pgACC/sgACC), insula, as well as brainstem nuclei including substantia nigra and ventral tegmental area in individuals that underwent compassion training (Klimecki et al., 2014). Thus, these findings suggest that LKM and CM may have markedly different neural substrates from the attentional family, as reported by Fox et al. (2014). On the other hand, other studies have implicated insula and cingulate/prefrontal cortices as possible functional substrates for CM (Lutz, Brefczynski-Lewis, Johnstone, & Davidson, 2008; Lutz, Greischar, Perlman, & Davidson, 2009), consistent with the Fox et al. meta-analysis. One possible reconciliation of these findings is that LKM and CM rely on both family-specific functions supporting the generation of affective states and suprafamiliar mechanisms related to meditation in general, possibly supporting general attentional function.

In this study, we tested this possibility by investigating structural changes in a sample of long-term meditators (LTMs), with extensive experience and preference for specifically LKM and CM meditation (mean ± SD = 40 ± 9k hours of prior practice). As meditation

practice is heterogeneous, this could bias investigations to pick up meditation-general effects. We therefore aimed to maximise the homogeneity of our sample by recruiting them only from a specific school of practice (the Nyingma school of Tibetan Buddhism), and who had participated in at least one three-year-long retreat in the same centre with the same teachers. Thus, by comparing the LTMs with matched, meditation-naïve controls, we could evaluate overall cortical thickness changes associated with expertise in meditation in general, and LKM and CM in particular. To differentiate these changes, we investigated the functional expression of LKM by investigating low-frequency amplitude fluctuations while our participants were meditating with that of a non-meditative resting-state. Functional amplitude increases during loving-kindness relative to non-meditative rest were mapped onto cortical surface models and overlap analyses with regions of cortical thickness changes were carried out, allowing us to identify regions that showed consistent increases in thickness, and were functionally involved in the generation of LKM.

5.3. Materials and methods

5.3.1. Sample

Participants were tested between November 2011 and March 2013. We studied a cohort of 17 long-term meditators (LTM; 5 women; 45-62 years, mean ± SD age = 56 ± 5 years). All meditators were adepts of the Nyingma tradition of Tibetan Buddhism (one of the schools focussing on the cultivation of loving-kindness, altruism, and compassion). Participants were included only if they had previously participated in a full-time meditation retreat of at least 3 years, which all carried out at the Songsen Chanteloube Retreat Center in Dordogne, France. LTMs reported having practiced meditation for an estimated total of 40k ± 9k hours (10k-62k hours) before our study, and to practice LKM and CM on a daily basis.

LTMs were compared to 15 healthy controls without prior meditation experience, recruited through advertisement. Controls were matched for age and sex (5 women; 46-63

years, mean ± SD age = 54 ± 6 years), as well as education level (LTM: mean ± SD years of education = 14 ± 3 years; Controls: 14 ± 3 years). We assessed the IQ of all participants using Raven's Progressive Matrices (Raven, 2000). LTMs and controls did not differ with respect to IQ (103.6 ± 20.0 *vs.* 102.4 ± 28.5). Furthermore, LTMs and controls scored similar on the Beck's Depression Inventory [BDI-2; (Beck, Steer, Ball, & Ranieri, 1996); mean ± SD in LTM: 5.9 ± 3.9; controls: 6.6 ± 7.0; $p > 0.7$;] and the Spielberger Trait Anxiety Scale [STAI-T; (Spielberger, Gorsuch, Lushene, & Vagg, 1983); mean ± SD in LTM: 37.3 ± 9.9; Controls: 37.0 ± 9.1; $p > 0.7$]. The study was approved by the Ethics Committee of the University of Leipzig and was carried out in compliance with the Declaration of Helsinki. All participants gave written informed consent, were paid for their participation, and were debriefed after the study.

5.3.2. Structural MRI

5.3.2.1 Acquisition parameters

Structural MRI data were acquired on a Siemens Verio scanner (Siemens Medical Systems, Erlangen, Germany) using a 32-channel head coil. Images were acquired using a T1-weighted 3D-MPRAGE sequence (repetition time [TR] = 2300ms, echo time [TE] = 2.98ms, flip angle = 7°, 176 sagittal slices, field of view [FOV] = 240×256 mm^2, matrix = 240×256, 1×1×1 mm^3 voxels).

5.3.2.2. Cortical thickness measurements.

FreeSurfer (5.0.0; http://surfer.nmr.mgh.harvard.edu) was used to generate cortical surface models and to measure cortical thickness on T1-weighted images. Previous work has validated FreeSurfer by comparing it with histological analysis (Rosas et al., 2002) and manual measurements (Kuperberg et al., 2003). Processing steps have been described in detail elsewhere (Dale, Fischl, & Sereno, 1999; Fischl, Sereno, & Dale, 1999; Han et al., 2006).

Following surface extraction, sulcal and gyral features across individuals were aligned to an average spherical representation that allows for accurate matching of cortical thickness measurement locations among participants. The entire cortex in each participant was visually inspected and a single rater manually corrected segmentation inaccuracies (LS). For whole-brain analysis, thickness data were smoothed using a 15 mm full-with-at-half-maximum surface-based Gaussian kernel. Surface-based smoothing reduces measurement noise while preserving the capacity for anatomical localization, as it respects cortical topology (Lerch, 2004).

5.3.3. Functional MRI

5.3.3.1. Paradigms

We analysed two fMRI sessions that were each seven minutes long. Participants were instructed to lie still in the scanner with open eyes, and to fixate a white cross in the centre of a black screen. *Session A* was a meditation-free *resting-state* run, where participants instructed to not think about anything in particular and to not meditate. *Session B* was a loving-kindness meditation session, similar to previous work (Klimecki et al., 2013; Kok et al., 2013; Lutz et al., 2008; Ricard, 2006; Salzberg, 2001). The specific instructions the LTMs implemented for the LKM session was: "*In this condition, we want you to cultivate loving-kindness, 'Metta' in Pali. Altruistic love, loving-kindness or, more simple, warm-heartedness, is the wish that others be happy and well. How to generate and cultivate that state of mind? The easiest way is to begin by bringing to the mind someone who is very dear to us, someone for whom this feeling of affection arises effortlessly and spontaneously. Imagine that a young child approaches you and gives you a look that is joyous, confident, and full of innocence. You stroke his head, look at him with tenderness, and take him in your arms. You feel a sense of unconditional benevolence and love. Let yourself be entirely pervaded by this love that wishes only for his well-being. Then cultivate, sustain, and nourish this feeling of loving-*

kindness. When it declines, revive it. Think repeatedly, 'May that person be safe, may that person be healthy, may that person find happiness and flourish in life'. You could also choose someone else toward whom you feel great tenderness and deep gratitude. Wish with all your heart that this person will find happiness and be well. "

Participants also performed an open-presence meditation *Session C*; this session was, however, not the focus of the current work and will be reported elsewhere. *Sessions A, B*, and *C* were presented in a counterbalanced order across subjects, and participants were allowed to rest between them for 1-2 minutes and to prepare themselves for the next session. As a check on the differentiability of the LKM and resting states, informal qualitative debriefing was performed after each scan, with participants reporting both subjective success in achieving a state of LKM in the scanner, and no difficulties with refraining from meditation in the resting state scan.

5.3.3.2. Acquisition parameters

Functional MRI data were acquired on the same scanner as the functional signals using a 12-channel head-coil. We recorded fMRI time-series using T2*-weighted gradient EPI sequence (TR = 2000ms, TE = 27ms, flip angle = 90°, 37 slices tilted at approximately 30° from an axial orientation, FOV = 210×210 mm^2, matrix = 70×70, 3×3×3 mm^3 voxels, 1 mm gap; 210 volumes per session). Data were available for all participants except one LTM.

5.3.3.3. Preprocessing

Processing was based on DPARSF/REST for Matlab [http://www.restfmri.net (Chao-Gan & Yu-Feng, 2010; Song et al., 2011)]. We discarded the first 5 volumes to ensure steady-state magnetization, performed slice-time correction, motion correction and realignment, and co-registered functional time series to the corresponding T1-weighted MRI. Images underwent DARTEL-based segmentation and registration, followed by nuisance covariate

regression to remove effects of average WM and CSF signal, as well as 6 motion parameters (3 translations and 3 rotations). To increase the robustness against motion confounds, we included a *scrubbing*(Power, Barnes, Snyder, Schlaggar, & Petersen, 2012). In brief, we modelled time-points with a frame-wise displacement > 0.5mm, together with one time-point before and one after each such time-point, as separate regressors during covariate correction. Time-series were band-pass filtered (0.01-0.08 Hz). We computed the amplitude of low frequency functional fluctuations in this frequency band, a marker of overall local functional activation. The amplitude of low frequency functional fluctuations has been suggested as a means to unveil regional functional changes in task-free paradigms (Zang et al., 2007; Z. Zhang et al., 2010). Moreover, the marker has been shown to differentiate between eyes open and eyes closed conditions (Yang et al., 2007)and to relate to the contents of thoughts in the resting state (Gorgolewski et al., 2014), suggesting that it provides a measure of the activation patterns supporting different brain states. Amplitude measures were z-scored within the brain mask of each participant prior to analysis.

5.3.3.4: Surface mapping.

Using a boundary-based registration, we mapped functional time-series to cortical surface models (Greve & Fischl, 2009). Like the cortical thickness data, functional data were registered to a surface-based template based on cortical folding patterns and smoothed on tessellated surfaces.

5.3.4. Statistical analysis

As in previous work (Bernhardt, Klimecki, Leiberg, & Singer, 2014), analysis was performed using SurfStat for Matlab(Worsley et al., 2009). Prior to analysis, we statistically corrected for effects of age and sex on thickness.

5.3.4.1. Cortical thickness comparisons.

We carried out a surface-based comparison of thickness between LTMs and controls, using Student's *t*-tests at each cortical point. In regions of findings, statistical group comparisons were complemented by individual analysis that provides prevalence estimates in LTMs for markedly different thickness relative to controls.

5.3.4.2. Functional analysis.

We applied random effects models to compare differences in amplitude between loving-kindness and resting-state conditions within LTMs.

5.3.4.3: Correction for multiple comparisons.

Surface-based structural and functional findings results were controlled at $p_{FWE} < 0.05$ (height threshold t = 2.1), using random-field theory for non-isotropic images.

5.4. Results

5.4.1. Cortical thickness increases in LTMs: surface-based findings

Surface-based analysis revealed increased cortical thickness in LTMs compared to controls in a right prefrontal cluster encompassing fronto-polar cortex and left ventrolateral (peak MNI-coordinate x/y/z = 24/60/2; cluster-wise $p_{FWE} < 0.05$) as well as fronto-insular cortices (peak MNI-coordinate x/y/z = -29/24/4; cluster-wise $p_{FWE} = 0.05$, Figure 5.1). There were no significant cortical thickness decreases in LTMs.

5.4.2. Loving-kindness related amplitude increases in LTMs

Comparing state-related differences in LTMs, we observed greater within-subject increases in LTMs when comparing loving-kindness meditation to non-meditative rest in medial and

anterior prefrontal regions (peak MNI x/y/z = -30/17/-22; $p_{FWE} < 0.05$), while amplitude in posterior regions decreased (Figure 5.2)

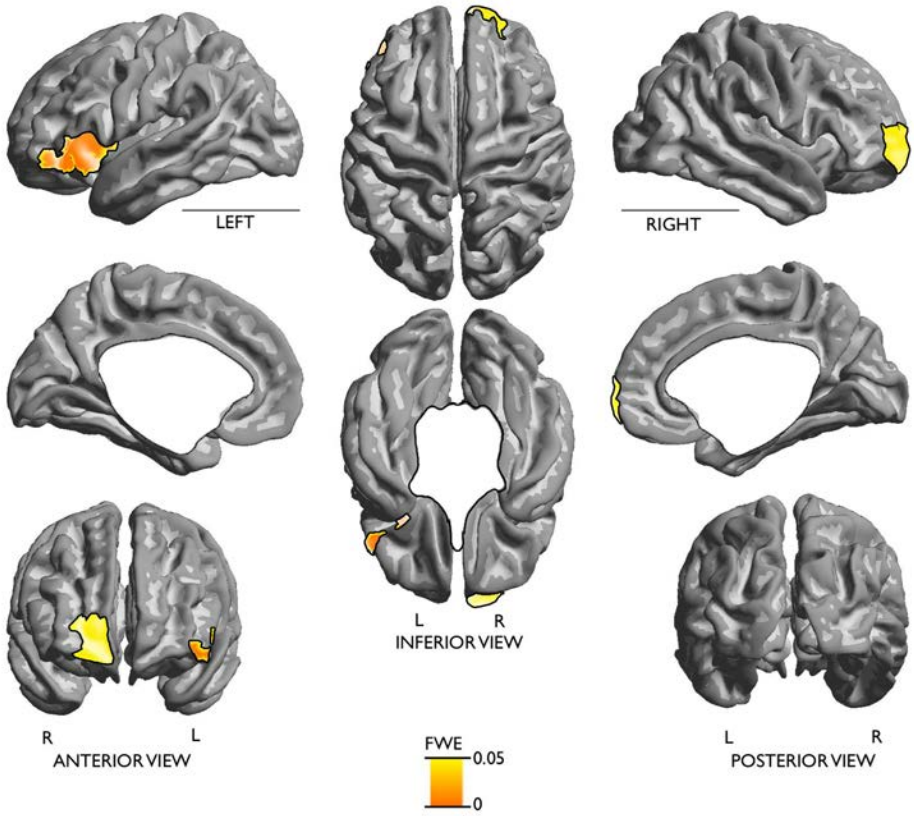


Figure 5.1: Cortical thickness increases in LTMs relative to controls: surface-based findings. Significant increases after multiple comparisons correction at a cluster-level of $p_{FWE} < 0.05$ are surrounded by bold black outlines.

5.4.3. Overlap of functional and structural findings

Overlaying FWE-corrected structural and functional findings (Figure 5.3), we observed overlaps in orbito-frontal/ inferior prefrontal regions (centroid coordinates MNI x/y/z = -41/41/-6, x/y/z = -46/32/-11) as well as anterior orbitofrontal/ventral anterior insular regions (centroid coordinate MNI x/y/z = -28/20/-11).

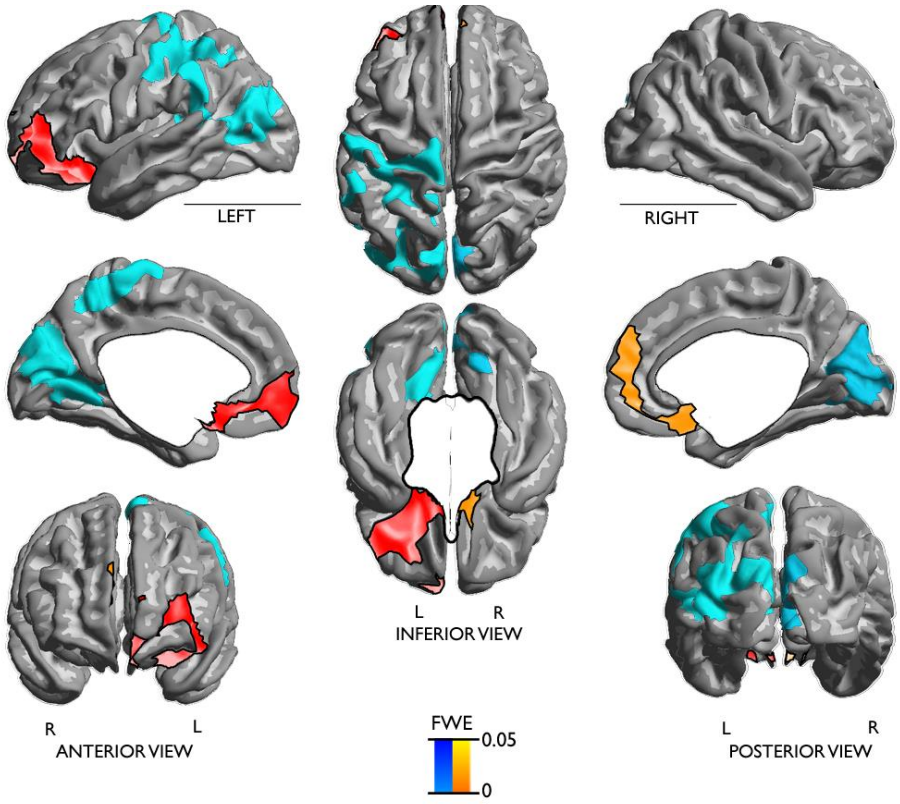


Figure 5.2: Functional amplitude correlates of loving-kindness meditation. Changes in functional amplitude during loving-kindness meditation relative to non-meditative rest in LTMs. Increases/decreases after multiple comparisons correction at a cluster-level of $p_{FWE} < 0.05$ are shown in red/blue.

5.5. Discussion

Our overall goal was to differentiate meditation-general effects from those specifically involved in loving-kindness meditation (LKM). We tested a homogenous group of long-term meditation practitioners (LTMs), who have practiced in developing compassion, loving-kindness, and altruism for many years prior to our study; this group was compared with an age- and sex-matched meditation-naïve control group. Our multi-method neuroimaging approach assessed morphological brain differences in LTMs compared to controls and subsequently evaluated whether these were functionally involved in loving-kindness

meditation, aiming to differentiate LKM-specific morphological changes from those associated with general meditation practice.

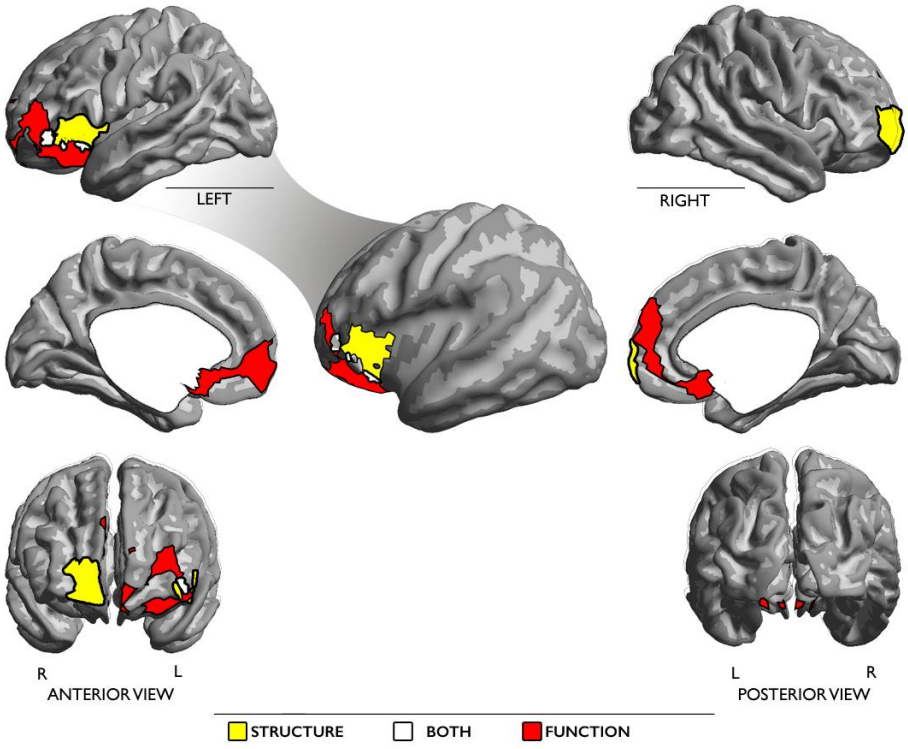


Figure 5.3: Overlap of structural and functional findings. Overlap analysis between structural and functional findings, showing overlap centred on left IFG and ventral anterior insula.

Cortical thickness analysis was chosen to evaluate differences in brain structure between LTMs and novices *in vivo* (Fischl & Dale, 2000). While this technique does not allow for the study of subcortical changes, it provides an anatomical meaningful marker of grey matter variations in the cortical mantle. Using an unconstrained surface-based approach, we observed thickness increases in LTMs relative to novices in a right-lateralised fronto-polar cluster as well as a left lateralised cluster ranging from ventrolateral prefrontal regions into anterior insula. These effects are consistent with those reported by Fox et al. (2014) in a recent meta-analysis of studies into the structural bases of meditation training in mainly

attentional meditation family practices (Dahl et al., 2015). Moreover, a previous study by Lazar and colleagues has suggested trends for increased thickness in participants with experience in insight meditation in the anterior insula (Lazar et al., 2005). A more recent study complemented these findings, by showing increased cortical folding complexity in the anterior insula in a group of meditators that followed different meditative practices, such as samata and Vipassana meditation (Luders et al., 2012). Interestingly, the anterior insula is heavily implicated in interoception (Bernhardt & Singer, 2012; Craig, 2009; Zaki, Davis, & Ochsner, 2012) and attentional processing (Nelson et al., 2010; Sridharan, Levitin, & Menon, 2008), suggesting the increases we observe here might reflect general effects of meditation practice involving attentional function. The Fox et al. meta-analyses also found evidence for grey matter alteration in frontopolar cortex (Fox et al., 2014), corresponding closely to our finding of thickness in the same region. Frontopolar cortex is thought to support higher-order integrative cognition related to metacognitive awareness, a skill that is heavily emphasised in all meditation practices (Dahl et al., 2015).

In the current assessment, structural MRI findings were complemented by a surface-based functional analysis of the amplitude of low-frequency fluctuations, a previously proposed local functional marker (Zang et al., 2007), during a LKM session relative to non-meditative rest. Although the majority of previous task-free fMRI studies have focused on time-series correlations between regions to study inter-regional connectivity, recent methodological work has proposed several markers that probe local functional characteristics of a given region (Song et al., 2011; Zuo et al., 2010). LTMs showed increases when engaging in loving-kindness practice in regions overlapping with a network previously shown to undergo functional plasticity after compassion training (Klimecki et al., 2013; 2014). Specifically, our analysis showed amplitude increases during LKM in portions of anterior and medial ventrolateral prefrontal cortex, medial and lateral OFC, together with sgACC/pgACC and insular subregions. Importantly, we also observed an overlap between our structural and

functional findings in the left ventrolateral PFC approximating BA 45/47. Thus, this region appears to play a central role in both acute and long-term implementations of LKM. While its precise function is still a subject of debate, this region has been implicated in processes requiring the internal generation of information and has been proposed to support controlled retrieval processes (Badre & Wagner, 2007). This is in line with its frequent involvement in emotion regulation, particularly forms of emotion regulation requiring the active generation of alternate meanings of emotional stimuli, such as cognitive reappraisal (Buhle et al., 2014; Wager et al., 2008). In a recent study (H. G. Engen & Singer, 2015), we found that a similar region was one of few areas showing activation during regulation of negative emotion using positive reappraisal and compassion mediation, suggesting the left IFG might be particularly important for the generation of emotional states. Interestingly, we also observed overlap of functional and structural findings in the ventral AI. While most commonly thought to have a similar function as the dorsal AI, recent work suggests that these regions partake in dissociable large-scale intrinsic connectivity networks that serve different aspects of orienting towards salient stimuli (Touroutoglou et al., 2012), with the dorsal AI being more closely related to attentional function while the ventral AI supports the generation of emotional experiences. Thus, our data could suggest that ventrolateral PFC/inferior frontal gyrus and ventral AI cooperate in the volitional generation of affective states and, potentially, meditation practices requiring the construction of affective states in general.

5.5.1. Limitations and conclusions

To circumvent high challenges and costs related to performing longitudinal studies that would directly probe causal effects of meditation training, the current work heuristically employed a cross-sectional comparison between practitioners and controls. Cross-sectional inference cannot rule a combination of selection (*i.e.* individuals like our LTMs are attracted to meditation), attrition (*i.e.* individuals like our LTMs are likely to continue meditating),

lifestyle (*i.e.,* long-term meditation may be associated with different lifestyle choices), and plasticity (*i.e.,* training indeed changed the brain in LTMs) driving our results. We assessed meditators with a mean of 40k hours of practice in life and participation in at least one three-year retreat in the same centre, all of them having specifically focused on cultivating socio-affective skills. While these criteria offered the opportunity to study a homogenous sample of long-term meditation practitioners, they have in effect restricted our analysis to older participants. Although possible age confounds in the current study were addressed by the use of age-matched controls as well as statistical corrections for age-effects, the inclusion criteria may have hampered generalizability given the narrow age range investigated. Moreover, the stringent inclusion criteria limited our sample size, which might have reduced statistical power to detect subtle structural changes.

Our findings may have implications for the general understanding of the brain basis of both constructive meditation practices and meditation in general, suggesting that the structural consequences of meditation training include both practice-specific and meditation-general processes that need to be dissociated in order to gain a comprehensive understanding of how meditation training engenders long-term changes in brain structure. Additionally, our findings suggest that care must be taken to dissociate functional and structural markers of plasticity, as demonstrated by the relatively circumscribed overlap between the structural changes we identified and the extended functional network found to support LKM state implementation. As such, our findings motivate future longitudinal studies that may directly address the causality between mental training, brain changes, and markers of affect and prosociality.

5.5.2. Acknowledgements

We would like to thank all meditators and control participants involved in this study. We are grateful to the support staff of the social neuroscience department, particularly Dr. Sandra Zurborg, for her help with the study logistics and organisation, Henrik Grunert for his

technical assistance, Elisabeth Murzik for her help with data archiving, as well as Sylvie Neubert and Nicole Pampus for their help with scanning. We also thank Sofie Valk and Bram Cordemans for their help in assessing questionnaire data. This work was supported by grants to TS from the European Research Council under the European Community's Seventh Framework Program (FP7/2007-2013)/ERC Grant agreement n◦ 205557 [EMPATHICBRAIN]. BCB received postdoctoral funding by the Canadian Institutes of Health Research

Chapter 6: Compassion-based emotion regulation up-regulates experienced positive affect and associated neural networks[5]

Haakon G. Engen, & Tania Singer

6.1. Abstract

Emotion regulation research has primarily focused on techniques that attenuate or modulate the impact of emotional stimuli. Recent evidence suggests that this mode regulation can be problematic in the context of regulation of emotion elicited by the suffering of others, resulting in reduced emotional connectedness. Here we investigated the effects of an alternative emotion regulation technique based on the up-regulation of positive affect via Compassion-meditation on experiential and neural affective responses to depictions of individuals in distress, and compared these with the established emotion regulation strategy of Reappraisal. Using fMRI, we scanned 15 expert practitioners of Compassion-meditation either passively viewing, or using Compassion-meditation or Reappraisal to modulate their emotional reactions to film clips depicting people in distress. Both strategies effectively, but differentially regulated experienced affect, with Compassion primarily *increasing* positive and Reappraisal primarily *decreasing* negative affect. Imaging results showed that Compassion, relative to both passive-viewing and Reappraisal increased activation in regions involved in affiliation, positive affect and reward processing including ventral striatum and medial OFC. This network was shown to be active *prior* to stimulus presentation, suggesting that the regulatory mechanism of Compassion is the stimulus-independent endogenous generation of positive affect.

[5] This chapter has previously been published as Engen, H. G., & Singer, T. (2015). Compassion-based emotion regulation up regulates experienced positive affect and associated neural networks. *Social Cognitive and Affective Neuroscience*, *10*, 1291–1301.

6.2. Introduction

Research has demonstrated that exposure to others' pain elicits negative affective responses on both an experiential and physiological level (Lamm et al., 2011). Consequently, being confronted with the suffering of others can be a potent source of personal distress. Prolonged exposure to suffering can therefore have deleterious mental health effects. This is seen in the high stress levels and burnout rates often reported for professionals tasked with caring for suffering individuals, such as physicians (Shanafelt et al., 2012) and nurses (Adriaenssens, De Gucht, & Maes, 2014). With this in mind, identifying effective emotion regulation strategies that can be employed to promote resilience for exposure to others' suffering is potentially of great help both for the individual and society at large.

One particularly effective strategy for regulating negative emotional responses involves the cognitive generation of alternate interpretations of an emotional event, thereby modulating their emotional meaning and impact (McRae, Ciesielski, & Gross, 2012a). This strategy, most frequently called Reappraisal, has been shown to be effective across a wide range of different emotional stimuli and contexts (J. J. Gross, 2007b). Furthermore, trait use of Reappraisal is a predictor of psychological well-being and resilience (McRae, Jacobs, Ray, John, & Gross, 2012b; Min, Yu, Lee, & Chae, 2013). As such, Reappraisal is as a strong candidate for an effective means of coping with exposure to the suffering of others. However, recent research has identified a potentially problematic side effect of using Reappraisal to regulate ones affective reactions to the suffering of others: Cameron & Payne (2011) demonstrated that Reappraisal can lead to decreased concern and willingness to help, especially when multiple individuals are suffering, and when helping is costly. One explanation for this is that Reappraisal involves discounting negative information as it is perceived, and substituting a more positive interpretation. While an effective panacea for personal distress, this mechanism in effect disconnects the Reappraiser from the communicated affective experience when

applied to stimuli signalling others' suffering. Thus, Reappraisal might not be the optimal strategy in contexts where an inter-individual emotional connection is required.

One promising way to supplement traditional emotion regulation strategies is the use of meditation techniques. In the context of suffering, one particularly promising technique is Compassion-meditation. The emotional state of compassion[6] can be defined as the emotion one experiences when feeling concern for another's suffering and desiring to enhance that individual's welfare (Goetz et al., 2010). Training in Compassion-meditation aims at enabling the individual to volitionally generate states of compassion, allowing them to encounter the suffering of others while maintaining a positive emotional state of benevolence, warmth and concern and a motivation to help (Klimecki et al., 2013; Kok et al., 2013; Lutz et al., 2008). Concretely, Compassion-meditation involves the initial generation of an emotional state of loving-kindness through directed imagery emphasising positive emotional qualities of warmth, care and other-related concern. Once this emotional state is achieved it can then be applied to the suffering of others, turning the initial state of loving-kindness into one of compassion (Singer & Klimecki, 2014). Behavioural research has shown that short-term training of Loving-Kindness and Compassion is associated with increases in daily positive affect (Kok et al., 2013), prosocial behaviour (Leiberg et al., 2011; Weng et al., 2013), resilience (Fredrickson et al., 2008), and empathy (Mascaro, Rilling, Negi, & Raison, 2013). Speaking to its potential efficacy as an explicit regulation strategy, Compassion training has been shown to alter the emotional experience of the individual when confronted with the suffering of others, by specifically increasing positive affect related to experiences of warmth and concern (Klimecki et al., 2013; 2014).

[6] In the following "compassion" refers to the emotional state, while the capitalised "Compassion" refers to the specific meditation technique employed in the current study.

Interestingly, the neural mechanisms supporting Compassion appear to be markedly different from those reported for Reappraisal. Increases in positive affect through Compassion-training have been shown to be paralleled by an increase in brain regions associated with reward and affiliation, such as the ventral striatum (VS) including nucleus accumbens (NACC) and medial orbitofrontal (mOFC), perigenual anterior cingulate (pgACC) and mid-insular cortices (Klimecki et al., 2013; 2014). In contrast, Reappraisal has consistently been associated with activation of regions involved in selective attention, conflict monitoring and cognitive control, including dorsal anterior cingulate (dACC), ventrolateral prefrontal cortex (vlPFC), dorsolateral PFC (dlPFC) and supramarginal gyrus/temporoparietal junction (SMG/TPJ) (Buhle et al., 2014; Ochsner, Silvers, & Buhle, 2012). As such, the neural implementation of Compassion and Reappraisal appears to mirror their conceptual differences, in that Compassion involves the volitional endogenous generation of an emotional and motivational state rather than alteration of exogenously triggered states through cognitive control and self-regulation, as seen in Reappraisal. These qualities suggest the appropriateness of Compassion as a regulation strategy in contexts where emotional connection is important since it does not involve the alteration of one's emotional reactions to stressors directly, but rather the counter-generation of a positive affective state.

In the current study, we tested this hypothesis by investigating how explicit employment of Compassion as an emotion regulation strategy modulates both subjective and neural reactions to emotional stimuli depicting individuals in distress, and how this compares to the modulatory effects of Reappraisal. From previous work (Klimecki et al., 2013; 2014) we know that the default response pattern to such stimuli is negative affect, presumably stemming from empathic distress reactions (see also Condon & Barrett, 2013), and that training is needed in order to generate loving-kindness and compassion when confronted with others' suffering. We therefore recruited a cohort of expert Compassion meditators who had undergone extensive instruction in a Buddhist tradition focusing on altruism and compassion.

Further, all meditators had participated in at least one 3-year full-time retreat at the same institution, in which they practiced these techniques and underwent instruction by the same teachers. This ensured homogenous and expert implementation of Compassion allowing us to describe the workings of the technique at its optimum.

We tested our hypotheses specifically in the context of negative emotion caused by exposure to the suffering of others by adapting the Socio-affective Video Task (SoVT; (Klimecki et al., 2014). This task has previously been successfully used in previous Compassion research, and is optimised to elicit extended negative affect of a social nature. We adapted the SoVT to an emotion-regulation setting by including explicit instructions for the participants to modulate their emotional reactions to the film clips, yielding a design similar to previous studies aimed at differentiating emotion regulation strategies (e.g. Goldin et al., 2008).

We hypothesised that Compassion and Reappraisal should be differentiable in terms of their effects on experienced affect. Provided that Compassion involves the direct generation of positive affect, we expected it to be particularly effective at increasing positive affect. Conversely, as Reappraisal involves re-interpreting the negative aspects of external emotion eliciting stimuli, we expected this to be more effective at decreasing negative affect. Neurally, we expected Compassion to rely less on lateral prefrontal regions thought to be important in top-down cognitive regulation of emotion (Buhle et al., 2014). Rather, we expected Compassion to engage networks known to be associated with affiliation and positive affect in general, including basal ganglia and VS/NACC, mOFC, sgACC/pgACC, and mid-insula (Berridge & Kringelbach, 2013; Kringelbach & Berridge, 2009; Rangel & Hare, 2010; Schultz, 2006; Strathearn, Fonagy, Amico, & Montague, 2009; Vrtička, Andersson, Grandjean, Sander, & Vuilleumier, 2008). Following our hypothesis that Compassion centrally involves the endogenous generation of positive affect, we expected to find evidence of activation of this network independently of stimulus presentation. Mirroring our

behavioural hypotheses, we expected that Compassion and Reappraisal would be differentiable in terms of their impact on core affective processing regions such as the amygdala and VS/NACC. As the regulation of affect through Reappraisal has been shown to be particularly noticeable by its influence on the amygdala (Buhle et al., 2014), we expected lower levels of amygdala activation during Reappraisal than Compassion. Conversely, given the focus of Compassion the generation of positive affect we expected to see higher activation of NACC/VS, a key region in positive affect, during Compassion relative to Reappraisal.

6.2. Methods

6.2.1. Participants

In total 18 long term practitioners of meditation in the Nyingma tradition of Tibetan Buddhism were recruited. This tradition is known for specifically focusing on the cultivation of loving-kindness, altruism, and compassion. Participants were included only if they had participated in a full-time meditation retreat of at least 3 years at the Songsen Chanteloube retreat centre in Dordogne, France. Of these 15 (5 Women; age range = 45-62 years, age mean ± SD = 56.1 ± 4.6 years) completed the current experiment. All participants were Western European Caucasians. Meditation experience was assessed through semi-structured interviews, showing an estimated cumulative total of 40k ± 9k hours of meditation (range = 10k-62k hours). Written and informed consent was obtained from all participants. The study was approved by the Ethics Committee of the University of Leipzig and was carried out in compliance with the Declaration of Helsinki. All participants gave written informed consent, were economically compensated, and debriefed after the study was completed.

6.2.2. MRI acquisition

Structural MRI data were acquired on a 3T Siemens Verio Scanner (Siemens Medical Systems, Erlangen, Germany) using a 32-channel head-coil. High-resolution structural images

were acquired using a T1-weighted 3D-MPRAGE sequence (TR = 2300ms, TE = 2.98ms, TI = 900ms, flip angle = 7°, iPat = 2; 176 sagittal slices, FOV = 256mm, matrix size = 240×256, 1^3 mm voxels; total acquisition time = 5.10 minutes). Functional volumes were collected using a 12-channel head-coil. We employed a T2*-weighted gradient EPI sequence that was optimised (Nichols, Brett, Andersson, Wager, & Poline, 2006) to minimise distortions in medial orbital and anterior temporal regions (TR = 2000ms, TE = 27ms, flip angle = 90°, iPat = 2; 37 slices tilted at ~30° from the AC/PC axial plane, FOV = 210 mm, matrix size = 70×70, 3^3 mm voxels, 1mm gap; 700 volumes per session).

6.2.3. Stimuli

The stimuli were short film clips (10-12 seconds in length; 40 negative and 20 neutral stimuli) taken from the previously validated SoVT stimulus set (for details see Klimecki et al., 2013). The negative film clips depicted people in distress, such as scenes from documentaries and newscasts of e.g. starving children, crying mothers, or hospitalised individuals. The matched neutral film clips depicted similar individuals in non-distressing situations doing everyday activities. The stimuli were back-projected using a mirror setup. Eyesight was corrected using goggles where appropriate.

6.2.4. Procedure

Prior to testing, subjects were sent short written descriptions of each of the conditions that they were requested to read and reflect upon. Immediately prior to scanning subjects were reminded of this text before they underwent a guided training session. In this session the different strategies were explained and discussed before the subjects performed a training session of 6 trials, allowing them to experience and practice each strategy employed in the experiment (Watch- Neutral, Watch- Negative, Reappraisal, Compassion, Distraction and Open-Presence). The Distraction and Open-Presence conditions will be the focus of another

forthcoming paper and do not figure in the current study. Subjects were instructed to start implementing the strategies immediately upon receipt of instruction. To ensure homogeneity in strategy execution, subjects were asked to describe each strategy in their own words prior to the start of training and to describe in detail exactly how they implemented the strategy after each practice trial, with misunderstandings corrected when apparent. For the Compassion condition subjects were asked to employ their Compassion-meditation technique, so as to generate a warm feeling of positive affect and caring towards the individuals depicted in the film. For Reappraisal, subjects were asked to reinterpret the clips by thinking about what was occurring in a way in which the narrative ended more positive than was immediately apparent, i.e. to employ a Reappraisal technique with positive emphasis (cf. Wager et al., 2008). We chose to use positive Reappraisal to ensure comparability of with

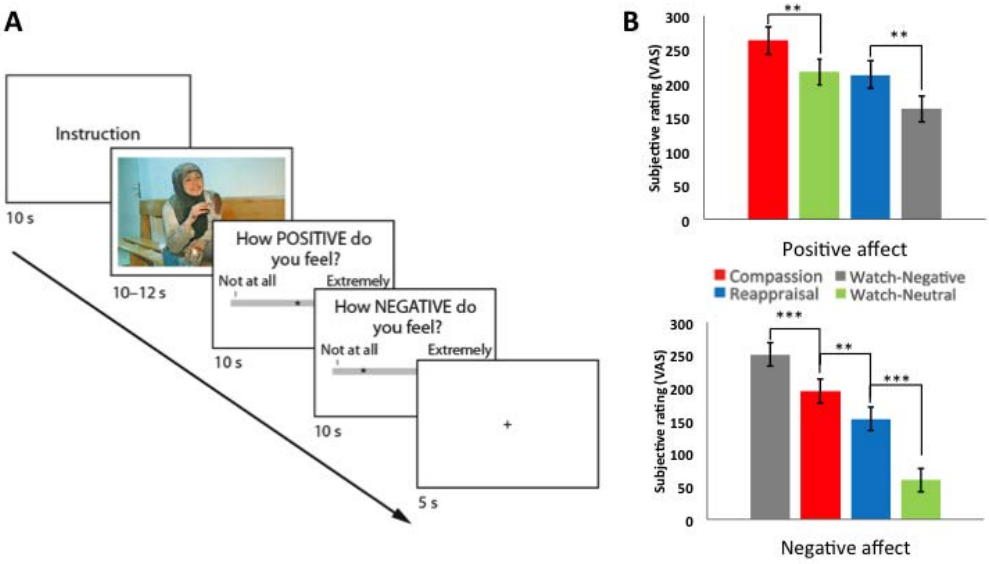


Figure 6.1: Trial structure and behavioural results. A) Schematic of a single trial. While being scanned, subjects were first instructed which strategy to employ, then they viewed a 10-12 second film clip while employing the strategy, whereupon they rated their experienced positive and negative affect on two serially presented scales. **B)** Behavioural results of experienced affect in each of the 4 conditions. Compassion was associated with significantly higher positive affect than all other conditions, while Reappraisal was associated with significantly higher positive affect than the Watch-Negative condition. Both compassion and Reappraisal decreased negative affect, though Reappraisal did so significantly more. ** = $p < .01$, *** = $p < .001$. All p-values Bonferroni corrected.

Compassion in terms of regulatory goal. For the Watch conditions, subjects were asked to respond naturally without trying to alter their reactions.

The subjects underwent two sessions of scanning. Emotion regulation strategies (Reappraisal & Distraction) and meditation techniques (Compassion & Open-Presence) were implemented in separate sessions. Each session consisted of 4 conditions (Session A: [Reappraisal, Distraction, Watch-Negative, Watch-Neutral], Session B: [Compassion, Open-Presence, Watch-Negative, Watch-Neutral]). 10 trials of each condition were performed in each session for a total of 40 trials. Stimulus and session order were counterbalanced across subjects. Within each session condition order was pseudo-randomised with the constraint that no more than 2 consecutive iterations of any condition could occur. Each trial (see Figure 1a) consisted of 1) 10 second instruction, 2) 10-12 second film clip presentation, 3) 10 second rating of experienced positive emotion, 4) 10 second rating of experienced negative emotion and 5) 5 second fixation cross. Ratings were given using a button box to move a cursor on a 600-point visual analogue scale (VAS) ranging from “Not at all” to “Extremely”. Subjects were instructed to rate their affect as it was at the moment of report rather than how they remembered it to be during the film clip.

6.2.5. fMRI preprocessing

Preprocessing was done using SPM8 (r5236, Wellcome Trust) and included slice time correction, combined realignment and field-map based unwarping, DARTEL-based normalization (Ashburner, 2007) and smoothing with an isotropic Gaussian kernel with FWHM of 8mm. As controlled breathing is a key component of meditation, we accounted for potential respiratory artefacts and confounds by despiking the data using the ArtRepair toolbox (version 4, http://cibsr.stanford.edu/tools/human-brain-project/artrepair-software.html) and removing the run-specific global signal for each voxel (P. M. Macey,

Macey, Kumar, & Harper, 2004) in line with previous work (e.g. Farb, Segal, & Anderson, 2013)

6.2.6. fMRI analysis

To ensure robustness of the analyses in the face of potential differences in temporal dynamics of the conditions, a Finite Impulse Response (FIR) deconvolution approach was used. Single subject models included both runs, and included regressors coding the onset of instruction in each trial and the following fourteen 2s time bins for each condition. Separate, non-orthogonalised parametric regressors for positive and negative affect ratings were included, as well as 7 nuisance regressors coding movement and linear temporal trend. Models were high-pass filtered at 0.005 Hz and temporal autocorrelations were modelled using an AR(1) process. Group repeated-measures analyses were performed using GLMFlex (http://mrtools.mgh.harvard.edu/) and constrained to voxels within a grey matter mask derived from the MNI-projected DARTEL-generated template, created using the optimised thresholding algorithm included in the Masking toolbox (Ridgway et al., 2009). Inference was performed on truncated AUC estimates of BOLD signal, with separate *t*-contrasts performed for the Preparation (0-10 seconds) and Implementation (10-22 seconds) phases of the trial. Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha < .05$ ($T > 3.36$, $p = .001$, $k > 30$) as determined by AFNI's *Alphasim* Monte Carlo simulation method. Cortical surface renderings were created using NeuroElf, while subcortical renderings were made using scripts provided by Tor Wager and colleagues (available at http://wagerlab.colorado.edu/tools). Anatomical labels were determined using a combination of the TD client implemented in NeuroElf, the Anatomy toolbox (Eickhoff et al., 2005) and stereotactic atlases (Duvernoy, 1999; Duvernoy, Cattin, & Risold, 2013; Naidich et al., 2009).

ROI analyses were done using the MarsBar toolbox (http://marsbar.sourceforge.net). To test our *a priori* hypotheses that Reappraisal and Compassion should differ in terms of

their temporal profiles and their effects on the neural substrates of positive and negative affect, we focused our analyses on subregions of the nucleus accumbens/ventral striatum and amygdala in which activation varied as a function of reported positive and negative affect, respectively (see *Appendix:* Supplemental methods A1.2.1 for details on the ROI selection procedure).

6.3. Results

6.3.1. Behavioural results

Figure 6.1 B shows the differences in subjective ratings as a function of employed strategy. Positive and negative emotion ratings were analysed separately using linear mixed modelling (LMM) as implemented in SPSS 21, with Condition (Compassion, Reappraisal, Watch-Negative, Watch-Neutral) as a fixed effect and subject-level random intercepts. This revealed a significant main effect of Condition for both Positive ($F(3, 882) = 26.66$, $p < .001$) and Negative ($F(3, 882) = 176.10$, $p < .001$) ratings. Post-hoc comparisons were corrected for multiple comparisons using the sequential Bonferroni method. These revealed that the Negative-Watch condition elicited significantly more negative ($\mu = 191.19$, SE = 8.47), $t(882) = 22.58$, $p < .001$) and significantly less positive ($\mu = -54.74$, SE = 9.71), $t(882) = -5.64$, $p < .001$) affect than the Neutral-Watch condition, demonstrating successful emotion induction. Further comparisons revealed that both Compassion and Reappraisal decreased negative (Compassion: ($\mu = -56.13$, SE = 10.37), $t(882) = -5.41$, $p < .001$); Reappraisal: ($\mu = -54.74$, SE = 9.71), $t(882) = -5.64$, $p < .001$) and increased positive affect (Compassion: $\mu = 101$, SE = 11,89, $t(882) = 8.50$, $p < .001$; Reappraisal: $\mu = -96.82$, SE = 10.37), $t(882) = -9.43$, $p < .001$) relative to the Negative-Watch condition, demonstrating their efficacy at regulating affective states. Importantly, direct comparison of Compassion and Reappraisal revealed that Compassion was associated with significantly higher positive affect ($\mu = 50.29$, SE = 13.73), $t(882) = 3.66$, $p < .005$) while Reappraisal was associated with significantly lower negative

affect (μ = -54.74, SE = 9.71), $t(882) = -5.64$, $p < .001$). Furthermore only Compassion increased positive affect *above* the Watch- Neutral condition (Compassion: μ = 46.28, SE = 11.89), $t(882) = 3.89$, $p < .001$; Reappraisal: μ = -4.01, SE = 11.89), $t(882) = -0.34$, $p > .1$) supporting the notion that Compassion is uniquely associated with an increase of positive affect, whereas Reappraisal occasioned a return to baseline positive affect.

6.3.2. fMRI results

6.3.2.1. Validation contrasts

In order to establish the efficacy of the emotion induction procedure used and the comparability of the Reappraisal implementation to previous work, several validation contrasts were performed (Reappraisal> Watch-Negative, Watch-Negative>Watch-Neutral). These contrasts are reported in *Appendix:* Tables A3.2.1 and A3.2.2. The Watch-Negative>Watch-Neutral contrast served as a validation contrast for successful emotion induction, and showed increased activation of core affective processing regions such as amygdala and insula, in addition to widespread activation of regions associated with both cognitive and perceptual components of affect. The Reappraisal>Watch-Negative contrast served to validate the implementation of Reappraisal in the current study and establish comparability to previous studies. This revealed a pattern of results closely resembling previous work (see e.g. Buhle et al., 2014; Goldin et al., 2008), including fronto-parietal activation and deactivation of amygdala (*Appendix*: Figure A2.2.1, Table A3.2.1).

6.3.2.2. Compassion vs. Watch- Negative

The contrast of Compassion over Watch-Negative assessed the neural correlates of the active employment of Compassion when preparing for and actively implementing Compassion to regulate one's affective response to negative stimuli. Results from these

contrasts are reported in Table 6.1. As we had no specific hypotheses regarding deactivations these were not interpreted, but are reported in *Appendix*: Table A3.2.3.

In the preparation phase (Figure 6.2A), activations were observed laterally in frontal regions, including middle frontal gyrus (MFG) and triangular/orbital junction of the inferior frontal gyrus (IFG), supplementary motor area (SMA), as well as superior and inferior parietal lobules. Medially, activations were observed in frontopolar and medial orbitofrontal cortex (mOFC) and ventromedial PFC (vmPFC), peri- and subgenual ACC (pgACC/sgACC) and posterior cingulate cortex (PCC). Subcortical activations were observed in pulvinar and medial nuclei of the thalamus, hypothalamus, ventral striatum, superior and inferior parietal lobules. Additionally, large portions of the cerebellum were activated.

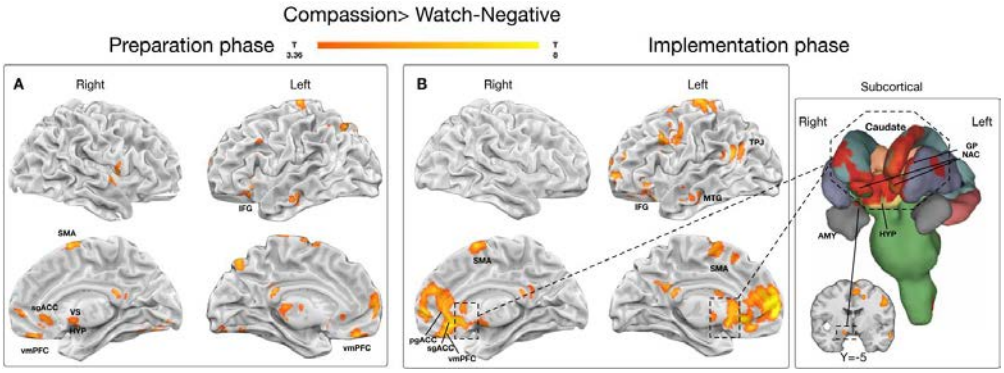


Figure 6.2: Compassion-related activation. Results from the whole brain contrast of Compassion> Watch-negative presented separately for the Preparation and Implementation phases. All results thresholded at FWEc $\alpha < .05$ as determined by AFNI's *Alphasim* ($p < .001$, extent threshold = 30 voxels).

In the Implementation phase (Figure 6.2B), substantial activations were observed in medial PFC, including vmPFC and mOFC, as well as genual ACC and SMA. Left dorsolateral and ventrolateral prefrontal activations were again observed, including middle and superior frontal gyri (SFG), as well as the orbital portion of the triangular and orbital IFG. Further activations were observed in SMG/TPJ and posterior cingulate, as well as middle and inferior temporal gyri, and portions of the cerebellum. Subcortical activations were observed

in VS including NACC, globus pallidus, caudate, putamen, hypothalamus, and superficial portions of the right amygdala.

6.3.2.3. Compassion vs. Reappraisal

The direct contrast of Compassion and Reappraisal over the course of the Preparation and Implementation phases was performed to identify differences in the neural underpinnings of Compassion and Reappraisal, as well as their temporal dynamics. The results from these contrasts are reported in Table 6.2. In the Preparation phase (Figure 6.3A), higher activation was found for the Compassion condition primarily in medial frontal regions, including vmPFC/gyrus rectus, pgACC, sgACC, dorsal ACC/SMA, precuneus. Additional activations

Region	Side	Extent (voxel)	*t* (max)	*t* (avg)	MNI x	MNI y	MNI z	Label/BA
Compassion> Watch-Negative								
Preparation phase								
Precuneus	**L**	**313**	**9.44**	**3.90**	**-12**	**-70**	**51**	**7**
Precuneus	*L*	*161*	*9.44*	*4.20*	*-12*	*-70*	*51*	*7*
Superior Parietal Lobule	*L*	*77*	*5.98*	*3.78*	*-33*	*-70*	*44*	*7*
Inferior Parietal Lobule	*L*	*31*	*4.43*	*2.96*	*-39*	*-46*	*39*	*19*
Cerebellum	***L***	***1164***	***8.66***	***4.21***	***-6***	***-67***	***-11***	***Declive***
Cerebellum	*L*	*161*	*8.66*	*4.92*	*-6*	*-67*	*-11*	*Declive*
Cerebellum	*R*	*138*	*8.30*	*4.84*	*6*	*-73*	*-26*	*Pyramis*
Cerebellum	*R*	*151*	*6.94*	*4.30*	*25*	*-74*	*-15*	*Declive*
Cerebellum	*L*	*129*	*6.26*	*4.25*	*-19*	*-64*	*-15*	*Declive*
Cerebellum	*R*	*57*	*6.93*	*4.28*	*41*	*-76*	*-40*	*Inferior Semi-Lunar*
Cerebellum	*R*	*119*	*6.56*	*4.12*	*38*	*-66*	*-20*	*Declive*
Cerebellum	*R*	*92*	*6.36*	*4.30*	*20*	*-83*	*-29*	*Tuber*
Cerebellum	*R*	*86*	*5.56*	*3.85*	*12*	*-81*	*-14*	*Declive*
Cerebellum	*L*	*51*	*5.06*	*3.38*	*-10*	*-84*	*-11*	*Lingual Gyrus*
Cerebellum	*R*	*46*	*4.98*	*3.63*	*46*	*-67*	*-28*	*Tuber*
Cerebellum	*L*	*39*	*4.78*	*3.12*	*-23*	*-84*	*-14*	*Declive*
Cerebellum	*L*	*45*	*4.65*	*3.34*	*-45*	*-70*	*-22*	*Declive*
Thalamus	**L**	**93**	**8.12**	**3.86**	**0**	**-27**	**2**	**Pulvinar**
Thalamus	*L*	*38*	*8.12*	*4.45*	*0*	*-27*	*2*	*Pulvinar*
Thalamus	*L*	*31*	*5.66*	*3.46*	*0*	*-14*	*12*	*Lateral-Dorsal*
Inferior Frontal Gyrus	**L**	**58**	**6.70**	**4.20**	**-42**	**30**	**-15**	**47**
Superior Frontal Gyrus	**R**	**107**	**6.87**	**3.55**	**3**	**8**	**60**	**6**

Superior Frontal Gyrus	*R*	*34*	*6.87*	*3.71*	*3*	*8*	*60*	*6*
Superior Frontal Gyrus	*L*	*53*	*5.27*	*3.31*	*-8*	*0*	*67*	*6*
Inferior Frontal Gyrus	**L**	**50**	**6.87**	**3.89**	**-53**	**12**	**1**	**45**
Precentral Gyrus	**L**	**50**	**6.50**	**3.51**	**-19**	**-23**	**71**	**6**
Middle Temporal Gyrus	**L**	**55**	**6.43**	**3.90**	**-61**	**-20**	**-9**	**21**
Posterior Cingulate	**L**	**51**	**6.43**	**3.90**	**0**	**-38**	**22**	**Posterior Cingulate**
Precentral Gyrus	**R**	**65**	**6.03**	**3.73**	**60**	**0**	**7**	**Precentral Gyrus**
Orbital Gyrus	**L**	**264**	**5.91**	**3.68**	**0**	**39**	**-20**	**11**
Orbital Gyrus	*L*	*68*	*5.91*	*3.81*	*0*	*39*	*-20*	*11*
Medial Frontal Gyrus	*L*	*46*	*5.77*	*3.94*	*-12*	*58*	*9*	*10*
Anterior Cingulate	*L*	*58*	*5.17*	*3.37*	*0*	*32*	*-2*	*24*
Medial Frontal Gyrus	*R*	*39*	*4.94*	*3.34*	*4*	*52*	*-2*	*10*
Inferior Frontal Gyrus	***L***	***53***	***5.21***	***3.41***	***-53***	***10***	***33***	***9***
Caudate	**L**	**77**	**4.88**	**3.42**	**0**	**4**	**1**	**Head**
Implementation phase								
Anterior Cingulate	**L**	**1651**	**10.59**	**4.40**	**-4**	**54**	**-2**	**10**
Anterior Cingulate	*L*	*125*	*10.59*	*5.80*	*-4*	*54*	*-2*	*10*
Medial Frontal Gyrus	*L*	*130*	*10.00*	*5.93*	*-12*	*55*	*9*	*10*
Anterior Cingulate	*L*	*103*	*6.74*	*4.91*	*-6*	*26*	*-3*	*24*
Middle Frontal Gyrus	*L*	*47*	*6.62*	*3.95*	*-25*	*45*	*-11*	*11*
Anterior Cingulate	*R*	*171*	*6.59*	*4.62*	*4*	*31*	*-10*	*32*
Inferior Frontal Gyrus	*L*	*48*	*6.31*	*4.39*	*-24*	*33*	*-9*	*47*
Superior Frontal Gyrus	*L*	*36*	*6.77*	*4.29*	*-25*	*57*	*-1*	*10*
Anterior Cingulate	*R*	*97*	*6.71*	*4.11*	*11*	*33*	*9*	*32*
Lentiform Nucleus	*L*	*74*	*6.67*	*4.27*	*-16*	*13*	*2*	*Putamen*
Ventral Striatum	*L*	*58*	*6.47*	*3.97*	*-13*	*13*	*-6*	*Nucleus Accumbens*
Medial Frontal Gyrus	*R*	*45*	*6.43*	*4.29*	*15*	*54*	*1*	*10*
Middle Frontal Gyrus	*L*	*99*	*6.33*	*4.16*	*-29*	*41*	*20*	*10*
Medial Frontal Gyrus	*L*	*126*	*6.26*	*4.19*	*-8*	*42*	*16*	*9*
Medial Frontal Gyrus	*R*	*96*	*6.08*	*3.88*	*12*	*44*	*19*	*9*
Caudate	*L*	*61*	*5.75*	*4.29*	*-16*	*23*	*3*	*Caudate*
Medial Frontal Gyrus	*R*	*38*	*5.50*	*4.06*	*8*	*55*	*15*	*10*
Lentiform Nucleus	*L*	*47*	*5.46*	*3.62*	*-23*	*5*	*19*	*Putamen*
Middle Frontal Gyrus	*L*	*33*	*5.45*	*3.72*	*-35*	*42*	*10*	*10*
Hypothalamus	*R*	*64*	*5.17*	*3.47*	*6*	*-3*	*-3*	*Anterior*
Caudate	*R*	*38*	*4.87*	*3.37*	*13*	*20*	*8*	*Caudate Body*
Anterior Cingulate	*L*	*34*	*4.75*	*3.58*	*-7*	*33*	*9*	*24*
Cerebellum	**R**	**1466**	**9.98**	**4.43**	**36**	**-47**	**-29**	**Culmen**
Cerebellum	*R*	*88*	*9.98*	*5.42*	*36*	*-47*	*-29*	*Culmen*
Cerebellum	*R*	*120*	*8.94*	*5.21*	*45*	*-64*	*-28*	*Tuber*
Cerebellum	*R*	*99*	*8.84*	*4.55*	*6*	*-73*	*-26*	*Pyramis*
Cerebellum	*L*	*127*	*8.44*	*5.07*	*0*	*-64*	*-26*	*Pyramis*
Cerebellum	*R*	*84*	*6.36*	*4.57*	*6*	*-53*	*-23*	*Anterior LobeDentate*
Cerebellum	*R*	*63*	*6.22*	*4.83*	*26*	*-81*	*-30*	*Tuber*
Cerebellum	*R*	*158*	*6.99*	*4.75*	*28*	*-65*	*-23*	*Uvula*

Cerebellum	*L*	*87*	*6.49*	*4.27*	*-6*	*-68*	*-9*	*Culmen*
Cerebellum	*R*	*115*	*6.27*	*4.76*	*19*	*-71*	*-30*	*Pyramis*
Cerebellum	*R*	*68*	*6.25*	*3.92*	*14*	*-54*	*-43*	*Cerebellar Tonsil*
Cerebellum	*R*	*39*	*5.91*	*3.49*	*20*	*-34*	*-19*	*Culmen*
Cerebellum	*R*	*54*	*5.90*	*4.26*	*25*	*-77*	*-15*	*Declive*
Cerebellum	*L*	*70*	*5.45*	*3.54*	*-13*	*-42*	*-34*	*Cerebellar Tonsil*
Cerebellum	*L*	*37*	*5.19*	*3.17*	*-22*	*-31*	*-17*	*Culmen*
Cerebellum	*R*	*60*	*5.15*	*4.01*	*33*	*-77*	*-39*	*Inferior Semi-Lunar*
Cerebellum	*L*	*71*	*5.13*	*3.73*	*-5*	*-48*	*-34*	*Cerebellar Tonsil*
Cerebellum	*L*	*60*	*4.87*	*3.41*	*-18*	*-64*	*-17*	*Declive*
Cerebellum	*L*	*35*	*4.47*	*3.27*	*-17*	*-48*	*-19*	*Culmen*
Inferior Frontal Gyrus	**L**	**81**	**8.90**	**3.77**	**-32**	**18**	**-17**	**47**
Inferior Frontal Gyrus	*L*	*32*	*6.64*	*4.34*	*-48*	*23*	*-8*	*47*
Precentral Gyrus	*L*	*357*	*6.82*	*3.67*	*-19*	*-23*	*71*	*4*
Precentral Gyrus	*L*	*103*	*6.82*	*3.97*	*-19*	*-23*	*71*	*4*
Medial Frontal Gyrus	*R*	*55*	*6.43*	*3.99*	*7*	*5*	*61*	*6*
Middle Frontal Gyrus	*L*	*50*	*5.85*	*3.46*	*-24*	*2*	*60*	*6*
Precentral Gyrus	*L*	*33*	*5.56*	*3.48*	*-20*	*-18*	*62*	*6*
Medial Frontal Gyrus	*L*	*58*	*5.31*	*3.65*	*-8*	*-3*	*55*	*6*
Precuneus	*L*	*73*	*6.61*	*3.08*	*-15*	*-70*	*51*	*7*
Precuneus	*L*	*42*	*6.61*	*3.28*	*-15*	*-70*	*51*	*7*
Precuneus	*L*	*31*	*4.28*	*2.81*	*-12*	*-53*	*58*	*7*
Middle Frontal Gyrus	**L**	**238**	**6.51**	**4.00**	**-49**	**2**	**40**	**6**
Middle Frontal Gyrus	*L*	*209*	*6.51*	*4.12*	*-49*	*2*	*40*	*6*
Posterior Cingulate	**L**	**120**	**6.40**	**3.69**	**-3**	**-42**	**19**	**29**
Posterior Cingulate	*L*	*70*	*6.40*	*4.02*	*-3*	*-42*	*19*	*29*
Precuneus	*L*	*50*	*4.45*	*3.23*	*0*	*-56*	*30*	*7*
Middle Temporal Gyrus	**L**	**73**	**6.14**	**3.70**	**-59**	**-8**	**-6**	**21**
Cerebellum	**L**	**90**	**5.57**	**3.68**	**-37**	**-57**	**-23**	**Culmen**
Cerebellum	*L*	*53*	*5.57*	*3.62*	*-37*	*-57*	*-23*	*Culmen*
Cerebellum	*L*	*37*	*5.30*	*3.78*	*-42*	*-67*	*-23*	*Tuber*
Supramarginal Gyrus	**L**	**173**	**5.39**	**3.47**	**-53**	**-52**	**23**	**40**
Precentral Gyrus	**L**	**61**	**5.31**	**3.35**	**-44**	**6**	**11**	**44**
Precentral Gyrus	*L*	*45*	*5.31*	*3.47*	*-44*	*6*	*11*	*44*

Table 6.1: Brain regions activated during Compassion relative to Watch-Negative. Cluster maxima are reported in bold. Local maxima (*italicised*) denote subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha < .05$ ($T > 3.36$, $p = .001$, $k > 30$) as determined by AFNI's *Alphasim* Monte Carlo simulation method. For consistency, subclusters smaller than the respective cluster thresholds are not reported.

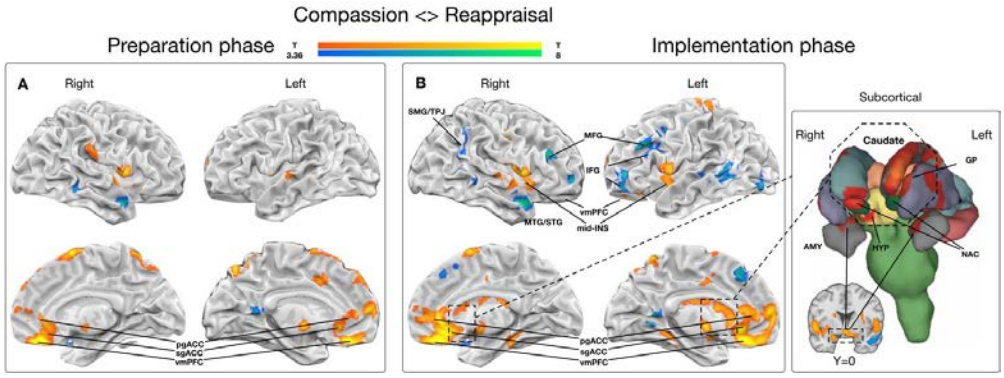


Figure 6.3: Differential activation of Compassion and Reappraisal. Results from the whole brain contrast of Compassion>Reappraisal and Reappraisal>Compassion presented separately for the Preparation and Implementation phases. Red-scaled blobs denote regions of significantly higher activation during Compassion than Reappraisal, while blue-scaled blobs denote regions with higher activation during Reappraisal than Compassion. All results thresholded at FWEc $\alpha < .05$ as determined by AFNI's *Alphasim* ($p < .001$, extent threshold = 30 voxels).

were observed in bilateral superior temporal gyrus (STG) and right IFG/operculum ranging into mid-insula. In contrast, higher activation for the Reappraisal condition was found in posterior and polar middle temporal gyrus (MTG), posterior cingulate/precuneus and cerebellum.

For the Implementation phase (Figure 6.3B), increased activation was found for the Compassion condition in medial PFC, including vmPFC, mOFC, sgACC, pgACC, frontopolar cortex, bilateral SMA and mid-cingulate. Laterally, activations were observed in bilateral operculum/ mid- insula, STG, precuneus and right fusiform gyrus. Subcortically, activations were observed in NACC/VS, bilateral amygdala, hypothalamus, caudate, globus pallidus, putamen, right hippocampus and ventral anterior portions of the thalamus. For Reappraisal, increased activation was observed in dorsolateral PFC, including bilateral MFG and left IFG, pre-SMA/medial SFG, right SMG/TPJ, right anterior MTG/STG and left posterior MTG, left calcarine gyrus and cerebellum.

Region	Side	Extent (voxel)	*t* (max)	*t* (avg)	MNI x	MNI y	MNI z	Label/BA
Compassion> Reappraisal								
Preparation phase								
Rectal Gyrus	**R**	**702**	**11.79**	**4.47**	**11**	**32**	**-21**	**11**
Rectal Gyrus	*R*	*173*	*11.79*	*5.52*	*11*	*32*	*-21*	*11*
Superior Orbital Gyrus	*L*	*91*	*9.20*	*4.45*	*-13*	*30*	*-20*	*11*
Superior Medial Gyrus	*L*	*143*	*6.95*	*4.34*	*-4*	*52*	*4*	*10*
Anterior Cingulate	*R*	*149*	*6.44*	*4.04*	*4*	*32*	*-8*	*32*
Middle Orbital Gyrus	*L*	*79*	*6.09*	*3.97*	*-21*	*41*	*-16*	*11*
SMA	**R**	**237**	**8.47**	**4.04**	**3**	**8**	**58**	**6**
SMA	*R*	*55*	*8.47*	*4.71*	*3*	*8*	*58*	*6*
Superior Medial Gyrus	*L*	*57*	*6.58*	*4.19*	*0*	*52*	*26*	*9*
Superior Medial Gyrus	*L*	*54*	*5.67*	*3.67*	*-4*	*40*	*43*	*8*
Precuneus	**L**	**302**	**6.43**	**4.37**	**-3**	**-70**	**42**	**7**
Precuneus	*L*	*192*	*6.43*	*4.57*	*-3*	*-70*	*42*	*7*
Precuneus	*L*	*66*	*6.67*	*4.35*	*0*	*-44*	*59*	*5*
Superior Temporal Gyrus	**R**	**150**	**6.97**	**4.01**	**60**	**0**	**4**	**22**
Implementation phase								
Rectal Gyrus	**R**	**2631**	**11.04**	**4.32**	**11**	**32**	**-21**	**11**
Rectal Gyrus	*R*	*181*	*11.04*	*5.84*	*11*	*32*	*-21*	*11*
Superior Orbital Gyrus	*L*	*102*	*9.94*	*5.55*	*-13*	*30*	*-20*	*11*
Anterior Cingulate	*R*	*212*	*8.95*	*5.61*	*4*	*32*	*-8*	*32*
Anterior Cingulate	*R*	*153*	*8.05*	*4.75*	*4*	*41*	*-1*	*32*
Superior Temporal Gyrus	*R*	*101*	*6.61*	*4.51*	*60*	*0*	*4*	*22*
Postcentral Gyrus	*R*	*86*	*6.12*	*3.83*	*43*	*-14*	*32*	*3A*
Superior Medial Gyrus	*L*	*163*	*6.05*	*4.81*	*-11*	*55*	*4*	*10*
Claustrum	*L*	*160*	*6.04*	*4.44*	*-23*	*22*	*3*	*Claustrum*
Superior Orbital Gyrus	*L*	*119*	*6.90*	*4.40*	*-14*	*44*	*-18*	*11*
Rolandic Operculum	*R*	*78*	*6.90*	*3.96*	*54*	*-12*	*13*	*43*
Anterior Cingulate	*R*	*94*	*5.94*	*4.00*	*7*	*27*	*5*	*24*
Insula	*R*	*107*	*5.90*	*4.08*	*41*	*11*	*-2*	*13*
Thalamus	*R*	*64*	*5.73*	*3.73*	*24*	*-31*	*11*	*Pulvinar*
Lentiform Nucleus	*L*	*106*	*5.73*	*3.65*	*-19*	*5*	*12*	*Putamen*
Anterior Cingulate	*R*	*107*	*5.61*	*4.07*	*18*	*41*	*0*	*10*
Anterior Cingulate	*L*	*74*	*5.51*	*3.88*	*-22*	*43*	*3*	*10*
Superior Temporal Gyrus	*R*	*47*	*5.38*	*3.86*	*50*	*10*	*-4*	*22*
Anterior Cingulate	*L*	*84*	*5.17*	*4.00*	*-3*	*27*	*7*	*24*
Lentiform Nucleus	*R*	*53*	*5.10*	*3.36*	*23*	*6*	*15*	*Putamen*
Superior Frontal Gyrus	*R*	*52*	*5.05*	*3.42*	*22*	*54*	*-1*	*10*
Claustrum	*R*	*51*	*4.36*	*3.56*	*27*	*25*	*3*	*Claustrum*
Thalamus	**L**	**96**	**8.01**	**4.34**	**-3**	**-8**	**2**	**Ventral Anterior**
Thalamus	*L*	*73*	*8.01*	*4.71*	*-3*	*-8*	*2*	*Ventral Anterior*
Precentral Gyrus	**L**	**93**	**6.60**	**3.63**	**-22**	**-23**	**68**	6/4

Fusiform Gyrus	**R**	**175**	**6.49**	**3.67**	**29**	**-33**	**-17**	**Fusiform Gyrus**
Fusiform Gyrus	*R*	*58*	*6.49*	*3.99*	*29*	*-33*	*-17*	*Fusiform Gyrus*
Superior Temporal Gyrus	**L**	**421**	**6.26**	**3.95**	**-49**	**4**	**-4**	**22**
Superior Temporal Gyrus	*L*	*86*	*6.26*	*4.11*	*-49*	*4*	*-4*	*22*
Rolandic Operculum	*L*	*112*	*6.17*	*4.22*	*-54*	*0*	*4*	*22*
Inferior Frontal Gyrus	*L*	*73*	*6.11*	*4.05*	*-47*	*6*	*6*	*44*
Amygdala	*L*	*47*	*5.29*	*3.73*	*-22*	*1*	*-15*	*Superficial*
Insula	*L*	*47*	*4.05*	*3.21*	*-35*	*1*	*3*	*Mid*
Precuneus	**L**	**220**	**5.93**	**3.58**	**-6**	**-56**	**52**	**7**
Precuneus	*L*	*153*	*5.93*	*3.67*	*-6*	*-56*	*52*	*7*
Cingulate Gyrus	**L**	**88**	**5.47**	**3.63**	**-22**	**-43**	**26**	**31**
Cingulate Gyrus	*L*	*77*	*5.47*	*3.64*	*-22*	*-43*	*26*	*31*
Reappraisal>Compassion								
Preparation phase								
Cerebellum	**R**	**1099**	**9.24**	**4.46**	**11**	**-40**	**-46**	**Cerebellar Tonsil**
Cerebellum	*R*	*111*	*9.24*	*5.15*	*11*	*-40*	*-46*	*Cerebellar Tonsil*
Cerebellum	*L*	*92*	*6.97*	*4.93*	*-2*	*-54*	*-51*	*Cerebellar Tonsil*
Cerebellum	*L*	*49*	*6.86*	*6.06*	*-10*	*-43*	*-48*	*Cerebellar Tonsil*
Brainstem	*R*	*137*	*6.36*	*4.88*	*1*	*-35*	*-37*	*Medulla*
Cerebellum	*R*	*116*	*6.28*	*4.29*	*12*	*-70*	*-37*	*Inferior Semi-Lunar Lobule*
Cerebellum	*L*	*63*	*6.12*	*4.74*	*-15*	*-40*	*-39*	*Cerebellar Tonsil*
Cerebellum	*R*	*124*	*6.47*	*4.59*	*6*	*-62*	*-26*	*Nodule*
Cerebellum	*L*	*92*	*6.11*	*4.35*	*-16*	*-52*	*-49*	*Cerebellar Tonsil*
Cerebellum	*R*	*60*	*4.92*	*3.65*	*16*	*-42*	*-29*	*Anterior Lobe*
Cerebellum	*L*	*170*	*4.86*	*3.69*	*-10*	*-54*	*-31*	*Cerebellar Tonsil*
Cerebellum	*R*	*55*	*4.54*	*3.64*	*23*	*-58*	*-51*	*Cerebellar Tonsil*
Middle Temporal Gyrus	**R**	**92**	**5.89**	**3.81**	**49**	**0**	**-17**	**21**
Inferior Frontal Gyrus	**L**	**95**	**5.37**	**3.50**	**-38**	**36**	**3**	**46**
Posterior Cingulate	**L**	**99**	**4.89**	**3.44**	**-9**	**-47**	**8**	**29**
Implementation phase								
Inferior Frontal Gyrus	**L**	**136**	**6.83**	**4.26**	**-51**	**25**	**25**	**45/44**
Inferior Frontal Gyrus	**R**	**93**	**6.20**	**4.26**	**49**	**25**	**20**	**45**
Cerebellum	**L**	**232**	**6.07**	**3.99**	**-20**	**-78**	**-37**	**Inferior Semi-Lunar**
Cerebellum	*L*	*60*	*6.07*	*4.72*	*-20*	*-78*	*-37*	*Inferior Semi-Lunar*
Cerebellum	*L*	*72*	*5.52*	*3.73*	*-20*	*-81*	*-26*	*Uvula*
Cerebellum	*L*	*67*	*5.22*	*3.74*	*-26*	*-70*	*-33*	*Pyramis*
Middle Temporal Gyrus	**L**	**245**	**6.97**	**3.94**	**-53**	**-34**	**-2**	**39**
Middle Temporal Gyrus	*L*	*61*	*6.97*	*4.42*	*-53*	*-34*	*-2*	*39*
Middle Temporal Gyrus	*L*	*63*	*5.79*	*4.00*	*-58*	*-57*	*7*	*21*
Middle Temporal Gyrus	*L*	*55*	*5.23*	*3.62*	*-60*	*-43*	*3*	*22*
Medial Temporal Gyrus	**R**	**133**	**6.68**	**4.20**	**53**	**5**	**-16**	**21**
Calcarine Gyrus	**L**	**95**	**6.66**	**3.77**	**-3**	**-57**	**4**	**17**

Inferior Frontal Gyrus	**L**	**123**	**6.61**	**4.13**	**-45**	**49**	**-3**	**45**
Superior Medial Gyrus	**L**	**90**	**5.47**	**3.88**	**-4**	**26**	**39**	**8**
Angular Gyrus	**R**	**117**	**5.07**	**3.59**	**56**	**-58**	**24**	**39**

Table 6.2. Brain regions differentially activated in Compassion and Reappraisal. Cluster maxima are reported in bold. Local maxima (*italicised*) denote subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha < .05$ ($T > 3.36$, $p = .001$, $k > 30$) as determined by AFNI's *Alphasim* Monte Carlo simulation method. For consistency, subclusters smaller than the respective cluster thresholds are not reported.

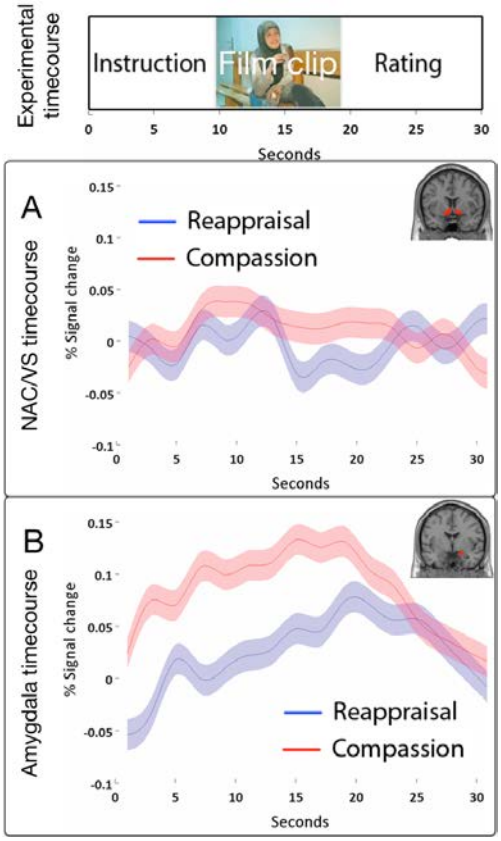


Figure 6.4: Dynamics of compassion-based emotion regulation. Event related time-courses in amygdala and NACC/VS ROIs for Compassion (red) and Reappraisal (blue), subtracted from their respective Watch-Negative baselines. Shaded areas denote within-subject standard errors (Loftus & Masson, 1994). Time points where shaded area does not overlap the abscissa denote significant effects relative to Watch-Negative baseline. Time courses are plotted relative to actual timing of experiment and have been interpolated for presentation purposes only.

6.3.3.4. Temporal Dynamics of Compassion and Reappraisal in Regions tracking Subjective Affect

To test our hypotheses that Reappraisal and Compassion would differ in terms of dynamics of core affective regions, we extracted the condition-wise time courses of regions in the NACC/VS and amygdala associated with trial-wise reported positive and negative affect (see Figure 6.4). First, we established the efficacy of Reappraisal and Compassion in modulating activity in these regions by submitting the extracted time series for these conditions and their respective Watch-Negative baselines to LMM analysis. Analyses were shifted to allow for hemodynamic lag by excluding the three first time points, corresponding to the 0-4 seconds following Instruction onset. The remaining time points were averaged for the Preparation (5 time points, 6-16 seconds following Instruction onset) and Implementation (6 time points, 16-28 seconds following Instruction onset) phases. Separate models were fitted for each technique and region, consisting of fixed factors for Condition (2 levels; Reappraisal/Compassion, Watch-Negative) and Period (2 levels; Preparation, Implementation) with subject-level random intercepts. For the Compassion condition, these analyses revealed a main effect of Condition in both VS/NAC ($F(1, 312) = 10.52$, $p < .001$) and amygdala ($F(1, 312) = 4.50$, $p < .05$) ROIs. Follow-up *T*-tests showed that this effect consisted of higher signal in the VS/NAC ($t(312) = 3.32$, $p =< .001$) and lower signal $t(342) = -2.17$, $p =< .05$) in the amygdala ROIs relative to the Watch-Negative condition. For the Reappraisal condition, only a main effect of Condition in the amygdala ($F(1, 312) = 28.83$, $p < .001$) ROI was observed. Follow-up *T*-tests showing these effects consisted of lower signal overall in the amygdala ($t(312) = -5.4$, $p =< .001$) in Reappraisal relative to the Watch-Negative condition. Thus, while both Compassion and Reappraisal was shown to decrease activation of amygdala relative to Watch-Negative, only Compassion was shown to increase activation of VS/NAC.

To enable direct comparison of modulatory effects, we subtracted their respective Watch-Negative baselines from the Compassion and Reappraisal time-courses and submitted

these to LMM analysis separately for each region using the same model as above. Figures 4a and 4b show time series of this subtraction for the NACC/VS and amygdala ROIs, respectively. For the VS/NAC, this revealed a main effect of Condition, ($F(1, 312) = 11.16$, $p < .001$) as well as a Condition*Period interaction effect ($F(1, 312) = 3.96$, $p < .05$). For the amygdala, only a main effect of Condition was observed ($F(1, 312) = 5.84$, $p < .05$). Follow-up *T*-tests revealed that these effects consisted of higher signal in the VS/NAC for Compassion relative to Reappraisal specifically during the Implementation period (Preparation: $t(134) = 3.79$, $p =< .0001$; Implementation: $t(164) = 0.97$, $p => .1$), and overall lower signal in the amygdala for Reappraisal than Compassion ($t(312) = -2.42$, $p =< .05$).

In summary, these results show that Compassion and Reappraisal are differentiable in terms of their modulatory effects on the neural correlates of subjective positive (VS/NAC) and negative (amygdala) affect.

6.4. Discussion

In this study we sought to identify the subjective and neuronal signatures of Compassion-meditation when employed to regulate emotional reactions to depictions of others' suffering and compare the mechanisms and effects of Compassion to those of the established emotion regulation strategy of Reappraisal. Behaviourally, we expected that the main regulatory effect of Compassion should be increased positive affect, reflecting the direct generation of positive affect hypothesised to underlie its regulatory effects. This in contradistinction to Reappraisal, which we expected to have a more pronounced effect on negative affect on account of its focus on altering the affective meaning of the stimuli. Neurally, we expected to find evidence of increased activation of core positive affect regions during Compassion. Further, consistent with our hypothesis that the regulatory mechanism of Compassion is the endogenous generation of positive affect we expected engagement of this network independent of stimulus presentation. This effect should also be distinguishable from

the modulatory mechanisms of Reappraisal, as Compassion to a lesser degree should down-regulate core negative affective regions, and not engage cognitive control regions shown to be important in regulating negative affect.

On the subjective level, we found that Compassion was associated with increased positive affect relative to Watch-Neutral, Watch-Negative and Reappraisal. Thus, Compassion was more effective at increasing positive affect than Reappraisal. Importantly, unlike Reappraisal, Compassion occasioned an increase of positive affect relative to baseline levels of positivity (Watch-Neutral). Mirroring these behavioural effects, our fMRI analyses revealed that Compassion, relative to both Watch-Negative and Reappraisal, was associated with increased activation in subcortical structures associated with positive affect such as ventral striatum/NACC and globus pallidus as well as midline cortical structures such as vmPFC, rgACC and pgACC (Figures 6.2 and 6.3). These regions have all previously been associated with positive affect, motivation and reward (Kringelbach & Berridge, 2009; Rangel & Hare, 2010; Schultz, 2010), and affiliation (Strathearn et al., 2009; Vrtička et al., 2008). Overall, these results are consistent with previous findings (Klimecki et al., 2013; 2014), demonstrating that Compassion is an effective regulation strategy and that a key mechanism underlying these regulatory effects is the engagement of neural systems associated with positive affect.

This interpretation is strengthened by the direct comparison between Reappraisal and Compassion: In line with previous work (Buhle et al., 2014), Reappraisal was characterised by activation of a fronto-parietal network, including ventral and dorsal PFC, dACC, and TPJ/SMG, regions known to be associated with cognitive control, attention regulation and working memory (Ochsner et al., 2012). Contrary to this, Compassion was shown to rely primarily on activation of the aforementioned medial and subcortical systems, with the strongest differentiation observable in mOFC and VS/NACC (Figure 6.3). Additionally, Compassion was shown to specifically increase activation in bilateral mid-insula, a region

previously associated with specifically affiliative types of positive affect, such as maternal love ("The neural correlates of maternal and romantic love.," 2004b). These different activation patterns appear to reflect the conceptual difference between Reappraisal and Compassion: Reappraisal involves the employment of cognitive control to modulate affective influences, whereas Compassion involves the generation of positive affect, without altering the processing of negative stimuli. In line with this, we found differential effects of Compassion and Reappraisal in regions specifically tracking experienced positive (VS/NACC) and negative (amygdala) affect: Compassion showed evidence for increased activation of VS/NACC compared to both Reappraisal and passive-viewing. Critically, this modulation was apparent prior stimulus presentation consistent with Compassion involving the endogenous generation of positive affect in a stimulus-independent fashion. Furthermore, Compassion was associated with overall higher amygdala activity than Reappraisal, suggesting that Compassion to a lesser degree modulated the primary affective processing of the negative stimulus material. Overall, these findings are in line with a view that the underlying mechanism of Compassion-based emotion regulation is the volitional and stimulus-independent engagement of neural systems supporting the endogenous generation of positive affect.

Our findings are largely consistent with earlier work on short-term effects of Compassion training by Klimecki and colleagues (Klimecki et al., 2013; 2014). However, unlike these studies, we found that Compassion had a small but significant regulatory effect on subjectively experienced negative affect. One possible explanation for this difference is the level of experience between the expert practitioners in our study and those subjects tested in the Klimecki studies, which had only one week of practice in Compassion. It is possible that extensive experience in Compassion affords a concomitant decrease in negative affect. Another, complementary, possibility is that these differences stem from the current design explicitly instructing participants to generate Compassion prior to exposure to the stimuli. It

could be that proactive generation of Compassion is particularly effective at dampening negative affect, presumably by providing a buffer of positive affect (Garland et al., 2010). The current design does not allow a direct test of this hypothesis, but future research could address this by comparing differences in Compassion efficacy as a function of whether it is generated proactively or reactively, as this has been shown to be an important determinant of the efficacy of other regulation strategies (Sheppes & Gross, 2011).

The capacity to be employed independently of specific stimuli and an underlying mechanism not involving attenuation or alteration of negative emotional responses potentially affords Compassion some unique advantages as an emotion regulation strategy. In the context of others' suffering, Compassion affords maintenance of an empathic connection while counteracting empathic distress (Singer & Klimecki, 2014). Thus, employment of Compassion as a regulation strategy could avoid of the decreased sensitivity and empathy reported when employing cognitive emotion regulation strategies to cope with others' suffering (Cameron & Payne, 2011). Furthermore, Compassion has been shown to be associated with an increase in prosocial motivation and helping (Leiberg et al., 2011; Weng et al., 2013) suggesting an additional beneficial effect if used as coping strategy in helping professions as it would not only increase resilience but also increase their willingness to assist individuals in need.

6.4.1. Conclusion

In this study we showed that Compassion can be employed as an effective emotion regulation strategy outside of its traditional meditative context, and that both experientially and neurally it is associated with the endogenous generation of positive affect. While the generalizability of our findings, especially with regards to the relative efficacy of Reappraisal and Compassion, is limited by the fact that our subjects had extensive experience in generating Compassion, recent work demonstrates that it is possible to elicit similar neural

and behavioural effects to what we observe here with short-term training (Klimecki et al., 2014; Leiberg et al., 2011; Weng et al., 2013). This suggests that Compassion might be an effective means to promote resilience to others' suffering also in the general population while at the same time promoting emotional connectedness and pro-sociality.

6.4.2. Acknowledgements

The authors would like to thank Tor Wager and Luka Ruzic for their help with the subcortical rendering scripts. We would furthermore like to thank Matthieu Ricard, who helped recruiting this very special population of long-term meditation practitioners, and the support staff of the Social Neuroscience Department, particularly Dr. Sandra Zurborg, for her help with the study logistics and organization, Henrik Grunert for his technical assistance, Elisabeth Murzik for her help with data archiving, and Sylvie Neubert and Nicole Pampus for their help with scanning. Finally, we thank all the long-term practitioners who were willing to fly to and spend a considerable time in our laboratory in Leipzig to serve as subjects for this large-scale project. This work was supported by grants to Tania Singer from the European Research Council under the European Community's Seventh Framework Program (FP7/2007-2013)/ ERC Grant agreement n◦ 205557 [EMPATHICBRAIN]

Chapter 7: Choosing how to feel: Endogenous emotion generation abilities mediate the relationship between trait affectivity and emotion management style[7]

Haakon G. Engen, Philipp Kanske, & Tania Singer

7.1. Abstract

Interventional research suggests that training the ability to self-generate emotion can have substantial mental health benefits, yet little is known about this ability in the normal population. We investigated individual differences in implementation of emotion generation and the association of this ability with trait affect and emotion management styles. In a novel task, participants (N = 288) generated positive, negative and neutral emotion using four primary modalities of emotional states (Semantic Analysis, Episodic Imagery, Auditory Imagery, Bodily Interoception) using self-chosen combinations optimising generation efficacy. Subjective report and psychophysiological indices of emotional arousal indicated participants successfully generated both positive and negative emotional states. While Episodic Imagery was the most preferred and efficacious modality, usage of all modalities except Auditory Imagery was effective, suggesting participants adopted idiosyncratically optimal means of implementation. Taking task performance as a measure of individual differences in emotion generation ability, we next investigated the relationship between emotion generation, trait affect, and emotion management styles. Using an extensive battery of questionnaires, we established composite measures of individual differences in trait affect and adaptive (Emotion-focused, Instrumental) and maladaptive emotion management, as defined by their correlations with trait affect. Both overall emotion generation ability and relative efficacy (positive vs. negative) emotion generation were positively correlated with

[7] The current chapter is presently under review, and the final, published version, might show discrepancies from the present version. Presented here is the submitted manuscript of 17.04.2016.

trait affect and Instrumental emotion management. Relative ability additionally correlated with Emotion-focused emotion management. However, only general generation abilities were found to causally mediate the relationship between emotion management and trait affect, suggesting that emotion generation abilities enable flexible adaptation to emotional stressors in a problem-focused manner.

7.2. Introduction

Emotions are often thought of as reactions to events in the world. However, emotional states are often caused by our own thoughts and memories without clear reference to external events. Recent evidence suggests that this ability to generate emotional states based on endogenous information can be used in strategic manner to facilitate self-regulation. For instance, it has recently been shown that different types of positive imagery can be used strategically to counteract both negative emotional reactions to external affairs and internally maintained mood states in both normal and clinical populations (H. G. Engen & Singer, 2015; 2016; Holmes et al., 2006; 2009; Poerio, Totterdell, Emerson, & Miles, 2015a). Moreover, it appears that an important aspect of resilience is the capacity to volitionally generate positive emotional states (Fredrickson, 2013; Tugade & Fredrickson, 2004). As such, the ability to self-generate emotional states appears to serve an important self-regulatory function with tangible mental health benefits.

Despite this very little is known about how EnGE abilities vary in the population. For one, as extant research on emotion generation has largely been interventional, it has focused on teaching individuals to generate emotions using episodic mental imagery (Holmes et al., 2009; Poerio, Totterdell, Emerson, & Miles, 2015a), on account of the attested efficacy of this modality in the generation of emotion (Holmes & Mathews, 2005). However, evidence suggests that emotional reactions can be generated in a volitional fashion using numerous different information modalities, including semantic analysis (Bergman & Craske, 2000;

Holmes et al., 2006), auditory imagery (Williamson et al., 2012) and bodily signals (Philippot et al., 2002). Indeed, given the multifaceted nature of emotional experiences (Barrett et al., 2007a), with clear semantic, imaginal and bodily concomitants it appears likely that naturalistic emotion generation involves combining multiple information modalities to create an embodied emotional states (Niedenthal et al., 2009). Thus, for instance, a given instance of a self-generated emotion is likely to include both thoughts and bodily sensations that might occur in combination with imagery, but is not necessarily tied to imagery as in e.g. rumination (Nolen-Hoeksema et al., 2008). One important hitherto unanswered question is therefore to what degree different methods of are utilised by the normal population, and, relatedly, whether particular generation methods or combinations thereof are particularly efficacious means of emotion generation. In addition to having important implications for future interventional research, this could potentially offer insight into how different approaches to generating emotion might be associated with adaptive or maladaptive affective styles.

The interventional focus of extant research has also left open questions regarding the mechanism supporting potential beneficial effects of emotion generation abilities. Naturally, given the focus of these studies on counteracting negative affect, participants were not trained in generating negative emotion, but rather were taught techniques to increase positive emotion. While evidence strongly supports the efficacy of such interventions (Fredrickson, 2013; Fredrickson & Branigan, 2005; Holmes et al., 2009; 2016) several questions are unanswered important about the mechanism by which emotion generation improves mental health outcomes. Most frequently assumed, one possibility is that emotion generation can allow individuals to experience positive emotions even in the absence of external appetitive stimuli, in effect acting as a counter to negative and stressful external stimuli (H. G. Engen & Singer, 2015; Singer & Klimecki, 2014). If this *buffering hypothesis* is correct, one would expect that specifically positive emotion generation would be associated with beneficial affect traits, like use of beneficial coping and reduced negative affect. Conversely, the capacity to

generate negative emotional states might predispose one to experience more negative affect, and maladaptive emotion management. Another possibility is that emotion generation abilities can enhance the capacity to deal with emotions through increased emotional complexity and differentiation between different emotional states. Thus, by allowing one to accurately simulate past and future emotional events (Baumgartner et al., 2008; S. E. Taylor & Schneider, 1989), emotion generation skills can be utilised in the service of instrumental and problem-focused emotion management. According to such a *flexibility hypothesis* overall emotion generation ability should be associated with beneficial emotional traits, irrespective of valence, and might particularly be helpful in enabling strategic and instrumental efforts to manage ones emotional states (Kashdan, Barrett, & McKnight, 2015).

The current study had two goals: First, we sought to describe how endogenous emotion generation is implemented in the normal population, with a special emphasis on investigating the relationship between modality usage and individual differences in emotion generation efficacy. Second, we sought to investigate the relationship between generation efficacy and aspects of emotional mental health, particularly trait affectivity and means of emotion management, operationalised here as the trait tendency to use different coping or emotion regulation techniques. This allowed us to directly test the *buffering* and *flexibility* hypotheses proposed above. We did this using a newly developed task aimed at gauging individual differences in emotion generation abilities. After an automatised training procedure, participants (N = 288) self-generated positive and negative emotional states using one or more of four modalities (Episodic Imagery, Semantic Analysis, Auditory Imagery, and Bodily Interoception) in whichever combination and relative degree they believed would facilitate successful emotion generation. Concurrently, participants' skin conductance levels were recorded. Thus, this design allowed an objectively validated assay of individual differences in the ability to self-generate emotional states. Thus, by investigating the relationship between generation efficacy and modality usage, we could establish the relative

efficacies of specific modalities or combinations of modalities. Further, participants completed an extensive battery of questionnaires gauging their trait affectivity and coping styles, enabling the investigation of how emotion generation was associated with affectivity and coping styles, allowing us to directly test the *buffering* and *flexibility* hypotheses suggested above. Specifically, if the *buffering hypothesis* was correct we expected that the differential capacity for generating positive to negative emotion would predict adaptive coping and trait positivity. If the *flexibility hypothesis* was correct we would conversely expect to find no valence effects, but rather that emotion generation on average would predict beneficial emotional traits.

7.3. Methods

7.3.1. Participants

Data was acquired in the context of an fMRI study, the neural data of which will be reported elsewhere. Participants were recruited in the context of the large-scale longitudinal *ReSource Project* (for details see Singer et al., 2016), with baseline data being used for the present study. Eligibility was determined using a screening procedure including SCID-I and II interviews performed by trained clinical psychologists, ensuring no ongoing mental health issues, and no life-time occurrence of psychotic or bipolar disorders, substance dependence, or any Axis-II disorders. Out of a recruited sample of 332, 305 participants completed the current paradigm. 5 participants were excluded on account of missing data and technical difficulties. 4 participants reported difficulties (e.g. nausea or sleepiness) during the experiment, and a further 3 participants were removed due to aberrant behaviour suggestive of task non-compliance, such as having low or no variance in behavioural ratings between conditions and trials. Finally, 5 participants had incomplete questionnaire data, leaving a final sample of 288 (168 female, mean age = 40.42, range: 20-55, SD = 9.29). The study was approved by the Research Ethics Committees of the University of Leipzig (number 376/12-ff)

and the Humboldt University in Berlin (numbers 2013-02, 2013-29, and 2014-10) and was carried out in compliance with the Declaration of Helsinki. All participants gave written informed consent and were debriefed and paid after the study was completed.

7.3.2. Training procedure

Before the experiment, participants underwent a supervised automated training session with two distinct portions. First, participants underwent a multimodal affect induction procedure that provided examples of high and low arousal positive and negative emotion. These inductions insured that participants had homogenous representations of the target emotional states. Second, participants were introduced to four different means of emotion generation using one of four information processing modalities (Episodic Imagery, Semantic Analysis, Auditory Imagery, and Bodily Sensations). In addition to corresponding to the induction procedure, these modalities were elected as they have been shown in previous literature to be effective means of self-inducing emotion (Mayberg et al., 1999; Philippot et al., 2002; Vrana et al., 1986; Wilson-Mendenhall et al., 2013).

Participants freely chose a modality or combination of modalities to generate emotions, and were also given the option to use self-formulated generation methods. Participants then trained generating positive and negative emotional states and were instructed to utilise the modality or combination of modalities that they experienced to best allow them to generate emotions in the main experiment. Further details of the procedure are reported in the *Appendix:* Supplemental Methods A1.3.1.

7.3.3. Experimental procedure

Each trial was split into a Generation phase, where the target emotion was elicited, and a Modulation phase, where the elicited emotion was either Maintained or Regulated. Here we focus on the Maintain conditions while the Regulation component will be reported elsewhere.

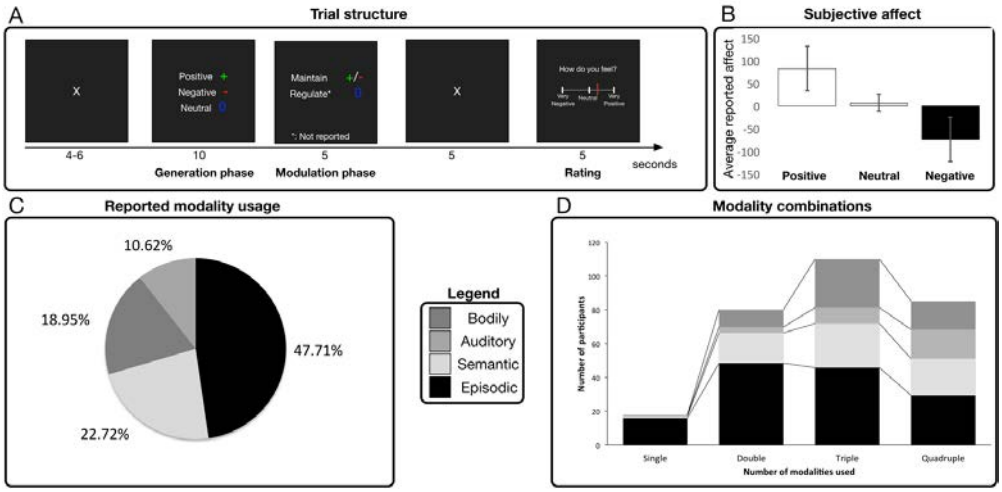


Figure 7.1: Trail structure, behavioural validation and modality usage. A) Schematic representation of the experiment. After an automatised procedure where participants trained and selected how to generate emotional states, participants were asked to generate positive, negative and neutral states. Each trial started with a fixation cross (4-6 s.). Participants were then presented with cues indicating what emotional state to generate (green plus-sign = generate positive, red minus-sign = generate negative, blue zero = generate neutral) for 10 seconds. For the emotional conditions, the cue then either changed to a blue zero indicating that participants should down-regulate their generated states or remained the same as in the generation phase indicating that they should maintain the emotional state for another 5 seconds. Only this maintain-condition was the topic of the current study. This was followed by a 5 second fixation cue, and a 5 second bipolar subjective affect rating. **B)** Effect of generation instructions on subjectively reported affect. **C)** Modality usage reported by participants to generate emotions. **D)** Reported combinations of modalities as a function of number of modalities participants reported using.

In the Neutral condition, participants were instructed to actively maintain a neutral state of mind. The order of conditions was pseudo-randomised with no more than two condition repetitions.

A trial (Figure 7.1A) started with a 4-6 second white fixation cross followed by a 10 second Inception phase, in which subjects were shown a cue indicating which emotional state to generate (Red minus = Negative, Green plus = Positive, Blue zero = Neutral). In the emotional conditions a 5 second Modulation phase followed in which the instruction symbol remained the same (Maintain condition) or changed to a blue 0 indicating that subjects should down-regulate the emotional state they were in (Regulate condition). After a 5 second fixation cross, a 5 second Visual Analogue Scale (VAS) ranging from “Extremely negative” via

"Neutral" to "Extremely positive" (range: +/- 250 from the neutral point (0)) was presented. Initial cursor position was jittered randomly (range: +/- 100 points relative to the Neutral point). Responses were given using the right hand index and middle finger. Participants were instructed to report their affective state as it was at the moment of report. Stimuli were back-projected in the MRI scanner using a mirror setup. Eyesight was corrected using goggles where appropriate.

After the experiment, participants were requested to report what modalities they used and to which degree they used them using 9-point Likert scales anchored with "Not used at all" and "Used a great deal"). Participants were also given the opportunity to specify that they used "Other" (i.e. self-defined) modalities, by describing them in a text box and giving a Likert-score for how much they used them.

7.3.4. Assessing trait affectivity and emotion management styles

In the context of the *ReSource* study, participants completed an extensive package of questionnaires measuring a range of socio-affective and cognitive traits (Singer et al., 2016). Important for present purposes, this package included multiple measures of the tendency to experience positive and negative emotions, as well as measures of the tendency to use different emotion regulation and coping strategies, and engage in effortful self-regulation. In the following we refer to this as *trait affectivity* and *emotion management style,* respectively. In order to have stable and comprehensive estimates of trait affectivity and emotion management styles, we submitted the relevant scales to separate principal component analyses (PCAs; varimax rotation; see *Appendix*: Supplemental methods A1.3.2-3, Figures A2.3.1-2 and Table A3.3.1-2 for more detail on scale selection, analyses and interpretation of PCA components). Individual loadings on the resultant components were used as our individual difference measures for further analysis.

For trait affectivity, we included all scales including questions regarding self-experienced positive and negative affect. Thus, we included the Positive and Negative Affect

Schedule (Krohne, Egloff, & Kohlmann, 1996), the Types of Positive Affect Scale (P. Gilbert et al., 2008), the Beck's Depression Inventory II (Hautzinger, Keller, Kühner, & Beck, 2009), the trait form of the State-Trait Anxiety Inventory (Laux, Glanzmann, Schaffner, & Spielberger, 1981), the negative and positive affect subscales of the NEO Five Factor Inventory (Borkenau & Ostendorf, 2008) and the negative affect and extraversion subscales of the Adult Temperament Questionnaire (Wiltink, Vogelsang, & Beutel, 2006). For emotion management, we included all scales measuring the tendencies to use different forms of coping or emotion regulation strategies, and the tendency to exercise self-control. This included the Cognitive Emotion Regulation Questionnaire (Loch, Hiller, & Witthöft, 2011); Brief COPE Inventory (Knoll, Rieckmann, & Schwarzer, 2005), Emotion Regulation of Self and Others (Niven, Totterdell, Stride, & Holman, 2011), and the effortful control subscale of the Adult Temperament Questionnaire. For trait affectivity, our analyses revealed a single component with scales measuring positive trait affect loading negatively and scales measuring negative trait affect loading positively (*Appendix:* Figure A2.3.1A, and Table A3.3.1). For ease of interpretation loadings were inverted for subsequent analyses, such that positive loadings reflect more trait positivity and vice versa. For emotion management styles, our analyses revealed three components (*Appendix:* Figure A2.3.1B). Component 1 accounted for 17.9% variance, with items measuring primarily the tendency to cognitively transform negative emotional situations to improve mood. Component 2 accounted

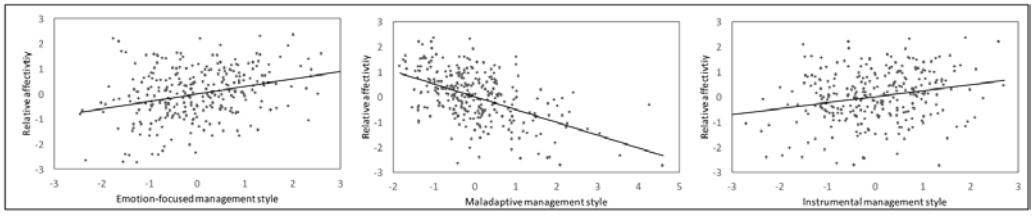


Figure 7.2: Correlation of trait affect and emotion management style. Scatterplots of the relationships between the composite scores calculated for average and relative trait-specific generation ability and the PCA components measuring trait affectivity (see *Appendix*: Table A3.3.1) and emotion management styles (see *Appendix:* Table A3.3.2). Relationship between the three emotion management styles and trait affectivity. Lines indicate linear regression lines of best fit and are provided for reference only. See Table 7.2 for corresponding correlation analyses.

for 12.11% variance, with loadings on items measuring the tendency to blame oneself, ruminate, catastrophise and other maladaptive tendencies resulting in mood worsening. Component 3 accounted for 8.61% variance with loadings on items measuring the tendency to take a problem-focused and instrumental approach to emotional stressors by seeking support, planning, and actively refocus efforts to improve mood. We labelled these components as *Emotion-focused*, *Maladaptive* and *Instrumental* means of emotion management. Importantly, these components showed an appropriate correlation structure (see Table 7.2 and Figure 7.2), such that individual tendencies to use *Emotion-focused* and *Instrumental* management styles positively correlated with trait affect. More specifically, these emotion management styles were associated with more positive and less negative affect. Conversely, *Maladaptive* usage negatively correlated with trait affect being was associated with less positive and more negative affect.

7.3.5. Psychophysiological data acquisition and pre-processing

Skin conductance responses were recorded during scanning using a BrainVision setup (BrainAmp ExG MR; Brain Products, Munich, Germany). Ag/AgCl electrodes with applied isotonic electrode paste as a conductance medium were attached to the index and middle finger of the left hand for each subject. The acquired data was filtered using a Butterworth 0.15 Hz low-pass filter of order 3 using custom MATLAB scripts. Time- courses were then visually inspected, excluding data-sets where overall data quality was poor (e.g. showing evidence of scanner artefacts after filtering or lack of variance for the entire time series). Of the 288 datasets included in this study, 215 also passed these criteria. The remaining data was analysed using the Ledalab toolbox (www.ledalab.de). The data was downsampled to 250 Hz, and continuous decomposition analysis (Benedek & Kaernbach, 2010b) was performed to create estimates of condition specific phasic (due to e.g. changing stimuli) and tonic (due to

e.g. changing emotional arousal) skin conductance responses. Here, tonic skin conductance levels (SCLs) were used as a measure of emotional arousal as this is the most frequently reported skin-conductance metric of emotional arousal (Kreibig, 2010).

7.4. Results

7.4.1. Validation and operationalization of emotion generation efficacy

Post-trial subjective ratings were analysed using paired *t* tests of the average reported affect in each condition (see Figure 7.1B). Relative to the neutral (mean = 7.49, SD = 19.11) baseline condition, increased affect was observed for both positive (mean = 82.86, SD = 49.00; paired $t(287) = 27.78$, $p < 0.001$, CI (95%) = 69.83/80.90) and negative (mean = -72.95, SD = 49.20; paired $t(287) = -27.74$, $p < 0.001$, CI (95%) = -87.15/-74.73) Maintain conditions.

We next sought to establish whether these generated emotions resulted in changes in psychophysiological measures of emotional arousal. We additionally investigated whether condition-specific correlation between ratings and SCL existed, as this would show that behavioural ratings can be taken as proxy for emotional arousal. To this end, trial-level linear mixed modelling of the Generation period was performed, predicting log-transformed estimates of SCL. The model included subject-level random intercepts, condition (Positive, Negative, Neutral) as a categorical fixed effect and trial-wise subjective ratings of affect as a continuous fixed effect. To control for potential learning/fatigue effects, trial number was entered as a nuisance covariate. Our analysis revealed a main effect of Condition($F(2,6224.38) = 3.77$, $p < .05$, and an interaction between Condition and Ratings ($F(2, 6227.00) = 14.07$, $p < .001$). Breaking this interaction down by condition, we found that Ratings predicted SCL in the Positive ($t(1937.99) = 4.63$, $p < .001$, CI(95) = -.020/.049) and Negative ($t(1938.06) = -3.16$, $p < .01$, CI(95) = -.037/-.009) conditions, but not the Neutral condition ($t(1937.67) = 1.43$, CI(95) = -.004/.025). Considering the bipolar rating scale used,

this shows that SCL increased with affect ratings for both positive and negative emotion, demonstrating that participants were capable of generating both subjective and physiological concomitants of positive and negative emotional states.

Having validated ratings as a proxy for emotional arousal, we generated composite efficacy scores for the remaining analyses based on the average ratings a participants had within each condition (Generate Positive, Generate Negative, Generate Neutral; see also Figure 7.1B). Average generation efficacy was calculated as ((Generate Positive + Generate Negative)/2 – Generate Neutral). Thus, a high loading on this score corresponds to reporting higher affect in both positive and negative conditions. Conversely, to investigate the effects of valence-specific generation efficacy we calculated a relative efficacy score as (Generate Positive - Generate Negative). Accordingly, a high positive score on this variable would correspond to high efficacy in generating positive relative to negative emotion and a high negative score high efficacy in generating negative relative to positive emotion.

7.4.2. Characterising the preference structure of emotion generation modalities

Next, we sought to establish whether our participants implemented emotion generation in an optimal manner by investigating their reported preferential use of modalities. 14 participants reported using "other" modalities. On closer inspection all except one of the participants' descriptions closely matched our primary modalities and so were included in the primary scores by averaging (see *Appendix:* Table A3.3.3). The final participant reported using several of the primary modalities to a large degree and so their "Other" score was ignored. To control for individual differences in rating tendency we calculated the proportion of reported usage of each modality relative to the sum total of usage reports. Reported modality usage is shown in Figure 7.1C, showing that Episodic Imagery was the most used (47.71%), followed by Semantic Imagery (22.72%), Bodily Interoception (18.95%) and Auditory Imagery (10.62%).

Next, we sought to establish the degree to which participants reported combining these modalities, and how they were combined. Usage of a given strategy was operationalised as giving a response other than "Did not use at all". Results (Table 7.1 and Figure 7.1D) showed that the majority of participants reported using two or more modalities, with the largest proportion combining three modalities. The dominant combination involved the using Episodic Imagery together with other modalities, suggesting that, while our participants tended to a multimodal implementation, this primarily involves using other modalities to supplement Episodic Imagery.

				Proportional composition (%)			
# of modalities	Combination	N	%	Episodic	Semantic	Bodily	Auditory
One	Episodic	16	88.89	100	--	--	--
	Semantic	1	5.56	--	100	--	--
	Bodily	0	0.00	--	--	N/A	--
	Auditory	1	5.56	--	--	--	100
	Total N	18	6.14				
Two	Episodic & Semantic	39	48.75	59.48	40.52	--	--
	Episodic & Bodily	28	35.00	66.54	--	33.46	--
	Episodic & Auditory	10	12.50	65.91	--	--	34.09
	Semantic & Bodily	3	3.75	--	69.48	30.52	--
	Semantic & Auditory	0	0.00	--	N/A	--	N/A
	Bodily & Auditory	0	0.00	--	--	N/A	N/A
	Total N	80	2730%				
Three	Episodic & Semantic & Bodily	68	61.82	38.42	32.12	29.46	--
	Episodic & Semantic & Auditory	15	13.64	48.49	26.01	--	25.50
	Episodic & Bodily & Auditory	26	23.64	48.09	--	21.32	30.60
	Semantic & Bodily & Auditory	1	0.91	--	18.00	9.00	73.00
	Total N	110	3754.27%				
Four	Episodic & Semantic & Bodily & Auditory	85	100.00	34.68	25.61	19.43	20.28
	Total N	85	2901.02%				

Table 7.1: Combinations of modalities. Usage and proportional composition of modality combinations as a function of number of modalities reported used. Number of participants reporting using each combination of modalities reported in the *N* column, whereas the self-reported degree to which each modality was used on average is reported in the Proportional Composition columns.

7.4.3. Efficacy of emotion generation modalities

Next we investigated the efficacy of these modalities by specifying two multiple regression models predicting average and relative generation efficacy (controlling for the respective other), including scaled self-reported modality usage scores as continuous, interacting predictors. Age and gender were included as nuisance covariates.

Average generation efficacy was significantly predicted by reported usage of Episodic Imagery (b = 10.79, $t(269) = 3.87$, $p < .001$, $F(1,269) = 15.01$, Partial $Eta^2 = .053$), Semantic Analysis (b = 7.45, $t(269) = 2.36$, $p =< .05$, $F(1, 269) = 5.59$, Partial $Eta^2 = .020$) and Bodily Interoception (b = 7.81, $t(269) = 2.49$, $p =< .05$, $F(1, 269) = 7.19$, Partial $Eta^2 = .023$), with no significant interactions (see Figure 7.3A). As the effect of Episodic Imagery was considerably larger than Semantic Analysis and Bodily Interoception, we used the *multcomp* R package to statistically contrast the parameters. Results indicated no significant difference between any of the combinations of modalities. When repeated for relative efficacy, no significant effects were observed (see Figure 7.3B). Thus, usage of all modalities except for Auditory Imagery predicted more effective emotion generation in general, but did not show evidence of being beneficial or detrimental for the generation of specific valences.

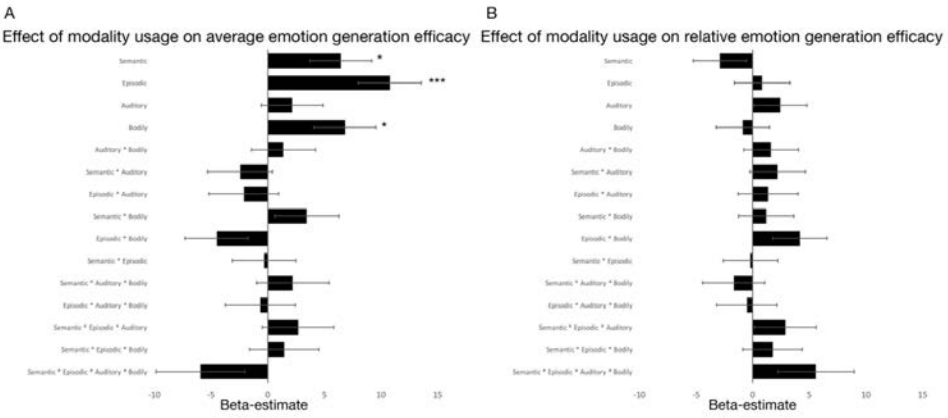


Figure 7.3: Relationship between modalities and generation efficacy. Plots of beta-estimates from multiple regression models investigating the effect of modality usage on emotion generation efficacy. **A)** Relationship between modality usage and average emotion generation efficacy. **B)** Relationship between modality usage and the relative ability to generate positive and negative emotion. Error bars denote +/- SE. * =< .05, ** =< .01, *** =< .001

7.4.4. Emotion generation efficacy, trait affect and emotion management styles

We next sought to investigate the relationship between individual differences in emotion generation efficacy and trait affect and emotion management styles. Using the R package *ppcor* (S. Kim, 2015), we calculated non-parametric (Spearman) partial correlations

between average and relative generation efficacy and loadings on the components described above, controlling for age and gender. Scatterplots of these comparisons are reported in *Appendix:* Figure A2.3.2, while Bonferroni-corrected results are reported in Table 7.2. Both absolute generation ability (*Appendix:* A2.3.2A), and relative ability to generate positive versus negative emotion (*Appendix:* A2.3.2C) was positively correlated with trait affect. Further, average generation efficacy was specifically correlated with usage of Instrumental emotion management (*Appendix:* A2.3.2B), whereas relative generation ability correlated with use of Emotion-focused and Instrumental emotion management (*Appendix:* A2.3.2D). Thus, while both average generation efficacy and the relative capacity to generate positive emotion were associated with trait affectivity and usage of Instrumental emotion management, only relative emotion generation ability was correlated with use of Emotion-focused emotion management styles.

	Average Generation Ability	**Relative Generation Ability**	**Trait Affect**
Relative Generation Ability	-0.03	1	
Trait affect	**0.18*** **	**0.12** **	1
Emotion-focused	0.063	**0.14*** **	**0.261*** **
Maladaptive	-0.02	0.01	**-0.527*** **
Instrumental	**0.128** **	**0.12** **	**0.219*** **

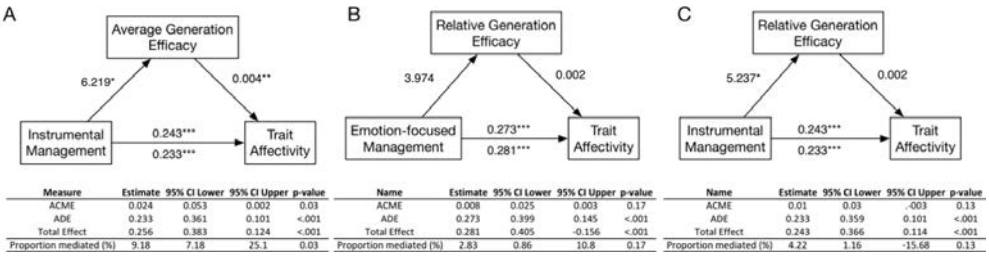
Table 7.2: Relationship between emotion generation and affective style. Bonferroni corrected non-parametric partial Spearman correlations (controlling for age and gender) between the composite generation efficacy scores for average and relative generation ability, and the single trait affect component and the three emotion management components revealed by our PCA analyses. See also *Appendix:* Figure A2.3.2 for scatterplots of these relationships. * = $p < .05$, ** = $p < .01$, *** = $p < .001$

7.4.5. Mediation analyses

Our final analyses sought to establish whether emotion generation abilities mediated the relationship between emotion management and trait affect using the *mediation* R toolbox (Tingley, Yamamoto, Hirose, Keele, & Imai, 2014) to perform nonparametric causal mediation analyses with bootstrapped confidence intervals (10000 samples). If such mediation

was in evidence, it would suggest that emotion generation skills are part of the mechanism by which (adaptive) emotion management influences trait affect.

Models were specified based on our correlation analyses (Table 7.2), investigating mediation of the relationship between Instrumental management style and trait affectivity both average and relative generation efficacy, and relationship between Emotion-focused management style and trait affectivity for relative generation efficacy only. In each model the other efficacy composite, age and gender were entered as covariates. This revealed that average generation efficacy partially mediated the relationship between instrumental emotion management and trait affect (Figure 7.4A), while no mediation was observed for valence-specific generation (Figures 7.4B and 7.4C). Thus, our results suggest that average, but not valence-specific, generation ability is part of the mechanism by which Instrumental emotion management improves trait affect.



Measure	Estimate	95% CI Lower	95% CI Upper	p-value
ACME	0.024	0.053	0.002	0.03
ADE	0.233	0.361	0.101	<.001
Total Effect	0.256	0.383	0.124	<.001
Proportion mediated (%)	9.18	7.18	25.1	0.03

Name	Estimate	95% CI Lower	95% CI Upper	p-value
ACME	0.008	0.025	0.003	0.17
ADE	0.273	0.399	0.145	<.001
Total Effect	0.281	0.405	-0.156	<.001
Proportion mediated (%)	2.83	0.86	10.8	0.17

Name	Estimate	95% CI Lower	95% CI Upper	p-value
ACME	0.01	0.03	-.003	0.13
ADE	0.233	0.359	0.101	<.001
Total Effect	0.243	0.366	0.114	<.001
Proportion mediated (%)	4.22	1.16	-15.68	0.13

Figure 7.4: Depiction of the causal mediation models tested. A) Mediation model showing that average generation efficacy partially mediates the relationship between instrumental emotion management styles and trait affectivity (9.18% of effect mediated). **B)** Mediation model showing that relative emotion generation efficacy does not mediate the relationship between emotion-focused management styles and trait affectivity. **C)** Mediation model showing that relative emotion generation efficacy does not mediate the relationship between instrumental emotion management styles and trait affectivity.

7.5. Discussion

The objective of the current study was twofold: First, we sought to describe how emotion generation is implemented in a naturalistic fashion in the normal population, with special emphasis on establishing the relative efficacy of different information modalities. Second, we wanted to test two competing hypotheses about emotion generation efficacy: the

buffering hypothesis proposing that enhanced ability to generate positive emotion facilitates increased trait positivity and emotion management skills and the *flexibility hypothesis* proposing that emotion generation in general enhances emotion management and thereby enhances trait positivity. We tested these hypotheses using a newly developed, minimally constrained paradigm where participants self-generated positive and negative emotion using one or more of four information modalities (Episodic Imagery, Semantic Analysis, Auditory Imagery, Bodily Interoception), and a comprehensive questionnaire-based assessment of trait affectivity and emotion management styles.

Overall, we found that participants were able to successfully generate both positive and negative emotional states, as indicated by both subjective report and physiological measures of emotional arousal. We found that participants freely used all four of the modalities we suggested in combination to achieve these states. Moreover, despite being free to use other modalities according to their own preferences, participant report did not indicate usage of modalities not captured by the four primaries, suggesting our results adequately capture the space of endogenous emotion generation modalities. Overall, and in line with previous research, we found that Episodic Imagery was the most frequently used modality (47.71%) followed by Semantic Analysis (22.72%), Bodily Interoception (18.95%), and Auditory Imagery (10.62%). In line with our expectations, the vast majority of participants reported using a combination of modalities to generate emotion. This most frequently took the form of using other modalities in adjunct to Episodic Imagery (see Table 7.1 and Figure 7.1D), with Semantic Analysis and Bodily Interoception being used in approximately equal measure. Despite this we did not find that any combination of modalities outperformed others, with Semantic Analysis, Episodic Imagery and Bodily Interoception usage all having comparable, significant, effects on generation efficacy. This suggests that proficiency with a given modality was reflected in better emotion generation abilities. The relative inefficacy and lack of use of the Auditory Imagery modality could be possibly be attributed to the loud

scanner environment the generation occurred in. That aside, these results support our contention that participants used the modalities that they were the most adept at using, meaning our experiment can be taken as reflecting individual differences in emotion generation abilities.

Investigating the relationship these abilities have with measures of emotion management style and trait affectivity, we found that both general emotion generation ability and the relative ability to generate positive over negative emotion were positively correlated with trait affect and beneficial emotion management styles. However, only general emotion generation ability was found to causally mediate the effect of emotion management styles on trait affect, suggesting that the relative ability to generate positive or negative affect is an *outcome* of trait affect and adaptive coping and does not partake in the mechanism by which adaptive emotion management facilitates mental health. Overall, our findings support the *flexibility hypothesis* of the mental health benefits of emotion generation, showing that the capacity to generate emotions in general supports the means by which adaptive, instrumental emotion management promotes positive affective tone. While the precise mechanism by which emotion generation abilities do this is a topic for future research, one possibility is that they enable the embodied episodic simulation of the emotional outcomes of future events (S. E. Taylor & Schneider, 1989), thereby enabling one to flexibly plan and adapt ones behaviour to negative and facilitate positive emotional events (Kappes, Singmann, & Oettingen, 2012; Kashdan et al., 2015; Kashdan & Rottenberg, 2010).

In addition to their theoretical importance, our findings inform the ongoing development of interventions aimed at harnessing different modes of emotion generation to facilitate emotional self-regulation, suggesting that these interventions should be aimed at the generation of both positive and negative emotion, and that there are multiple means by which generation can be implemented. This contrasts with current research that tends to highlight the ability to generate of positive emotion as a means to improve mental health (H. G. Engen &

Singer, 2015; Holmes et al., 2006; e.g. Poerio, Totterdell, Emerson, & Miles, 2015a). Implicit in these studies is a *buffering* hypothesis, along the lines that increasing access to positive emotion will undo negative emotion. Our results that relative emotion generation abilities do not mediate the relationship between trait affect and emotion management styles could be taken as evidence against such a buffering account. However, it should be remembered here that the current results pertain to the normal, untrained individual. In support of this, recent evidence suggests that in the normal population beneficial effects of positive emotion generation are phasic, and can, indeed, be deleterious to mental health over time if they are divorced from behaviours aimed at achieving the eliciting end-state (Oettingen, Mayer, & Portnow, 2016). Thus, while valence-specific emotion generation abilities might not enable an untrained individual to manage their emotions, this might be made possible with training (Fredrickson, 2013).

Speaking to how to train emotion generation, while the vast majority of extant research has focused on Episodic Imagery (Murphy et al., 2015), our results suggest that focusing interventions based on Episodic Imagery alone might not be optimal for everyone. Indeed, we found that, while Episodic Imagery was the most frequently used, the large majority of participants spontaneously combined Episodic Imagery with other modalities when instructed to optimally generate emotions. This is interesting because it appears that a sequela of major affective disorders like anxiety and depression is impaired ability to engage in Episodic Imagery (Morina et al., 2011). Possibly, this can be counteracted by devising interventions that emphasise the use of supporting modalities to enable flexible generation of emotions in both pathology and normal function.

7.5.1. Conclusion

In this study we provided evidence that endogenous emotion generation enables emotion management, causally mediating the relationship between trait affect and specifically

instrumental means of dealing with negative affect. Importantly, evidence suggests that this ability is related to individual differences in the flexibility of emotion management rather than an increased ability to create endogenous buffers against exogenous stressors, possibly related to the capacity to simulate and thereby predict future affective states. As such, endogenous emotion generation appears to be an important part of emotional self-regulation that should be a focus of future studies.

7.5.2. Acknowledgements

Tania Singer, as principal investigator, received funding for the *ReSource Project* from a) the European Research Council under the European Community's Seventh Framework Program (FP7/2007-2013/ ERC Grant Agreement Number 205557 to T.S.), and b) from the Max Planck Society. We are thankful to all the members of the Department of Social Neuroscience involved in the ReSource study over the many years, to Astrid Ackermann, Christina Bochow, Matthias Bolz, and Sandra Zurborg for managing the large-scale longitudinal study, to Hannes Niederhausen, Henrik Grunert, and Torsten Kästner for their technical support, to Sylvia Tydeks, Elisabeth Murzick, Manuela Hofmann, Sylvie Neubert, and Nicole Pampus for their help with recruitment and data collection.

Part III: General discussion

Chapter 8: Summary and discussion

8.1. Chapter overview

The two goals of this thesis were 1) to explore the mechanisms supporting the endogenous generation of emotion and 2) how these can be used for emotion management. Pursuing the first goal, Chapters 4 and 5 explored the neural bases of EnGE. Chapter 4 sought to establish the neural component process architecture of EnGE in two large representative samples, using a novel paradigm that enabled dissociation of general from implementation-specific neural mechanisms. Chapter 5 sought to verify this architecture by investigating morphological differences in a sample of long term meditators with expertise in EnGE-based practices. Pursuing the second goal, Chapters 6 and 7 investigated the functional consequences of EnGE as an emotion management technique. Chapter 6 investigated the neural mechanisms of using EnGE-based meditation techniques for to regulate emotional reactions to external stressors in a population of expert meditators. Chapter 7 widened the scope of investigation and explored how EnGE-abilites in general are associated with emotion management and trait affect in the normal population. In the following, the results from these studies are summarised starting with a characterisation of the phenomenon of EnGE as the current studies reveal it, followed by an account of what the findings suggest about how EnGE can be used as a means of emotion management.

8.2. Characterising endogenous emotion generation

8.2.1. Behaviour and psychophysiology

In the working model outlined in Chapter 2, it was proposed that EnGE should be conceptualised as an embodied phenomenon, resulting (when successfully implemented) in emotional reactions with subjective and physiological responses similar to those concomitant exogenously generated emotions. Overall, results showed that participants were capable of

self-generating strong emotional states, as measured by self-report in the RAGE paradigm (Chapters 4 and 7; Figure 4.1). Moreover, a significant relationship was found between reported affect and psychophysiological responses, such that ratings of positive or negative affect were associated with higher skin conductance levels (SCL). Given the known association between emotional arousal and SCL (Kreibig, 2010) this both validates the experimental approach taken, and supports the operationalisation offered in Chapter 2.2 in which EnGE was defined as a process by which embodied emotional experiences are elicited.

8.2.2. Naturalistic EnGE: Implementation forms and efficacy

As was discussed in Chapter 2, the vast majority of research on the efficacy of EnGE has focused on mental imagery, with only a few comparative studies investigating the relative efficacy of mental imagery versus e.g. semantic analysis as a means of self-induction. Summarising, this research (see Holmes & Pictet, 2013 for a recent review) has found that mental imagery appears to be the most efficacious technique for generation of both positive and negative emotion. Conversely, approaches using semantic analysis apparently leads to paradoxical effects, such as attempts at generating positive emotion resulting in mood decreases (Holmes & Mathews, 2010). In support of this, the current results show that analogue of mental imagery (i.e. episodic imagery) was the most frequently reported of the four modality-options participants reported using in the RAGE (Chapter 7, see Figure 7.1). However, unlike previous studies, participants were allowed to freely combine different modalities, with the objective of gaining a more naturalistic measure of how different modalities might be combined. When investigating the proportional combinations of modalities, (Chapter 7, Figure 7.1D, Table 7.1) it was found that the overwhelming majority of participants reported using combinations of modalities to generate emotion. While episodic imagery was clearly the most preferred modality overall, both semantic analysis and bodily interoception were frequently used in conjunction with it (see Table 7.1). Thus, it appears that

using multiple modalities constitutes the standard approach to generating emotion in the normal population. Moreover, considerable variation was observed in which and to what degree participants combined modalities, likely reflecting individual differences in the ability to access information from different modalities. This source of variability is important to consider in future work investigating EnGE, in particular for interventional research where populations might have decreased ability to use given modalities (e.g. depression tends to decrease the ability to engage in mental imagery Bergman & Craske, 2000) such that training in other modalities might enable circumvention or compensation for deficits. This conclusion is strengthened when considering the relative efficacy of strategies: The current findings suggest that, while imagery is the most effective means of generating emotion, both semantic analysis and bodily interoception can be used to self-induce emotional states effectively. Indeed, when combined with the fact that participants spontaneously chose to combine modalities when given the option, this suggests that effective emotion generation is best afforded by combining multiple streams of information. This is in line with the operationalisation, proposed in Chapter 2, of EnGE as a process by which rich, multi-modal, embodied representations of emotional events are constructed. While imagery of course is central to this by allowing one to imagine emotional scenarios, both bodily indicators of emotion (G. A. Miller et al., 1987) and different semantic appraisals (Kross et al., 2005; 2009; Kross & Ayduk, 2008) impact on the emotional consequence of such imagery. Thus, combining modalities is likely to result in more vividly experienced emotional states, explaining the current findings.

8.2.3: The neural component-process architecture of EnGE

In Chapter 2, a working model of EnGE was proposed in which EnGE is supported by the cooperation of three major intrinsic connectivity networks, the Default Mode (DMN), Salience (SN) and Frontoparietal Control Networks (FPCN). Based on an analysis of the

psychological mechanisms likely involved in EnGE, it was proposed that these networks implement distinct component processes during EnGE. Specifically, it was proposed that SN was a candidate network for the generation of core affective states. DMN was proposed supporting the generation of internal representations of the conceptual context that gives meaning to these core affective states, resulting in emotional experiences that can be characterised as embodied simulations. Finally, FPCN was proposed to serve a particular role in volitional EnGE, supporting both controlled retrieval processes and supporting goal-directed coordination of SN and DMN. In Chapter 4, this model was tested in two fMRI experiments using the same paradigm (the RAGE). Overall, it was found that EnGE of both positive and negative emotional states (relative to maintaining a neutral state) was associated with activation of nodes of the three intrinsic connectivity networks proposed (Figure 8.1).

In two separate experiments in Chapters 5 and 6, replication of these findings were sought by investigating the neural correlates of loving kindness and compassion meditation – two meditation techniques that both are known to centrally involve active generation of, specifically positive, affect (Klimecki et al., 2013; 2014). As such, this allowed the investigation of a "naturally occurring" technique by which to train emotion generation. Importantly, participants in these studies were expert, long term meditators that had

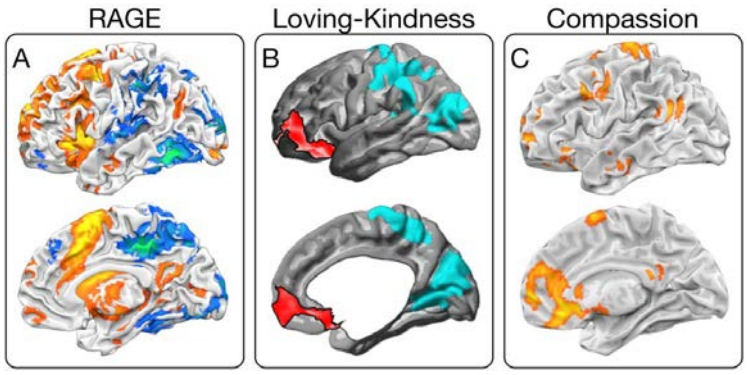


Figure 8.1: The main functional neuroimaging findings from the current series of experiments. **A)** Results from Chapter 4, showing the contrast of Generate > Neutral. **B)** Results from Chapter 5, showing increases in amplitude of low-frequency fluctuations during loving-kindness meditation relative to rest. **C)** Results from Chapter 6, showing the contrast Compassion-meditation > Watch-Negative. For details see respective chapters. All results FWEc < .05.

specialised in loving-kindness and compassion meditation, allowing us to investigate the effects of emotion generation expertise on brain morphology. Summarising, the results from these two studies showed a great deal of overlap with the results described above. Loving-kindness meditation was associated with increased activation of left ventrolateral prefrontal cortex, including inferior frontal gyrus, overlapping with the FPCN nodes reported above. Further, activation of perigenual, frontopolar and ventromedial prefrontal cortices was observed, corresponding closely to the frontal portion of the DMN. Evidence was also found that compassion meditation was associated with increased activation of several nodes of the extended Salience Network, including anterior insula and several subcortical regions, including parts of the basal ganglia (most notably ventral striatum and nucleus accumbens). Thus, these studies suggest that loving-kindness and compassion meditation has a similar neural implementation to endogenous emotion generation as observed in Chapter 4, supporting the contention that these regions implement core component processes of volitional emotion generation. Importantly, Chapter 6 demonstrated that this was the case also when participants were employing emotion generation to cope with external stimuli, meaning that the engagement of this network generalises outside of purely self-focused generation contexts.

Using both model-based and data-driven analysis approaches, the distinct component processes supported by these networks were probed: Model-based decomposition of time-series (Figure 4.3) indicated that the initial generation of emotional states was associated with activation of the SN, while the representation of such emotional states was associated with activation of DMN, together with ventral AI and core limbic structures. FPCN nodes, centred on pre-SMA and left-lateralised. Finally, left lateralised nodes of the Frontoparietal Control Network, in particular the triangular and orbital portions of the inferior frontal gyrus, middle frontal gyrus and the pre-supplementary motor area had activation dynamics consistent with them supporting the maintenance of the emotion generation process. Complementary data-

driven network-centric analyses showed that FPCN partook in two distinct functional components predictive of EnGE-success. Interestingly, in addition to FPCN, these two components consisted of nodes of (primarily cortical) SN and DMN (together with limbic structures) respectively. Thus, these findings suggest FPCN coordinates activation of these networks EnGE, and thus plays a part in implementing both core affect generation and representation formation component processes. When viewed in the context of the finding that FPCN appears to be centrally involved in the effortful maintenance of EnGE, this suggests that the FPCN supports EnGE by maintaining generated emotional states over time. Interestingly, Chapter 5 found evidence of cortical thickening in the IFG portion of this network in long-term meditators with expertise in practices requiring the endogenous generation of emotion (Figure 5.1). This thickness increase overlapped with the functional activations supporting the state of loving-kindness meditation, and differs from structural changes frequently reported for kinds of meditation less reliant on the generation of internal representations (Fox et al., 2014). Thus, extensive training in the generation of emotional states appears to alter brain morphology in the left IFG, consistent with this region playing a key role in EnGE. Summarising, the current results support the hypothesised three-process, three-network model of EnGE, albeit with a number of discrepancies with respect to the exact regions found to be involved, and how they functionally interact. These are discussed in detail below.

8.2.4. Neural mechanisms of valence-specific EnGE

In addition to establishing the overall neural architecture of EnGE, Chapter 4 explored the neural correlates of the generation of different valences. This was done by investigating parametric modulations of BOLD signal by subjective reports following each trial (see Figure 4.5). Overall, there was a remarkable overlap between regions correlating with positive and negative affect, most notably in the frontal portions of the left FPCN (IFG, MFG and pre-

SMA) and SN (dorsal AI, dorsal ACC, basal ganglia, thalamus). This finding is consistent with recent work showing that cortical and limbic representations of emotion are mainly bivalent (K. A. Lindquist, Satpute, Wager, Weber, & Barrett, 2016). Thus, it appears that the generation of subjective states of positive and negative emotion largely relies on the same neural mechanisms.

However, a clear dissociation between positive and negative emotion was observed, centred on the brainstem: Positive emotion ratings correlated with activation of ventral aspects of the mesencephalon corresponding to the substantia nigra/ventral tegmental area (SN/VTA). Conversely, negative emotion ratings correlated with dorsal aspects of the mesencephalon, corresponding to the periaqueductal grey. Interestingly, these regions are perhaps the evolutionarily oldest structures known to be associated with hedonic experiences (Berridge & Kringelbach, 2013). For instance, the SN/VTA is heavily implicated in reward processing, and is known to be the originator of the dopamine-based reward circuit (Fields, Hjelmstad, Margolis, & Nicola, 2007). Important for present purposes, the integrity of this reward circuit is associated with trait differences in anhedonia, or the inability to experience positive emotion (Alcaro, Huber, & Panksepp, 2007; J. Keller et al., 2013), consistent with it being involved in the generation of the positive core of endogenous affective experiences. The PAG, conversely, is involved in the processing of negative reinforcers ranging from painful stimulation (Roy et al., 2014) to negative emotion (Satpute et al., 2013). Indeed, a recent study (Buhle et al., 2013) found that the PAG was a convergence zone for the experience of both pain and negative emotion elicited by negative picture viewing. This is consistent with the present findings, suggesting that recruitment of the PAG is involved in the generation of core affective qualities of endogenous negative emotional states. While usually overshadowed by the amygdala in the human literature on the neural bases of negative affect, it should be noted that the PAG features heavily in animal models of motivation and affect (Behbehani, 1995; Panksepp, 2004). Importantly, the connectivity patterns (Coulombe, Erpelding, Kucyi,

& Davis, 2016; Tomasi & Volkow, 2014) of the SN/VTA and PAG are consistent with the current observations, with both regions showing intrinsic functional connectivity with insula, basal ganglia and dorsomedial prefrontal – all regions found to show bivalent correlations with subjective experience in the current results.

Interestingly, it does not appear that these midbrain regions are functionally connected to FPCN. This suggests a model in which frontal FPCN engages (particularly cortical) SN to enact the generation of valenced, core affective states that in turn are expressed by activation of the mesencephalon. In support of this model, CPCA results (Figure 4.4) suggest that cortical and thalamic SN, but not mesencephalon, form a functional network. The precise causal chain of this influence is unclear however, with both dorsal ACC (Beckmann, Johansen-Berg, & Rushworth, 2009) and insula (Cauda et al., 2011) being intrinsically connected to the frontal nodes of the FPCN observed, meaning that FPCN could be enacting these effects via both dorsal and ventral routes. Future work could adopt a model-based effective connectivity approach, using e.g. dynamic causal modelling to investigate this (Cha et al., 2016).

At this point, the possibility should be mentioned that the correlations reported here might resolve into distinct subregions coding for positive and negative valences if data was acquired with higher resolution. For instance, evidence suggests that, while the nucleus accumbens (NACC) is involved in the processing of both reward and aversion, there is a distinct topography to this, such that anterior NACC supports rewards and posterior NACC supports aversion processing (see Berridge & Kringelbach, 2015 for a review). Unfortunately, the current investigations do not have sufficient spatial resolution to resolve subcomponents of the NACC, so valence specificity at a higher level than mesencephalon cannot be ruled out. Similarly, as the current results are based on univariate methods (specifically parametric modulation of region activation), it cannot be ruled out that there are differential, multivariate activation patterns supporting positive and negative emotion generation. However, even if this

were the case this would not invalidate the general conclusion from the current work that EnGE engages even deep and evolutionarily old circuits known to subserve the generation of core affective sensations. This strongly suggests that volitionally generated endogenous emotions should be considered "real emotions", on line with both spontaneous endogenous and exogenously elicited emotional reactions.

On a theoretical level, it should be noted that this finding diverges from the process model of EnGE proposed in Chapter 2 (Figure 2.3), where control processes were proposed to support recall of modality-specific information and the multimodal associative construction process, in line with previous work on self-generated thought and planning (Gerlach et al., 2014; Spreng et al., 2010). On this model, one would expect control networks (i.e. the FCPN) to primarily interact with systems supporting the generation of representations (supported by the DMN), which in turn would couple to systems supporting the generation of core affect (supported by the SN). While the current results do support a role of the FPCN in both representation formation and recall of modality-specific information (see Chapter 8.2.5), FPCN activation both scales with subjective affect intensity and forms a functional subcomponent with the SN. This is consistent with FPCN initialising both the generation of core affective states and representation generation. Seemingly, this finding is at odds with the modal model of emotion generation (see Figure 2.3 A; J. J. Gross, 2007b), where emotional reactions are posited to stem from appraisal of affectively relevant situations. It does, however, cohere with what is predicted from a constructivist account of emotion generation (Barrett, 2011), which posits that emotional experiences occur when feelings of physiological changes in the body are interpreted by relating them to conceptual knowledge about the world (i.e. situation). On this model, EnGE could not occur without coordinated generation of both sensations of the bodily concomitants of emotions, as well as the formation of a representational context in which these sensations are made meaningful. Thus, the co-activation of, respectively, SN and DMN, with FPCN could plausibly reflect this generation

process, with the SN-FPCN subcomponent generating the core affective states of emotions, while FPCN-DMN supports the (goal-directed) generation of simulations used to provide context to these states.

8.2.5. Supramodal and modality-specific neural systems of EnGE

Supporting this account, evidence overall supported a role of DMN nodes in specifically the formation of elaborated representations. Reported usage of modalities was correlated with activation in largely domain-relevant regions (see Figure 4.6): Semantic analysis usage correlated with activation of regions overlapping with the proposed semantic system, including Wernicke's area and premotor cortex (Binder et al., 2009). Conversely, episodic imagery usage correlated with activation of the retrosplenial cortex/posterior cingulate cortex, known to be involved in episodic retrieval processes (Andrews-Hanna et al., 2010). Finally, bodily interoception usage correlated with activation of bilateral anterior insulae, known to be involved in a wide range of interoceptive processes (Craig, 2002; Seth, 2013; Singer, Critchley, & Preuschoff, 2009).

Next, regions were identified whose relationship with reported usage was mediated by activation of these regions. As this is essentially a correlational method, this analysis allowed us to identify the extended functional connectivity networks supporting modality usage. As above, the networks thus identified were largely consistent with expectations: Semantic analysis was supported by large components of the extended semantic system (Binder et al., 2009), including Broca's area (BA44), a region known to be essential for generative aspects of language. Episodic imagery was found to most centrally be supported by precuneus/anterior PCC and ventromedial/orbitofrontal regions overlapping with the medial-temporal subsystem of the DMN, most closely associated with episodic retrieval and mental imagery (Andrews-Hanna et al., 2010; 2014; Hassabis & Maguire, 2007; Schacter & Addis, 2007). Finally, bodily interoception was supported by a more constrained network, but centrally including 1)

posterior insula, thought to correspond to primary interoceptive cortex (Craig, 2002), and 2) extrastriate and fusiform body areas, known to support representations of bodies and body parts (Lamm & Decety, 2008; J. C. Taylor et al., 2007).

In addition to these modality-specific networks, a supramodal pathway (Figure 4.6C) was identified. This pathway included key nodes of the DMN, including dorsomedial PFC and middle temporal gyrus ranging into the temporal pole. Additionally, the pathway included key nodes of the FPCN, including bilateral IFG, and left lateral MFG and premotor cortex, as well as components of the SN, including ventral anterior insula and portions of the caudate and pallidum of the basal ganglia. The fact that this pathway supported multimodal implementation suggests that it is at the functional core of EnGE. Interestingly, this pathway to a large degree overlaps with the dorsal-medial subsystem of the DMN, known to have close associations with domain-general integration of endogenous information into internal simulations (Andrews-Hanna, 2012). Moreover, the FPCN nodes partaking in this pathway roughly correspond to the inferior frontal junction, known to be involved in a wide range of self-control processes (Brass & Eickhoff, 2016), and the pars triangularis (BA 45) and opercularis (BA 47) portions of the inferior frontal gyrus. Interestingly, these two regions are thought to be central to the cognitive control of mnemonic retrieval (Badre & Wagner, 2007), particularly if such retrieval is based on semantic cues (Thompson, Henshall, & Jefferies, 2016). Notably, there was a marked overlap between these components and those found to predict individual ratings of emotional intensity (discussed above in Chapter 8.2.4), suggesting that ventrolateral FPCN supports the controlled retrieval of information relevant to the emotional target state, which is then used to construct detailed simulations of emotional states, presumably supported by the dorsal-medial DMN. Supporting this interpretation, constrained principal components analysis of the EnGE process revealed that the same ventral and dorsolateral components of the FPCN partook in two functional components whose activation predicted generation success (see Figure 4.4). Importantly, in the remainder, these

components consisted of regions largely overlapping with the DMN and the SN, respectively. Overall, these findings strongly suggest that the left frontal FPCN is pivotal in organising the EnGE process, engaging both core affective regions, domain-general systems involved in the generation of representations, and modality-specific networks, in line with the degree to which the generated representations rely on different sources of information.

8.2.6. The role of limbic structures in EnGE

In the working model presented in Chapter 2, it was proposed that the extended Salience Network (SN) worked in unison to elicit core affective states. One of the more unexpected findings in the current thesis was evidence that two key structures of the extended SN, the amygdala (AMY) and NACC appeared to not be involved in the initial generation phase of EnGE, but rather in its extended representation (see Figures 4.3, 4.4). Based on extensive evidence regarding the important role of these structures in the generation of emotional reactions (Berridge & Kringelbach, 2013; Kober et al., 2008; K. A. Lindquist et al., 2016; K. A. Lindquist & Barrett, 2012; Wager et al., 2008) the involvement of these limbic structures was primarily thought to be in the generation of affective responses. Activation patterns of these regions were not consistent with them supporting the generation of core affective qualities of endogenous emotions, but rather suggested they were involved in the elaboration of these states into emotional experiences (see Figure 4.3). Moreover, data-driven multivariate network analysis of the EnGE process revealed that AMY and NACC formed a functional component with DMN and not SN (see Figure 4.4), a finding that is surprising given their intrinsic connectivity patterns of these regions (Menon & Uddin, 2010; Seeley et al., 2007). Overall, this suggests that AMY and NACC supports relatively higher-order processes related to the experience of emotion in EnGE, and not the generation of hedonic states as originally expected.

As previously mentioned, recent constructivist models of emotion generation (K. A. Lindquist & Barrett, 2008; e.g. Russell, 2014), propose that the experience of an emotional state only occurs when core affective sensations are interpreted relative to the context the individual finds itself in. This act of contextualization is thought to be supported mainly by the DMN (K. A. Lindquist et al., 2012), instantiating constructive memory processes (Schacter et al., 2012; Schacter & Addis, 2007). Their activation during EnGE could thus reflect a role of NACC and AMY in guided search for affective memory traces in service of elaborating core affective states, in turn produced by the activation of mesencephalic structures and more posterior parts of the basal ganglia by cortical SN. Supporting this interpretation, animal models suggest that interaction of AMY and NACC plays an important role in the encoding and consolidation of emotional memories (LaLumiere, LaLumiere, Nawar, & McGaugh, 2005; Roozendaal et al., 2009; Setlow, Roozendaal, & McGaugh, 2008). Moreover, both NACC and AMY are known to play a key role in effecting behaviours based on instrumental learning (Albertin, Mulder, Tabuchi, Zugaro, & Wiener, 2000; Balleine & Killcross, 2006; Corbit, Corbit, & Balleine, 2005; Corbit et al., 2001), a process that critically involves linking stimuli to affective responses via the intermediary of internal representations of causal relationships.

8.2.7. The role of anterior insula in EnGE

However, fractionation of the cortical portion of the SN was also observed, with an apparent dissociation evidenced between the ventral and dorsal portions of the anterior insula (AI), with dorsal AI apparently supporting the initial generation of core affect (as reviewed in Chapter 8.2.4), and the ventral AI forming a part of the supramodal pathway (described in Chapter 8.2.5), together with limbic structures and DMN in support of experience representation (see Figure 4.6). Interestingly, this differentiation mimics a recent study reporting that dorsal and ventral AI are functionally dissociable, and that the connectivity

strength of this region with other components of the SN (notably hypothalamus, basal ganglia and perigenual ACC) is predictive of individual differences in the vividness of emotional experiences (Touroutoglou et al., 2012). Conversely, the dorsal AI connected to posterior regions of dACC, overlapping with the pre-SMA, MFG and IFG, roughly corresponding to the portions of the FPCN found to support the executive maintenance of EnGE in the current work. Moreover, connectivity strength of this subnetwork predicted performance on the Trail Making Test (TMT; Reitan, 1958), a frequently used measure of processing speed and cognitive flexibility. Interestingly, it appears that the TMT is sensitive to individual differences in the capacity to implement and shield task sets in a top-down manner (Arbuthnott & Frank, 2000; Kortte, Horner, & Windham, 2002; Sanchez-Cubillo et al., 2009). Thus, these findings hint at a functional organisation by which the FPCN entrains dorsal AI, which in turn implements the attentional set guiding the EnGE process. Ventral AI, conversely, appears more closely involved in the contextualization of this core affect and experience of emotion proper, in line with the proposed role of the anterior insula in generating experience states (Craig, 2002; 2011). Supporting this interpretation, Chapter 5 demonstrated that LKM expertise was associated with increased cortical thickness of both dorsal and ventral portions of the AI. However, only ventral AI overlapped with the functional activation pattern supporting LKM, suggesting it is a unique structural correlate of EnGE expertise, together with left IFG. Moreover, dorsal AI is frequently reported as a structural correlate of meditation practice in general (Fox et al., 2014), suggesting its involvement here might reflect general effects of meditation related to the volitional regulation of attention, in line with the known role of the dorsal AI in attention regulation (Dosenbach et al., 2006).

One problem with this account is that ventral AI is thought to partake in a network supporting core affective processing in general (Touroutoglou et al., 2012). Contrary to this, the current results indicated that dorsal, but not ventral, AI is correlated with intensity of

generated emotional experiences and that it co-activates with mesencephalic structures. Moreover, unlike Touroutoglou and colleagues, the current data indicated that dorsal AI and genual ACC co-activate and form functional network during EnGE suggesting that dorsal AI still serves important affective functions for generating emotional reactions to internal experiences. Supporting this notion, it has been found that dorsal and ventral AI have subtly different functions in risk processing, with dorsal AI activation being involved in the prediction of risk, whereas the ventral AI is involved in processing prediction errors (Preuschoff et al., 2008). Important for present purposes, risk prediction centrally involves simulating the future outcomes of behaviours and estimating their hedonic value, while prediction error involves evaluating the situation based on one's expectations. Importantly, both can be thought of as affective states, but differ critically in their reliance on internal and external sources of information: Risk prediction involves the projection of potential future affective outcomes (i.e. potential gain or loss). Prediction error involves integrating internal and external information to form an evaluation, frequently resulting in an affective state (Jensen et al., 2007). Finally, the intrinsic connectivity of mesencephalon, found in the current work to be associated with the generation of core affective states, appears to be centred on dorsal aspects of the AI (Coulombe et al., 2016; Tomasi & Volkow, 2014). This could suggest that the dorsal AI is central in generating core affective states based on internal representations or goals, while ventral AI is involved in generating the reaction to these states, i.e. the emotional experiences proper.

8.2.8. Disinhibition as a possible component process of EnGE

Unexpectedly, strong lateralisation effects were observed in Chapter 4, with emotion generation ostensibly involving the activation of left and deactivation of right FPCN, together with a collection of regions known to partake in the dorsal attention and visual networks. The latter finding can be explained by reference to the fact that the dorsal attention network most

frequently is associated with attention to the external world, whereas endogenous emotion generation by its very nature requires attention to be directed internally, away from the outside world. Supporting this interpretation, this pattern of deactivation was not apparent when compassion meditation based emotion generation was used as an emotion regulation strategy (Chapter 6), where attention is required towards the external world. However, a similar explanation cannot as readily be offered for the FPCN effects, also in evidence. Similar activations have frequently been reported in previous studies (Liotti et al., 2000; Mayberg et al., 1999). Interestingly, unlike most large scale intrinsic networks, there is evidence for the functional independence of left and right frontoparietal networks (Harrison et al., 2008; Laird et al., 2011; K. L. Ray et al., 2013). Moreover, right, but not left, frontoparietal networks are traditionally thought to be closely related to a specific form of cognitive control pertaining to response monitoring and inhibition (Aron, Robbins, & Poldrack, 2014). Conversely, as previously discussed, left frontoparietal control regions (particularly in the ventrolateral aspect found to be central here) is more closely related to modes of control involving e.g. cognitive control of memory (Badre & Wagner, 2007), semantics (Noonan, Jefferies, Visser, & Lambon Ralph, 2013), verbal fluency (Costafreda et al., 2006) and other processes that require active efforts to construct representations of meaning based on internal information. While speculative, it is possible that the lateralisation differences might reflect the conceptual divide between inhibition and generation as general modes of processing.

Three points of evidence in the current data support this conclusion: First, when participants were requested to down-regulate their endogenously generated emotions in Chapter 4, right frontoparietal regions went from being deactivated relative to baseline to becoming strongly activated, suggestive of right frontoparietal regions supporting the suppression of existing emotional states. Second, Chapter 6 showed that use of reappraisal lead to higher activation of bilateral frontoparietal networks relative to the watch-negative

baseline, but only the right frontoparietal component when contrasted with compassion. This is important because one can think of reappraisal as requiring two distinct component processes in order to effect the transformation of the one emotional meaning of a stimulus into another: 1) inhibiting extant meaning and 2) the active generation of a new interpretation (Ochsner & Gross, 2008). Recent evidence suggests that left ventrolateral prefrontal activation is both robust (Buhle et al., 2014) and unique to reappraisal as an emotion regulation strategy (Dörfel et al., 2014). Conversely, while right FPCN activation is also frequently reported in reappraisal (Buhle et al., 2014) at large and it appears to be a core network of emotion regulation in general (Dörfel et al., 2014), consistent with a general role of the right FPCN as a suppressor of emotional information. Supporting this, deactivation of right FPCN was observed compared to both reappraisal and passively viewing neutral videos (cf. Tables 6.1-2). Importantly, this deactivation was observed independently of external stimulation (i.e. in the *preparation* phase, see *Appendix*: Table A3.2.3, Figure 6.1), suggesting it plays a key role in the generation of the state of compassion itself. Thus, it appears that EnGE involves activation of left ventrolateral PFC (VLPFC) with complimentary deactivation of right VLPFC and that this balance is inverted when the objective is suppressing already existing emotions. Thus, one possibility is that they involve some sort of "disinhibition" process, by which emotional information is allowed to take place in working memory (Mayberg et al., 1999). Future studies could investigate this by e.g. performing a principled comparison of the neural systems supporting the generation of states that are matched in content with the exception of emotional content, using e.g. a guided imagery approach (e.g. Wilson-Mendenhall et al., 2013).

8.2.9. Summary: Endogenously constructing emotional states

The neural component process architecture indicated by the current findings largely cohere with the proposed working model, demonstrating that EnGE is implemented by

activation of the extended Salience Network, both core and dorsomedial subsystems of the Default Mode Network (Andrews-Hanna et al., 2010), and left lateralised Frontoparietal Control Network across a variety of implementations and for both positive and negative target states. Moreover, findings suggest that these networks implement dissociable processes in EnGE. In particular, evidence was found for a neural distinction between processes of core affect generation (centred on the Salience Network), and processes supporting the integration of this core affective state with memories and conceptual knowledge to create a full-fledged emotional experience (centred on the Default Mode Network, but supported by limbic structures and ventral portions of the insula). As such, the current findings are compatible with recent constructivist account of the component processes of emotion (Barrett, 2014) and their neural implementation (Kober et al., 2008; K. A. Lindquist et al., 2012).

8.3. EnGE-based emotion management

The second goal of the thesis was to investigate the utility of EnGE as a means of emotion management. Specifically, it was hypothesised that positive EnGE could be used as a means to manage emotional reactions to negative stressors and that this should be supported by engagement of the same neural mechanisms as described above. As little work exists on the regulatory mechanism of reactive EnGE-based emotion management, three differential hypotheses were proposed: 1) *distraction* (Dörfel et al., 2014; Kanske et al., 2011; McRae et al., 2010), by which EnGE-based emotion management should result in decreased negative and increased positive emotion, 2) *facilitation* (Diedrich et al., 2016), which would have similar subjective outcomes, but additional engagement of other emotion regulation circuits, and 3) *buffering,* by which increased positive emotion would enhance the ability to endure negative emotion, leading primarily to increased positive affect, and little impact on negative emotion. Chapter 6 tested this by investigating the neural and behavioural effects of using compassion meditation to modulate emotional responses to negative stimuli. As compassion

meditation centrally involves the generation of positive affect, this served as an experimental analogy to the situation described above. The practitioners also regulated their emotions using reappraisal, a widely investigated effective cognitive emotion regulation strategy that is recognised as being highly effective (Ochsner & Gross, 2008) and to be neurally representative for emotion regulation techniques that involve altering one's emotional reactions (Dörfel et al., 2014). Importantly, as reappraisal involves the active generation of mental content in the form of alternate interpretations of emotion-eliciting stimuli (Ochsner & Gross, 2008), it is comparable to compassion in that they both require active effort and reference to the eliciting stimulus (Mascaro et al., 2013). Further, to avoid confounding potential regulatory effects with differences in regulatory goals, participants were instructed in positive reappraisal, where the objective is to regulate emotional reactions to negative stimuli by generating a positive interpretation. Thus, the critical conceptual difference between compassion and reappraisal as it was here implemented is their reliance on *de novo* generation of emotion versus changing how one processes emotion-eliciting stimuli. As such, Chapter 6 provided a direct test of the reactive-mode of EnGE-based emotion management discussed in Chapter 2.8.5, and investigated how this neurally and behaviourally is comparable to forms of cognitive emotion regulation.

8.3.1. Subjective effects of EnGE-based emotion regulation

The current results speak against the active mechanism of EnGE-based regulation being either *inhibition* or *facilitation* of transformation of already existing emotional states, since the involvement of such mechanisms would predict alterations to emotional state being regulated: Compassion was associated with both decreased negative affect and increased positive affect. Moreover, the increase in positive affect being considerably more pronounced than the decrease of negative affect, though the latter decrease was, indeed, significant. The subjective regulatory effect of positive reappraisal, conversely, was characterised by a

reduction of negative affect, following the literature at large (e.g. Wager et al., 2008). Thus, the regulatory effect of compassion appears to have been brought about by the counter generation of positive affect. This had the effect enabling mixed subjective feeling state (J. T. Larsen, McGraw, & Cacioppo, 2001) whereby both ongoing negative and *de novo* generated positive emotional states coexist, leading to a reduction of experienced negative affect. As such, the supposition that emotion generation can be used as an affect-based regulation strategy was supported. Moreover, evidence suggests that the means of regulation are markedly different from that observed in even closely related cognitive strategies, such as positive reappraisal.

8.3.2. Neural bases of EnGE-based emotion management

Chapter 6 also investigated the neural bases of this difference. Overall, the behavioural findings were mirrored by neural dynamics: Compassion selectively increased activation of neural correlates of subjective positive affect (notably nucleus accumbens), and reappraisal decreased activation in neural correlates of subjectively negative affect (notably amygdala). Moreover, clear dissociation was apparent in the overall activation patterns associated with these techniques with reappraisal activations centring on frontoparietal regions shown to support general implementation of emotion regulation across strategies (Dörfel et al., 2014). Conversely, compassion activations were centred on the representative (medial prefrontal) and core affective (insula, basal ganglia, pre-supplementary motor area) subsystems of the EnGE architecture proposed above (see Figure 6.2).

These findings demonstrate the feasibility of using endogenous emotion generation as an emotion management technique. Furthermore, they show that the behavioural consequences and neural mechanisms supporting the regulatory effects of generation-based regulation are clearly dissociable from other forms of emotion regulation, including the closely comparable strategy of positive cognitive reappraisal.

Returning to the hypotheses proposed in Chapter 2, the behavioural and neural results from Chapter 6 are consistent with a buffering account of reactive EnGE-based emotion management (discussed in Chapter 2.5.2), whereby emotion generation facilitates affective well-being by allowing one to spontaneously elicit positive emotional states (Fredrickson, 2013), in effect allowing one to maintain a positive hedonic balance and buffer against negative stressors.

8.3.3. The role of the subgenual anterior cingulate in EnGE-based regulation

One thing that it left unclear in Chapter 6 is the precise mechanism by which such buffering occurs. Interestingly, Chapter 6 saw robust activation of the subgenual ACC, which further was one of the main differences between compassion-related activations and those observed for EnGE in general (compare Figures 8.1A and C). The subgenual ACC has long been implicated in emotion regulation research, with dense connections to regions involved in core affect and physiological regulation, such as the amygdala, hypothalamus, insula and brainstem. This connectivity is thought to enable the subgenual ACC to regulate affective responses at a deep level, and importantly to support contextual or implicit modes of regulation that occur outside of explicit awareness and intention to regulate (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; M. L. Phillips, Ladouceur, & Drevets, 2008; Stein et al., 2007). The subgenual ACC, or more specifically imbalance of relative subgenual ACC and amygdala activation (Mayberg et al., 1999) is thought to be a neural substrate of severe forms of treatment-resistant depression (Mayberg et al., 2005). Evidence shows that correcting this imbalance through deep brain stimulation of the subgenual ACC results in marked betterment of symptoms (Mayberg et al., 2005), suggesting that the subgenual ACC, and by extension implicit emotion regulation in general, plays a central role in maintaining healthy hedonic balance. In line with its role in treatment-resistant depression, such implicit emotion regulation is largely thought to be trait-like and relatively hard-wired (Gyurak, Gross, &

Etkin, 2011), a notion supported by the absence of subgenual ACC activation in evidence for explicit, strategic modes of emotion regulation (Buhle et al., 2014; Dörfel et al., 2014). Thus, a potentially unique benefit of EnGE abilities is that they enable one to volitionally engage implicit emotion regulation mechanisms.

A central function of the subgenual ACC appears to be resolution of conflicts between the immediate evaluation of a stimulus and a contextualised understanding of them, be they due to stimulus properties making a task difficult (Etkin et al., 2006; Ochsner, Hughes, Robertson, Cooper, & Gabrieli, 2009a), dynamic changes in the environment (Mobbs et al., 2007), evolving experience with a stimulus (Schiller, Levy, Niv, LeDoux, & Phelps, 2008), or even internal motivational states (Nili, Goldberg, Weizman, & Dudai, 2010). The study by Nili and colleagues is perhaps most relevant for present purposes, showing that ventral cingulate is associated with the ability to overcome fear. However, much as in the current study, this effect was not driven by decreases in negative affect. Rather, overcoming appeared to involve the decoupling of the experience of negative affect from physiological indices of distress. Thus, subgenual ACC appears to support the overcoming of immediate reactions to stimuli in the service of goal-attainment. Possibly, by eliciting a positive emotional state, one in effect forces a re-evaluation of negative stressors, enabling access to a more flexible behavioural repertoire (Fredrickson & Branigan, 2005).

While speculative, this observation could provide a neural mechanism for the efficacy of EnGE-based interventions for depression (Holmes et al., 2006; 2009; 2016), suggesting that EnGE-based interventions could have analogous effects to deep-brain stimulation of subgenual ACC (Mayberg et al., 2005). It should be noted that while I here champion the idea that subgenual ACC implements specifically regulatory functions, that an alternate hypothesis is that subgenual ACC supports the generation of emotional states directly. Supporting this, Chapter 5 found that loving-kindness meditation alone, without external stressors, also activated subgenual ACC (See Figure 8.1B). As the samples of Chapters 5 and 6 were experts

in EnGE, it is possible that training, and especially training in CM and LKM, could enable one to recruit subgenual ACC in the service of emotion generation, unlike the representative samples in Chapter 4. Speaking against this, no evidence of cortical thickness increases in subgenual ACC was found in Chapter 5. Thus, deciding between these two hypotheses is ultimately a topic for future longitudinal research into the neural bases of EnGE-training.

8.3.4. EnGE as a mediator of trait affect and emotion management abilities

Having established that EnGE-based emotion management could be used for reactive emotion management in EnGE-experts, the final topic of the thesis was to establish whether this was also the case in the normal population. Specifically, it was proposed that EnGE-based emotion management could take one of two forms: 1) reactive, like that explored in Chapter 6, based on the generation of positive emotion in response to negative stressors, and 2) a proactive form, associated with the ability to predict and pre-emptively adapt to potential emotional stressors before they occur. It was reasoned that, to the degree that either of these accounts were correct, one would expect to see a relationship between EnGE-abilities and usage of reactive, emotion-focused or pro-active, instrumental emotion management techniques. Importantly, if EnGE- abilities are indeed used for emotion management in the population at large, one would expect them to mediate the known relationship between adaptive emotion management and trait affect.

Chapter 7 addressed this hypothesis by investigating the relationship between endogenous emotion generation abilities and trait affect and emotion management styles. Emotion generation abilities were operationalised as the degree to which participants reported being able to generate emotions in the RAGE paradigm (also used in Chapter 4). Generation abilities were found to be predictive of adaptive coping skills and trait affectivity in a large, representative sample. Specifically, the capacity to generate emotions in general (irrespective of valence) was associated with both trait positivity and the tendency to use problem-focused

coping skills (see Table 7.2). Moreover, the capacity to generate emotions was found to mediate the relationship between coping skills and trait affect, suggesting that instrumental emotion management is facilitated by the capacity to generate emotional states. Possibly this could be because of an increased ability to simulate potential emotional states in the future, or what was termed pre-emptive, simulation-based emotion management in Chapter 2.5.3.

However, despite the documented efficacy of reactive, buffering-based, EnGE emotion management in Chapter 6 (discussed above), limited support was found for the spontaneous occurrence of this in the normal population. If this was the case, one would expect that the ability to specifically generate positive emotional states would be predictive of mental health. While the relative ability to generate positive emotion was associated with usage of emotion-focused forms of emotion management, and increased positive trait affect, relative emotion generation skills did not mediate the relationship between emotion management and trait affect. This suggests that the capacity to generate specifically positive emotion might be an outcome of beneficial affective styles, rather than being causal in facilitating them.

In summary, in the normal population, the ability to generate emotion appears to be causal in adaptive emotional functioning by enabling individuals to adopt instrumental emotion management techniques. The fact that no clear valence effects were observed could suggest that this effect is driven in large by domain-general abilities, such as the capacity to form internal representations. This explanation is supported by the extant neuroimaging findings, showing a significant degree of overlap between the neural regions supporting EnGE and those supporting a range of mental simulation phenomena most notably in the dorsal medial subnetwork of the DMN, as discussed above. An important question for future research is to what degree individual differences in the component processes of EnGE (i.e. core affect generation, representation abilities, executive maintenance or, as discussed above, inhibition) predict emotion management styles.

8.3.5. Summary: The utility of emotion generation

Chapter 2 proposed two complementary means by which EnGE could facilitate adaptive emotion management. On the one hand, EnGE could be used in an acute manner to generate (primarily positive) emotional states, thereby buffering against and diffusing the impact of negative events. On the other, EnGE could enable one to accurately simulate future emotional events, thereby enabling one to take pre-emptive action to diffuse the impact of negative events and increase the likelihood of the occurrence of positive events. In the studies presented here, evidence was found for both these hypotheses, showing that EnGE is both an effective means of dealing with negative stressors as they occur (Chapter 6) and that the capacity to engage in EnGE enables one to engage in instrumental forms of emotion management. One caveat to these findings is that trait-level EnGE abilities in a normal population did not appear to support reactive, emotion-focused forms of emotion management. This is in line with extensive research showing that the ability to engage in positive fantasising alone appears to be maladaptive in the long run (Oettingen et al., 2016; Oettingen & Mayer, 2002), unless the thus generated positive fantasies are contrasted with the actual reality of one's situation (Oettingen et al., 2009). Interestingly, a key feature of compassion and loving-kindness meditation is that these practices not only involve generating an emotional state of warmth and caring, but also of the motivation to alleviate suffering and to enable other individuals to experience positive emotional states (Singer & Klimecki, 2014). Thus, as the sample in Chapter 6 were experts in EnGE, it is possible that engaging in buffering-forms of regulation might require some training.

8.4. Conclusion

The overarching motivation for this series of studies was to highlight the often overlooked aspect of emotions as volitional entities that we actively enlist to govern ourselves. Supporting this view, we found that individuals are readily able to generate in themselves both positive and negative emotional states, and that the implementation thereof is

highly idiosyncratic, and involves the combination of multiple sources of emotion-related information. Moreover, this ability is robustly supported by a neural architecture closely related to the generation of vivid mental simulations and the regulation of physiological states. This suggests that endogenous generation of emotion results in *de facto* embodied simulations of emotional states (Gallese & Caruana, 2016). Thus, the capacity to volitionally generate emotional states based on endogenous information appears to be a highly complex, constructive mental act, related one of the pinnacles of human cognition: the capacity to create and manipulate models of the external world in lieu of actual experience to enable adaptive behaviour. Supporting this, evidence suggests that the ability to generate emotional states is mechanistically related to the ability to predict and pre-emptively manage emotional states. However, as Descartes suggested, we also find that this ability can be used to substitute or counteract external stressors with more appetitive positive emotional states, enabling one to achieve one's goals and endure negative stressors. Such resilience does, however, appear to require some cultivation, suggesting that training is required to unlock the true self-regulatory potential of endogenous emotion generation.

Chapter 9: Limitations, implications, and future directions

9.1. Chapter overview

In this final chapter, I provide a critical view on the current results, highlighting limitations of the current findings and how future research can address these shortcomings. Based on these caveats, the implications of the current findings are discussed in terms of how they inform affective science, emotion theory, and emotion regulation research. Finally, an outlook is provided, discussing how the current findings can evolve into and inform theory in affective science and other fields of inquiry.

9.2. Limitations

9.2.1. Is naturalistic EnGE volitional?

This work was guided by an operationalisation of EnGE as an emotional reaction that comes into being based on endogenous sources of information (see Chapter 2.2). However, due to the constraints of the experimental method, the investigation was forced to limit this further by only investigating EnGE as it was volitionally implemented. While this is fundamentally in agreement with the goal of investigating how EnGE can be employed as a means of self-regulation, it leaves open the question of whether the neural model developed here reflects EnGE is ecologically valid. For instance, to the degree that EnGE is involved in instrumental forms of emotion management, it is likely that its role is not that of consciously generating emotional states like in the RAGE. Rather, it is probable that EnGE abilities play a role in colouring projections and simulations of the future (or past) in a manner that enhances their accuracy (Szpunar, 2010; S. E. Taylor & Schneider, 1989; S. E. Taylor, Pham, Rivkin, & Armor, 1998). While Chapter 7 found that volitional emotion generation abilities did predict this form of emotion management, it could be that spontaneous tendencies to generate

emotion more accurately predict this ability. Similarly, while the proposed three-network, three-process structure of EnGE appears to hold for goal-directed self-generation of emotion, it is still an open question to what degree this holds for spontaneous generation, or tasks where the generation of emotion is secondary to goal-achievement. For instance, while the current results suggest that FPCN coordinates SN and DMN in the directed generation of emotional states, this might be contingent on the goal-directed nature of the task (Spreng et al., 2010), congruent with the known role of the FPCN in implementing cognitive control (Banich et al., 2009). On the other hand, recent models suggest that cooperation of DMN and FPCN is a key feature of all internally generated mentation (Smallwood et al., 2012; Smallwood & Schooler, 2015), indicating that the three-network structure should be applicable to the spontaneous case as well. One way this question could be addressed is to investigate the neural implementation of spontaneously occurring emotional experiences during e.g. resting state scans. While traditionally thought of as a kind of blank or default mode of processing (Raichle & Snyder, 2007), recent evidence suggests that the phenomenology of being subjected to a resting state scan is characterised by vivid internally generated experiences (Delamillieure et al., 2010; Kühn, Fernyhough, Alderson-Day, & Hurlburt, 2014a), that often have affective qualities (Tusche, Smallwood, Bernhardt, & Singer, 2014). In addition to resolving the question of the generalisability of the three-network structure, comparing the neural underpinnings of spontaneous and volitional EnGE could be a useful individual differences measure to investigate the behavioural and neural bases of affective intrusive thoughts like seen in rumination (Kühn et al., 2012; Kühn, Vanderhasselt, De Raedt, & Gallinat, 2014b).

9.2.2. What is a proper baseline for EnGE

Closely related to the question of the relationship between volitional and spontaneous EnGE is the question of what constitutes a proper baseline to investigate EnGE. The current

series of studies deliberately adopted a relatively loose baseline as the kind of processing that occurs in the absence of emotion-focused processing. For instance, Chapter 5 contrasted loving-kindness meditation to a resting state where participants were instructed to refrain from meditation. Psychologically (and presumably neurally) these conditions are similar in as much as the task-free resting state has no clear emotional connotations. By contrasting active generation efforts with these purported states of neutrality one should be left with specifically the regions involved in EnGE, or so the reasoning goes. However, as mentioned above, there is no true resting state, if by resting state one means an absolute absence of brain activation. Rather, the experience of a resting state scan is rich, and highly variable between individuals (Delamillieure et al., 2010), meaning that any statistical comparison with this as a baseline will be noisy. Moreover, on a neural level, this problem is aggravated by the key role of precisely the networks thought to drive task-unrelated experiences proposed in EnGE (Smallwood et al., 2012). In addition to this causing a problem for the estimation of neural activation, this potentially has implications for the claim that the RAGE can provide an individual difference measure of EnGE abilities. For instance, in the RAGE paradigm participants were shown neutral pictures prior to the experiment and requested to adopt the emotional state these pictures elicited when requested to generate neutral emotional states. While this had the advantage of having participants actively attempt to achieve a specific level of emotionality in both neutral and generation conditions, this also meant that participants' responses in the neutral condition is also a measure of the degree to which they were able to avoid experiencing emotion. Thus, the neutral condition could be thought of as an active regulation condition, which could challenge the validity of the psychological variables investigated here. Moreover, when considering the neural activation patterns this could mean that any differences seen were driven by deactivations induced by the regulation condition. In the current case, data-driven CPCA and model based temporal decomposition analyses (Figures 4.3 and 4.4) speak against this interpretation, showing that the neutral

condition simply did not activate the relevant networks overall. Moreover, this critique is not applicable to the approach taken in Chapter 6, where the critical baseline was passive viewing of negative emotional stimuli. Despite this, there was a substantial overlap between activations seen here (mOFC/vmPFC, PCC, IFG, MFG, PMC, pre-SMA) and those reported in Chapters 4 and 5.

However, one possibility not controlled for in the present set of studies is that the EnGE effects seen could be driven by a third variable, such as the ability to generate vivid internal representations in general (Zeman et al., 2015). Future research could address this by including active baseline conditions such as future simulation, varying the emotional content of the representations systematically (Szpunar & Schacter, 2013). In addition to possibly providing a better assay of EnGE abilities, this could enable the validation of the component-process architecture here proposed by e.g. comparing neutral and emotional episodic imagery, with the assumption that DMN should be similarly activated for both, but SN only showing activation during emotional imagery (but see Benoit et al., 2014; Oosterwijk et al., 2012).

9.2.3. Qualitative characteristics of emotion states

In the current research a dimensional model of emotions was implicitly adopted by having participants report their emotional states on a sliding scale ranging from neutral to highly positive or negative. As such, participant reports were based on information that was abstracted from the concrete emotional states they generated. Possibly, better measures of EnGE abilities can be afforded by more nuanced assessment of the emotional states generated. For instance, it is possible that investigating specific emotional states could provide a better differentiation of valence than the current results (but see K. A. Lindquist et al., 2016). Moreover, better insight into the contributions and efficacies of different modalities in EnGE could be investigated by implementing trial-wise qualitative report of the emotional states as they were generated.

9.2.4. Cause and effect of emotion management, trait affect, and generation skills

Following on the preceding discussions, it should be noted that the extant results on the relationship between emotion management, trait affect, and emotion generation skills cannot rule out the existence of mediating variables such as imagery abilities, erudition, general *g*-factor of intelligence etc. Future research could investigate this by investigating how EnGE abilities correlates with individual differences in "classical" cognitive factors like working memory capacity, executive function, and general intelligence. In addition to providing a better understanding of what the constituent skills supporting EnGE are, this could provide an interesting avenue for tailoring interventions aimed at enhancing EnGE. For instance, it could be that the capacity to utilise different modalities to generate emotion is predicated on overall abilities, meaning that optimal generation training should aim at either compensating for deficient abilities or playing to one's strengths.

9.2.5. Meditation practice as a model of EnGE

Finally, the use of loving-kindness and compassion meditators as expert models for EnGE should be scrutinised. While the current results suggest that both these practices rely on largely overlapping neural mechanisms as EnGE in the normal population, the fact remains that meditation practice — especially in highly trained experts — involves intensive training of attention and other functions perhaps not directly involved in EnGE. Indeed, Chapter 5 found that the expert meditators had significant changes of brain morphology in regions involved in attentional and meta-cognitive processing that did not feature centrally in EnGE (Figure 5.3). This observation must be taken into account when drawing the conclusion that EnGE can be used for reactive forms of emotion regulation, such that this might not be a viable management approach for the untrained individual. Moreover, as Chapter 7 indicates (Figure 7.4), the ability to use EnGE to enable the sort of emotion-focused buffering enabled by compassion meditation does not appear to occur spontaneously. As discussed in the

previous chapter, one possibility is that such regulation requires directed training. Future research could address both the problem of generalising from meditation experts and test this hypothesis by investigating the neural and behavioural consequences of directed EnGE training (e.g. Holmes et al., 2006; 2009).

9.3. Implications

9.3.1. Exogenous versus endogenous emotion generation

Perhaps the most theoretically interesting aspect of the current findings is the overlap between the neural bases of endogenous emotion and that identified by studies primarily investigating exogenously generated emotional states (Kober et al., 2008; K. A. Lindquist et al., 2012 compare Figure 9.1 A and B). As discussed in Chapter 2, these meta-analyses indicate that ExGE is supported by the coordinated activation of several functionally distinct groups of regions. Importantly, these largely overlap with the neural bases of EnGE as explored in the current thesis. The notable exception to this is the lack of visual association areas (Group VI in 9.1B) in the current data, a finding that is explainable the fact that the large majority of the studies included in the Kober (2008) meta-analysis used visual emotional stimuli. Supporting this interpretation, the Kober (2008) meta-analysis shows that visual regions form a functional grouping relatively distinct from core networks centred around medial PFC, and limbic and paralimbic regions (Groups I, II, IV, V in Figure 9.2B; Figure 9.2C). This aside, findings suggest that EnGE is largely similar in neural implementation to ExGE: Groups I and II in Figure 9.2B correspond roughly to the extended Salience Network (SN), including ventral cortical SN (i.e. insula and posterior OFC, Group I) and subcortical SN including amygdala, thalamus, basal ganglia and brainstem (Group II). On the other hand, groups IV and V together correspond roughly to the posterior and anterior

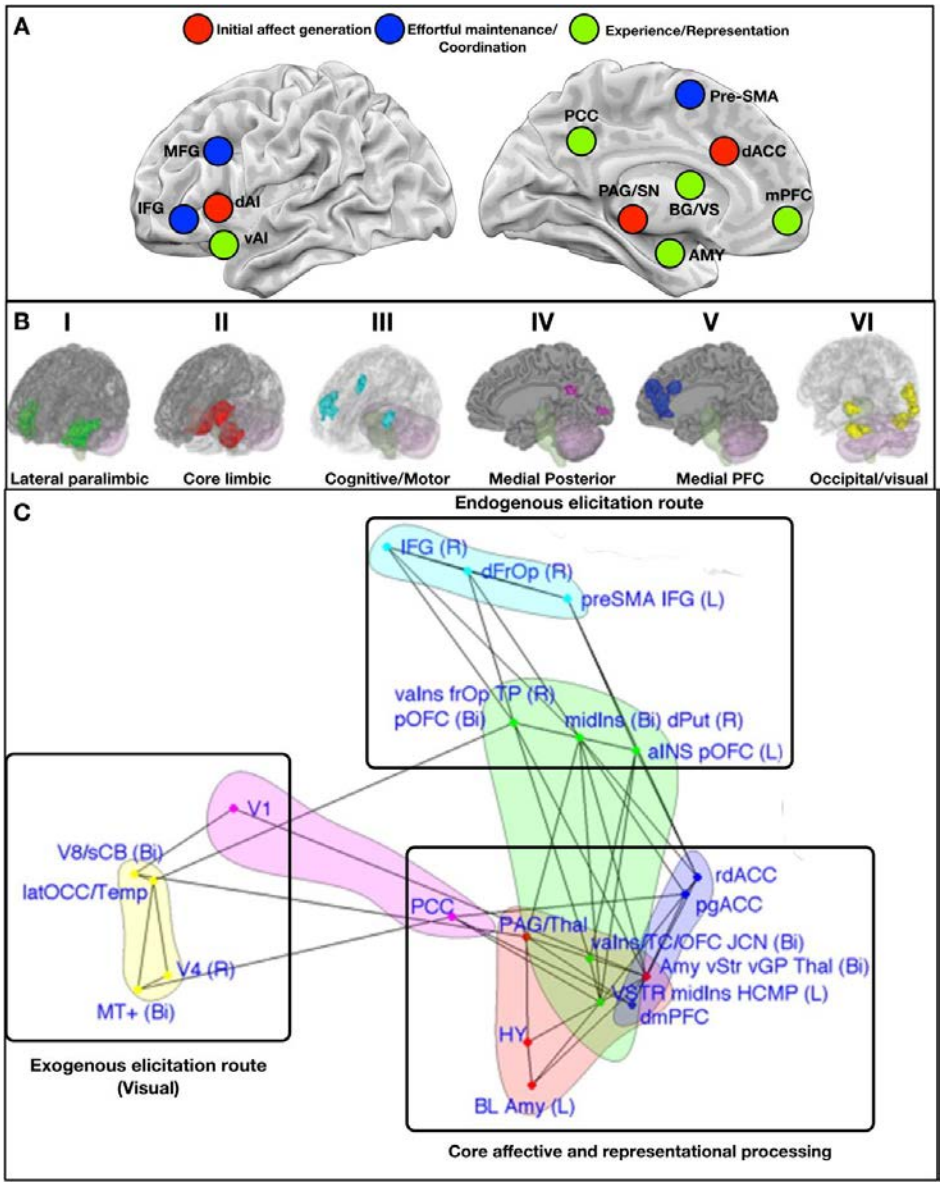


Figure 9.1: Comparison of current findings with previous meta-analysis. A) Synthesis of the current findings, with the suggested component process mapping of networks denoted. **B)** Functional neural components of emotion generation identified in previous meta-analyses. **C)** The functional connectivity space of these components. Shadings correspond to component colouring in B) and line length is inversely related to connectivity strength. Boxes denote three proposed functional domains of these groupings based on current results. Panels B and C adapted with permission from Kober et al. (2008).

portions of the DMN. This could suggest that there are two distinct neural routes of emotion generation: A bottom-up route requiring modal sensory association systems (e.g. group VI in Figure 9.1B) and a top-down route focused on cognitive control systems (i.e. group III in Figure 9.1 B; see Ochsner et al., 2009b for a similar distinction). Interestingly, strong evidence was found that a set of regions (left lateral PFC and pre-SMA) played a central role in enabling the effortful maintenance of EnGE (Figure 4.3), overlapping with the fronto-lateral portion of the FPCN. This network is closely related to executive processing and cognitive control, suggesting it could be involved in setting large-scale neural configurations to facilitate internal or external mentation (Andrews-Hanna et al., 2014). Supporting this interpretation, these regions were involved in a pathway mediating usage of multiple modalities (Figure 4.6), in addition to being coupled to both DMN and SN to form functional networks whose activation predicted generation success (Figure 4.4). Moreover, this set of regions closely resembles the "cognitive-motor" group identified in the Kober meta-analysis (Group II in Figure 9.1B). Consistent with the present findings, co-activation analyses indicate that this group is closely coupled to the lateral paralimbic network (cyan and green fields in Figure 9.1C). Thus, it appears that top-down generation of emotion crucially involves left lateral and medial PFC (see Ochsner et al., 2009b for similar findings).

The findings presented in the current thesis also provide information about what the psychological functions supported by these groupings are. For instance, Chapter 4 showed that the SN formed two distinct functional components during EnGE: Cortical SN (dorsal AI, dACC) together with PAG and SN/VTA both uniquely activated early in the EnGE process (Figure 4.3A) and showed a parametric relationship with experienced positive and negative affect. Thus, these regions appear to support the generation of the core affective properties of endogenous emotional states. Conversely, core limbic structures, most notably the nucleus accumbens (NACC) and amygdala (AMY), were in the current data found to be associated

with the extended elaboration of emotional states, and to co-activate with the DMN (Figure 4.3C), consistent with them primarily supporting the elaboration of core affective sensations into representations of emotional experiences. One intriguing possibility hinted at by these results is that their involvement might reflect the degree to which the generation process involves focus on the bodily feeling aspects of emotion (Figure 4.6D). This is interesting because the limbic system is traditionally defined as including the hippocampus (Papez, 1937), a structure known to be heavily implicated in mnemonic processes, particularly in the context of episodic memory (Martinelli et al., 2013) and navigation (Buzsáki & Moser, 2013), a process that centrally involves creating simulations of the world. Moreover, both NACC (Ito et al., 2008) and AMY (LaLumiere et al., 2005) are known to be involved in emotional memory processes and to be strongly coupled to the hippocampus. Thus, the reactivation seen here might reflect their involvement in the process of recalling emotional memories in an embodied fashion. Interestingly, while the overall engagement of structures in the data presented here overlaps with the Kober study, the precise patterns of co-activation differ, such that brainstem systems (in the Kober (2008) study assigned to the core limbic group; II in Figure 9.1B) were found to co-activate with the lateral paralimbic group, which here included dorsal ACC. This could suggest that the coupling of brainstem to either limbic or to lateral paralimbic systems could be an essential configural difference between ExGE and EnGE.

Summarising, it appears that exogenous and endogenous emotions are highly similar in terms of neural implementation. Moreover, they suggest that ExGE in large part relies on endogenous sources of information. Thus, these findings strongly support a constructivist model of emotion (Barrett, 2014; K. A. Lindquist & Barrett, 2012), in which the occurrence of emotional states depend on the interplay of events as they occur in the world and the subsequent interpretation of them as emotionally meaningful (J. J. Gross & Barrett, 2011; K. A. Lindquist & Barrett, 2012). Critically, such interpretation requires the integration of current sensory inputs with previous experiences. Moreover, this process is highly inferential,

with the actual sensory datum only providing the initial impetus for the generation process. As such, one would on this theory expect a large degree of overlap between the processing of endogenous and exogenous emotions.

9.3.2. The neural bases of endogenous positive and negative emotion

Another interesting observation made in the current research is the large degree of overlap between the neural bases of positive and negative emotion (Figure 4.5). While initially surprising, given the strong subjective differences between negative and positive emotional states, this finding is largely in agreement with previous meta-analyses (K. A. Lindquist et al., 2016). One notable discrepancy with previous work is that little evidence was found for the involvement of the ventromedial prefrontal cortex in the generation of emotional valences: Meta-analyses have revealed that this region appears to track with experienced affect (K. A. Lindquist et al., 2016). Moreover, it has convincingly been argued that the activation patterns and anatomical connectivity of this region makes it ideally suited to support the generation of affective meaning when such meaning is based on conceptual, or endogenous sources of information (Roy et al., 2012). As all current experiments involved such endogenous generation, one would have expected to see some relationship between activation of this region and the valence of experienced emotion, something not apparent in Chapter 4 (see Figure 4.5). One possible explanation for this discrepancy is that the current set of experiments did not require the integration of twin streams of information (sensory vs. conceptual), but rather the top-down entrainment of processing to facilitate EnGE. Supporting this, the strongest evidence for differentiation of positive and negative emotion was observed in evolutionarily old systems in the brainstem involved in core somatic regulation. Thus, it appears that the process of EnGE involves utilising top-down influences to generate a bottom-up signal at low levels of processing, which is subsequently elaborated into emotional experiences, a process which is supported by DMN (of which the VMPFC is part) and ventral

SN. It is interesting to note that an important distinction between EnGE and ExGE is that the latter necessarily requires some form of interpretation as sensory information is always ambiguous and context dependent (Barrett & Bar, 2009). Conversely, endogenously generated states are, by necessity, not subject to such uncertainty, especially in the context of goal-directed generation like that investigated here. Evidence suggests an involvement of the VMPFC in uncertainty processing, specifically relating to subjective confidence in one's decisions (White, N. H. Engen, Sørensen, Overgaard, & Shergill, 2014). Thus, the data presented here suggests that endogenous emotion is centrally differentiable into positive and negative qualities at early levels of processing. However, it is unlikely that this is unique to endogenous emotion, suggesting that future research aimed at differentiating positive and negative emotion should attempt to do so by investigating relatively basic processing, related to fundamental approach and withdrawal behaviours.

9.3.3. The relationship of emotion generation and regulation

The results presented here also provide an interesting insight into the relationship between emotion generation and emotion regulation, a topic of some debate in current affective science (J. J. Gross et al., 2011; J. J. Gross & Barrett, 2011). Chapter 7 investigated the neural and behavioural effects of emotion-regulation using compassion meditation (CM), and compared this with reappraisal (RE) – perhaps the most investigated "classical" cognitive emotion regulation strategy (Buhle et al., 2014). Evidence strongly suggested a clear differentiation of the two: Behaviourally, CM lead primarily to increases in positive affect and only secondarily decreases in experienced negative affect. Conversely, RE primarily decreased negative affect and only increased positive affect to a level comparable to being exposed to neutral emotional stimuli (Figure 7.1). Neurally, this difference was equally pronounced, with CM showing stronger activation in activating classical emotion regions, including midline PFC, the basal ganglia, and insular regions. RE, on the other hand, showed

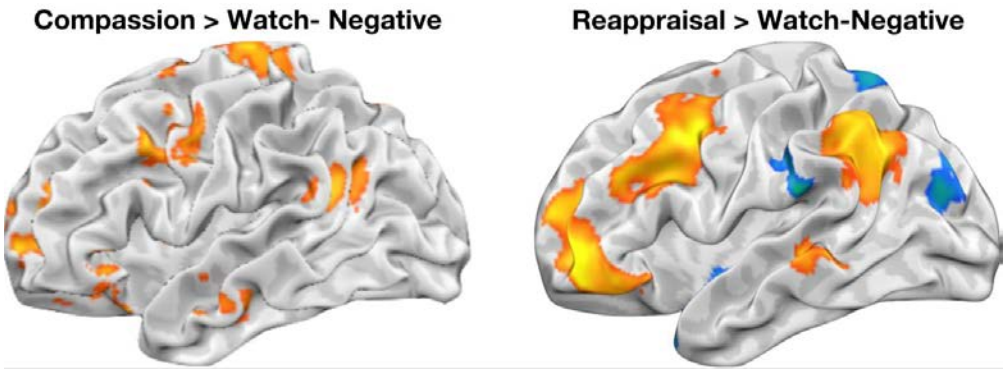


Figure 9.2: Shared circuits of Compassion and Reappraisal. Results from Chapter 6, showing the contrast of Compassion and Reappraisal versus the passive-viewing baseline. Note the overlap of activations in dorsal PFC and TPJ.

stronger activation of the lateral PFC and parietal regions known to be associated with cognitive control and inhibitory processes. These results, then, suggest that there is a clear distinction between the generation and regulation of emotion, both in terms of behavioural consequences and neural activation patterns. However, when comparing both CM and RE to passively viewing negative stimuli, marked degree of neural overlap was observed, most notably in left ventral and dorsal lateral PFC (Figure 9.2). Thus, this suggests a degree of similarity between, at least on a neural level.

Interestingly, recent evidence suggests that the left lateralised PFC activation might be unique to reappraisal when compared to other emotion regulation strategies (Dörfel et al., 2014). Notably, RE involves inventing alternate constraints to one's interpretations of stimuli (Barrett, Ochsner, & Gross, 2007b). As such, RE has a clear generative aspect that sets it aside from other forms of emotion regulation, like distraction, distancing or expressive suppression, that to varying degrees primarily involve neutralising one's emotional reactions and attaining some form of equanimity. Supporting this notion, extant evidence suggests that reappraisal efficacy is primarily supported by the ability to effortfully generate cognitive contents (Gyurak, Goodkind, Kramer, Miller, & Levenson, 2012). Thus, the results in Chapter 7 could point at a neural (and psychological) basis for distinguishing between the generation

and regulation of emotion, with the critical distinction being the occurrence of active efforts to elicit in oneself emotional contents, contrasted with efforts to achieve states of equanimity.

While this fits the working operationalisation proposed in Chapter 2 of the difference between EnGE and emotion regulation, one important caveat is that the current evidence only pertains to the voluntary generation of emotion. This is clearly a boundary case, as the majority of emotion generation and regulation is implicit (Gyurak et al., 2011), happening without conscious effort. As such, it is an empirical question whether this distinction holds for the neural implementation of implicit regulatory efforts. A larger problem, however, is that without reference to an eliciting event and conscious goal to modulate emotion the distinction is also blurred on a theoretical level: As pointed out by Gross and Barrett (2011), drawing a distinction between generation and regulation relies on one's admission of the existence of clear emotion-eliciting events. In the case of implicit, or spontaneous, regulation and generation of emotion specifying such events are difficult since any event resulting in the ostensive generation of emotion, can equally be seen as an act of regulating one's previous emotional state to something more appropriate for one's ongoing behaviours. To my knowledge, there is no obvious way of resolving the issue aside from the somewhat unsatisfactory, and ultimately pragmatic, operationalisation adopted in the present work.

9.3.4. The utility of emotion generation as a means of emotion management

Further blurring the issue of distinguishing emotion generation from emotion regulation, the current work also indicates that, while neurally distinct from inhibitory modes of emotion regulation, emotion generation can in fact be used to counteract exogenous stressors. Traditional accounts of coping and emotion regulation tend to pivot on notions of adaptive regulation as a means of diffusing the emotional impact of external stressors (J. J. Gross, 2007b; Lazarus & Folkman, 1984). Central to this diffusion process is, in some way or another, denying the emotional reality of the stressor. Take a situation like the experiment

used in Chapter 6, where participants were confronted with film depictions of individuals clearly suffering, evoking a negative emotional reaction known as empathic distress (Singer & Klimecki, 2014). To implement successful reappraisal of such stimuli it is necessary to somehow question the depicted situation, either in terms of the seriousness of the consequences of the issue at hand or, alternately, whether the situation is real and not just staged for camera. Similarly, most other strategies described in the literature rely on some sort of denial of a given aspect of a situation, involving either one's own engagement to the situation (as in the techniques of distancing and perspective taking), or whether the situation is worth paying attention to (as in distraction). Thus, these strategies essentially involve the transformation of aspects of one's relationship to the emotional stimuli. While such transformative strategies are effective in many situations where emotional reactions somehow interfere with e.g. goal-achievement or immediate well-being (Webb et al., 2012), there are situations where such cognitive transformation might be less effective, or even counterproductive.

Consider, for instance, a situation where one encounters such suffering in real life and, instead of being a passive observer, one has the responsibility to provide care for this person, as is the case for health professionals and caretakers. In such situations, transformative strategies that emphasise down-regulating one's negative reactions can be inimical to one's goal or obligation of providing care, at least to the degree that such care requires being attuned to the actual causes and experiences of suffering. Indeed, recent evidence suggests that reappraising the suffering of others could have an increased tendency towards callousness as a side-effect (Cameron & Payne, 2012). Thus, these strategies appear unsuited to dealing with negative emotions in contexts such as healthcare, where retaining an empathic link with patients is essential to providing quality care. However, these situations still require a degree of emotion regulation on the hand of the healthcare provider: Constant exposure to the suffering of others, often without positive outcomes, is a substantial stress factor, which could

contribute to the high rates of burnout in both lower and higher status members of such professions (Adriaenssens et al., 2014; Dyrbye et al., 2014; Gundersen, 2001).

Using EnGE to regulate one's emotion offers a mean to circumvent several of the issues with inhibition-based emotion regulation. For one, neural and behavioural data indicate that emotion generation does not require the inhibition of ongoing emotional states, but can be used to elicit mixed emotional states including both positive and negative elements, as is the case with the compassion meditation technique investigated in Chapter 6. Possibly, this positive emotional state could counter empathic distress reactions, preventing them from becoming overwhelming (Singer & Klimecki, 2014). Outside of chronic exposure to negative events, it could facilitate long term resilience and coping by enabling one to remain in a positive emotional state, in effect allowing one to endure the impact of stressors by providing a buffer against persistent negative emotion. Importantly, while the work presented here suggests that such buffering can be a highly effective means of emotion regulation, it also suggests that some degree of training is required to enable this. Future intervention studies could investigate this, promising to not only deepen the understanding of EnGE as a phenomenon, but also potentially offer an avenue by which to increase resilience and coping in both normal (Fredrickson, 2013; Tugade & Fredrickson, 2004) and pathological (Holmes et al., 2006; 2009) populations.

9.3.5. Designing interventions to train EnGE

The work presented here also provides important pointers on how to optimise such interventions. First, current evidence suggests that capacity to generate emotions in general is a better predictor of beneficial emotion management than the capacity to generate specifically positive emotion. This is in line with recent theoretical developments arguing for the importance of fluency with negative emotions in enabling psychological and affective flexibility (Kashdan & Rottenberg, 2010). One possible mechanism for this is that the

capacity to generate emotional states enables better emotion differentiation or granularity, thereby giving more leverage for regulatory efforts (Kashdan et al., 2015). Thus, training programs should aim to improve the capacity to generate both positive and negative emotion. As the current work suggests, such training should emphasise multiple modalities, yet centre around training the capacity to generate episodic mental imagery. One source of inspiration for such trainings could be the meditation practices of loving-kindness and compassion meditation, as these centrally involve the generation of mental imagery (e.g. anchored on thinking about a loved one), that is supplemented by focus on bodily sensations of warmth and caring and verbal mantras (Goetz et al., 2010). As such, these practices are essentially multimodal EnGE practices around which there exists an extensive tradition of pedagogy (Singer et al., 2016). However, as these practices are focused on training the capacity to generate a specific form of positive emotion (i.e. loving-kindness and compassion), some modification might be needed to enhance emotional fluency in general (Kashdan et al., 2015; Kashdan & Rottenberg, 2010). However, a key consideration in such an intervention would be to maximise adherence to the training regime in order to enable generalisation of such skills to daily life (H. G. Engen & Kanske, 2013). Thus, focusing on the (arguably more appetitive) training of positive emotion generation might be an overall more effective protocol. Ultimately, this is an empirical question and future research should investigate the benefits of both generalised fluency-based interventions and specifically positive generation training.

9.4. Future directions

9.4.1. Neural signatures of emotion

One promising avenue for future research into EnGE is utilising machine learning methods to identify the signatures of different affective phenomena. Recently, Chang et al. (2015) presented a signature of neural activation patterns to picture-based emotional

reactions. Moreover, this pattern was clearly distinguishable from activation patterns associated with pain processing (Wager et al., 2013), suggesting that this pattern specifically represented processing of emotional information. Interestingly, this pattern included several regions overlapping with the current work, including amygdala, periaqueductal grey (PAG), dorsal AI, dorsomedial prefrontal cortex, pre-SMA, ventromedial temporal lobe, and posterior cingulate cortex. However, it also included ventral occipital cortex, most likely reflecting visual processing. As such, it is unclear whether the contributions of these different systems are key to the generation of emotional states or, for instance, interpretation of stimuli. By comparing endogenously and exogenously elicited emotion it would be possible to clarify this, and differentiate the neural systems involved in EnGE and ExGE. This comparison would allow one to test the hypothesis proposed above of two routes of emotion generation. Moreover, by investigating similarities between the two, it would be possible to identify the neural core of emotion generation in general, a topic of high importance for affective science.

9.4.2. Towards an integrated model of emotion control

Taking this approach would also allow one to deepen the understanding of the generation/regulation distinction. Based on sensitive and specific signatures of emotion generation, it would then be possible to differentiate neural mechanisms of emotion generation from active efforts to regulate them by establishing discrete, predictive signatures of emotion regulation. Thus it would be possible to extend this line of research into developing an integrated neural model of emotion control. On a theoretical level, this could provide a unified approach to understanding the relationship between different modes or techniques for emotion management (Webb et al., 2012). Importantly, this could provide an understanding of the mechanistic foundations of emotion management in general, differentiating neural networks supporting domain-general goal-directed regulatory efforts (J.

J. Gross, 2015a), from those supporting the specific implementation of those efforts (McRae, Ciesielski, & Gross, 2012a).

9.4.3. Pathological endogenous emotion generation

Furthermore, as machine learning models are essentially predictive, establishing and validating such signatures would allow one to move beyond theory-crafting and enhance our understanding of the neural mechanisms of pathology. This is of some importance because current diagnostic systems are relatively agnostic regarding the underlying mechanisms of affective disorders, a problem compounded by the substantial heterogeneity in symptomatology (Lieblich et al., 2015; Nitschke & Heller, 2005; X. Wang, Zhang, Chaim, Zanetti, & Davatzikos, 2015) and underlying genetics (Ripke et al., 2012). Importantly, at the heart of most, if not all, affective disorders are disturbances in the generation and regulation of emotion (D. A. Clark & Beck, 2010; McLaughlin, Mennin, & Farach, 2007). By investigating how such signatures differ in populations with affective disorders it would be possible to establish the relative ætiological role of differences in neural mechanisms supporting emotion generation and regulation mechanisms. Moreover, such investigations could form the basis for the development of brain-based classification and diagnoses systems that are both mechanistically informed and capable of accounting for heterogeneity of these disorders. This could allow understanding the underlying neural mechanisms of such disorders, on both a population and an individual level. Thus, signature-based diagnostics could potentially enable tailoring of interventions, possibly allowing enhanced treatment of some of the most debilitating and socio-economically costly mental disorders in existence.

9.4.4. Training emotion generation to regulate emotion

Finally, such a mechanistic understanding could further the understanding of how to formulate appropriate interventions to alleviate these disorders. Critically, the findings described here indicate that such interventions could benefit from teaching skills at both

endogenously generating emotional states, as well how to regulate or manage negative stressors. Thus, interventions could fruitfully be devised aimed at increasing general affective flexibility (Kashdan et al., 2015; Kashdan & Rottenberg, 2010) and the ability to actively use emotions in the service of goal-achievement.

9.5. Concluding remarks

It is common to think of emotions as things that happen to us, as essentially external influences perturbing our thoughts (Nussbaum, 2008). However, as the current data indicates, emotions also have volitional aspects, such that we can, if we are so inclined, instil in ourselves vivid and real emotional states. This ability to harness the fire of our passions can be used both to counter the emotional vagaries of the external world and to predict our emotional reactions to future, unlived events. As such, the capacity to endogenously generate emotional states appears to be an oft overlooked, but highly important, part of our self-regulatory toolkit.

Part IV: Appendix

A1. Supplemental methods

A1.1. Supplemental methods: Chapter 4

A1.1.1. ReSource eligibility criteria

Eligibility to participate in the study was determined using a comprehensive screening procedure, culminating in full SCID-I and II interviews performed by trained clinical psychologists, ensuring no ongoing mental health issues, and no life-time occurrence of psychotic disorders or schizophrenia, or any Axis-II disorders. Furthermore, participants were screened for alexithymia as measured by the Toronto Alexithymia Scale (Kupfer, Brosig, & Brähler, 2000 cutoff 60), and trait anxiety as measured by the State Trait Anxiety Inventory (Laux, L., Glanzmann, P.S., Schaffner, P., & Spielberger, C.D., 1981 cutoff 56).

A1.1.2. Training procedure

A1.1.2.1. Experiment 1

Before scanning, participants underwent a supervised automated training session. In order to ensure that participants had a homogenous representation of the affective states they were requested to generate, they first underwent a multimodal affect induction procedure combining emotional music (adopted from the norms in Eerola & Vuoskoski, 2010) and affectively charged pictures, preceded by verbal descriptions of the bodily and affective sensations associated with these states. The inductions aimed at inducing states of sadness, happiness or neutrality in the participants. Each emotional state was induced twice. The participants were then instructed to identify what means they themselves thought would be the most efficient at eliciting similar emotional states in themselves. No constraints were

given as to how they should do this, as long as it was possible to do so while in the scanner environment, avoiding overt movement.

Participants then underwent three Generation trials identical to the experimental procedure generating both positive, negative emotional states, as well as the neutral baseline state. Subjects were then asked to identify means by which they could regulate the emotional states they generated, so as to attain a state of neutrality, and performed seven training trials corresponding to the full set of conditions (i.e. 3 (Modulation; Maintain, Cease, Regulate) X 2 (Emotion; Positive, Negative) + 1(Baseline; Neutral).

A1.1.2.2. Experiment 2

In Experiment 2, participants underwent the same training procedure as Experiment 1, with the exception that they were given the option to generate either high or low arousal exemplars of positive (e.g. joy vs. tenderness) and negative (e.g. fear vs. sadness) emotion in order to enhance ecological validity, and that they were constrained in their use of strategies based on feedback from the participants from Experiment 1 and theoretical considerations. Two key alterations of the training procedure was thus performed. First, the emotion induction procedure was changed by including both high and low arousal inductions (with no repetitions). Second, rather than being given free choice to select strategies, following the induction phase, immediately before generation training, participants were presented with a list of four emotion generation technique (Verbal, Visual, Auditory, Bodily) with short descriptions detailing how to implement each technique and descriptions of each technique (see *Pre-Scan Training Instructions* below for details). Participants were then given the option of which arousal level to generate (High/Low) for each emotion, and asked to select one or more techniques to employ.

Participants then underwent three Generation trials identical to the experimental procedure (see *fMRI experimental procedure* and Figure 1) generating both positive and negative

emotional states, as well as the neutral baseline state. After this, subjects were given the option to change their initial selection of strategies and arousal levels, in case they discovered the unsuitability of employed strategy and/or arousal level during training.

Finally, subjects were presented a list of six emotion regulation techniques and their descriptions, and again asked to select one or more of these techniques to employ to down-regulate their self-generated emotions according their own preference. The regulation condition and the strategies employed are the focus of a future study. They then trained regulating two Regulation trials (see Figure 6.1), in which they both generated and down-regulated positive and negative affect. Participants were instructed to use only the techniques they chose during the experiment, and to employ them in equal measure to generate and regulate both positive and negative affect.

A1.1.3. Psychophysiological data acquisition and preprocessing

In Experiment 2 only, skin conductance responses were recorded during scanning using a BrainVision setup (BrainAmp ExG MR; Brain Products, Munich, Germany). Ag/AgCl electrodes with applied isotonic electrode paste as a conductance medium were attached to the index and middle finger of the left hand for each subject. The acquired data was filtered using a Butterworth 0.15 Hz low-pass filter of order 3 using custom MATLAB scripts. Overall time-courses were then visually inspected, excluding data-sets where overall data quality was noisy, showing evidence of scanner artefacts after filtering, or lack of variance for the entire time series. Of the 293 data sets passing the fMRI preprocessing quality control, 225 also passed these criteria, forming the subset used for our psychophysiological analyses. The remaining data was analysed using the Ledalab toolbox (www.ledalab.de). After downsampling the data to 250 Hz, continuous decomposition analysis (Benedek & Kaernbach, 2010a) was performed to identify task-related modulations of skin conductance levels.

A1.1.4. Generation strategy descriptions

In the following the description of the different generation modalities that were presented to the participants are reproduced, translated from the original German. Presentation order of the modalities were randomised in the actual experiment.

Verbal[8]: Internal monologue. E.g. telling yourself things that you believe will make you feel different things. Instead of statements you can also tell yourself short stories. The important thing is that you use verbal abilities.

Visual[9]: Using visual imagery to imagine real or hypothetical situations or events that are emotional. Importantly, this technique is associated with a visual experience and not just e.g. retelling a story like in the Verbal).

Auditory[10]: As Visual, but with a focus on sounds, such as voices, music or other emotionally evocative sounds. Again, the important thing is that emotions should be elicited through auditory means.

Bodily[11]: Creating and/or amplifying bodily manifestations of emotions, such as breathing, facial expressions, muscular tension, heart rate, thereby changing one's emotional experience.

Other: If you will be using a technique (either alone, or in combination with the others) that is not similar to any of the above, please indicate this by selecting E. You will be asked to describe this in writing.

[8] Corresponds to the *Semantic Analysis* modality
[9] Corresponds to the *Episodic Imagery* modality
[10] Corresponds to the *Auditory Imagery* modality
[11] Corresponds to the *Bodily Interoception* modality

A1.1.5. Assessing the effect of fatigue on emotion generation

In our SCL analyses we included trial number in order to control for potential fatigue or habituation effects in the relationship between ratings and SCL reactivity. As we wanted to ensure that our analyses were not confounded with these effects, we performed additional analyses investigating the effect of fatigue on the current results, using a similar trial-wise analysis with random intercepts as in the main analyses.

As expected given the effortful nature of the task, we found effects of time-on-task on reported ratings [$F(11017.96)=34.099$, $p<.001$], that took the form of less extreme ratings with time for both Positive [$t(4272.97) = -12.86$; $p<.001$] and Negative [$t(4272.97) = -4.40$, $p<.001$] conditions. This was also the case for SCL levels [$F(11012.142) = 474.05$, $p<.001$], with the effects manifesting as lower SCL with time for both positive [$t(4271.97) = -11.80$, $p<.001$], negative [$t(4271.85) = -12.39$, $p<.001$] and neutral [$t(2022.41) = -13.54$, $p<.001$] conditions. Thus, these results suggest that efficacy of emotion generation diminishes with time on task, in terms of both subjective and physiological indices of emotion. This is consistent with a general fatigue/habituation effect. In an extended model, we investigated whether this effect was moderated by modality usage, and found no interaction between time on task and reported modality usage. Thus, it appears that all modalities were equally fatiguing.

Next, we investigated what the effect of fatigue/habituation on neural activation patterns in Experiment 2, by including a centered condition-wise parametric modulator coding the trial number in the first level models. Overall, this regressor had little effect on the main findings. Investigating the neural activation patterns associated with time on task revealed increased activation of dorsal attention regions with time, and correspondingly decreased activation of DMN, SN, and FPCN (see Figure S3). Notably, this decreased activation was observable in the Generation condition and not the Neutral condition, suggesting a selective task-dependent decrease of activation in largely overlapping regions to those found in the

main analyses. Importantly, we did not find any differential effects of time-on-task for modalities, nor did we find any strong differential effects of time-on-task in either Generation or Modulation phases, with the exception of a stronger effect of time on task on DMN (mOFC and PCC) in the generation part of the trials. Importantly, while the Neutral baseline condition showed similar increases of activation with time as the active conditions, corresponding deactivation patterns were not observed. Thus, in line with the behavioral and psychophysiological results, it appears that time on task decreased the activation of emotion generation networks in a condition-specific manner. However, as the activation and deactivation patterns were as would be expected based on the main results, main analyses omitted the temporal regressor, to avoid interpretation issues stemming from serial orthogonalisation of multiple parametric modulators, or alternately, the interpretation of the main rating parametric modulator in a model including a highly correlated regressor coding time.

A1.2. Supplemental methods: Chapter 6

A1.2.1: ROI selection procedure

While often associated with positive and negative affect respectively, the VS/NAC and amygdala are known to be functionally heterogeneous. To ensure that the time courses as closely as possible reflected subjective affect we used a three-step procedure ensuring the specificity of our ROIs to subjective affect: First, the NACC/VS ROI was defined anatomically using the Oxford-GSK-Imanova Structural Striatal Atlas (Tziortzi et al., 2011), encompassing NACC and its junction with medial caudate and rostro-ventral putamen, while the amygdala ROI was defined using the amygdala template from the Anatomy toolbox. We then identified voxels within these regions where BOLD amplitude parametrically varied as a function of subjective affect across all conditions at a threshold of $p < .005$ uncorrected. This step ensured that our ROIs did not reflect condition specific mechanisms associated with e.g.

the generation of positive and negative affect, but rather general activity correlated with subjective affect. This procedure allowed us to identify three regions associated with negative and positive affect. Negative affect subjective affect ratings were found to be correlated with activity in the left (peak MNI_{xyz} = -18 0 -20) and right (peak MNI_{xyz} = 21 -6 -16) central amygdala, while positive ratings were correlated with a region in the left VS/NAC (MNI_{xyz} = -10 18 -4). Using the MarsBaR toolbox, median, condition and run specific time courses were extracted from a 5 mm sphere centred on the peak voxel of each region, corrected for serial autocorrelation using an AR(1) process, converted to percent signal change, and submitted to separate analyses using LMM modelling.

A1.3. Supplemental Methods: Chapter 7

A1.3.1. Training procedure

Before scanning, participants underwent a supervised, automated training procedure. In order to ensure that participants had a homogenous representation of the affective states they were requested to generate, they first underwent a multimodal affect induction procedure combining emotional music, affectively charged pictures and verbal descriptions of bodily and psychological sensations associated with the different emotion states, ensuring that participants were not biased towards any particular information modality. Inductions aimed at inducing positive and negative emotional states of both high (e.g. happiness and fear) and low (e.g tenderness and sadness) arousal as well as give subjects an example of a neutral state by presenting them with emotionally neutral visual stimuli (neutral induction).

Participants were then presented with a list of four emotion generation modalities with short descriptions. Briefly, *semantic analysis* was specified as a form of non-imagery based inner monologue, *episodic imagery* as imagining hypothetical or real situations or events of an emotional nature, *auditory imagery* as imagining emotionally evocative sounds, like music or the timbre of voices, while *bodily sensations* was specified as generating or amplifying

bodily sensations associated with emotional states. The order of descriptions was counterbalanced. Participants were also given the option of using techniques or modalities that were not specified and asked to describe this in writing. Participants then chose which arousal level to generate (High/Low) separately for positive and negative emotion, and asked to select one or more techniques to employ. Three Generation trials identical to the experimental procedure followed in which participants generated positive, negative and neutral emotional states. After this, they were given the option to revise their initial selection of strategies in case they discovered the unsuitability of employed strategy and/or arousal level during training.

A1.3.2. Assessment of trait affectivity

As part of the *ReSource* study, participants completed a comprehensive package of questionnaires. To generate a nuanced measure of trait affectivity we combined scales that where participants judge their own positive and negative affect. In the following the scales used and the rational for their inclusion will be provided.

A1.3.2.1. Positive and Negative Affect Schedule

The PANAS (PANAS; Krohne et al., 1996) is a very frequently used questionnaire that measures the tendency to experience positive and negative forms of affect. Importantly, studies indicate that the measure is stable over time (Watson, Clark, & Tellegen, 1988), suggesting that it measures differences in emotional traits. Moreover, positive and negative affectivity as measured by the PANAS are largely independent constructs.

A1.3.2.2. The State-Trait Anxiety inventory

Another very frequently used measure, the STAI (STAI; Laux et al., 1981) indicates to what degree an individual endorses feeling anxiety (i.e. a high-arousal negative emotional

state) either in the moment (state) or on a regular basis (trait version). Here we focused on the trait measure of anxiety.

A1.3.2.3. Beck's Depression Inventory II

The BDI-II (BDI-II; Hautzinger et al., 2009) is perhaps the most frequently used measure of depressive symptoms (i.e. a low-arousal negative state), both in clinical and research settings. While the scale provided measures of both somatic and affective symptoms, we here combine these.

A1.3.2.4. The Types of Positive Affect Scale

TTPAS (TTPAS; P. Gilbert et al., 2008) is a relatively new scale that measures positive affect in a nuanced way, providing measures of high (active) and low (relaxation, compassionate warmth) arousal emotional states characteristic to different individuals that are typically not captured by traditional valence-focused measurements.

A1.3.2.5. NEO-Five Factor Inventory: Positive and Negative subscales

The NEO-FFI (NEO-FFI; Borkenau & Ostendorf, 2008) is perhaps the predominant measure of personality traits used in research. In addition to providing overall measurement of broad personality traits, the FFI includes a number of facets measuring specific personality characteristics (McCrae & Costa, 1992). Here we opted to focus on the subscales measuring the tendency to experience positive and negative affect rather than the main parent factors of extraversion and neuroticism. We elected to focus on subscales because the extraversion and neuroticism measure a wide range of behaviours not necessarily affective in nature.

A1.3.2.6. Adult Temperament Questionnaire

Finally, we included the negative affect and extraversion subscales from the ATQ (ATQ; Wiltink et al., 2006). Emotional reactivity is a defining feature of temperament and can be thought of as being the physiological base of personality. The ATQ provides measures of the tendency to react to positive and negative emotion, including both high and low arousal exemplars.

A1.3.2.7.. PCA procedure: Trait affect

To get a measure of trait affectivity we combined these scales using principal components analysis (PCA). Scree plotting (see *Appendix*: Figure A2.3.1) indicated that these scales could be combined into a single component explaining 40.08% of the variance in reports. Scales measuring positive affect (PANAS Positive, the three TTPAS subscales, the Positive affect subscale of the NEO-FFI and the extraversion subscale of the ATQ) loaded negatively on this component, while the remainder scales measuring negative affect loaded positively (see *Appendix*: Figure A2.3.1). For subsequent analyses, individual loadings on this component served as our measure of trait affectivity. For ease of interpretation loadings were inverted, such that positive loadings corresponded to relative tendency to experience positive emotion and *vice versa*.

A1.3.3. Assessment of coping styles

In addition to trait affect, we sought to measure individual differences in emotion management styles. To this end, we focused on scales measuring the tendency to use different coping and emotion regulation strategies, as well as general measures of self-regulation tendencies.

A1.3.3.1. Brief COPE

The brief COPE (Knoll et al., 2005) inventory measures the degree to which an individual engages in different behaviours to cope with emotional stressors, and includes both adaptive and counterproductive coping strategies (e.g. self-blame or drug-use). Extensively used, the brief COPE has been shown to be associated with mental fortitude in the face of numerous challenges ranging from health issues to natural disasters. As such, the brief COPE provides a nuanced measure of the individual coping styles in applied settings. In the current study, all scales were included with the exception of the subscale measuring the tendency to find comfort in spiritual or religious practices, on account of the poor communality (< .2) of this subscale.

A1.3.3.2. Cognitive Emotion Regulation Questionnaire

Like the COPE, the CERQ (CERQ; Loch et al., 2011) measures the tendency to use different strategies in the face of negative emotional stressors. However, as the name suggests, it focuses on cognitive approaches to emotion management, i.e. those that involve thinking or attending to specific aspects of an emotional situation, thereby altering its emotional quality. The CERQ includes subscales measuring both adaptive (i.e. those that reduce or transform negative emotions) and dysfunctional (i.e. those that tend to intensify the negative emotion) strategies of varying cognitive complexity. Importantly, usage of these strategies have been shown to have an appropriate developmental trajectory, while the relative balance of adaptive and dysfunctional strategy use has been shown to be predictive of psychopathology (Garnefski & Kraaij, 2006).

A1.3.3.3. Emotion Regulation of Self and Other scale

Unlike the COPE and CERQ, the EROS (EROS; Niven et al., 2011) measures individual differences in the *goal* of emotion regulation (i.e. worsening or improving), and differentiates between intrinsic and extrinsic means of regulation. As such, the EROS

provides a more abstract measure of emotion management across numerous specific strategies and behaviours, and critically include a social dimension frequently overlooked in the literature at large.

A1.3.3.4. Adult Temperament Questionnaire: Effortful Control subscale

Finally, we included the Effortful Control subscale of the ATQ. Effortful control is thought to be at the centre of both cognitive and behavioural self-regulation, and has been shown to be inversely related to both negative affect, interpersonal problems , psychiatric symptoms and general distress (Wiltink et al., 2006). The effortful control subscale of the ATQ can be thought of as measuring the core dispositional ability to override emotional impulses and achieve behaviours in the face of e.g. inherent avoidance tendencies.

A1.3.3.5. PCA procedure: Emotion management styles

We combined these scales using PCA to create composite measures of emotion management using the scree criterion. Our analyses revealed a three component structure to emotion management styles explaining a total of 37% of variance in reports (see *Appendix*: Figure A2.3.1). Based on the loading structures and extant coping and emotion regulation theory, we interpreted these as reflecting Emotion-focused (16.90% variance explained), Maladaptive (12.11% variance explained), and Instrumental (8.61% variance explained) emotion management. Characteristic of the Emotion-focused component was positive loadings on scales pertaining to cognitively altering emotional states or taking different perspectives, notably with the aim of improving one's emotional states. The Maladaptive component had strong loadings on scales including self-blame, catastrophising and rumination, notably with an emphasis on worsening one's emotional states, and negatively loading on effortful control. Finally, the Instrumental component had loadings on scales measuring the tendency to take an active, instrumental approach to managing emotions,

seeking emotional support and having active plans, aimed at improving one's own and others emotional states.

A2. Supplemental figures

A2.1 Supplemental figures: Chapter 4

Figure A2.1.1. Previous studies of endogenous emotion generation

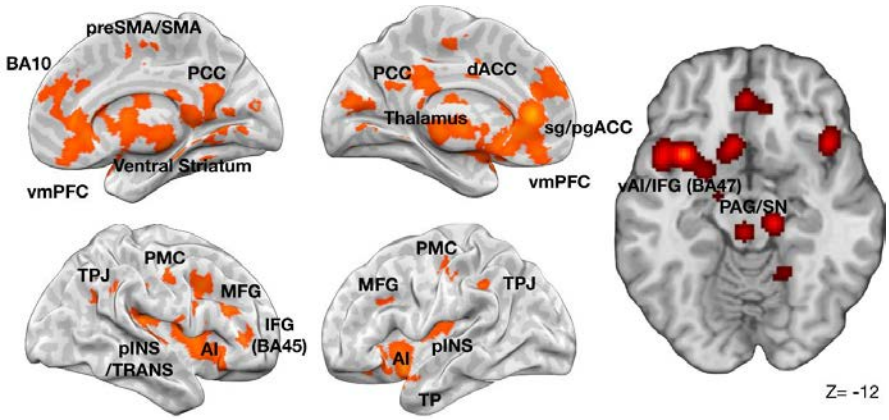


Figure A2.1.1: Previous studies of endogenous emotion generation. Related to Introduction
An overview of previous studies of emotion generation (Damasio et al., 2000; Gemar et al., 1996; George et al., 1996; Kimbrell et al., 1999; Liotti et al., 2000; Mayberg et al., 1999; Reiman et al., 1997), derived from the GingerALE meta-analysis software package (Eickhoff et al., 2009), thresholded at $p < .05$, uncorrected. Note: Figure is presented for descriptive purposes only. See Phan et al. (2002) for a quantitative summary of differences between exogenous and endogenous emotion elicitation

Figure A2.1.2. Main contrasts for model-based decomposition analyses.

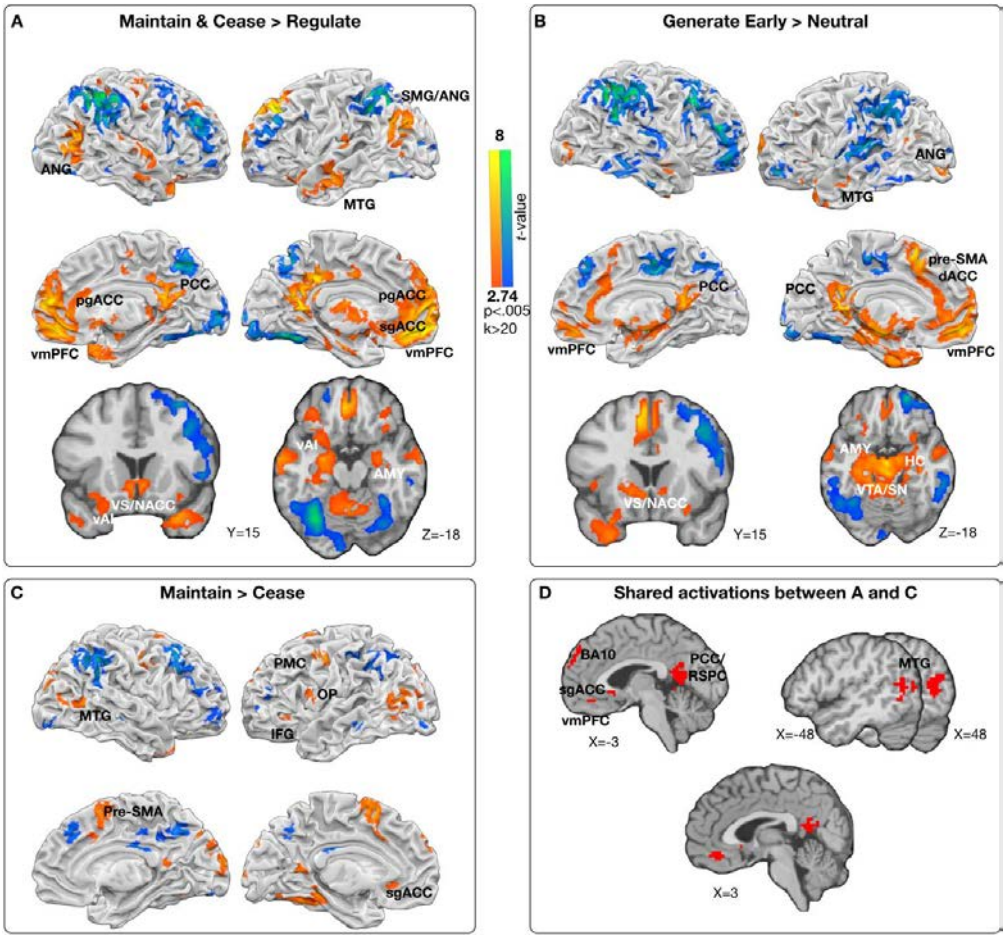


Figure A2.1.2: Main contrasts for model-based decomposition analyses. Related to Figure 6.3; Model-based component process mapping. The constituent contrasts were used in the model-based component process decomposition analysis adopted in Experiment 1. Results were thresholded at T > 2.74, $p < .005$ and k > 20.

Figure A2.1.3. Effect of fatigue/habituation on neural signatures of EGE

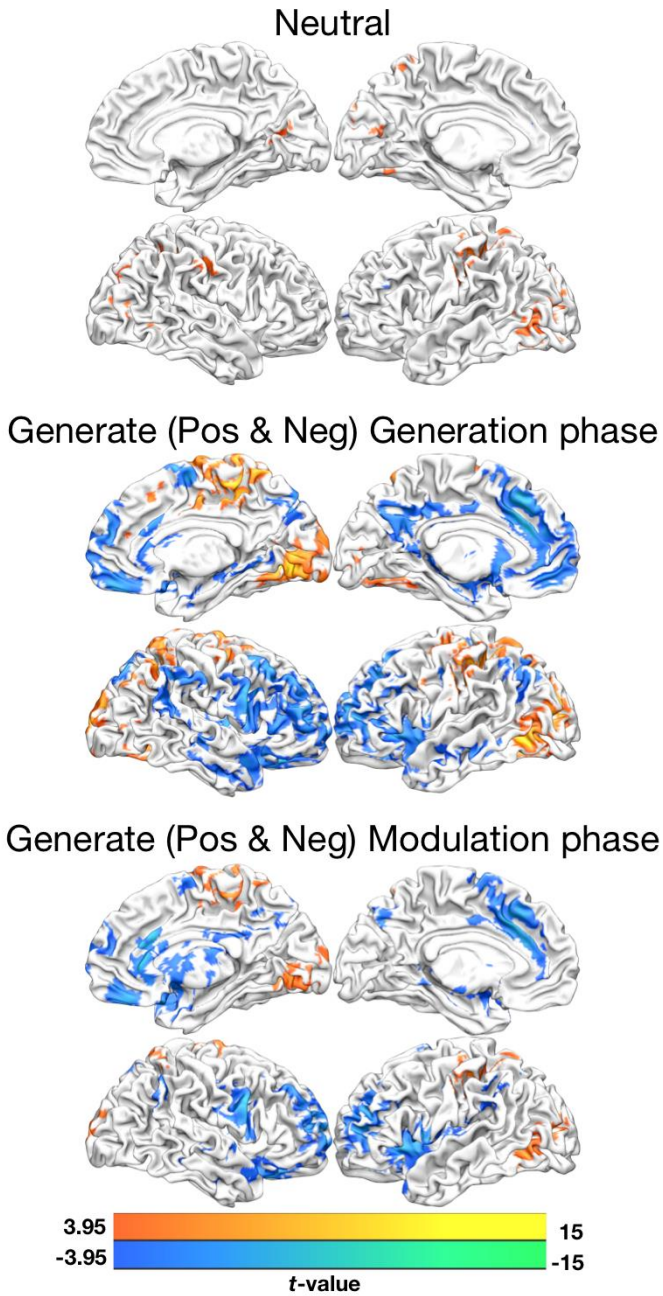


Figure A2.1.3: Regions parametrically modulated by time on task in Experiment 2 for the Neutral and Generation conditions. All results corrected at FWEc α<.05 (T>3.95, k>10). See Supplemental Analysis for details.

A2.2 Supplemental figures: Chapter 6

Figure A2.2.1. Reappraisal-related activations

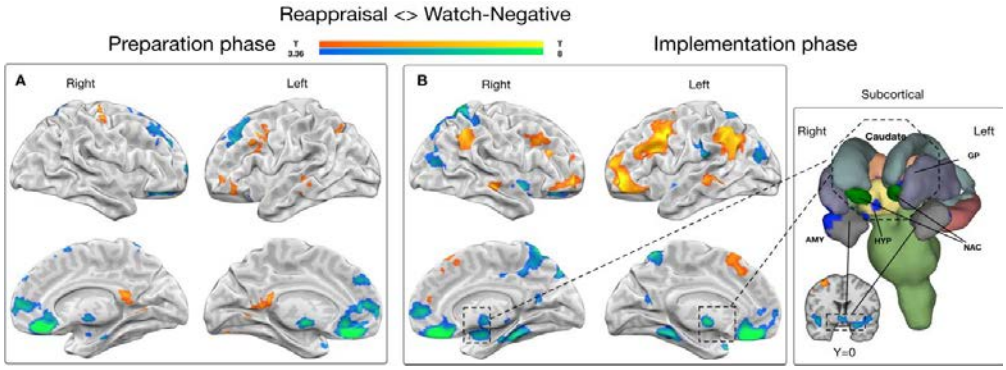


Figure A2.2.1: Reappraisal-related activations. Results from the whole brain contrast of Compassion>Watch-negative presented separately for the Preparation and Implementation phases. All results thresholded at FWEc α < .05 as determined by AFNI's *Alphasim* ($p < .001$, extent threshold = 30 voxels).

A2.3 Supplemental figures: Chapter 7

Figure A2.3.1. Scree plots from the two PCA analyses performed

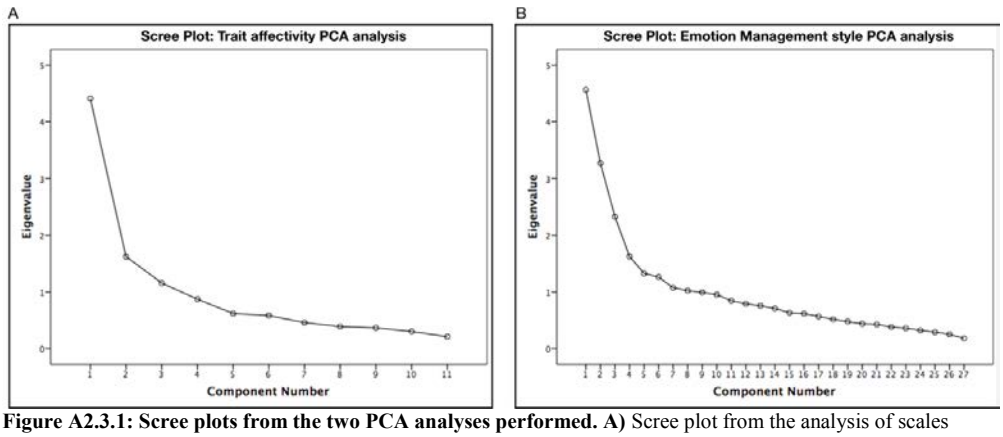


Figure A2.3.1: Scree plots from the two PCA analyses performed. A) Scree plot from the analysis of scales measuring trait affect, indicating the optimality of a single component solution. See *Appendix*: Table A3.3.1 for scale loadings. **B)** Scree plot from the analysis of scales measuring emotion management styles, indicating the optimality of a three-component solution. See *Appendix*: Table A3.3.2 for scale loadings.

Figure A2.3.2. Correlation of emotion generation ability and affective traits

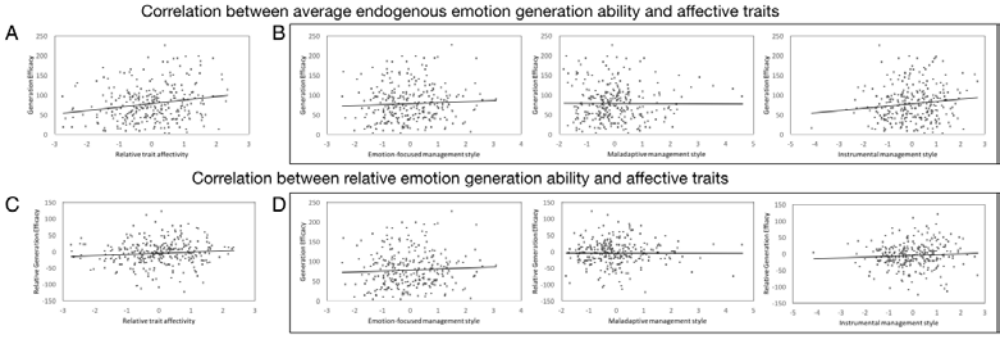


Figure A2.3.2: Correlation of emotion generation ability and affective traits. Scatterplots of the relationships between the composite scores calculated for average and relative trait-specific generation ability and the PCA components measuring trait affectivity (see *Appendix*: Table A3.3.1) and emotion management styles (see *Appendix*: Table A3.3.2). **A)** Relationship between average generation efficacy and trait affectivity. **B)** Relationship between average generation efficacy and three emotion management styles. **C)** Relationship between the relative ability to generate positive and negative emotion and trait affect. **D)** Relationship between relative ability to generation positive and negative emotion and emotion management styles. Lines indicate linear regression lines of best and are provided for reference only (see Table 7.3 for correlation analyses).

A3. Supplemental tables

A3.1. Supplemental tables: Chapter 4

Table A3.1.1. Core networks of emotion generation

Region	Label/BA	Side	MNI x	MNI y	MNI z	Extent (voxel	*t*-value (max	*t*-value (avg
		Experiment 1 (*N*=32)						
Activations								
Medial Frontal Gyrus	**32**	**L**	**-11**	**11**	**47**	**696**	**8.27**	**4.27**
Medial Frontal Gyrus	*32*	*L*	*-11*	*11*	*47*	*125*	*8.27*	*4.64*
Medial Frontal Gyrus	*11*	*L*	*-7*	*51*	*-13*	*105*	*6.89*	*4.97*
Medial Frontal Gyrus	*10*	*L*	*-10*	*37*	*-11*	*40*	*5.45*	*4.24*
Anterior Cingulate	*33*	*R*	*0*	*33*	*4*	*108*	*5.42*	*4.13*
Medial Frontal Gyrus	*11*	*R*	*4*	*42*	*-15*	*83*	*5.37*	*4.29*
Medial Frontal Gyrus	*10*	*L*	*-12*	*58*	*6*	*127*	*5.06*	*3.89*
Anterior Cingulate	*24*	*R*	*3*	*27*	*21*	*61*	*4.89*	*3.89*
Posterior Cingulate	**29**	**L**	**-6**	**-45**	**17**	**2031**	**7.89**	**4.24**
Posterior Cingulate	*29*	*L*	*-6*	*-45*	*17*	*135*	*7.89*	*5.49*
Globus Pallidus	*Lateral*	*R*	*25*	*-9*	*-1*	*58*	*6.90*	*4.17*
Cerebellum	*Culmen*	*R*	*11*	*-34*	*-13*	*128*	*6.78*	*4.19*
Midbrain	*Mammilary/Red Nucleus*	*L*	*0*	*-13*	*-6*	*104*	*6.17*	*4.31*
Cerebellum	*Lingual*	*R*	*0*	*-47*	*-19*	*72*	*6.15*	*4.31*
Hippocampus	*Posterior*	*L*	*-33*	*-27*	*-8*	*72*	*6.12*	*4.14*
Cerebellum	*Lingual*	*R*	*3*	*-42*	*-8*	*146*	*6.07*	*4.32*
Putamen	*Lentiform*	*L*	*-26*	*7*	*5*	*81*	*5.96*	*4.43*
Midbrain	*Substantia Nigra/VTA*	*L*	*-14*	*-20*	*-6*	*93*	*5.87*	*4.15*
Retrosplenial Cortex	*30*	*L*	*-12*	*-37*	*6*	*97*	*5.82*	*4.63*
Midbrain	*Substantia Nigra*	*R*	*14*	*-24*	*-10*	*42*	*5.79*	*3.86*
Caudate	*Head*	*L*	*-9*	*7*	*-2*	*60*	*5.79*	*4.18*
Thalamus	*Anterior*	*L*	*-3*	*-2*	*4*	*77*	*5.78*	*4.10*
Thalamus	*Anterior*	*R*	*6*	*-3*	*0*	*46*	*5.68*	*4.18*
Putamen	*Lentiform*	*R*	*22*	*0*	*1*	*61*	*5.60*	*3.97*
Cerebellum	*Anterior Lobe*	*R*	*16*	*-37*	*-30*	*60*	*6.53*	*4.48*
Lentiform Nucleus	*Pallidus*	*L*	*-24*	*-13*	*-7*	*59*	*5.41*	*4.06*
Posterior Cingulate	*23*	*R*	*6*	*-50*	*22*	*98*	*5.30*	*4.28*
Midbrain	*Red Nucleus*	*L*	*-5*	*-23*	*-3*	*114*	*5.14*	*3.97*
Posterior Cingulate	*30*	*L*	*-13*	*-55*	*16*	*48*	*4.99*	*4.00*
Parahippocampal Gyrus	*30*	*L*	*-14*	*-31*	*-7*	*86*	*4.91*	*3.91*
Parahippocampal Gyrus	*30*	*R*	*15*	*-14*	*-7*	*59*	*4.69*	*3.92*
Parahippocampal Gyrus	*27*	*R*	*9*	*-37*	*4*	*74*	*4.60*	*3.90*
Cerebellum	*Culmen*	*L*	*-6*	*-42*	*-1*	*51*	*4.50*	*3.88*
Cerebellum	*Culmen*	*L*	*-17*	*-45*	*-18*	*46*	*4.15*	*3.69*
Middle Temporal Gyrus	**21**	**L**	**-62**	**-8**	**-9**	**66**	**5.49**	**4.21**
Inferior Frontal Gyrus	**13**	**L**	**-41**	**27**	**7**	**63**	**5.14**	**3.87**
Middle Temporal Gyrus	**39**	**L**	**-50**	**-71**	**20**	**42**	**4.32**	**3.66**
Deactivations								

Inferior Parietal Lobule	**7**	**R**	**36**	**-57**	**42**	**1674**	**-11.98**	**-5.05**
Inferior Parietal Lobule	*7*	*R*	*36*	*-57*	*42*	*440*	*-11.98*	*-5.94*
Inferior Parietal Lobule	*40*	*R*	*45*	*-40*	*40*	*321*	*-11.36*	*-5.77*
Precuneus	*7*	*R*	*9*	*-64*	*47*	*108*	*-8.95*	*-4.39*
Middle Occipital Gyrus	*19*	*R*	*27*	*-87*	*10*	*64*	*-8.37*	*-4.77*
Inferior Parietal Lobule	*40*	*R*	*50*	*-28*	*46*	*115*	*-7.67*	*-5.10*
Cingulate Gyrus	*31*	*R*	*7*	*-26*	*38*	*134*	*-7.31*	*-4.60*
Precuneus	*7*	*R*	*9*	*-63*	*37*	*56*	*-6.46*	*-4.62*
Cingulate Gyrus	*31*	*R*	*13*	*-37*	*38*	*56*	*-5.75*	*-4.14*
Cuneus	*17*	*R*	*11*	*-97*	*0*	*67*	*-5.51*	*-4.20*
Supramarginal Gyrus	*40*	*R*	*59*	*-49*	*26*	*106*	*-5.39*	*-4.08*
Superior Occipital Gyrus	*39*	*R*	*31*	*-72*	*29*	*78*	*-5.06*	*-4.14*
Inferior Parietal Lobule	*40*	*R*	*61*	*-25*	*33*	*80*	*-4.72*	*-3.78*
Cuneus	*7*	*R*	*19*	*-74*	*31*	*42*	*-3.96*	*-3.63*
Inferior Parietal Lobule	**40**	**L**	**-43**	**-42**	**49**	**762**	**-7.60**	**-4.31**
Inferior Parietal Lobule	*40*	*L*	*-43*	*-42*	*49*	*420*	*-7.60*	*-4.65*
Postcentral Gyrus	*2*	*L*	*-54*	*-29*	*39*	*161*	*-5.10*	*-4.08*
Insula	*40*	*L*	*-47*	*-27*	*19*	*75*	*-4.84*	*-3.81*
Precuneus	*7*	*L*	*-19*	*-70*	*35*	*50*	*-4.50*	*-3.79*
Angular Gyrus	*39*	*L*	*-28*	*-55*	*36*	*40*	*-3.95*	*-3.65*
Cerebellum	**Inf. Semi-lunar**	**L**	**-43**	**-68**	**-42**	**110**	**-7.46**	**-4.62**
Precentral Gyrus	**9**	**R**	**46**	**19**	**35**	**620**	**-7.04**	**-4.29**
Precentral Gyrus	*9*	*R*	*46*	*19*	*35*	*124*	*-7.04*	*-4.33*
Middle Frontal Gyrus	*46*	*R*	*53*	*39*	*16*	*128*	*-6.92*	*-4.59*
Middle Frontal Gyrus	*10*	*R*	*48*	*51*	*-6*	*90*	*-6.10*	*-4.48*
Inferior Frontal Gyrus	*10*	*R*	*42*	*53*	*1*	*122*	*-5.59*	*-4.22*
Superior Frontal Gyrus	*8*	*R*	*40*	*17*	*49*	*93*	*-5.42*	*-4.16*
Superior Frontal Gyrus	*11*	*R*	*29*	*52*	*-17*	*47*	*-4.76*	*-3.74*
Middle Occipital Gyrus	**19**	**L**	**-25**	**-88**	**11**	**756**	**-6.99**	**-4.27**
Middle Occipital Gyrus	*19*	*L*	*-25*	*-88*	*11*	*77*	*-6.99*	*-4.71*
Inferior Temporal Gyrus	*20*	*L*	*-53*	*-45*	*-13*	*97*	*-6.82*	*-4.23*
Lingual Gyrus	*17*	*L*	*-7*	*-95*	*-9*	*98*	*-6.27*	*-4.80*
Cerebellum	*Declive*	*L*	*-9*	*-71*	*-21*	*50*	*-5.49*	*-4.27*
Fusiform Gyrus	*19*	*L*	*-43*	*-66*	*-6*	*89*	*-5.44*	*-4.09*
Fusiform Gyrus	*19*	*L*	*-35*	*-74*	*-9*	*80*	*-5.30*	*-4.09*
Middle Occipital Gyrus	*37*	*L*	*-51*	*-63*	*-8*	*101*	*-5.09*	*-4.18*
Lingual Gyrus	*18*	*L*	*-13*	*-84*	*-8*	*60*	*-5.03*	*-3.98*
Fusiform Gyrus	*37*	*L*	*-43*	*-57*	*-12*	*53*	*-5.00*	*-4.06*
Lingual Gyrus	*18*	*L*	*-26*	*-77*	*-6*	*50*	*-4.86*	*-4.06*
Lingual Gyrus	**18**	**R**	**31**	**-73**	**-7**	**322**	**-6.69**	**-4.30**
Lingual Gyrus	*18*	*R*	*31*	*-73*	*-7*	*114*	*-6.69*	*-4.40*
Fusiform Gyrus	*37*	*R*	*56*	*-50*	*-13*	*100*	*-6.07*	*-4.24*
Middle Temporal Gyrus	*37*	*R*	*58*	*-45*	*-5*	*108*	*-5.87*	*-4.23*
Middle Frontal Gyrus	**11**	**L**	**-45**	**46**	**-11**	**40**	**-4.79**	**-3.80**
	Experiment 2 (*N*=293)							
Activations								
Superior Frontal Gyrus	**6**	**R**	**3**	**8**	**58**	**9887**	**20.42**	**7.51**
Superior Frontal Gyrus	*6*	*R*	*3*	*8*	*58*	*294*	*20.42*	*10.90*
Superior Frontal Gyrus	*6*	*L*	*-4*	*11*	*49*	*184*	*20.25*	*11.40*
Cingulate Gyrus	*32*	*L*	*-8*	*21*	*37*	*375*	*18.99*	*9.11*
Medial Frontal Gyrus	*6*	*R*	*3*	*14*	*44*	*193*	*16.15*	*10.17*
Insula	*13*	*L*	*-47*	*9*	*6*	*229*	*15.11*	*8.74*
Cerebellum	*Tuber*	*R*	*37*	*-60*	*-23*	*355*	*15.07*	*8.83*
Inferior Frontal Gyrus	*45*	*L*	*-46*	*21*	*5*	*271*	*14.79*	*9.11*

Middle Frontal Gyrus	*6*	*L*	*-40*	*0*	*52*	*355*	*14.12*	*8.55*
Inferior Frontal Gyrus	*45*	*L*	*-31*	*25*	*6*	*94*	*14.04*	*10.48*
Cerebellum	*Culmen*	*L*	*-27*	*-52*	*-20*	*166*	*14.01*	*8.15*
Cerebellum	*Declive*	*L*	*-15*	*-55*	*-14*	*246*	*13.91*	*8.93*
Midbrain	*Substantia Nigra* *VTA* *PAG*	*L*	*0*	*-18*	*-4*	*147*	*13.64*	*8.17*
Middle Frontal Gyrus	*9*	*L*	*-40*	*18*	*28*	*306*	*13.23*	*7.01*
Cerebellum	*Declive*	*R*	*21*	*-58*	*-17*	*242*	*13.14*	*9.41*
Basal Ganglia	*Caudate Body*	*L*	*-20*	*5*	*19*	*63*	*12.88*	*8.92*
Posterior Cingulate	*31*	*L*	*-6*	*-52*	*20*	*320*	*12.87*	*7.24*
Basal Ganglia	*Caudate Body*	*R*	*19*	*4*	*19*	*95*	*12.78*	*9.54*
Basal Ganglia	*Putamen*	*L*	*-22*	*2*	*12*	*80*	*12.52*	*9.06*
Basal Ganglia	*Putamen*	*R*	*26*	*3*	*13*	*143*	*12.47*	*7.80*
Thalamus	*Anterior*	*L*	*-3*	*-4*	*8*	*173*	*12.24*	*7.60*
Superior Frontal Gyrus	*9*	*L*	*-12*	*55*	*29*	*214*	*12.23*	*8.59*
Cerebellum	*Declive*	*L*	*0*	*-58*	*-11*	*321*	*12.23*	*8.82*
Basal Ganglia	*Putamen*	*L*	*-32*	*4*	*5*	*284*	*12.17*	*8.17*
Basal Ganglia	*Caudate Body*	*L*	*-16*	*-9*	*23*	*38*	*11.93*	*8.50*
Midbrain	*Substantia Nigra*	*L*	*-11*	*-20*	*-5*	*75*	*11.68*	*7.46*
Insula	*13*	*R*	*47*	*7*	*6*	*161*	*11.68*	*7.21*
Cerebellum	*Declive*	*L*	*-44*	*-61*	*-22*	*109*	*11.59*	*7.12*
Hippocampus	*Anterior*	*L*	*-18*	*-18*	*-6*	*184*	*11.47*	*6.56*
Precentral Gyrus	*6*	*R*	*55*	*-4*	*42*	*87*	*10.86*	*6.74*
Cerebellum	*Pyramis*	*R*	*10*	*-79*	*-26*	*210*	*10.67*	*6.77*
Cerebellum	*Lingual*	*R*	*6*	*-42*	*-8*	*229*	*10.64*	*8.03*
Basal Ganglia	*Caudate Head*	*R*	*3*	*1*	*6*	*35*	*10.40*	*7.10*
Medial Frontal Gyrus	*11*	*L*	*-4*	*54*	*-11*	*224*	*10.14*	*6.83*
Cerebellum	*Tonsil*	*R*	*34*	*-59*	*-42*	*247*	*10.00*	*6.08*
Posterior Cingulate	*29*	*L*	*-6*	*-47*	*10*	*135*	*9.50*	*6.89*
Superior Frontal Gyrus	*10*	*L*	*-35*	*48*	*22*	*239*	*9.03*	*6.54*
Cerebellum	*Declive*	*R*	*29*	*-82*	*-22*	*268*	*8.94*	*6.69*
Basal Ganglia	*Lateral Globus Pallidus*	*R*	*22*	*-6*	*-1*	*123*	*8.76*	*6.01*
Basal Ganglia	*Caudate Body*	*R*	*13*	*4*	*10*	*43*	*8.73*	*6.11*
Hypothalamus	*Anterior*	*L*	*-9*	*-5*	*-3*	*64*	*8.66*	*5.82*
Middle Frontal Gyrus	*8*	*L*	*-22*	*34*	*42*	*196*	*8.60*	*6.07*
Cerebellum	*Culmen*	*L*	*0*	*-33*	*2*	*49*	*8.29*	*5.19*
Cerebellum	*Culmen*	*R*	*17*	*-37*	*-17*	*101*	*8.12*	*6.45*
Basal Ganglia	*Lateral Globus Pallidus*	*R*	*13*	*6*	*0*	*86*	*7.97*	*5.44*
Hippocampus	*Head*	*R*	*18*	*-17*	*-5*	*112*	*7.73*	*5.47*
Cerebellum	*Anterior Lobe*	*R*	*3*	*-47*	*-26*	*167*	*7.69*	*5.76*
Middle Frontal Gyrus	*6*	*L*	*-27*	*2*	*60*	*135*	*7.59*	*5.66*
Inferior Frontal Gyrus	*13*	*R*	*43*	*23*	*5*	*175*	*7.53*	*5.61*
Precentral Gyrus	*6*	*R*	*63*	*2*	*21*	*42*	*7.47*	*5.22*
Cerebellum	*Tonsil*	*R*	*37*	*-53*	*-33*	*82*	*7.06*	*5.27*
Basal Ganglia	*Caudate Tail*	*R*	*30*	*-38*	*6*	*20*	*6.99*	*5.53*
Inferior Frontal Gyrus	*45*	*R*	*54*	*21*	*6*	*55*	*6.91*	*5.53*
Cuneus	*17*	*L*	*-17*	*-78*	*9*	*113*	*6.82*	*4.99*
Thalamus	*Pulvinar*	*L*	*-12*	*-28*	*13*	*56*	*6.80*	*5.45*
Cuneus	*17*	*R*	*22*	*-77*	*8*	*79*	*6.78*	*4.99*
Medial Frontal Gyrus	*11*	*R*	*4*	*48*	*-16*	*47*	*6.63*	*5.09*
Superior Frontal Gyrus	*10*	*L*	*-29*	*63*	*5*	*105*	*6.49*	*5.08*
Basal Ganglia	*Caudate Tail*	*R*	*36*	*-35*	*-1*	*14*	*6.44*	*5.17*
Parahippocampal Gyrus	*30*	*R*	*28*	*-54*	*8*	*14*	*6.37*	*5.06*
Inferior Frontal Gyrus	*47*	*L*	*-34*	*30*	*-5*	*18*	*6.32*	*5.04*

Posterior Cingulate	*29*	*R*	*6*	*-40*	*6*	*18*	*6.16*	*4.90*
Precentral Gyrus	*6*	*R*	*44*	*-7*	*55*	*20*	*6.14*	*4.80*
Posterior Cingulate	*30*	*L*	*-26*	*-67*	*9*	*47*	*6.13*	*4.74*
Precentral Gyrus	*4*	*L*	*-45*	*-12*	*37*	*62*	*6.08*	*4.77*
Cerebellum	*Anterior Lobe*	*L*	*-7*	*-47*	*-25*	*19*	*6.07*	*5.04*
Superior Frontal Gyrus	*8*	*L*	*-8*	*37*	*48*	*24*	*6.04*	*5.44*
Posterior Cingulate	*30*	*R*	*6*	*-51*	*6*	*29*	*6.03*	*4.69*
Postcentral Gyrus	*3*	*R*	*23*	*-25*	*58*	*30*	*6.01*	*5.00*
Basal Ganglia	*Caudate Body*	*L*	*-10*	*20*	*3*	*40*	*5.96*	*4.75*
Precentral Gyrus	*6*	*R*	*43*	*-14*	*35*	*33*	*5.83*	*4.96*
Parahippocampal Gyrus	*18*	*L*	*-25*	*-55*	*8*	*26*	*5.71*	*4.68*
Thalamus	*Pulvinar*	*R*	*15*	*-35*	*8*	*13*	*5.70*	*4.91*
Basal Ganglia	*Putamen*	*L*	*-28*	*-18*	*9*	*10*	*5.64*	*4.86*
Basal Ganglia	*Caudate Tail*	*L*	*-28*	*-36*	*8*	*27*	*5.60*	*4.60*
Superior Frontal Gyrus	*6*	*R*	*20*	*-10*	*71*	*25*	*5.39*	*4.49*
Precentral Gyrus	*6*	*R*	*32*	*-11*	*64*	*15*	*5.12*	*4.45*
Middle Frontal Gyrus	*6*	*L*	*-20*	*-15*	*59*	*53*	*5.05*	*4.53*
Cerebellum	*Inf. Semi-Lunar*	*R*	*8*	*-60*	*-49*	*37*	*4.99*	*4.42*
Parahippocampal Gyrus	*28*	*L*	*-21*	*-10*	*-20*	*21*	*4.93*	*4.37*
Parahippocampal Gyrus	*28*	*R*	*27*	*-14*	*-20*	*27*	*4.89*	*4.24*
Inferior Temporal Gyrus	*20*	*R*	*39*	*-16*	*-29*	*11*	*4.62*	*4.20*
Parahippocampal Gyrus	*36*	*L*	*-29*	*-15*	*-24*	*10*	*4.49*	*4.22*
Inferior Occipital Gyrus	**18**	**R**	**30**	**-94**	**-5**	**96**	**12.68**	**7.54**
Inferior Occipital Gyrus	**18**	**L**	**-28**	**-93**	**-3**	**88**	**10.24**	**6.76**
Cerebellum	**Tonsil**	**L**	**-27**	**-59**	**-42**	**86**	**8.55**	**5.57**
Middle Temporal Gyrus	**39**	**L**	**-40**	**-61**	**23**	**152**	**7.78**	**5.35**
Middle Temporal Gyrus	*39*	*L*	*-40*	*-61*	*23*	*85*	*7.78*	*5.59*
Angular Gyrus	*39*	*L*	*-50*	*-68*	*29*	*67*	*6.23*	*5.06*
Middle Temporal Gyrus	**21**	**L**	**-56**	**-11**	**-9**	**72**	**7.51**	**5.23**
Superior Temporal Gyrus	**22**	**R**	**45**	**-37**	**2**	**54**	**7.50**	**5.41**
Medial Frontal Gyrus	**10**	**R**	**4**	**57**	**17**	**37**	**7.02**	**5.12**
Middle Temporal Gyrus	**21**	**L**	**-46**	**8**	**-25**	**36**	**5.94**	**4.66**
Precentral Gyrus	**4**	**L**	**-19**	**-23**	**59**	**22**	**5.61**	**4.86**
Precentral Gyrus	**6**	**R**	**26**	**-13**	**53**	**10**	**5.01**	**4.33**
Deactivations								
Precuneus	**19**	**R**	**34**	**-69**	**32**	**7336**	**-18.26**	**-7.42**
Precuneus	*19*	*R*	*34*	*-69*	*32*	*286*	*-18.26*	*-11.55*
Inferior Temporal Gyrus	*37*	*L*	*-53*	*-57*	*-3*	*484*	*-17.70*	*-8.73*
Middle Temporal Gyrus	*37*	*R*	*58*	*-51*	*-3*	*443*	*-16.90*	*-8.47*
Cingulate Gyrus	*31*	*R*	*6*	*-36*	*38*	*491*	*-16.56*	*-8.00*
Inferior Parietal Lobule	*40*	*R*	*39*	*-54*	*42*	*378*	*-15.62*	*-10.58*
Inferior Parietal Lobule	*40*	*R*	*47*	*-45*	*45*	*567*	*-15.32*	*-9.55*
Cuneus	*18*	*R*	*14*	*-95*	*9*	*92*	*-13.66*	*-7.85*
Cuneus	*18*	*L*	*-13*	*-99*	*8*	*49*	*-13.54*	*-8.34*
Middle Occipital Gyrus	*18*	*L*	*-19*	*-93*	*10*	*146*	*-13.36*	*-7.87*
Lingual Gyrus	*18*	*L*	*-23*	*-79*	*-6*	*97*	*-12.79*	*-7.64*
Lingual Gyrus	*18*	*R*	*34*	*-73*	*-7*	*179*	*-12.72*	*-7.17*
Middle Occipital Gyrus	*18*	*R*	*27*	*-90*	*12*	*155*	*-12.51*	*-7.12*
Inferior Occipital Gyrus	*19*	*L*	*-35*	*-74*	*-7*	*201*	*-11.96*	*-6.79*
Lingual Gyrus	*18*	*R*	*12*	*-86*	*-4*	*57*	*-11.80*	*-6.65*
Inferior Parietal Lobule	*40*	*L*	*-54*	*-32*	*39*	*225*	*-11.30*	*-7.12*
Superior Occipital Gyrus	*19*	*L*	*-31*	*-77*	*25*	*243*	*-11.08*	*-7.20*
Precuneus	*7*	*L*	*-28*	*-67*	*28*	*33*	*-11.04*	*-9.74*
Lingual Gyrus	*18*	*R*	*21*	*-81*	*-4*	*62*	*-10.98*	*-7.38*

Precuneus	*7*	*R*	*12*	*-70*	*43*	*398*	*-10.60*	*-6.88*
Middle Temporal Gyrus	*39*	*R*	*42*	*-74*	*24*	*251*	*-10.56*	*-6.25*
Cuneus	*17*	*L*	*-13*	*-93*	*6*	*32*	*-10.05*	*-6.05*
Precuneus	*7*	*L*	*-25*	*-67*	*38*	*154*	*-10.01*	*-6.67*
Middle Temporal Gyrus	*21*	*R*	*61*	*-28*	*-2*	*286*	*-9.99*	*-6.04*
Lingual Gyrus	*18*	*L*	*-10*	*-85*	*-6*	*71*	*-9.86*	*-6.04*
Inferior Parietal Lobule	*40*	*L*	*-44*	*-43*	*41*	*218*	*-9.51*	*-6.30*
Fusiform Gyrus	*37*	*L*	*-31*	*-47*	*-7*	*142*	*-9.50*	*-5.65*
Superior Temporal Gyrus	*42*	*L*	*-58*	*-27*	*12*	*200*	*-8.82*	*-6.16*
Superior Temporal Gyrus	*42*	*R*	*56*	*-30*	*13*	*289*	*-8.70*	*-5.62*
Inferior Parietal Lobule	*40*	*R*	*50*	*-38*	*30*	*67*	*-8.64*	*-5.56*
Transverse Temporal Gyrus	*42*	*L*	*-62*	*-15*	*8*	*89*	*-8.64*	*-6.15*
Insula	*13*	*L*	*-40*	*-14*	*2*	*128*	*-8.41*	*-5.52*
Cerebellum	*Pyranus*	*L*	*-12*	*-71*	*-24*	*67*	*-8.40*	*-5.63*
Parahippocampal Gyrus	*36*	*R*	*30*	*-39*	*-8*	*110*	*-7.94*	*-5.39*
Inferior Parietal Lobule	*40*	*L*	*-49*	*-46*	*36*	*81*	*-7.48*	*-5.66*
Cerebellum	*Declive*	*R*	*33*	*-52*	*-9*	*87*	*-7.43*	*-5.20*
Superior Parietal Lobule	*7*	*L*	*-25*	*-58*	*41*	*35*	*-6.58*	*-4.99*
Paracentral Lobule	*5*	*R*	*7*	*-39*	*60*	*92*	*-6.45*	*-5.02*
Posterior Cingulate	*23*	*R*	*6*	*-32*	*24*	*24*	*-6.28*	*-4.97*
Postcentral Gyrus	*1*	*L*	*-64*	*-16*	*22*	*42*	*-6.01*	*-4.82*
Superior Temporal Gyrus	*41*	*L*	*-42*	*-35*	*16*	*79*	*-5.85*	*-4.75*
Superior Temporal Gyrus	*22*	*R*	*63*	*-5*	*1*	*43*	*-5.68*	*-4.75*
Paracentral Lobule	*5*	*L*	*-3*	*-38*	*57*	*29*	*-5.54*	*-4.64*
Precuneus	*7*	*L*	*-25*	*-49*	*41*	*33*	*-5.34*	*-4.66*
Postcentral Gyrus	*5*	*L*	*-13*	*-41*	*66*	*21*	*-5.32*	*-4.40*
Insula	*13*	*L*	*-34*	*-25*	*16*	*52*	*-5.03*	*-4.46*
Precuneus	*7*	*L*	*-9*	*-53*	*56*	*26*	*-4.60*	*-4.18*
Middle Frontal Gyrus	**46**	**R**	**53**	**36**	**16**	**1326**	**-12.81**	**-7.32**
Middle Frontal Gyrus	*46*	*R*	*53*	*36*	*16*	*217*	*-12.81*	*-8.07*
Middle Frontal Gyrus	*47*	*R*	*48*	*45*	*-6*	*257*	*-12.77*	*-8.69*
Middle Frontal Gyrus	*8*	*R*	*41*	*29*	*40*	*333*	*-11.47*	*-7.36*
Superior Frontal Gyrus	*8*	*R*	*34*	*18*	*50*	*230*	*-10.73*	*-7.45*
Middle Frontal Gyrus	*10*	*R*	*32*	*60*	*8*	*149*	*-8.66*	*-5.91*
Middle Frontal Gyrus	*11*	*R*	*24*	*35*	*-15*	*63*	*-6.65*	*-5.03*
Medial Frontal Gyrus	*8*	*R*	*8*	*38*	*39*	*47*	*-6.61*	*-4.77*
Middle Frontal Gyrus	*11*	*R*	*18*	*47*	*-20*	*30*	*-4.91*	*-4.28*
Cerebellum	**Tonsil**	**L**	**-32**	**-66**	**-34**	**223**	**-9.76**	**-6.22**
Insula	**13**	**R**	**40**	**-13**	**3**	**62**	**-6.64**	**-4.94**
Cerebellum	**Tonsil**	**L**	**-38**	**-41**	**-33**	**16**	**-6.02**	**-4.74**
Middle Frontal Gyrus	**11**	**L**	**-17**	**41**	**-22**	**21**	**-5.67**	**-4.65**
Middle Frontal Gyrus	**47**	**L**	**-48**	**46**	**-8**	**19**	**-5.13**	**-4.35**

Table A3.1.1: Brain regions activated in the Maintain condition for both positive and negative valence relative to the Neutral baseline in Experiment 1 (N=32) and 2 (N=293). Cluster maxima are reported in bold. Italicised entries denote local maxima of subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha<.05$ using AFNIs alphasim procedure (Experiment 1: $T>3.38$, $p<.001$, $k>38$; Experiment 2: $T>3.95$, $p<.00005$, $k>10$).

Table A3.1.2. Main contrasts: model based component process deconstruction

Region	Label/BA	Side	MNI x	MNI y	MNI z	Extent (voxel)	*t*-value (max)	*t*-value (avg)
		Generate (Positive & Negative)> Neutral						
Activations								
Posterior Cingulate	**29**	**L**	**-3**	**-44**	**13**	**3878**	**9.04**	**3.84**
Posterior Cingulate	*29*	*L*	*-3*	*-44*	*13*	*292*	*9.04*	*4.99*
Midbrain	*Red nucleus*	*L*	*0*	*-16*	*-4*	*190*	*8.87*	*4.91*
Medial Frontal Gyrus	*32*	*L*	*-11*	*11*	*47*	*114*	*7.81*	*4.86*
Medial Frontal Gyrus	*11*	*L*	*-7*	*48*	*-13*	*135*	*6.94*	*4.78*
Basal Ganglia	*Putamen*	*L*	*-27*	*-15*	*-7*	*74*	*6.60*	*4.01*
Cerebellum	*Culmen*	*L*	*-8*	*-36*	*-9*	*132*	*6.16*	*3.82*
Medial Frontal Gyrus	*11*	*R*	*4*	*45*	*-16*	*109*	*6.11*	*3.97*
Hippocampus	*Anterior/Mid*	*L*	*-33*	*-27*	*-8*	*91*	*6.07*	*4.10*
Parahippocampal Gyrus	*35*	*L*	*-17*	*-34*	*-8*	*78*	*5.94*	*3.84*
Thalamus	*Posterior*	*L*	*-3*	*-25*	*1*	*162*	*5.93*	*3.93*
Medial Frontal Gyrus	*10*	*L*	*-12*	*60*	*3*	*132*	*5.84*	*3.91*
Parahippocampal Gyrus	*35*	*L*	*-20*	*-26*	*-11*	*60*	*5.79*	*3.80*
Thalamus	*Anterior Ventral Lateral*	*L*	*-3*	*-7*	*6*	*70*	*5.69*	*3.75*
Thalamus	*Nucleus*	*R*	*18*	*-11*	*4*	*50*	*5.43*	*3.66*
Parahippocampal Gyrus	*Uncus*	*L*	*-30*	*-6*	*-22*	*95*	*5.43*	*3.65*
Hippocampus	*Anterior/Mid*	*R*	*36*	*-26*	*-10*	*33*	*5.42*	*3.64*
Parahippocampal Gyrus	*30*	*R*	*12*	*-38*	*8*	*76*	*5.33*	*3.56*
Superior Frontal Gyrus	*10*	*L*	*-8*	*63*	*17*	*47*	*5.33*	*3.77*
Midbrain	*Substantia Nigra*	*R*	*12*	*-11*	*-9*	*96*	*5.29*	*3.92*
Parahippocampal Gyrus	*28*	*L*	*-15*	*-13*	*-8*	*112*	*5.26*	*4.00*
Precuneus	*31*	*L*	*-13*	*-59*	*20*	*129*	*5.24*	*3.61*
Lentiform Nucleus	*Putamen*	*L*	*-16*	*10*	*0*	*69*	*5.09*	*3.50*
Lentiform Nucleus	*Lateral Globus Pallidus*	*R*	*16*	*3*	*1*	*41*	*5.07*	*3.48*
Anterior Cingulate	*24*	*R*	*0*	*36*	*3*	*109*	*5.04*	*3.68*
Superior Temporal Gyrus	*38*	*L*	*-32*	*13*	*-30*	*93*	*4.95*	*3.48*
Cerebellum	*Anterior Lobe*	*R*	*1*	*-45*	*-23*	*54*	*4.89*	*3.51*
Basal Ganglia	*Putamen*	*R*	*25*	*-6*	*-4*	*42*	*4.87*	*3.54*
Medial Frontal Gyrus	*6*	*R*	*3*	*14*	*47*	*38*	*4.81*	*3.47*
Anterior Cingulate	*24*	*L*	*0*	*29*	*-4*	*53*	*4.77*	*3.33*
Inferior Frontal Gyrus	*47*	*L*	*-32*	*13*	*-19*	*30*	*4.75*	*3.31*
Uncus	*20*	*L*	*-33*	*-6*	*-30*	*70*	*4.74*	*3.51*
Anterior Cingulate	*32*	*R*	*7*	*27*	*21*	*72*	*4.68*	*3.39*
Uncus	*28*	*L*	*-23*	*-10*	*-31*	*40*	*4.66*	*3.51*
Parahippocampal Gyrus	*28*	*R*	*20*	*-29*	*-9*	*33*	*4.43*	*3.16*
Basal Ganglia	*Caudate Tail*	*R*	*37*	*-14*	*-12*	*38*	*4.42*	*3.31*
Cingulate Gyrus	*32*	*L*	*-15*	*21*	*32*	*53*	*4.41*	*3.56*
Amygdala	*Centromedial*	*L*	*-24*	*-7*	*-9*	*39*	*4.40*	*3.28*
Inferior Frontal Gyrus	*47*	*L*	*-27*	*24*	*-18*	*32*	*4.39*	*3.25*
Inferior Frontal Gyrus	*47*	*L*	*-22*	*10*	*-19*	*28*	*4.36*	*3.22*
Basal Ganglia	*Nucleus Accumbens*	*L*	*-9*	*1*	*-1*	*32*	*4.33*	*3.23*
Parahippocampal Gyrus	*34*	*R*	*29*	*3*	*-15*	*45*	*4.32*	*3.20*
Cingulate Gyrus	*32*	*R*	*14*	*20*	*31*	*33*	*4.30*	*3.41*
Anterior Cingulate	*32*	*L*	*-4*	*36*	*17*	*58*	*4.27*	*3.40*
Cingulate Gyrus	*32*	*R*	*3*	*21*	*37*	*45*	*4.25*	*3.39*

Cerebellum	*Anterior Lobe*	*R*	*16*	*-34*	*-29*	*37*	*4.44*	*3.44*
Medial Frontal Gyrus	*10*	*R*	*4*	*57*	*0*	*49*	*4.06*	*3.29*
Superior Temporal Gyrus	*38*	*L*	*-43*	*10*	*-21*	*35*	*4.04*	*3.22*
Cerebellum	*Lingual*	*R*	*0*	*-48*	*-15*	*55*	*4.00*	*3.27*
Parahippocampal Gyrus	*27*	*R*	*27*	*-28*	*-5*	*40*	*3.99*	*3.33*
Cerebellum	*Culmen*	*L*	*-17*	*-48*	*-16*	*35*	*3.94*	*3.13*
Hippocampus	*Anterior*	*R*	*31*	*-9*	*-11*	*41*	*3.92*	*3.15*
Thalamus	*Posterior*	*R*	*14*	*-29*	*-2*	*49*	*3.90*	*3.14*
Parahippocampal Gyrus	*Posterior*	*R*	*27*	*-20*	*-4*	*25*	*3.80*	*3.20*
Cerebellum	*Culmen*	*R*	*11*	*-34*	*-13*	*34*	*3.59*	*3.14*
Cerebellum	*Declive*	*R*	*18*	*-61*	*-19*	*35*	*3.39*	*2.95*
Lingual Gyrus	**17**	**R**	**24**	**-89**	**-2**	**142**	**6.66**	**3.99**
Inferior Occipital Gyrus	**18**	**L**	**-29**	**-93**	**-6**	**55**	**6.38**	**3.78**
Cerebellum	**Culmen**	**R**	**37**	**-54**	**-20**	**112**	**4.63**	**3.22**
Cerebellum	*Culmen*	*R*	*37*	*-54*	*-20*	*76*	*4.63*	*3.31*
Cerebellum	*Culmen*	*R*	*32*	*-44*	*-24*	*29*	*3.84*	*3.06*
Middle Temporal Gyrus	**21**	**L**	**-62**	**-8**	**-9**	**56**	**4.46**	**3.37**
Cerebellum	**Anterior Lobe**	**L**	**-15**	**-34**	**-30**	**45**	**4.39**	**3.54**
Superior Temporal Gyrus	**22**	**R**	**54**	**-7**	**-8**	**50**	**4.32**	**3.31**
Insula	**13**	**L**	**-34**	**25**	**9**	**79**	**4.20**	**3.21**
Insula	*13*	*L*	*-34*	*25*	*9*	*39*	*4.20*	*3.39*
Inferior Frontal Gyrus	*44*	*L*	*-42*	*15*	*13*	*32*	*3.48*	*3.08*
Middle Temporal Gyrus	**39**	**L**	**-48**	**-68**	**20**	**81**	**3.73**	**3.12**
Middle Frontal Gyrus	**8**	**L**	**-25**	**29**	**40**	**29**	**3.47**	**3.08**
Deactivations								
Inferior Parietal Lobule	**40**	**R**	**47**	**-37**	**40**	**1964**	**-11.68**	**-4.43**
Inferior Parietal Lobule	*40*	*R*	*47*	*-37*	*40*	*429*	*-11.68*	*-5.13*
Inferior Parietal Lobule	*40*	*R*	*42*	*-52*	*37*	*304*	*-10.04*	*-5.87*
Superior Parietal Lobule	*7*	*R*	*27*	*-64*	*46*	*129*	*-8.73*	*-3.98*
Supramarginal Gyrus	*40*	*R*	*53*	*-52*	*26*	*194*	*-7.47*	*-4.58*
Precuneus	*19*	*R*	*31*	*-63*	*39*	*163*	*-6.59*	*-4.40*
Precuneus	*7*	*R*	*9*	*-65*	*39*	*108*	*-5.90*	*-3.68*
Transverse Temporal Gyrus	*41*	*R*	*45*	*-23*	*12*	*131*	*-5.12*	*-3.81*
Cuneus	*19*	*R*	*12*	*-76*	*36*	*89*	*-4.63*	*-3.42*
Cuneus	*7*	*R*	*24*	*-79*	*30*	*103*	*-4.60*	*-3.40*
Transverse Temporal Gyrus	*42*	*R*	*59*	*-15*	*8*	*108*	*-4.38*	*-3.36*
Inferior Parietal Lobule	*40*	*R*	*61*	*-25*	*33*	*135*	*-4.13*	*-3.34*
Insula	*13*	*R*	*37*	*-13*	*12*	*49*	*-3.82*	*-3.18*
Insula	*13*	*R*	*56*	*-36*	*17*	*21*	*-3.29*	*-2.95*
Middle Frontal Gyrus	**9**	**R**	**48**	**15**	**35**	**1168**	**-8.65**	**-4.03**
Middle Frontal Gyrus	*9*	*R*	*48*	*15*	*35*	*212*	*-8.65*	*-4.48*
Middle Frontal Gyrus	*46*	*R*	*50*	*40*	*18*	*188*	*-7.25*	*-4.37*
Superior Frontal Gyrus	*11*	*R*	*25*	*50*	*-17*	*117*	*-6.68*	*-3.82*
Middle Frontal Gyrus	*47*	*R*	*48*	*45*	*-8*	*151*	*-6.04*	*-4.32*
Middle Frontal Gyrus	*6*	*R*	*40*	*14*	*47*	*157*	*-5.57*	*-3.94*
Inferior Frontal Gyrus	*44*	*R*	*55*	*8*	*23*	*44*	*-5.55*	*-3.52*
Inferior Frontal Gyrus	*44*	*R*	*53*	*12*	*16*	*37*	*-5.26*	*-3.65*
Inferior Frontal Gyrus	*9*	*R*	*55*	*5*	*30*	*34*	*-5.05*	*-3.65*
Middle Frontal Gyrus	*10*	*R*	*32*	*60*	*13*	*52*	*-4.92*	*-3.48*
Middle Frontal Gyrus	*10*	*R*	*42*	*56*	*7*	*86*	*-4.35*	*-3.58*
Middle Frontal Gyrus	*6*	*R*	*25*	*24*	*54*	*68*	*-4.25*	*-3.52*
Inferior Frontal Gyrus	*47*	*R*	*55*	*30*	*-9*	*22*	*-3.85*	*-3.33*
Paracentral Lobule	**31**	**R**	**3**	**-20**	**41**	**463**	**-7.26**	**-3.84**

Paracentral Lobule	*31*	*R*	*3*	*-20*	*41*	*369*	*-7.26*	*-3.99*
Paracentral Lobule	*5*	*R*	*10*	*-36*	*54*	*60*	*-4.16*	*-3.38*
Medial Frontal Gyrus	*6*	*R*	*3*	*-26*	*64*	*32*	*-3.74*	*-3.10*
Superior Frontal Gyrus	**8**	**R**	**11**	**34**	**46**	**102**	**-6.94**	**-3.70**
Superior Frontal Gyrus	*8*	*R*	*11*	*34*	*46*	*74*	*-6.94*	*-3.82*
Medial Frontal Gyrus	*9*	*R*	*8*	*41*	*25*	*28*	*-4.08*	*-3.39*
Inferior Temporal Gyrus	**20**	**R**	**56**	**-12**	**-23**	**64**	**-6.70**	**-3.96**
Superior Temporal Gyrus	**41**	**L**	**-37**	**-31**	**14**	**2091**	**-6.70**	**-3.81**
Superior Temporal Gyrus	*41*	*L*	*-37*	*-31*	*14*	*139*	*-6.70*	*-4.19*
Superior Temporal Gyrus	*22*	*L*	*-63*	*-38*	*10*	*179*	*-6.53*	*-3.83*
Transverse Temporal Gyrus	*42*	*L*	*-59*	*-10*	*10*	*132*	*-6.17*	*-3.84*
Superior Temporal Gyrus	*41*	*L*	*-53*	*-26*	*9*	*170*	*-6.01*	*-4.09*
Inferior Parietal Lobule	*40*	*L*	*-38*	*-53*	*50*	*97*	*-5.92*	*-3.79*
Superior Temporal Gyrus	*41*	*L*	*-45*	*-35*	*14*	*67*	*-5.84*	*-4.27*
Inferior Parietal Lobule	*40*	*L*	*-49*	*-46*	*43*	*166*	*-5.79*	*-4.34*
Inferior Parietal Lobule	*40*	*L*	*-57*	*-35*	*36*	*75*	*-5.59*	*-4.25*
Inferior Parietal Lobule	*40*	*L*	*-44*	*-32*	*34*	*62*	*-5.40*	*-3.92*
Supramarginal Gyrus	*40*	*L*	*-55*	*-49*	*28*	*118*	*-5.25*	*-4.07*
Inferior Parietal Lobule	*40*	*L*	*-46*	*-31*	*54*	*68*	*-5.13*	*-3.66*
Inferior Parietal Lobule	*40*	*L*	*-63*	*-32*	*26*	*53*	*-5.11*	*-3.49*
Postcentral Gyrus	*1*	*L*	*-61*	*-16*	*29*	*65*	*-5.08*	*-3.60*
Fusiform Gyrus	*37*	*L*	*-48*	*-57*	*-14*	*103*	*-4.94*	*-3.47*
Fusiform Gyrus	*37*	*L*	*-37*	*-57*	*-12*	*58*	*-4.94*	*-3.69*
Superior Parietal Lobule	*7*	*L*	*-35*	*-61*	*50*	*60*	*-4.89*	*-3.48*
Inferior Parietal Lobule	*40*	*L*	*-36*	*-34*	*37*	*36*	*-4.69*	*-3.85*
Postcentral Gyrus	*2*	*L*	*-55*	*-24*	*36*	*107*	*-4.68*	*-3.75*
Inferior Parietal Lobule	*40*	*L*	*-33*	*-36*	*45*	*88*	*-4.62*	*-3.44*
Lingual Gyrus	*18*	*L*	*-29*	*-71*	*-7*	*59*	*-4.30*	*-3.33*
Middle Temporal Gyrus	*20*	*L*	*-52*	*-37*	*-10*	*51*	*-4.10*	*-3.24*
Superior Temporal Gyrus	*22*	*L*	*-63*	*-41*	*18*	*30*	*-3.85*	*-3.30*
Cerebellum	*Declive*	*L*	*-35*	*-69*	*-15*	*23*	*-3.78*	*-3.34*
Inferior Temporal Gyrus	*20*	*L*	*-58*	*-45*	*-12*	*32*	*-3.55*	*-3.06*
Postcentral Gyrus	*3*	*L*	*-36*	*-30*	*63*	*23*	*-3.21*	*-2.96*
Middle Temporal Gyrus	**21**	**R**	**61**	**-43**	**0**	**296**	**-6.47**	**-3.53**
Middle Temporal Gyrus	*21*	*R*	*61*	*-43*	*0*	*55*	*-6.47*	*-3.58*
Inferior Temporal Gyrus	*20*	*R*	*53*	*-50*	*-13*	*73*	*-6.16*	*-3.49*
Middle Temporal Gyrus	*21*	*R*	*67*	*-23*	*-2*	*33*	*-5.48*	*-3.39*
Middle Temporal Gyrus	*20*	*R*	*58*	*-39*	*-12*	*51*	*-5.06*	*-3.76*
Middle Temporal Gyrus	*21*	*R*	*61*	*-34*	*0*	*46*	*-4.69*	*-3.48*
Middle Temporal Gyrus	*21*	*R*	*58*	*-31*	*-7*	*36*	*-4.56*	*-3.47*
Cerebellum	**Pyramis**	**L**	**-12**	**-71**	**-24**	**347**	**-6.46**	**-3.65**
Cerebellum	*Pyramis*	*L*	*-12*	*-71*	*-24*	*103*	*-6.46*	*-3.93*
Cerebellum	*Inf. Semi-Lunar*	*L*	*-34*	*-78*	*-39*	*116*	*-6.31*	*-3.86*
Cerebellum	*Tonsil*	*L*	*-38*	*-61*	*-42*	*37*	*-4.45*	*-3.28*
Cerebellum	*Inf. Semi-Lunar*	*L*	*-48*	*-62*	*-38*	*31*	*-4.40*	*-3.45*
Cerebellum	*Tuber*	*L*	*-42*	*-85*	*-30*	*34*	*-4.37*	*-3.07*
Cerebellum	*Inf. Semi-Lunar*	*L*	*-15*	*-69*	*-41*	*23*	*-3.63*	*-3.09*
Middle Frontal Gyrus	**47**	**L**	**-48**	**46**	**-8**	**108**	**-6.13**	**-3.59**
Lingual Gyrus	**18**	**L**	**-4**	**-93**	**-8**	**153**	**-6.02**	**-3.68**
Lingual Gyrus	*18*	*L*	*-4*	*-93*	*-8*	*68*	*-6.02*	*-3.89*
Cuneus	*18*	*L*	*-16*	*-99*	*6*	*60*	*-5.65*	*-3.56*
Middle Occipital Gyrus	*18*	*L*	*-16*	*-99*	*15*	*21*	*-4.45*	*-3.47*
Cerebellum	**Inf. Semi-Lunar**	**R**	**30**	**-79**	**-39**	**20**	**-4.23**	**-3.22**
Inferior Frontal Gyrus	**46**	**L**	**-47**	**41**	**13**	**21**	**-4.05**	**-3.20**

Maintain (Positive & Negative)> Cease

Activations								
Precentral Gyrus	**4**	**L**	**-48**	**-6**	**45**	**108**	**7.49**	**3.93**
Precentral Gyrus	*4*	*L*	*-48*	*-6*	*45*	*87*	*7.49*	*4.11*
Middle Frontal Gyrus	*6*	*L*	*-37*	*-2*	*50*	*21*	*3.84*	*3.16*
Superior Frontal Gyrus	**6**	**L**	**-4**	**6**	**58**	**315**	**6.10**	**3.62**
Superior Frontal Gyrus	*6*	*L*	*-4*	*6*	*58*	*131*	*6.10*	*3.88*
Superior Frontal Gyrus	*6*	*R*	*11*	*8*	*60*	*55*	*5.53*	*3.58*
Superior Frontal Gyrus	*6*	*L*	*-7*	*-3*	*67*	*20*	*5.07*	*3.52*
Superior Frontal Gyrus	*6*	*R*	*3*	*8*	*49*	*40*	*4.59*	*3.47*
Cingulate Gyrus	*32*	*L*	*0*	*15*	*36*	*68*	*4.21*	*3.26*
Middle Temporal Gyrus	**19**	**L**	**-43**	**-61**	**11**	**835**	**5.78**	**3.38**
Middle Temporal Gyrus	*19*	*L*	*-43*	*-61*	*11*	*54*	*5.78*	*3.83*
Cerebellum	*Culmen*	*L*	*-30*	*-50*	*-15*	*54*	*5.58*	*3.58*
Middle Temporal Gyrus	*39*	*L*	*-53*	*-68*	*12*	*74*	*5.13*	*3.72*
Middle Temporal Gyrus	*39*	*L*	*-45*	*-71*	*18*	*39*	*4.86*	*3.47*
Parahippocampal Gyrus	*36*	*L*	*-24*	*-37*	*-6*	*60*	*4.80*	*3.36*
Fusiform Gyrus	*37*	*L*	*-29*	*-35*	*-13*	*63*	*4.79*	*3.42*
Precuneus	*31*	*L*	*-22*	*-80*	*29*	*72*	*4.50*	*3.30*
Cerebellum	*Declive*	*L*	*-35*	*-63*	*-10*	*55*	*4.45*	*3.33*
Posterior Cingulate	*29*	*L*	*-6*	*-50*	*6*	*61*	*4.26*	*3.39*
Middle Temporal Gyrus	*39*	*L*	*-45*	*-76*	*11*	*71*	*4.25*	*3.35*
Posterior Cingulate	*30*	*L*	*0*	*-48*	*15*	*56*	*4.23*	*3.17*
Middle Temporal Gyrus	*37*	*L*	*-43*	*-60*	*-4*	*31*	*4.09*	*3.19*
Parahippocampal Gyrus	*27*	*L*	*-11*	*-36*	*1*	*32*	*4.07*	*3.18*
Middle Occipital Gyrus	*19*	*L*	*-36*	*-85*	*17*	*47*	*3.76*	*3.11*
Middle Occipital Gyrus	*19*	*L*	*-28*	*-75*	*20*	*34*	*3.75*	*3.20*
Cerebellum	*Culmen*	*L*	*-14*	*-42*	*-13*	*23*	*3.66*	*3.11*
Middle Temporal Gyrus	**39**	**R**	**42**	**-58**	**9**	**454**	**5.65**	**3.46**
Middle Temporal Gyrus	*19*	*R*	*40*	*-61*	*13*	*1*	*2.76*	*2.76*
Middle Occipital Gyrus	*18*	*R*	*11*	*-97*	*13*	*83*	*5.53*	*3.75*
Middle Temporal Gyrus	*39*	*R*	*42*	*-58*	*9*	*100*	*5.65*	*3.37*
Middle Occipital Gyrus	*19*	*R*	*39*	*-85*	*21*	*48*	*5.45*	*3.58*
Middle Occipital Gyrus	*18*	*R*	*27*	*-87*	*14*	*36*	*5.06*	*3.65*
Cuneus	*19*	*R*	*8*	*-86*	*27*	*48*	*4.90*	*3.33*
Middle Occipital Gyrus	*18*	*R*	*20*	*-90*	*14*	*24*	*4.79*	*3.61*
Cuneus	*7*	*R*	*21*	*-77*	*33*	*57*	*4.39*	*3.36*
Middle Occipital Gyrus	*19*	*R*	*45*	*-79*	*10*	*28*	*3.84*	*3.11*
Precentral Gyrus	**4**	**R**	**48**	**-12**	**40**	**91**	**5.46**	**3.30**
Precentral Gyrus	*4*	*R*	*48*	*-12*	*40*	*61*	*5.46*	*3.42*
Postcentral Gyrus	*4*	*R*	*37*	*-18*	*45*	*21*	*3.70*	*3.06*
Precentral Gyrus	**6**	**L**	**-54**	**2**	**16**	**69**	**4.87**	**3.33**
Precentral Gyrus	*6*	*L*	*-54*	*2*	*16*	*44*	*4.87*	*3.34*
Precentral Gyrus	*44*	*L*	*-52*	*5*	*9*	*25*	*4.13*	*3.32*
Superior Frontal Gyrus	**9**	**L**	**-8**	**52**	**32**	**40**	**4.41**	**3.32**
Anterior Cingulate	**24**	**L**	**-6**	**29**	**-1**	**24**	**3.97**	**3.15**
Inferior Frontal Gyrus	**47**	**L**	**-49**	**23**	**3**	**21**	**3.92**	**3.09**
Cuneus	**18**	**L**	**-10**	**-99**	**8**	**28**	**3.84**	**3.14**
Medial Frontal Gyrus	**11**	**L**	**0**	**34**	**-14**	**20**	**3.63**	**3.04**
Fusiform Gyrus	**37**	**R**	**28**	**-68**	**-2**	**21**	**3.43**	**2.98**
Deactivations								
Superior Temporal Gyrus	**39**	**R**	**45**	**-55**	**34**	**921**	**-6.04**	**-3.86**
Superior Temporal Gyrus	*39*	*R*	*45*	*-55*	*34*	*182*	*-6.04*	*-4.59*

Precuneus	*7*	*R*	*9*	*-63*	*35*	*55*	*-5.91*	*-3.82*
Supramarginal Gyrus	*40*	*R*	*56*	*-52*	*31*	*116*	*-5.58*	*-4.05*
Inferior Parietal Lobule	*7*	*R*	*39*	*-60*	*42*	*160*	*-5.54*	*-4.08*
Superior Parietal Lobule	*7*	*R*	*25*	*-62*	*42*	*46*	*-5.09*	*-3.65*
Cingulate Gyrus	*31*	*R*	*3*	*-30*	*41*	*62*	*-4.72*	*-3.56*
Inferior Parietal Lobule	*40*	*R*	*47*	*-42*	*50*	*81*	*-4.37*	*-3.51*
Precuneus	*7*	*R*	*3*	*-62*	*42*	*46*	*-4.28*	*-3.41*
Supramarginal Gyrus	*40*	*R*	*53*	*-50*	*22*	*43*	*-4.25*	*-3.45*
Cingulate Gyrus	*31*	*R*	*13*	*-34*	*35*	*26*	*-3.88*	*-3.14*
Superior Parietal Lobule	*7*	*R*	*36*	*-58*	*51*	*31*	*-3.79*	*-3.18*
Precuneus	*7*	*L*	*-9*	*-59*	*35*	*26*	*-3.76*	*-3.17*
Precuneus	*31*	*R*	*13*	*-47*	*35*	*32*	*-3.45*	*-3.10*
Middle Frontal Gyrus	**9**	**R**	**46**	**19**	**33**	**413**	**-5.88**	**-3.69**
Middle Frontal Gyrus	*9*	*R*	*46*	*19*	*33*	*155*	*-5.88*	*-3.76*
Middle Frontal Gyrus	*6*	*R*	*43*	*13*	*49*	*79*	*-5.61*	*-3.99*
Middle Frontal Gyrus	*8*	*R*	*28*	*18*	*50*	*92*	*-4.98*	*-3.59*
Middle Frontal Gyrus	*46*	*R*	*44*	*38*	*16*	*86*	*-4.74*	*-3.40*
Inferior Parietal Lobule	**40**	**L**	**-36**	**-49**	**39**	**496**	**-5.85**	**-3.59**
Inferior Parietal Lobule	*40*	*L*	*-36*	*-49*	*39*	*151*	*-5.85*	*-4.07*
Superior Parietal Lobule	*7*	*L*	*-22*	*-66*	*43*	*34*	*-5.41*	*-3.38*
Inferior Parietal Lobule	*40*	*L*	*-41*	*-34*	*39*	*67*	*-4.74*	*-3.59*
Supramarginal Gyrus	*40*	*L*	*-47*	*-49*	*33*	*82*	*-4.65*	*-3.41*
Inferior Parietal Lobule	*40*	*L*	*-46*	*-31*	*47*	*76*	*-4.49*	*-3.33*
Precuneus	*39*	*L*	*-31*	*-64*	*36*	*84*	*-3.85*	*-3.26*
Inferior Occipital Gyrus	**18**	**R**	**30**	**-92**	**-2**	**118**	**-5.56**	**-3.98**
Inferior Occipital Gyrus	*18*	*R*	*30*	*-92*	*-2*	*76*	*-5.56*	*-4.17*
Middle Occipital Gyrus	*18*	*R*	*34*	*-81*	*-7*	*42*	*-4.92*	*-3.64*
Cerebellum	**Tuber**	**L**	**-43**	**-70**	**-25**	**111**	**-5.35**	**-3.18**
Cerebellum	*Tuber*	*L*	*-43*	*-70*	*-25*	*33*	*-5.35*	*-3.36*
Cerebellum	*Tonsil*	*L*	*-48*	*-62*	*-41*	*49*	*-3.68*	*-3.12*
Cerebellum	*Tuber*	*L*	*-41*	*-61*	*-30*	*24*	*-3.68*	*-3.14*
Cerebellum	**Declive**	**L**	**-6**	**-68**	**-21**	**84**	**-5.10**	**-3.60**
Middle Frontal Gyrus	**10**	**L**	**-41**	**42**	**18**	**53**	**-4.79**	**-3.37**
Middle Frontal Gyrus	**11**	**R**	**35**	**53**	**-10**	**162**	**-4.66**	**-3.20**
Middle Frontal Gyrus	*11*	*R*	*35*	*53*	*-10*	*74*	*-4.66*	*-3.43*
Middle Frontal Gyrus	*47*	*R*	*51*	*45*	*-5*	*27*	*-3.44*	*-3.05*
Middle Frontal Gyrus	*10*	*R*	*35*	*56*	*1*	*35*	*-3.41*	*-3.02*
Sub-Gyral	*10*	*R*	*42*	*44*	*-1*	*26*	*-3.15*	*-2.95*
Medial Frontal Gyrus	**8**	**R**	**7**	**32**	**39**	**54**	**-4.52**	**-3.38**
Middle Temporal Gyrus	**21**	**R**	**61**	**-25**	**-7**	**52**	**-4.27**	**-3.20**
Middle Temporal Gyrus	*21*	*R*	*61*	*-25*	*-7*	*26*	*-4.27*	*-3.27*
Middle Temporal Gyrus	*21*	*R*	*64*	*-29*	*0*	*26*	*-3.77*	*-3.13*
Middle Temporal Gyrus	**21**	**L**	**-58**	**-37**	**-7**	**43**	**-4.09**	**-3.18**
Middle Occipital Gyrus	**18**	**L**	**-29**	**-87**	**-3**	**67**	**-4.03**	**-3.16**
Middle Frontal Gyrus	**10**	**R**	**29**	**52**	**22**	**21**	**-3.58**	**-3.00**
Maintain & Cease (Positive & Negative)> Regulate								
Activations								
Middle Frontal Gyrus	**8**	**L**	**-26**	**34**	**39**	**3972**	**12.76**	**4.13**
Middle Frontal Gyrus	*8*	*L*	*-26*	*34*	*39*	*216*	*12.76*	*5.11*
Medial Frontal Gyrus	*10*	*L*	*-8*	*63*	*3*	*126*	*9.77*	*4.62*
Medial Frontal Gyrus	*10*	*L*	*-3*	*48*	*-10*	*203*	*9.46*	*5.74*
Medial Frontal Gyrus	*10*	*R*	*12*	*52*	*10*	*164*	*8.48*	*4.69*
Posterior Cingulate	*29*	*L*	*-6*	*-50*	*6*	*123*	*8.21*	*4.50*
Posterior Cingulate	*23*	*L*	*-6*	*-55*	*18*	*151*	*7.77*	*4.84*

Medial Frontal Gyrus	*10*	*L*	*-10*	*40*	*-12*	*167*	*7.76*	*5.07*
Cingulate Gyrus	*31*	*L*	*-6*	*-35*	*30*	*113*	*7.72*	*4.82*
Precuneus	*31*	*L*	*-12*	*-49*	*25*	*45*	*7.43*	*4.98*
Anterior Cingulate	*32*	*L*	*-4*	*47*	*1*	*180*	*7.29*	*4.82*
Posterior Cingulate	*29*	*R*	*6*	*-46*	*17*	*111*	*6.94*	*4.50*
Posterior Cingulate	*29*	*L*	*-6*	*-42*	*20*	*26*	*6.94*	*5.86*
Cingulate Gyrus	*31*	*L*	*-9*	*-36*	*38*	*53*	*6.70*	*4.10*
Thalamus	*Anterior*	*L*	*-3*	*-7*	*6*	*55*	*6.44*	*4.36*
Superior Frontal Gyrus	*9*	*L*	*-12*	*52*	*35*	*95*	*6.43*	*4.09*
Middle Frontal Gyrus	*8*	*L*	*-25*	*20*	*37*	*69*	*6.29*	*4.50*
Sub-lobarAmygdala		*L*	*-24*	*-10*	*-9*	*212*	*6.04*	*3.60*
Superior Frontal Gyrus	*9*	*L*	*-8*	*57*	*26*	*90*	*5.91*	*4.20*
Basal Ganglia	*N. Accumbens*	*R*	*6*	*10*	*2*	*54*	*5.72*	*3.71*
Middle Temporal Gyrus	*21*	*L*	*-62*	*-8*	*-4*	*98*	*5.45*	*3.81*
Anterior Cingulate	*32*	*R*	*11*	*44*	*-1*	*119*	*5.26*	*4.26*
Superior Temporal Gyrus	*41*	*L*	*-45*	*-32*	*9*	*56*	*5.14*	*3.43*
Cerebellum	*Culmen*	*L*	*0*	*-51*	*-11*	*146*	*5.12*	*3.50*
Thalamus	*Medial*	*L*	*-3*	*-11*	*16*	*58*	*4.82*	*3.62*
Cerebellum	*Culmen*	*L*	*-8*	*-48*	*-9*	*132*	*4.65*	*3.40*
Thalamus	*Medial*	*L*	*-6*	*-15*	*4*	*24*	*4.65*	*3.20*
Middle Temporal Gyrus	*21*	*L*	*-61*	*-22*	*-9*	*42*	*4.65*	*3.62*
Thalamus	*Lateral*	*R*	*3*	*-4*	*12*	*27*	*4.64*	*3.58*
Middle Temporal Gyrus	*21*	*L*	*-53*	*-14*	*-7*	*76*	*4.57*	*3.53*
Superior Frontal Gyrus	*9*	*R*	*4*	*49*	*32*	*61*	*4.57*	*3.45*
Cerebellum	*Culmen*	*R*	*3*	*-42*	*-5*	*83*	*4.48*	*3.25*
Anterior Cingulate	*25*	*R*	*3*	*3*	*-3*	*43*	*4.44*	*3.40*
Anterior Cingulate	*25*	*L*	*-3*	*16*	*-6*	*70*	*4.43*	*3.24*
Hippocampus	*Posterior*	*L*	*-33*	*-30*	*-6*	*23*	*4.42*	*3.40*
Insula	*Ventral Anterior*	*L*	*-19*	*4*	*-7*	*33*	*4.39*	*3.19*
Inferior Frontal Gyrus	*47*	*L*	*-28*	*7*	*-13*	*82*	*4.24*	*3.23*
Basal Ganglia	*Putamen*	*L*	*-31*	*-13*	*0*	*22*	*4.19*	*3.25*
Middle Temporal Gyrus	*21*	*L*	*-54*	*-1*	*-16*	*76*	*4.18*	*3.28*
Anterior Cingulate	*24*	*L*	*0*	*33*	*15*	*94*	*4.12*	*3.36*
Anterior Cingulate	*33*	*R*	*7*	*21*	*21*	*21*	*4.03*	*3.25*
Inferior Frontal Gyrus	*47*	*L*	*-37*	*29*	*-13*	*68*	*3.98*	*3.17*
Hippocampus	*Posterior*	*L*	*-29*	*-26*	*-13*	*51*	*3.80*	*3.17*
Superior Temporal Gyrus	*13*	*L*	*-45*	*-20*	*7*	*40*	*3.74*	*3.14*
Superior Temporal Gyrus	*41*	*L*	*-56*	*-24*	*10*	*45*	*3.66*	*3.18*
Thalamus	*Pulvinar*	*R*	*15*	*-32*	*8*	*68*	*3.66*	*3.10*
Superior Temporal Gyrus	*38*	*L*	*-47*	*16*	*-20*	*24*	*3.37*	*3.04*
Middle Temporal Gyrus	*21*	*L*	*-36*	*-6*	*-28*	*22*	*3.34*	*3.02*
Middle Temporal Gyrus	**39**	**R**	**53**	**-69**	**17**	**349**	**8.21**	**3.93**
Middle Temporal Gyrus	*39*	*R*	*53*	*-69*	*17*	*96*	*8.21*	*4.93*
Middle Temporal Gyrus	*39*	*R*	*59*	*-63*	*11*	*40*	*6.01*	*3.96*
Middle Occipital Gyrus	*19*	*R*	*48*	*-76*	*6*	*53*	*5.62*	*3.91*
Middle Temporal Gyrus	*39*	*R*	*51*	*-71*	*24*	*42*	*5.15*	*3.51*
Inferior Temporal Gyrus	*37*	*R*	*51*	*-68*	*1*	*54*	*4.50*	*3.37*
Middle Temporal Gyrus	*39*	*R*	*40*	*-67*	*17*	*42*	*4.29*	*3.16*
Middle Temporal Gyrus	*37*	*R*	*45*	*-60*	*1*	*22*	*3.86*	*3.17*
Middle Temporal Gyrus	**39**	**L**	**-39**	**-71**	**27**	**400**	**7.42**	**4.30**
Middle Temporal Gyrus	*39*	*L*	*-39*	*-71*	*27*	*372*	*7.42*	*4.36*
Superior Occipital Gyrus	*19*	*L*	*-36*	*-84*	*26*	*28*	*4.41*	*3.49*
Middle Frontal Gyrus	**8**	**R**	**26**	**31**	**40**	**65**	**5.68**	**3.52**
Superior Temporal Gyrus	**38**	**R**	**36**	**11**	**-19**	**322**	**5.60**	**3.44**
Superior Temporal Gyrus	*38*	*R*	*36*	*11*	*-19*	*132*	*5.60*	*3.65*

Superior Temporal Gyrus	*38*	*R*	*45*	*17*	*-21*	*54*	*5.03*	*3.62*
Hippocampus	*Anterior*	*R*	*28*	*-11*	*-11*	*78*	*4.92*	*3.30*
Inferior Frontal Gyrus	*47*	*R*	*41*	*31*	*-13*	*20*	*3.43*	*3.03*
Superior Temporal Gyrus	*38*	*R*	*26*	*-3*	*-24*	*31*	*3.18*	*2.95*
Sub-Gyral	**40**	**R**	**28**	**-41**	**51**	**62**	**5.37**	**3.26**
Insula	**13**	**R**	**43**	**-14**	**6**	**382**	**5.03**	**3.33**
Insula	*13*	*R*	*43*	*-14*	*6*	*78*	*5.03*	*3.59*
Middle Temporal Gyrus	*21*	*R*	*62*	*-16*	*-11*	*38*	*4.79*	*3.53*
Superior Temporal Gyrus	*22*	*R*	*63*	*-6*	*4*	*78*	*4.49*	*3.30*
Superior Temporal Gyrus	*41*	*R*	*51*	*-20*	*7*	*102*	*4.36*	*3.31*
Middle Temporal Gyrus	*21*	*R*	*63*	*-5*	*-6*	*24*	*3.76*	*3.25*
Transverse Gyrus	*41*	*R*	*42*	*-31*	*13*	*23*	*3.64*	*3.04*
Insula	*13*	*R*	*43*	*-17*	*19*	*34*	*3.63*	*2.99*
Postcentral Gyrus	**3**	**R**	**34**	**-21**	**45**	**148**	**4.77**	**3.29**
Postcentral Gyrus	*3*	*R*	*34*	*-21*	*45*	*43*	*4.77*	*3.46*
Precentral Gyrus	*6*	*R*	*35*	*-8*	*58*	*69*	*4.39*	*3.28*
Postcentral Gyrus	*3*	*R*	*43*	*-16*	*52*	*34*	*3.58*	*3.11*
Cerebellum	**Uvula**	**R**	**16**	**-76**	**-34**	**92**	**4.64**	**3.37**
Cerebellum	*Uvula*	*R*	*16*	*-76*	*-34*	*52*	*4.64*	*3.50*
Cerebellum	*Pyramis*	*R*	*26*	*-73*	*-28*	*40*	*3.77*	*3.19*
Cingulate Gyrus	**24**	**L**	**0**	**-8**	**35**	**78**	**4.63**	**3.26**
Cerebellum	**Tonsil**	**R**	**13**	**-43**	**-42**	**33**	**4.59**	**3.13**
Postcentral Gyrus	**3**	**R**	**57**	**-12**	**42**	**23**	**4.19**	**3.12**
Precentral Gyrus	**4**	**R**	**23**	**-22**	**68**	**38**	**4.14**	**3.24**
Cerebellum	**Tonsil**	**L**	**-16**	**-46**	**-41**	**37**	**4.02**	**3.04**
Paracentral Lobule	**6**	**R**	**7**	**-27**	**52**	**49**	**3.63**	**3.00**
Medial Frontal Gyrus	*6*	*R*	*3*	*-23*	*52*	*1*	*2.76*	*2.76*
Medial Frontal Gyrus	*6*	*L*	*-7*	*-17*	*59*	*20*	*3.46*	*2.99*
Deactivations								
Inferior Parietal Lobule	**40**	**R**	**39**	**-45**	**40**	**2208**	**-11.02**	**-4.85**
Inferior Parietal Lobule	*40*	*R*	*39*	*-45*	*40*	*589*	*-11.02*	*-5.57*
Inferior Parietal Lobule	*40*	*L*	*-39*	*-49*	*36*	*238*	*-10.09*	*-5.46*
Inferior Parietal Lobule	*40*	*L*	*-43*	*-42*	*49*	*249*	*-8.53*	*-4.65*
Inferior Parietal Lobule	*40*	*R*	*53*	*-44*	*27*	*144*	*-8.13*	*-4.63*
Precuneus	*7*	*R*	*9*	*-65*	*39*	*235*	*-7.92*	*-4.24*
Inferior Parietal Lobule	*40*	*R*	*53*	*-35*	*43*	*177*	*-7.64*	*-5.07*
Inferior Parietal Lobule	*7*	*L*	*-33*	*-57*	*46*	*236*	*-7.53*	*-4.58*
Supramarginal Gyrus	*40*	*R*	*56*	*-38*	*27*	*94*	*-6.54*	*-4.48*
Precuneus	*7*	*L*	*-6*	*-67*	*42*	*140*	*-5.71*	*-3.86*
Precuneus	*7*	*L*	*-6*	*-62*	*52*	*78*	*-4.85*	*-3.61*
Precuneus	*31*	*L*	*-25*	*-67*	*25*	*28*	*-3.80*	*-3.29*
Cerebellum	**Declive**	**L**	**-35**	**-66**	**-12**	**1262**	**-7.30**	**-3.86**
Cerebellum	*Declive*	*L*	*-35*	*-66*	*-12*	*118*	*-7.30*	*-4.65*
Cuneus	*17*	*R*	*14*	*-95*	*7*	*82*	*-7.22*	*-4.23*
Lingual Gyrus	*18*	*R*	*25*	*-75*	*-7*	*113*	*-7.15*	*-3.87*
Cuneus	*18*	*L*	*-16*	*-99*	*4*	*111*	*-6.83*	*-4.17*
Cerebellum	*Declive*	*L*	*-34*	*-57*	*-12*	*82*	*-6.66*	*-4.35*
Lingual Gyrus	*18*	*L*	*-32*	*-71*	*-4*	*85*	*-6.38*	*-3.86*
Fusiform Gyrus	*37*	*R*	*33*	*-48*	*-14*	*40*	*-6.05*	*-3.57*
Lingual Gyrus	*18*	*R*	*5*	*-86*	*-1*	*110*	*-5.39*	*-3.63*
Lingual Gyrus	*18*	*L*	*-10*	*-84*	*-8*	*119*	*-5.39*	*-3.70*
Cerebellum	*Uvula*	*L*	*-32*	*-65*	*-23*	*54*	*-5.19*	*-3.68*
Cerebellum	*Declive*	*L*	*-41*	*-63*	*-20*	*68*	*-5.15*	*-3.97*
Middle Occipital Gyrus	*18*	*R*	*20*	*-98*	*9*	*23*	*-5.10*	*-3.67*

Middle Occipital Gyrus	*19*	*L*	*-28*	*-85*	*11*	*29*	*-4.85*	*-3.50*
Fusiform Gyrus	*37*	*R*	*37*	*-58*	*-12*	*64*	*-4.35*	*-3.27*
Inferior Temporal Gyrus	*20*	*L*	*-51*	*-54*	*-13*	*59*	*-4.33*	*-3.45*
Fusiform Gyrus	*37*	*L*	*-43*	*-54*	*-6*	*50*	*-4.13*	*-3.44*
Cerebellum	*Culmen*	*L*	*-29*	*-46*	*-26*	*20*	*-3.47*	*-3.10*
Cerebellum	*Culmen*	*L*	*-42*	*-48*	*-26*	*27*	*-3.42*	*-3.07*
Middle Frontal Gyrus	**46**	**R**	**55**	**33**	**22**	**936**	**-7.16**	**-3.76**
Middle Frontal Gyrus	*46*	*R*	*55*	*33*	*22*	*46*	*-7.16*	*-4.65*
Superior Frontal Gyrus	*9*	*R*	*44*	*35*	*29*	*141*	*-6.53*	*-4.50*
Middle Frontal Gyrus	*6*	*R*	*37*	*14*	*52*	*94*	*-6.03*	*-3.72*
Precentral Gyrus	*44*	*R*	*53*	*9*	*9*	*89*	*-5.46*	*-3.54*
Middle Frontal Gyrus	*46*	*R*	*44*	*38*	*16*	*63*	*-5.34*	*-3.94*
Inferior Frontal Gyrus	*9*	*R*	*53*	*5*	*28*	*63*	*-5.28*	*-3.61*
Precentral Gyrus	*9*	*R*	*42*	*7*	*33*	*62*	*-5.27*	*-3.74*
Superior Frontal Gyrus	*9*	*R*	*36*	*51*	*25*	*69*	*-4.89*	*-3.68*
Middle Frontal Gyrus	*6*	*R*	*50*	*6*	*40*	*57*	*-4.52*	*-3.29*
Inferior Frontal Gyrus	*46*	*R*	*53*	*42*	*1*	*51*	*-4.39*	*-3.49*
Superior Frontal Gyrus	*6*	*R*	*25*	*7*	*58*	*68*	*-4.35*	*-3.36*
Middle Frontal Gyrus	*10*	*R*	*48*	*51*	*-6*	*28*	*-4.29*	*-3.38*
Middle Frontal Gyrus	*46*	*R*	*51*	*46*	*8*	*54*	*-4.26*	*-3.55*
Superior Frontal Gyrus	*6*	*R*	*15*	*17*	*54*	*26*	*-4.02*	*-3.15*
Inferior Frontal Gyrus	*9*	*R*	*61*	*14*	*23*	*25*	*-3.94*	*-3.20*
Cerebellum	**Inf. Semi-Lunar**	**L**	**-26**	**-71**	**-42**	**152**	**-7.14**	**-3.59**
Cerebellum	*Inf. Semi-Lunar*	*L*	*-26*	*-71*	*-42*	*47*	*-7.14*	*-3.62*
Cerebellum	*Inf. Semi-Lunar*	*L*	*-36*	*-67*	*-49*	*62*	*-6.87*	*-3.68*
Cerebellum	*Tonsil*	*L*	*-39*	*-51*	*-44*	*43*	*-4.73*	*-3.43*
Middle Frontal Gyrus	**46**	**L**	**-44**	**41**	**18**	**270**	**-6.25**	**-3.85**
Middle Frontal Gyrus	*46*	*L*	*-44*	*41*	*18*	*172*	*-6.25*	*-3.87*
Middle Frontal Gyrus	*9*	*L*	*-46*	*35*	*32*	*98*	*-5.43*	*-3.82*
Superior Frontal Gyrus	**11**	**L**	**-28**	**53**	**-14**	**62**	**-5.47**	**-3.35**
Superior Frontal Gyrus	*11*	*L*	*-28*	*53*	*-14*	*34*	*-5.47*	*-3.46*
Middle Frontal Gyrus	*10*	*L*	*-38*	*56*	*-10*	*25*	*-4.32*	*-3.25*
Precentral Gyrus	**6**	**L**	**-47**	**5**	**35**	**46**	**-4.58**	**-3.28**
Middle Frontal Gyrus	**6**	**L**	**-32**	**-2**	**48**	**20**	**-3.90**	**-3.20**
Middle Temporal Gyrus	**21**	**R**	**58**	**-37**	**-5**	**33**	**-3.86**	**-3.20**

Table A3.1.2: Main contrasts for the model based component process deconstruction analyses (Experiment 1 only). Cluster maxima are reported in bold. Italicised entries denote local maxima of subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the *splitclustercoords* function). Results were thresholded at $p<.005$ and $k>20$.

Table A3.1.3. Results from contrast-based decomposition approach

Region	Label/BA	Side	MNI x	MNI y	MNI z	Extent (voxel)	*t*-value (max)	*t*-value (avg)
		Generation						
Midbrain	**Red Nucleus**	**L**	**0**	**-16**	**-4**	**1591**	**8.87**	**3.75**
Midbrain	*Red Nucleus*	*L*	*0*	*-16*	*-4*	*169*	*8.87*	*5.03*
Cerbellum	*Culmen*	*L*	*-8*	*-36*	*-9*	*54*	*6.16*	*4.07*
Hippcampus	*Mid*	*L*	*-33*	*-21*	*-8*	*48*	*6.08*	*4.18*
Thalamus	*Pulvinar*	*L*	*-3*	*-25*	*1*	*100*	*5.93*	*4.06*
Parahippocampal Gyrus	*35*	*L*	*-17*	*-26*	*-9*	*65*	*5.46*	*3.69*
Thalamus	*Ventral Lateral*	*R*	*18*	*-11*	*4*	*51*	*5.43*	*3.70*
Hippcampus	*Mid*	*R*	*36*	*-26*	*-10*	*31*	*5.42*	*3.62*
Midbrain	*Substantia Nigra*	*R*	*12*	*-11*	*-9*	*90*	*5.29*	*3.99*
Midbrain	*Substantia Nigra*	*L*	*-12*	*-13*	*-7*	*60*	*5.25*	*4.11*
Thalamus	*Pulvinar*	*L*	*-5*	*-36*	*2*	*32*	*5.19*	*3.93*
Basal Ganglia	*Putamen*	*L*	*-16*	*10*	*0*	*52*	*5.09*	*3.49*
Basal Ganglia	*Lat. Globus Pallidus*	*R*	*16*	*3*	*1*	*22*	*5.07*	*3.55*
Thalamus	*Anterior*	*L*	*-3*	*-8*	*2*	*27*	*5.03*	*3.48*
Superior Temporal Gyrus	*38*	*L*	*-32*	*13*	*-30*	*88*	*4.95*	*3.48*
Thalamus	*Pulvinar*	*L*	*-12*	*-34*	*8*	*20*	*4.91*	*3.52*
Lentiform Nucleus	*Putamen*	*R*	*25*	*-6*	*-4*	*37*	*4.87*	*3.54*
Parahippocampal Gyrus	*36*	*L*	*-27*	*-4*	*-27*	*142*	*4.84*	*3.53*
Inferior Frontal Gyrus	*47*	*L*	*-32*	*13*	*-19*	*18*	*4.75*	*3.31*
Cerebellum	*Anterior Lobe*	*L*	*-8*	*-50*	*-25*	*20*	*4.71*	*3.59*
Midbrain	*Periaqueductal grey*	*L*	*0*	*-36*	*-9*	*41*	*4.65*	*3.52*
Parahippocampal Gyrus	*27*	*L*	*-24*	*-32*	*-3*	*28*	*4.55*	*3.52*
Parahippocampal Gyrus	*28*	*R*	*20*	*-29*	*-9*	*25*	*4.43*	*3.19*
Basal Ganglia	*Caudate Tail*	*R*	*37*	*-14*	*-12*	*22*	*4.42*	*3.34*
Cerebellum	*Tonsil*	*R*	*3*	*-45*	*-36*	*19*	*4.34*	*3.33*
Basal Ganglia	*Med. Globus Pallidus*	*L*	*-9*	*1*	*-1*	*15*	*4.33*	*3.28*
Parahippocampal Gyrus	*30*	*R*	*6*	*-37*	*8*	*17*	*4.30*	*3.32*
Cerebellum	*Anterior Lobe*	*R*	*1*	*-45*	*-26*	*24*	*4.06*	*3.24*
Superior Temporal Gyrus	*38*	*L*	*-43*	*10*	*-21*	*27*	*4.04*	*3.27*
Inferior Frontal Gyrus	*13*	*R*	*29*	*9*	*-11*	*21*	*4.01*	*3.26*
Cerebellum	*Lingual*	*R*	*0*	*-48*	*-15*	*16*	*4.00*	*3.51*
Parahippocampal Gyrus	*27*	*R*	*27*	*-28*	*-5*	*38*	*3.99*	*3.34*
Thalamus	*Pulvinar*	*R*	*14*	*-29*	*-2*	*45*	*3.90*	*3.16*
Middle Temporal Gyrus	*21*	*L*	*-39*	*-3*	*-30*	*27*	*3.90*	*3.36*
Basal Ganglia	*Lat. Globus Pallidus*	*R*	*27*	*-20*	*-4*	*23*	*3.80*	*3.21*
Cerebellum	*Culmen*	*R*	*14*	*-43*	*-13*	*15*	*3.70*	*2.99*
Cerebellum	*Culmen*	*R*	*11*	*-34*	*-13*	*25*	*3.59*	*3.15*
Superior Temporal Gyrus	*38*	*R*	*35*	*3*	*-15*	*14*	*3.46*	*3.04*
Amygdala	*Centromedial*	*R*	*28*	*-6*	*-16*	*16*	*3.18*	*2.94*
Medial Frontal Gyrus	**32**	**L**	**-11**	**11**	**47**	**225**	**7.81**	**3.68**
Medial Frontal Gyrus	*32*	*L*	*-11*	*11*	*47*	*37*	*7.81*	*5.04*
Medial Frontal Gyrus	*6*	*R*	*3*	*14*	*47*	*21*	*4.81*	*3.40*
Cingulate Gyrus	*32*	*L*	*-15*	*21*	*32*	*43*	*4.41*	*3.60*
Cingulate Gyrus	*32*	*R*	*14*	*20*	*31*	*36*	*4.30*	*3.45*
Cingulate Gyrus	*32*	*R*	*3*	*21*	*37*	*27*	*4.25*	*3.36*
Anterior Cingulate	*32*	*L*	*-4*	*33*	*20*	*46*	*4.13*	*3.31*
Anterior Cingulate	*33*	*L*	*-4*	*18*	*21*	*15*	*3.89*	*3.14*
Lingual Gyrus	**17**	**R**	**24**	**-89**	**-2**	**142**	**6.66**	**3.99**

Inferior Occipital Gyrus	**18**	**L**	**-29**	**-93**	**-6**	**55**	**6.38**	**3.78**
Cerebellum	**Culmen**	**R**	**37**	**-54**	**-20**	**110**	**4.63**	**3.22**
Cerebellum	*Culmen*	*R*	*37*	*-54*	*-20*	*62*	*4.63*	*3.39*
Cerebellum	*Culmen*	*R*	*32*	*-44*	*-24*	*27*	*3.84*	*3.07*
Cerebellum	*Tuber*	*R*	*51*	*-65*	*-25*	*18*	*3.14*	*2.93*
Cerebellum	**Anterior Lobe**	**R**	**16**	**-34**	**-29**	**15**	**4.44**	**3.40**
Cerebellum	**Anterior Lobe**	**L**	**-15**	**-34**	**-30**	**36**	**4.39**	**3.51**
Inferior Frontal Gyrus	**47**	**L**	**-27**	**24**	**-18**	**20**	**4.39**	**3.27**
Superior Temporal Gyrus	**22**	**R**	**54**	**-7**	**-8**	**28**	**4.32**	**3.35**
Insula	**13**	**L**	**-34**	**25**	**9**	**71**	**4.20**	**3.22**
Insula	*13*	*L*	*-34*	*25*	*9*	*36*	*4.20*	*3.39*
Inferior Frontal Gyrus	*44*	*L*	*-42*	*15*	*13*	*33*	*3.48*	*3.05*
Posterior Cingulate	**30**	**L**	**-19**	**-51**	**8**	**15**	**4.09**	**3.24**
Superior Frontal Gyrus	**10**	**L**	**-19**	**63**	**15**	**18**	**4.08**	**3.32**
Precuneus	**31**	**L**	**-13**	**-62**	**23**	**11**	**3.99**	**3.32**
Cerebellum	**Declive**	**R**	**18**	**-61**	**-19**	**28**	**3.39**	**3.00**
	Maintenance							
Precentral Gyrus	**4**	**L**	**-48**	**-6**	**45**	**108**	**7.49**	**3.93**
Precentral Gyrus	*4*	*L*	*-48*	*-6*	*45*	*79*	*7.49*	*4.21*
Middle Frontal Gyrus	*6*	*L*	*-37*	*-2*	*50*	*18*	*3.84*	*3.22*
Precentral Gyrus	*6*	*L*	*-37*	*-8*	*43*	*11*	*3.52*	*3.02*
Superior Frontal Gyrus	**6**	**L**	**-4**	**6**	**58**	**315**	**6.10**	**3.62**
Superior Frontal Gyrus	*6*	*L*	*-4*	*6*	*58*	*131*	*6.10*	*3.88*
Superior Frontal Gyrus	*6*	*R*	*11*	*8*	*60*	*55*	*5.53*	*3.58*
Superior Frontal Gyrus	*6*	*L*	*-7*	*-3*	*67*	*20*	*5.07*	*3.52*
Superior Frontal Gyrus	*6*	*R*	*3*	*8*	*49*	*40*	*4.59*	*3.47*
Cingulate Gyrus	*32*	*L*	*0*	*15*	*36*	*57*	*4.21*	*3.31*
Cingulate Gyrus	*32*	*R*	*14*	*14*	*34*	*11*	*3.32*	*3.01*
Middle Temporal Gyrus	**39**	**R**	**42**	**-58**	**9**	**391**	**5.65**	**3.52**
Middle Temporal Gyrus	*39*	*R*	*42*	*-58*	*9*	*17*	*5.65*	*3.57*
Middle Occipital Gyrus	*18*	*R*	*11*	*-97*	*13*	*83*	*5.53*	*3.75*
Middle Temporal Gyrus	*37*	*R*	*45*	*-66*	*8*	*29*	*5.48*	*3.59*
Middle Occipital Gyrus	*19*	*R*	*39*	*-85*	*21*	*41*	*5.45*	*3.66*
Middle Occipital Gyrus	*18*	*R*	*27*	*-87*	*14*	*36*	*5.06*	*3.65*
Cuneus	*19*	*R*	*8*	*-86*	*27*	*48*	*4.90*	*3.33*
Middle Occipital Gyrus	*18*	*R*	*20*	*-90*	*14*	*24*	*4.79*	*3.61*
Cuneus	*7*	*R*	*21*	*-77*	*33*	*57*	*4.39*	*3.36*
Middle Occipital Gyrus	*19*	*R*	*37*	*-77*	*10*	*18*	*3.94*	*3.18*
Middle Occipital Gyrus	*19*	*R*	*45*	*-79*	*10*	*13*	*3.84*	*3.18*
Precuneus	*31*	*R*	*28*	*-75*	*21*	*25*	*3.79*	*3.21*
Middle Temporal Gyrus	**39**	**L**	**-45**	**-61**	**9**	**309**	**5.62**	**3.39**
Middle Temporal Gyrus	*39*	*L*	*-45*	*-61*	*9*	*28*	*5.62*	*3.75*
Cerebellum	*Culmen*	*L*	*-30*	*-50*	*-15*	*52*	*5.58*	*3.59*
Parahippocampal Gyrus	*36*	*L*	*-24*	*-37*	*-6*	*46*	*4.80*	*3.42*
Fusiform Gyrus	*37*	*L*	*-29*	*-35*	*-13*	*47*	*4.79*	*3.50*
Cerebellum	*Declive*	*L*	*-35*	*-63*	*-10*	*42*	*4.45*	*3.36*
Middle Temporal Gyrus	*37*	*L*	*-43*	*-60*	*-4*	*31*	*4.09*	*3.19*
Parahippocampal Gyrus	*27*	*L*	*-11*	*-36*	*1*	*15*	*4.07*	*3.06*
Lingual Gyrus	*18*	*L*	*-26*	*-71*	*-9*	*19*	*3.64*	*3.12*
Cerebellum	*Culmen*	*L*	*-16*	*-39*	*-15*	*18*	*3.61*	*3.02*
Superior Temporal Gyrus	*22*	*L*	*-56*	*-60*	*14*	*10*	*3.52*	*3.06*
Precentral Gyrus	**4**	**R**	**48**	**-12**	**40**	**62**	**5.46**	**3.39**
Precentral Gyrus	*4*	*R*	*48*	*-12*	*40*	*38*	*5.46*	*3.61*
Middle Frontal Gyrus	*6*	*R*	*38*	*-7*	*45*	*10*	*3.85*	*3.05*

Precentral Gyrus	*6*	*R*	*55*	*-4*	*42*	*14*	*3.29*	*3.03*
Precentral Gyrus	**6**	**L**	**-54**	**2**	**16**	**69**	**4.87**	**3.33**
Precentral Gyrus	*6*	*L*	*-54*	*2*	*16*	*44*	*4.87*	*3.34*
Precentral Gyrus	*44*	*L*	*-52*	*5*	*9*	*25*	*4.13*	*3.32*
Middle Temporal Gyrus	**39**	**L**	**-51**	**-73**	**14**	**242**	**4.63**	**3.30**
Middle Temporal Gyrus	*39*	*L*	*-51*	*-73*	*14*	*44*	*4.63*	*3.54*
Precuneus	*31*	*L*	*-22*	*-80*	*29*	*70*	*4.50*	*3.31*
Middle Occipital Gyrus	*19*	*L*	*-31*	*-80*	*9*	*14*	*4.04*	*3.31*
Middle Occipital Gyrus	*19*	*L*	*-40*	*-84*	*9*	*32*	*3.94*	*3.33*
Middle Occipital Gyrus	*19*	*L*	*-36*	*-85*	*17*	*48*	*3.76*	*3.11*
Middle Occipital Gyrus	*19*	*L*	*-28*	*-75*	*20*	*34*	*3.75*	*3.20*
Inferior Frontal Gyrus	**47**	**L**	**-49**	**23**	**3**	**21**	**3.92**	**3.09**
Cuneus	**18**	**L**	**-10**	**-99**	**8**	**28**	**3.84**	**3.14**
Anterior Cingulate	**24**	**R**	**7**	**29**	**-1**	**11**	**3.54**	**3.11**
Cerebellum	**Lingual**	**R**	**28**	**-68**	**-2**	**21**	**3.43**	**2.98**
	Representation							
Middle Frontal Gyrus	**8**	**L**	**-26**	**34**	**39**	**3730**	**12.76**	**4.08**
Middle Frontal Gyrus	*8*	*L*	*-26*	*34*	*39*	*207*	*12.76*	*5.15*
Medial Frontal Gyrus	*10*	*L*	*-8*	*63*	*3*	*119*	*9.77*	*4.63*
Medial Frontal Gyrus	*10*	*L*	*-3*	*48*	*-10*	*199*	*9.46*	*5.72*
Medial Frontal Gyrus	*10*	*R*	*12*	*52*	*10*	*164*	*8.48*	*4.69*
Posterior Cingulate	*23*	*L*	*-6*	*-55*	*18*	*112*	*7.77*	*4.40*
Medial Frontal Gyrus	*10*	*L*	*-10*	*40*	*-12*	*148*	*7.76*	*5.03*
Cingulate Gyrus	*31*	*L*	*-6*	*-35*	*30*	*113*	*7.72*	*4.82*
Precuneus	*31*	*L*	*-12*	*-49*	*25*	*45*	*7.43*	*4.98*
Anterior Cingulate	*32*	*L*	*-4*	*47*	*1*	*169*	*7.29*	*4.85*
Posterior Cingulate	*29*	*R*	*6*	*-46*	*17*	*67*	*6.94*	*4.56*
Posterior Cingulate	*29*	*L*	*-6*	*-42*	*20*	*30*	*6.94*	*5.77*
Cingulate Gyrus	*31*	*L*	*-9*	*-36*	*38*	*53*	*6.70*	*4.10*
Thalamus	*Anterior*	*L*	*-3*	*-7*	*6*	*55*	*6.44*	*4.36*
Middle Frontal Gyrus	*8*	*L*	*-25*	*20*	*37*	*69*	*6.29*	*4.50*
Amygdala	*Laterobasal*	*L*	*-24*	*-10*	*-9*	*169*	*6.04*	*3.72*
Superior Frontal Gyrus	*8*	*L*	*-12*	*49*	*35*	*88*	*6.00*	*3.85*
Basal Ganglia	*Caudate Head*	*R*	*6*	*10*	*2*	*51*	*5.72*	*3.69*
Middle Temporal Gyrus	*21*	*L*	*-62*	*-8*	*-4*	*83*	*5.45*	*3.93*
Anterior Cingulate	*32*	*R*	*11*	*44*	*-1*	*119*	*5.26*	*4.26*
Superior Temporal Gyrus	*41*	*L*	*-45*	*-32*	*9*	*48*	*5.14*	*3.50*
Cerebellum	*Culmen*	*L*	*0*	*-51*	*-11*	*146*	*5.12*	*3.50*
Posterior Cingulate	*29*	*R*	*3*	*-48*	*10*	*42*	*5.07*	*3.60*
Medial Frontal Gyrus	*10*	*L*	*-4*	*55*	*20*	*85*	*5.04*	*4.01*
Cerebellum	*Culmen*	*L*	*-9*	*-50*	*4*	*29*	*5.03*	*3.37*
Thalamus	*Medial Dorsal*	*L*	*-3*	*-11*	*16*	*55*	*4.82*	*3.66*
Cerebellum	*Culmen*	*L*	*-8*	*-48*	*-9*	*111*	*4.65*	*3.41*
Thalamus	*Medial Dorsal*	*L*	*-6*	*-15*	*4*	*17*	*4.65*	*3.31*
Middle Temporal Gyrus	*21*	*L*	*-61*	*-22*	*-9*	*42*	*4.65*	*3.62*
Thalamus	*Medial Dorsal*	*R*	*3*	*-4*	*12*	*27*	*4.64*	*3.58*
Middle Temporal Gyrus	*21*	*L*	*-53*	*-14*	*-7*	*76*	*4.57*	*3.53*
Superior Frontal Gyrus	*9*	*R*	*4*	*49*	*32*	*61*	*4.57*	*3.45*
Cerebellum	*Culmen*	*R*	*3*	*-42*	*-5*	*78*	*4.48*	*3.27*
Parahippocampal Gyrus	*30*	*L*	*-9*	*-37*	*6*	*19*	*4.48*	*3.45*
Anterior Cingulate	*25*	*R*	*3*	*3*	*-3*	*43*	*4.44*	*3.40*
Anterior Cingulate	*25*	*L*	*-3*	*16*	*-6*	*63*	*4.43*	*3.24*
Basal Ganglia	*Putamen*	*L*	*-19*	*4*	*-7*	*32*	*4.39*	*3.20*
Inferior Frontal Gyrus	*47*	*L*	*-28*	*7*	*-13*	*82*	*4.24*	*3.23*

Basal Ganglia	*Putamen*	*L*	*-31*	*-13*	*0*	*21*	*4.19*	*3.28*
Middle Temporal Gyrus	*21*	*L*	*-54*	*-1*	*-16*	*58*	*4.18*	*3.35*
Anterior Cingulate	*24*	*L*	*0*	*33*	*15*	*93*	*4.12*	*3.37*
Superior Temporal Gyrus	*22*	*L*	*-52*	*0*	*-2*	*17*	*4.11*	*3.15*
Anterior Cingulate	*33*	*R*	*7*	*21*	*21*	*21*	*4.03*	*3.25*
Inferior Frontal Gyrus	*47*	*L*	*-37*	*29*	*-13*	*68*	*3.98*	*3.17*
Amygdala	*Laterobasal*	*L*	*-8*	*-20*	*-5*	*17*	*3.96*	*3.08*
Parahippocampal Gyrus	*28*	*L*	*-23*	*-12*	*-28*	*10*	*3.92*	*3.28*
Hippocampus	*Posterior*	*L*	*-33*	*-33*	*-6*	*47*	*4.06*	*3.18*
Superior Temporal Gyrus	*13*	*L*	*-45*	*-20*	*7*	*36*	*3.74*	*3.18*
Superior Temporal Gyrus	*41*	*L*	*-56*	*-24*	*10*	*45*	*3.66*	*3.18*
Thalamus	*Pulvinar*	*R*	*15*	*-32*	*8*	*41*	*3.66*	*3.14*
Superior Temporal Gyrus	*38*	*L*	*-55*	*10*	*-17*	*18*	*3.51*	*3.02*
Hippocampus	*Posterior*	*L*	*-30*	*-32*	*-6*	*13*	*3.82*	*3.15*
Basal Ganglia	*Caudate Tail*	*R*	*33*	*-41*	*1*	*12*	*3.40*	*3.05*
Thalamus	*Pulvinar*	*R*	*24*	*-33*	*2*	*11*	*3.38*	*3.01*
Superior Temporal Gyrus	*38*	*L*	*-47*	*16*	*-20*	*24*	*3.37*	*3.04*
Middle Temporal Gyrus	*21*	*L*	*-36*	*-6*	*-28*	*22*	*3.34*	*3.02*
Insula	*13*	*L*	*-37*	*-17*	*14*	*16*	*3.14*	*2.88*
Parahippocampal Gyrus	*34*	*L*	*-21*	*-4*	*-23*	*13*	*3.11*	*2.87*
Basal Ganglia	*Putamen*	*L*	*-25*	*-2*	*0*	*10*	*3.09*	*2.89*
Middle Temporal Gyrus	**39**	**R**	**53**	**-69**	**15**	**286**	**7.70**	**3.90**
Middle Temporal Gyrus	*39*	*R*	*53*	*-69*	*15*	*97*	*7.70*	*4.75*
Middle Occipital Gyrus	*19*	*R*	*48*	*-76*	*6*	*37*	*5.62*	*4.00*
Middle Temporal Gyrus	*39*	*R*	*51*	*-71*	*24*	*45*	*5.15*	*3.55*
Inferior Temporal Gyrus	*37*	*R*	*51*	*-68*	*1*	*46*	*4.50*	*3.35*
Middle Temporal Gyrus	*39*	*R*	*40*	*-67*	*17*	*42*	*4.29*	*3.16*
Middle Temporal Gyrus	*37*	*R*	*45*	*-60*	*1*	*19*	*3.86*	*3.20*
Middle Temporal Gyrus	**39**	**L**	**-39**	**-71**	**27**	**286**	**7.42**	**4.54**
Middle Temporal Gyrus	*39*	*L*	*-39*	*-71*	*27*	*260*	*7.42*	*4.64*
Superior Occipital Gyrus	*19*	*L*	*-36*	*-84*	*26*	*26*	*4.41*	*3.54*
Middle Frontal Gyrus	**8**	**R**	**26**	**31**	**40**	**65**	**5.68**	**3.52**
Superior Temporal Gyrus	**38**	**R**	**36**	**11**	**-19**	**322**	**5.60**	**3.44**
Superior Temporal Gyrus	*38*	*R*	*36*	*11*	*-19*	*122*	*5.60*	*3.71*
Superior Temporal Gyrus	*38*	*R*	*45*	*17*	*-21*	*54*	*5.03*	*3.62*
Amygdala	*Laterobasal*	*R*	*28*	*-11*	*-11*	*78*	*4.92*	*3.30*
Inferior Frontal Gyrus	*47*	*R*	*41*	*31*	*-13*	*15*	*3.43*	*3.01*
Inferior Frontal Gyrus	*47*	*R*	*40*	*22*	*-15*	*10*	*3.32*	*3.03*
Middle Temporal Gyrus	*38*	*R*	*35*	*1*	*-28*	*12*	*3.22*	*2.92*
Parahippocampal Gyrus	*38*	*R*	*26*	*-3*	*-24*	*31*	*3.18*	*2.95*
Parietal Lobe	**40**	**R**	**28**	**-41**	**51**	**62**	**5.37**	**3.26**
Parietal Lobe	*40*	*R*	*28*	*-41*	*51*	*43*	*5.37*	*3.41*
Superior Parietal Lobule	*7*	*R*	*28*	*-49*	*61*	*19*	*3.35*	*2.91*
Insula	**13**	**R**	**43**	**-14**	**6**	**382**	**5.03**	**3.33**
Insula	*13*	*R*	*43*	*-14*	*6*	*78*	*5.03*	*3.59*
Middle Temporal Gyrus	*21*	*R*	*62*	*-16*	*-11*	*24*	*4.79*	*3.68*
Superior Temporal Gyrus	*22*	*R*	*63*	*-6*	*4*	*78*	*4.49*	*3.30*
Superior Temporal Gyrus	*41*	*R*	*51*	*-20*	*7*	*102*	*4.36*	*3.31*
Middle Temporal Gyrus	*21*	*R*	*54*	*-10*	*-11*	*19*	*4.12*	*3.14*
Middle Temporal Gyrus	*21*	*R*	*63*	*-5*	*-6*	*24*	*3.76*	*3.25*
Transverse Temporal Gyrus	*41*	*R*	*42*	*-31*	*13*	*23*	*3.64*	*3.04*
Insula	*13*	*R*	*43*	*-17*	*19*	*34*	*3.63*	*2.99*
Cerebellum	**Uvula**	**R**	**16**	**-76**	**-34**	**92**	**4.64**	**3.37**
Cerebellum	*Uvula*	*R*	*16*	*-76*	*-34*	*52*	*4.64*	*3.50*
Cerebellum	*Pyramis*	*R*	*26*	*-73*	*-28*	*40*	*3.77*	*3.19*

Cingulate Gyrus	**24**	**L**	**0**	**-8**	**35**	**78**	**4.63**	**3.26**
Cingulate Gyrus	*24*	*L*	*0*	*-8*	*35*	*65*	*4.63*	*3.32*
Cingulate Gyrus	*24*	*R*	*10*	*-6*	*44*	*11*	*3.56*	*2.97*
Cerebellum	**Tonsil**	**R**	**13**	**-43**	**-42**	**33**	**4.59**	**3.13**
Cerebellum	*Tonsil*	*R*	*13*	*-43*	*-42*	*15*	*4.59*	*3.33*
Cerebellum	*Anterior Lobe*	*R*	*8*	*-42*	*-30*	*16*	*3.18*	*2.97*
Precentral Gyrus	**6**	**R**	**35**	**-8**	**58**	**126**	**4.39**	**3.22**
Precentral Gyrus	*6*	*R*	*35*	*-8*	*58*	*53*	*4.39*	*3.27*
Postcentral Gyrus	*3*	*R*	*37*	*-24*	*50*	*26*	*4.02*	*3.23*
Precentral Gyrus	*4*	*R*	*40*	*-18*	*63*	*18*	*3.86*	*3.27*
Postcentral Gyrus	*3*	*R*	*43*	*-16*	*52*	*27*	*3.58*	*3.12*
Postcentral Gyrus	**3**	**R**	**57**	**-12**	**42**	**16**	**4.19**	**3.17**
Precentral Gyrus	**4**	**R**	**23**	**-22**	**68**	**38**	**4.14**	**3.24**
Cerebellum	**Tonsil**	**L**	**-16**	**-46**	**-41**	**37**	**4.02**	**3.04**
Paracentral Lobule	**6**	**R**	**7**	**-27**	**52**	**49**	**3.63**	**3.00**
Medial Frontal Gyrus	*6*	*L*	*-7*	*-17*	*59*	*20*	*3.46*	*2.99*
Overlap between Representation and Maintenance								
Middle Temporal Gyrus	**39**	**R**	**56**	**-68**	**11**	**48**	**N/A**	**N/A**
Middle Occipital Gyrus	**19**	**R**	**48**	**-77**	**15**	**13**	**N/A**	**N/A**
Precentral Gyrus	**4**	**R**	**43**	**-16**	**45**	**21**	**N/A**	**N/A**
Posterior Cingulate	**29**	**R**	**6**	**-45**	**15**	**130**	**N/A**	**N/A**
Medial Frontal Gyrus	**11**	**R**	**7**	**39**	**-15**	**20**	**N/A**	**N/A**
Anterior Cingulate	**25**	**L**	**0**	**17**	**1**	**11**	**N/A**	**N/A**
Medial Frontal Gyrus	**9**	**L**	**-4**	**49**	**37**	**40**	**N/A**	**N/A**
Cerebellum	**Culmen**	**L**	**-8**	**-51**	**-11**	**15**	**N/A**	**N/A**
Parahippocampal Gyrus	**35**	**L**	**-23**	**-29**	**-10**	**23**	**N/A**	**N/A**
Middle Occipital Gyrus	**19**	**L**	**-40**	**-64**	**11**	**108**	**N/A**	**N/A**

Table A3.1.3: Results from the contrast-based decomposition approach taken in Experiment 1 (N=32) to identify regions with temporal response profiles corresponding to hypothesised component processes of endogenous emotion generation. See text for details on the masking approach adopted. Cluster maxima are reported in bold. Unbolded entries denote local maxima of subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Component contrasts are reported in Table S2. Size threshold k>10.

Table A3.1.4. Results from the data-driven decomposition

			MNI			Extent	Loading	Loading
Region	**Label/BA**	**Side**	**x**	**y**	**z**	(voxel)	(max)	(avg)
Component 1								
Posterior Cingulate	**23**	**L**	**-3**	**-52**	**17**	**6085**	**0.26**	**0.19**
Posterior Cingulate	*23*	*L*	*-3*	*-52*	*17*	*1096*	*0.26*	*0.20*
Middle Frontal Gyrus	*8*	*L*	*-29*	*28*	*42*	*539*	*0.25*	*0.20*
Superior Frontal Gyrus	*6*	*L*	*0*	*8*	*60*	*403*	*0.25*	*0.20*
Superior Frontal Gyrus	*9*	*L*	*0*	*57*	*26*	*585*	*0.24*	*0.19*
Precentral Gyrus	*4*	*R*	*26*	*-25*	*61*	*298*	*0.23*	*0.20*
Paracentral Lobule	*5*	*L*	*0*	*-41*	*57*	*387*	*0.23*	*0.19*
Paracentral Lobule	*31*	*L*	*0*	*-29*	*47*	*341*	*0.23*	*0.19*
Medial Frontal Gyrus	*10*	*L*	*0*	*51*	*-8*	*416*	*0.22*	*0.19*
Cingulate Gyrus (Middle)	*24*	*L*	*0*	*-8*	*35*	*228*	*0.22*	*0.19*
Medial Frontal Gyrus	*6*	*R*	*3*	*-12*	*65*	*328*	*0.22*	*0.20*

Precentral Gyrus	*4*	*R*	*43*	*-17*	*40*	*233*	*0.22*	*0.19*
Posterior Cingulate	*29*	*R*	*6*	*-40*	*6*	*228*	*0.21*	*0.19*
Precentral Gyrus	*4*	*L*	*-16*	*-26*	*59*	*127*	*0.21*	*0.18*
Paracentral Lobule	*6*	*L*	*-6*	*-32*	*67*	*124*	*0.21*	*0.19*
Paracentral Lobule	*6*	*R*	*7*	*-30*	*67*	*154*	*0.21*	*0.19*
Cingulate Gyrus	*32*	*L*	*0*	*21*	*31*	*246*	*0.21*	*0.18*
Postcentral Gyrus	*2*	*R*	*26*	*-37*	*63*	*112*	*0.20*	*0.18*
Middle Frontal Gyrus	*6*	*L*	*-41*	*3*	*50*	*88*	*0.19*	*0.18*
Middle Frontal Gyrus	*10*	*L*	*-32*	*60*	*8*	*140*	*0.19*	*0.18*
Angular Gyrus	**39**	**L**	**-45**	**-68**	**27**	**422**	**0.24**	**0.20**
Thalamus	**Dorsal**	**L**	**0**	**-7**	**12**	**327**	**0.21**	**0.18**
Thalamus	*Dorsal*	*L*	*0*	*-7*	*12*	*154*	*0.21*	*0.19*
Caudate	*Head/NACC*	*L*	*-13*	*2*	*12*	*87*	*0.20*	*0.18*
Caudate	*Head/NACC*	*R*	*13*	*1*	*12*	*74*	*0.19*	*0.18*
Superior Temporal Gyrus	**22**	**L**	**-55**	**6**	**4**	**787**	**0.21**	**0.18**
Superior Temporal Gyrus	*22*	*L*	*-55*	*6*	*4*	*156*	*0.21*	*0.19*
Superior Temporal Gyrus	*22*	*L*	*-53*	*15*	*-2*	*152*	*0.21*	*0.18*
Inferior Frontal Gyrus	*45/47*	*L*	*-49*	*29*	*15*	*246*	*0.19*	*0.18*
Middle Temporal Gyrus	*21*	*L*	*-56*	*-11*	*-7*	*102*	*0.19*	*0.18*
Transverse Temporal Gyrus	*41*	*L*	*-56*	*-21*	*10*	*117*	*0.18*	*0.18*
Superior Temporal Gyrus	**22**	**R**	**60**	**-3**	**9**	**483**	**0.21**	**0.18**
Superior Temporal Gyrus	*22*	*R*	*60*	*-3*	*9*	*323*	*0.21*	*0.19*
Transverse Temporal Gyrus	*41*	*R*	*51*	*-26*	*11*	*141*	*0.19*	*0.18*
Precentral Gyrus	**4**	**L**	**-42**	**-15**	**35**	**64**	**0.20**	**0.18**
Hippocampus	**Head**	**L**	**-24**	**-18**	**-9**	**39**	**0.19**	**0.18**
Cerebellum	**Declive**	**L**	**-15**	**-55**	**-12**	**48**	**0.19**	**0.18**
Middle Temporal Gyrus	**19**	**R**	**53**	**-63**	**17**	**30**	**0.18**	**0.18**
	Component 2							
Positive loading								
Inferior Frontal Gyrus	**45/47**	**L**	**-53**	**17**	**1**	**386**	**0.15**	**0.10**
Medial Frontal Gyrus	**32**	**L**	**0**	**17**	**44**	**400**	**0.13**	**0.10**
Medial Frontal Gyrus	*32*	*L*	*0*	*17*	*44*	*153*	*0.13*	*0.10*
Superior Frontal Gyrus	*6*	*L*	*-1*	*17*	*57*	*247*	*0.12*	*0.10*
Lingual Gyrus	**18**	**L**	**-4**	**-83**	**-3**	**1252**	**0.12**	**0.09**
Lingual Gyrus	*18*	*L*	*-4*	*-83*	*-3*	*296*	*0.12*	*0.10*
Lingual Gyrus	*18*	*R*	*6*	*-80*	*-4*	*432*	*0.12*	*0.10*
Cerebellum	*Declive*	*R*	*37*	*-60*	*-20*	*94*	*0.11*	*0.09*
Cerebellum	*Declive*	*R*	*28*	*-69*	*-15*	*238*	*0.10*	*0.09*
Cerebellum	*Declive*	*R*	*51*	*-71*	*-20*	*74*	*0.10*	*0.09*
Lingual Gyrus	*18*	*L*	*-23*	*-74*	*-9*	*105*	*0.09*	*0.09*
Inferior Frontal Gyrus	**47**	**R**	**56**	**15**	**4**	**148**	**0.12**	**0.09**
Middle Frontal Gyrus	**6**	**L**	**-40**	**0**	**54**	**90**	**0.10**	**0.09**
Thalamus	**Pulvinar**	**L**	**-3**	**-33**	**6**	**50**	**0.09**	**0.09**
Negative loading								
Postcentral gyrus	**40**	**R**	**23**	**-37**	**57**	**1333**	**-0.13**	**-0.09**

Postcentral gyrus	*40*	*R*	*23*	*-37*	*57*	*749*	*-0.13*	*-0.10*
Postcentral gyrus	*40*	*L*	*-19*	*-38*	*58*	*308*	*-0.12*	*-0.09*
Precuneus	*7*	*R*	*7*	*-30*	*44*	*273*	*-0.10*	*-0.09*
Middle Frontal Gyrus	**8**	**R**	**32**	**27**	**40**	**163**	**-0.10**	**-0.09**
Middle Temporal Gyrus	**3**	**R**	**45**	**-69**	**24**	**48**	**-0.09**	**-0.08**
	Conjunction of both components							
Superior Frontal Gyrus	**6/pre-SMA**	**R**	**11**	**14**	**63**	**218**	**N/A**	**N/A**
Inferior Frontal Gyrus	**47/45**	**L**	**-43**	**27**	**5**	**163**	**N/A**	**N/A**
Precentral Gyrus	**44**	**R**	**61**	**9**	**7**	**69**	**N/A**	**N/A**
Thalamus	**Pulvinar**	**R**	**6**	**-34**	**6**	**43**	**N/A**	**N/A**
Cingulate Gyrus	**32**	**R**	**3**	**14**	**39**	**36**	**N/A**	**N/A**

Table A3.1.4: Results from the data-driven decomposition approach taken in Experiment 2 (N=293), using constrained principle components analysis (CPCA), showing the regions corresponding to the two components shown to correlate with individual differences in emotion generation ability, as well as their conjunction. Cluster maxima for the main components are reported in bold, while conjunction results report the centroid for each cluster. Italicised entries denote local maxima of subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the *splitclustercoords* function). Results were thresholded to the 10% strongest loadings of each component and k>30.

Table A3.1.5. Parametric modulation by subjective affect.

Region	Label/BA	Side	MNI x	MNI y	MNI z	Extent (voxel)	*t*-value (max)	*t*-value (avg)
			Experiment 1 (N=32)					
			Parametric modulation: Generate Positive					
Caudate	**Head**	**R**	**13**	**16**	**4**	**39**	**5.49**	**3.89**
			Parametric modulation: Generate Negative					
Putamen	**Anterior**	**L**	**-25**	**4**	**-2**	**79**	**-5.39**	**-4.05**
Putamen	**Anterior**	**R**	**19**	**6**	**3**	**30**	**-4.79**	**-3.78**
Putamen	**Anterior**	**R**	**19**	**6**	**3**	**12**	**-4.79**	**-3.92**
Putamen	**Posterior**	**R**	**29**	**5**	**3**	**18**	**-4.02**	**-3.70**
			Conjunction: positive and negative parametric modulation (NB! MNI = Center coordinate)					
Putamen	**Anterior**		**21**	**15**	**-3**	**1**	**N/A**	**N/A**
			Experiment 2 (N=293)					
			Parametric modulation by affect ratings: Positive Generation					
Putamen	**Anterior**	**L**	**-26**	**7**	**0**	**1443**	**7.84**	**5.07**
Putamen	*Anterior*	*L*	*-26*	*7*	*0*	*170*	*7.84*	*5.44*
Thalamus	*Anterior*	*L*	*-3*	*-10*	*10*	*87*	*7.76*	*5.84*
Putamen	*Anterior*	*R*	*26*	*9*	*3*	*206*	*7.24*	*5.28*
Caudate	*Head/NAC*	*L*	*-16*	*10*	*2*	*125*	*6.99*	*5.35*

Middle Frontal Gyrus	*9*	*L*	*-48*	*17*	*30*	*171*	*6.82*	*4.80*
Caudate	*Body*	*R*	*20*	*13*	*14*	*76*	*6.81*	*5.41*
Thalamus	*Posterior*	*L*	*-3*	*-19*	*11*	*80*	*6.73*	*5.01*
Caudate	*Body*	*R*	*19*	*4*	*19*	*59*	*6.66*	*5.32*
Caudate	*Body*	*L*	*-19*	*-1*	*19*	*26*	*6.47*	*5.22*
Caudate	*Head*	*R*	*3*	*1*	*6*	*45*	*6.38*	*4.91*
Caudate	*Body*	*L*	*-9*	*-1*	*12*	*15*	*6.00*	*5.18*
Caudate	*Body*	*L*	*-17*	*11*	*19*	*36*	*5.97*	*5.18*
Inferior Frontal Gyrus	*45*	*L*	*-46*	*21*	*5*	*100*	*5.52*	*4.54*
Middle Frontal Gyrus	*46*	*L*	*-47*	*38*	*6*	*94*	*5.41*	*4.54*
Putamen	*Posterior Lateral*	*R*	*31*	*-15*	*8*	*23*	*5.34*	*4.49*
Insula	*Dorsal Anterior*	*L*	*-47*	*12*	*6*	*59*	*5.26*	*4.50*
Thalamus	*Pulvinar*	*L*	*-9*	*-31*	*8*	*15*	*5.17*	*4.33*
Middle Frontal Gyrus	*46*	*L*	*-50*	*46*	*5*	*20*	*5.02*	*4.44*
Hippocampus	*Posterior*	*L*	*-21*	*-15*	*-6*	*25*	*4.95*	*4.29*
Medial Frontal Gyrus	**Pre-SMA**	**R**	**7**	**5**	**55**	**65**	**6.97**	**5.29**
Medial Frontal Gyrus	**Pre-SMA**	**L**	**-4**	**8**	**57**	**56**	**6.52**	**4.94**
Midbrain	**Red Nucleus Substantia Nigra**	**L**	**-6**	**-16**	**-4**	**121**	**6.31**	**4.67**
Midbrain	*Red Nucleus Substantia Nigra*	*L*	*-6*	*-16*	*-4*	*31*	*6.31*	*5.03*
Midbrain	*Substantia Nigra Ventral tegmentum*	*R*	*3*	*-18*	*-2*	*26*	*5.96*	*4.80*
Midbrain	*Substantia Nigra Ventral tegmentum*	*L*	*-5*	*-28*	*-8*	*27*	*5.36*	*4.61*
Hippocampus	*Anterior*	*R*	*21*	*-14*	*-3*	*25*	*5.01*	*4.35*
Midbrain	*Substantia Nigra Ventral tegmentum*	*R*	*11*	*-23*	*-3*	*12*	*4.66*	*4.24*
Cerebellum	**Declive**	**R**	**34**	**-57**	**-20**	**147**	**5.88**	**4.47**
Cerebellum	*Declive*	*R*	*34*	*-57*	*-20*	*67*	*5.88*	*4.64*
Cerebellum	*Inferior Semi-Lunar*	*R*	*38*	*-63*	*-37*	*47*	*5.30*	*4.41*
Cerebellum	*Inferior Semi-Lunar*	*R*	*38*	*-63*	*-46*	*12*	*4.76*	*4.26*
Cerebellum	*Tuber*	*R*	*42*	*-73*	*-23*	*21*	*4.42*	*4.16*
Middle Frontal Gyrus	**6**	**L**	**-43**	**3**	**47**	**61**	**5.31**	**4.43**
Middle Frontal Gyrus	*6*	*L*	*-43*	*3*	*47*	*48*	*5.31*	*4.45*
Middle Frontal Gyrus	*Premotor cortex*	*L*	*-36*	*9*	*54*	*13*	*5.08*	*4.35*
Cingulate Gyrus	**32**	**L**	**-4**	**20**	**39**	**45**	**5.29**	**4.49**
Caudate	**Tail**	**R**	**27**	**-35**	**11**	**12**	**4.96**	**4.46**
Superior Frontal Gyrus	**10**	**L**	**-22**	**58**	**10**	**32**	**4.81**	**4.32**

Parametric modulation by affect ratings: Negative Generation

Caudate	**Body**	**L**	**-16**	**5**	**14**	**774**	**-7.06**	**-4.88**
Caudate	*Body*	*L*	*-16*	*5*	*14*	*97*	*-7.06*	*-5.25*
Putamen	*Anterior*	*L*	*-23*	*13*	*4*	*70*	*-6.46*	*-5.24*
Putamen	*Anterior*	*R*	*22*	*3*	*12*	*66*	*-6.27*	*-5.08*
Putamen	*Lateral*	*L*	*-32*	*4*	*3*	*70*	*-6.18*	*-5.09*
Thalamus	*Dorslal*	*L*	*0*	*-7*	*10*	*72*	*-6.02*	*-4.81*
Caudate	*Body*	*R*	*19*	*4*	*19*	*40*	*-5.96*	*-4.86*
Caudate	*Body*	*L*	*-17*	*14*	*14*	*34*	*-5.86*	*-5.01*
Putamen	*Lateral*	*R*	*28*	*2*	*-2*	*90*	*-5.83*	*-4.62*
Putamen	*Lateral*	*L*	*-25*	*-7*	*-2*	*45*	*-5.70*	*-4.67*
Putamen	*Anterior*	*L*	*-22*	*7*	*-3*	*47*	*-5.69*	*-4.88*
Putamen	*Posterior*	*R*	*31*	*-12*	*3*	*29*	*-5.47*	*-4.53*
Putamen	*Lateral*	*R*	*28*	*-9*	*12*	*16*	*-5.38*	*-4.59*

Caudate	*Head*	*R*	*16*	*10*	*7*	*70*	*-5.30*	*-4.49*
Insula	*Dorsal Anterior*	*L*	*-33*	*13*	*2*	*13*	*-5.24*	*-4.39*
Caudate	*Body*	*L*	*-16*	*-9*	*19*	*13*	*-5.02*	*-4.43*
Inferior Frontal Gyrus	**45**	**L**	**-46**	**23**	**8**	**523**	**-6.42**	**-4.62**
Inferior Frontal Gyrus	*45*	*L*	*-46*	*23*	*8*	*74*	*-6.42*	*-4.88*
Inferior Frontal Gyrus	*44*	*L*	*-50*	*14*	*13*	*83*	*-5.88*	*-4.72*
Inferior Frontal Gyrus	*9*	*L*	*-53*	*19*	*23*	*45*	*-5.72*	*-4.66*
Middle Frontal Gyrus	*9*	*L*	*-48*	*17*	*30*	*69*	*-5.50*	*-4.64*
Middle Frontal Gyrus	*6*	*L*	*-47*	*5*	*47*	*40*	*-5.47*	*-4.56*
Inferior Frontal Gyrus	*47*	*L*	*-56*	*22*	*11*	*36*	*-5.45*	*-4.51*
Middle Frontal Gyrus	*8*	*L*	*-47*	*8*	*40*	*56*	*-5.44*	*-4.71*
Precentral Gyrus	*Premotor cortex*	*L*	*-36*	*6*	*33*	*24*	*-5.22*	*-4.55*
Inferior Frontal Gyrus	*47*	*L*	*-55*	*31*	*-1*	*63*	*-4.84*	*-4.32*
Inferior Frontal Gyrus	*47*	*L*	*-54*	*23*	*-5*	*33*	*-4.79*	*-4.34*
Cerebellum	**Declive**	**R**	**25**	**-65**	**-20**	**190**	**-5.36**	**-4.45**
Cerebellum	*Declive*	*R*	*25*	*-65*	*-20*	*58*	*-5.36*	*-4.52*
Cerebellum	*Uvula*	*R*	*16*	*-74*	*-24*	*72*	*-5.26*	*-4.51*
Cerebellum	*Culmen*	*R*	*33*	*-53*	*-25*	*29*	*-4.97*	*-4.36*
Cerebellum	*Inferior Semi-Lunar*	*R*	*29*	*-73*	*-36*	*17*	*-4.85*	*-4.32*
Cerebellum	*Declive*	*R*	*35*	*-75*	*-18*	*14*	*-4.61*	*-4.12*
Middle Temporal Gyrus	**39**	**L**	**-47**	**-66**	**27**	**50**	**-5.16**	**-4.36**
Superior Frontal Gyrus	**6**	**R**	**3**	**11**	**55**	**22**	**-5.04**	**-4.38**
Cerebellum	**Culmen**	**L**	**-19**	**-34**	**-17**	**22**	**-4.96**	**-4.21**
Cerebellum	*Culmen*	*L*	*-19*	*-34*	*-17*	*12*	*-4.96*	*-4.20*
Cerebellum	*Culmen*	*L*	*-17*	*-45*	*-14*	*10*	*-4.50*	*-4.22*
Medial Frontal Gyrus	**8**	**L**	**-4**	**23**	**44**	**38**	**-4.79**	**-4.28**
Cerebellum	**Declive**	**L**	**-27**	**-56**	**-18**	**20**	**-4.76**	**-4.19**
Cerebellum	**Culmen**	**L**	**0**	**-33**	**-9**	**14**	**-4.52**	**-4.25**
Superior Frontal Gyrus	**9**	**L**	**-8**	**52**	**26**	**14**	**-4.36**	**-4.15**
Conjunction: positive and negative parametric modulation (NB! MNI = Center coordinate)								
Caudate	**Body**	**L**	**-1**	**4**	**7**	**618**	**N/A**	**N/A**
Middle Frontal Gyrus	**9**	**L**	**-47**	**16**	**30**	**114**	**N/A**	**N/A**
Inferior Frontal Gyrus	**45**	**L**	**-47**	**19**	**6**	**109**	**N/A**	**N/A**
Cerbellum	**Declive**	**R**	**30**	**-60**	**-20**	**36**	**N/A**	**N/A**
Middle	**Premotor Cortex**	**L**	**-43**	**5**	**46**	**23**	**N/A**	**N/A**
Cingulate Gyrus	**32**	**L**	**-6**	**22**	**41**	**20**	**N/A**	**N/A**
Inferior Frontal Gyrus	**47**	**L**	**-49**	**34**	**6**	**19**	**N/A**	**N/A**
Superior Frontal Gyrus	**Pre-SMA**	**R**	**5**	**10**	**57**	**18**	**N/A**	**N/A**

Table A3.1.5: Brain regions showing significant parametric modulation by affect ratings in positive and negative generation conditions as well as their conjunction, for both experiments. Cluster maxima are reported in bold for main contrasts, while conjunction results report coordinates of the cluster centroid. I entries denote local maxima of subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction using AFNIs alphasim procedure at $\alpha<.05$ (Experiment 1: $T>3.38$, $p<.001$, $k>24$; Experiment 2, $T>3.95$, $p<.00005$, $k>10$).

Table A3.1.6. Modality-specific activations.

Region	Label/BA	Side	MNI x	MNI y	MNI z	Extent (voxel)	*t*-value (max)	*t*-value (avg)
Semantic Analysis								
Positive correlation								
Superior Temporal Gyrus	**22/Wernicke**	**L**	**-52**	**-41**	**8**	**80**	**4.69**	**3.81**
Precentral Gyrus	**6**	**L**	**-47**	**-1**	**45**	**56**	**5.08**	**3.93**
Negative correlation								
Inferior Temporal Gyrus	**37**	**L**	-55	-60	-4	42	-4.18	-3.65
Episodic Imagery								
Posterior Cingulate	**23**	**L**	**-6**	**-57**	**24**	**86**	**3.89**	**3.51**
Bodily Interoception								
Anterior Insula	**13**	**L**	**-44**	**9**	**3**	**72**	**4.36**	**3.69**
Anterior Insula	**13**	**R**	**44**	**7**	**4**	**95**	**4.31**	**3.62**

Table A3.1.6: Results from robust regression analysis of the main Maintain (Positive & Negative) > Neutral contrast, showing regions whose activation correlated with post-scanning reports of generation modality usage. Multiple comparisons were controlled for using cluster-level FWE correction using AFNIs *alphasim* procedure at $\alpha<.05$ ($T>3.32$, $p<.0005$, $k>38$).

Table A3.1.7. Results from mediation analyses

Region	Label/BA	Side	MNI x	MNI y	MNI z	Extent (voxel)	*z*-value (max)	*z*-value (avg)
Mediated pathway for Semantic Analysis								
Insula	**13**	**L**	**-34**	**-22**	**14**	**328**	**4.18**	**3.69**
Insula	*13*	*L*	*-34*	*-22*	*14*	*40*	*4.18*	*3.82*
Insula	*13*	*L*	*-42*	*-32*	*19*	*42*	*4.05*	*3.77*
Superior Temporal Gyrus	*38*	*L*	*-49*	*10*	*-12*	*44*	*3.92*	*3.71*
Superior Temporal Gyrus	*42*	*L*	*-58*	*-29*	*12*	*33*	*3.70*	*3.57*
Putamen	**Posterior**	**L**	**-31**	**-2**	**-2**	**139**	**4.17**	**3.77**
Putamen	*Anterior*	*L*	*-19*	*7*	*2*	*31*	*3.99*	*3.83*
Precentral Gyrus	**6**	**L**	**-37**	**-13**	**61**	**62**	**4.11**	**3.78**
Superior Temporal Gyrus	**13**	**R**	**48**	**-44**	**18**	**68**	**4.06**	**3.69**
Inferior Frontal Gyrus	**47**	**R**	**41**	**32**	**-5**	**97**	**4.05**	**3.74**
Inferior Frontal Gyrus	*45*	*R*	*49*	*22*	*5*	*32*	*4.02*	*3.76*
Middle Temporal Gyrus	**21**	**R**	**52**	**5**	**-9**	**74**	**4.04**	**3.68**
Middle Temporal Gyrus	*21*	*R*	*52*	*5*	*-9*	*64*	*4.04*	*3.67*
Superior Temporal Gyrus	*21*	*R*	*60*	*-8*	*-3*	*10*	*3.79*	*3.71*
Precentral Gyrus	**6**	**L**	**-38**	**0**	**38**	**72**	**4.00**	**3.67**
Superior Frontal Gyrus	**8**	**L**	**-26**	**40**	**39**	**133**	**3.96**	**3.66**
Posterior Cingulate	**23**	**R**	**6**	**-57**	**20**	**66**	**3.94**	**3.63**

Posterior Cingulate	*23*	*R*	*6*	*-57*	*20*	*30*	*3.94*	*3.69*
Precentral Gyrus	**6**	**L**	**-51**	**-2**	**25**	**98**	**3.93**	**3.69**
Inferior Frontal Gyrus	*44*	*L*	*-48*	*14*	*13*	*32*	*3.92*	*3.71*
Medial Frontal Gyrus	**6**	**L**	**-11**	**3**	**52**	**60**	**3.88**	**3.66**
Medial Frontal Gyrus	*6*	*L*	*-11*	*3*	*52*	*30*	*3.88*	*3.64*
Middle Frontal Gyrus	**8**	**L**	**-45**	**14**	**45**	**65**	**3.74**	**3.62**
Middle Frontal Gyrus	*8*	*L*	*-45*	*14*	*45*	*51*	*3.74*	*3.62*
Mediated pathway for Visual Imagery								
Thalamus	**Pulvinar**	**L**	**0**	**-25**	**1**	**168**	**3.97**	**3.69**
Thalamus	*Pulvinar*	*L*	*0*	*-25*	*1*	*84*	*3.97*	*3.71*
Hippocampus	*Posterior*	*L*	*-27*	*-35*	*-1*	*31*	*3.93*	*3.69*
Thalamus	*Medial Dorsal*	*R*	*9*	*-15*	*10*	*40*	*3.87*	*3.65*
Fusiform Gyrus	**37**	**R**	**48**	**-49**	**-16**	**30**	**3.95**	**3.72**
Superior Temporal Gyrus	**22**	**R**	**59**	**-55**	**17**	**231**	**3.95**	**3.70**
Superior Temporal Gyrus	*22*	*R*	*59*	*-55*	*17*	*34*	*3.95*	*3.73*
Superior Temporal Gyrus	*39*	*R*	*53*	*-58*	*31*	*40*	*3.91*	*3.71*
Supramarginal Gyrus	*40*	*R*	*51*	*-52*	*20*	*32*	*3.89*	*3.70*
Angular Gyrus	*39*	*R*	*45*	*-74*	*31*	*55*	*3.85*	*3.70*
Middle Temporal Gyrus	*39*	*R*	*48*	*-69*	*19*	*32*	*3.83*	*3.70*
Angular Gyrus	*39*	*R*	*45*	*-60*	*34*	*37*	*3.78*	*3.68*
Cerebellum	**Pyramis**	**R**	**16**	**-71**	**-27**	**130**	**3.95**	**3.70**
Cerebellum	*Pyramis*	*R*	*16*	*-71*	*-27*	*72*	*3.95*	*3.71*
Cerebellum	*Uvula*	*R*	*29*	*-70*	*-25*	*57*	*3.90*	*3.70*
Middle Temporal Gyrus	**22**	**R**	**58**	**-34**	**2**	**55**	**3.94**	**3.76**
Superior Frontal Gyrus	**6**	**R**	**18**	**22**	**54**	**1025**	**3.94**	**3.68**
Superior Frontal Gyrus	*6*	*R*	*18*	*22*	*54*	*66*	*3.94*	*3.78*
Middle Frontal Gyrus	*8*	*R*	*40*	*20*	*47*	*73*	*3.92*	*3.73*
Middle Frontal Gyrus	*10*	*R*	*23*	*54*	*21*	*74*	*3.91*	*3.68*
Middle Frontal Gyrus	*10*	*L*	*-29*	*63*	*8*	*104*	*3.90*	*3.68*
Anterior Cingulate	*25*	*R*	*4*	*16*	*-6*	*55*	*3.90*	*3.68*
Anterior Cingulate	*24*	*L*	*-3*	*23*	*-3*	*79*	*3.87*	*3.67*
Medial Frontal Gyrus	*10*	*L*	*-11*	*52*	*7*	*82*	*3.85*	*3.70*
Medial Frontal Gyrus	*11*	*L*	*-7*	*59*	*-14*	*65*	*3.84*	*3.64*
Middle Frontal Gyrus	*9*	*R*	*32*	*25*	*35*	*53*	*3.84*	*3.67*
Anterior Cingulate	*32*	*L*	*-10*	*41*	*0*	*91*	*3.83*	*3.70*
Anterior Cingulate	*32*	*R*	*11*	*46*	*-7*	*58*	*3.82*	*3.68*
Middle Frontal Gyrus	*10*	*L*	*-39*	*54*	*9*	*30*	*3.81*	*3.67*
Medial Frontal Gyrus	*11*	*R*	*8*	*59*	*-17*	*30*	*3.80*	*3.63*
Medial Frontal Gyrus	*10*	*R*	*11*	*54*	*1*	*30*	*3.78*	*3.65*
Anterior Insula	*13/Ventral*	*R*	*35*	*2*	*1*	*30*	*3.78*	*3.64*
Caudate	*Head/NACC*	*L*	*-6*	*7*	*-2*	*33*	*3.76*	*3.63*
Middle Temporal Gyrus	**39**	**L**	**-48**	**-73**	**20**	**267**	**3.92**	**3.68**
Middle Temporal Gyrus	*39*	*L*	*-53*	*-68*	*25*	*1*	*3.54*	*3.54*
Superior Temporal Gyrus	*39*	*L*	*-45*	*-58*	*33*	*108*	*3.87*	*3.70*
Supramarginal Gyrus	*40*	*L*	*-55*	*-54*	*28*	*54*	*3.87*	*3.69*
Middle Temporal Gyrus	*39*	*L*	*-48*	*-73*	*20*	*54*	*3.92*	*3.68*
Precuneus	**31**	**R**	**19**	**-61**	**25**	**433**	**3.90**	**3.68**
Precuneus	*31*	*R*	*19*	*-61*	*25*	*82*	*3.90*	*3.72*
Lingual Gyrus	*18*	*R*	*12*	*-51*	*4*	*43*	*3.89*	*3.66*
Cingulate Gyrus	*23*	*L*	*-3*	*-25*	*24*	*59*	*3.87*	*3.69*
Cingulate Gyrus	*31*	*L*	*-6*	*-39*	*44*	*33*	*3.86*	*3.71*
Precuneus	*7*	*R*	*13*	*-50*	*38*	*53*	*3.85*	*3.68*
Precuneus	*7*	*R*	*6*	*-62*	*42*	*31*	*3.82*	*3.68*
Cingulate Gyrus	*31*	*L*	*-3*	*-26*	*38*	*56*	*3.78*	*3.66*

Cingulate Gyrus	*31*	*R*	*6*	*-36*	*32*	*60*	*3.76*	*3.66*
Middle Temporal Gyrus	**22**	**L**	**-58**	**-37**	**5**	**102**	**3.90**	**3.71**
Middle Temporal Gyrus	*22*	*L*	*-58*	*-37*	*5*	*35*	*3.90*	*3.78*
Middle Temporal Gyrus	*21*	*L*	*-63*	*-28*	*-11*	*60*	*3.88*	*3.68*
Cuneus	**17**	**R**	**11**	**-97**	**0**	**38**	**3.88**	**3.68**
Middle Temporal Gyrus	**21**	**L**	**-48**	**-1**	**-15**	**33**	**3.88**	**3.68**
Middle Frontal Gyrus	**9**	**L**	**-48**	**20**	**35**	**97**	**3.85**	**3.65**
Middle Frontal Gyrus	*9*	*L*	*-48*	*20*	*35*	*33*	*3.85*	*3.68*
Middle Frontal Gyrus	*9*	*L*	*-35*	*28*	*35*	*62*	*3.81*	*3.64*
Cuneus	**17**	**L**	**-13**	**-96**	**-1**	**32**	**3.83**	**3.64**
Middle Temporal Gyrus	**21**	**R**	**59**	**-22**	**-4**	**30**	**3.83**	**3.68**
Inferior Frontal Gyrus	**47**	**L**	**-29**	**16**	**-12**	**42**	**3.79**	**3.68**
Mediated pathway for Bodily Interoception								
Middle Frontal Gyrus	**9**	**R**	**32**	**42**	**36**	**634**	**4.98**	**3.74**
Middle Frontal Gyrus	*9*	*R*	*32*	*42*	*36*	*101*	*4.98*	*3.86*
Middle Frontal Gyrus	*9*	*R*	*32*	*34*	*27*	*87*	*4.45*	*3.84*
Precentral Gyrus	*9*	*R*	*39*	*11*	*33*	*79*	*4.36*	*3.82*
Middle Frontal Gyrus	*9*	*R*	*48*	*12*	*35*	*100*	*4.36*	*3.76*
Middle Frontal Gyrus	*9*	*R*	*41*	*23*	*33*	*44*	*4.27*	*3.73*
Precentral Gyrus	*6*	*R*	*48*	*-15*	*29*	*30*	*4.20*	*3.72*
Middle Frontal Gyrus	*10*	*R*	*39*	*45*	*12*	*82*	*3.93*	*3.64*
Precentral Gyrus	*6*	*R*	*52*	*-1*	*25*	*49*	*3.86*	*3.61*
Inferior Frontal Gyrus	*44*	*R*	*56*	*17*	*18*	*43*	*3.75*	*3.56*
Postcentral Gyrus	**43**	**L**	**-62**	**-10**	**15**	**442**	**4.96**	**3.67**
Postcentral Gyrus	*43*	*L*	*-62*	*-10*	*15*	*76*	*4.96*	*3.80*
Transverse Temporal Gyrus	*42*	*L*	*-59*	*-18*	*10*	*65*	*4.37*	*3.74*
Inferior Parietal Lobule	*40*	*L*	*-63*	*-30*	*26*	*31*	*3.98*	*3.65*
Transverse Temporal Gyrus	*41*	*L*	*-48*	*-18*	*10*	*57*	*3.90*	*3.64*
Superior Temporal Gyrus	*42*	*L*	*-53*	*-30*	*16*	*63*	*3.83*	*3.64*
Insula	*13*	*L*	*-40*	*-11*	*15*	*65*	*3.83*	*3.60*
Inferior Parietal Lobule	*40*	*L*	*-58*	*-24*	*29*	*33*	*3.81*	*3.65*
Supramarginal Gyrus	*40*	*L*	*-57*	*-43*	*30*	*40*	*3.73*	*3.56*
Medial Frontal Gyrus	**9**	**R**	**8**	**41**	**28**	**326**	**4.84**	**3.67**
Medial Frontal Gyrus	*9*	*R*	*8*	*41*	*28*	*44*	*4.84*	*3.82*
Cingulate Gyrus	*24*	*R*	*3*	*-6*	*47*	*95*	*4.23*	*3.66*
Cingulate Gyrus	*24*	*L*	*0*	*-11*	*41*	*54*	*4.07*	*3.70*
Anterior Cingulate	*32*	*R*	*4*	*47*	*7*	*44*	*4.05*	*3.67*
Anterior Cingulate	*32*	*L*	*-8*	*33*	*23*	*49*	*3.65*	*3.56*
Superior Temporal Gyrus	**22**	**R**	**62**	**-12**	**6**	**494**	**4.50**	**3.68**
Superior Temporal Gyrus	*22*	*R*	*62*	*-12*	*6*	*135*	*4.50*	*3.79*
Postcentral Gyrus	*43*	*R*	*65*	*-16*	*17*	*38*	*4.28*	*3.70*
Postcentral Gyrus	*2*	*R*	*65*	*-22*	*28*	*37*	*4.25*	*3.67*
Superior Temporal Gyrus	*41*	*R*	*53*	*-30*	*13*	*83*	*4.24*	*3.65*
Insula	*13*	*R*	*42*	*-26*	*14*	*51*	*4.06*	*3.63*
Claustrum		*R*	*37*	*-13*	*10*	*45*	*3.88*	*3.65*
Inferior Parietal Lobule	*40*	*R*	*56*	*-33*	*30*	*64*	*3.76*	*3.61*
Insula	*13*	*R*	*40*	*-10*	*-2*	*37*	*3.75*	*3.60*
Middle Frontal Gyrus	**6**	**R**	**30**	**-5**	**45**	**222**	**4.21**	**3.67**
Middle Frontal Gyrus	*6*	*R*	*30*	*-5*	*45*	*104*	*4.21*	*3.69*
Middle Frontal Gyrus	*6*	*R*	*27*	*-8*	*53*	*57*	*4.20*	*3.71*
Middle Frontal Gyrus	*6*	*R*	*22*	*13*	*60*	*52*	*3.89*	*3.62*
Middle Frontal Gyrus	**9**	**L**	**-29**	**43**	**36**	**113**	**4.14**	**3.59**
Middle Frontal Gyrus	*9*	*L*	*-29*	*43*	*36*	*46*	*4.14*	*3.62*
Middle Frontal Gyrus	*9*	*L*	*-43*	*26*	*35*	*30*	*3.96*	*3.60*

Middle Frontal Gyrus	*9*	*L*	*-41*	*39*	*34*	*36*	*3.62*	*3.54*
Cerebellum	**Culmen**	**R**	**36**	**-45**	**-19**	**58**	**4.08**	**3.69**
Middle Frontal Gyrus	**6**	**L**	**-23**	**-9**	**50**	**188**	**4.07**	**3.64**
Middle Frontal Gyrus	*6*	*L*	*-23*	*-9*	*50*	*74*	*4.07*	*3.69*
Middle Frontal Gyrus	*6*	*L*	*-26*	*-7*	*61*	*61*	*3.91*	*3.61*
Middle Frontal Gyrus	*6*	*L*	*-37*	*-5*	*45*	*43*	*3.75*	*3.61*
Fusiform Gyrus	**19**	**L**	**-25**	**-56**	**-10**	**94**	**3.90**	**3.63**
Fusiform Gyrus	*19*	*L*	*-25*	*-56*	*-10*	*41*	*3.90*	*3.62*
Fusiform Gyrus	*37*	*L*	*-33*	*-44*	*-12*	*50*	*3.81*	*3.65*
Precuneus	**31**	**L**	**-7**	**-74**	**29**	**93**	**3.89**	**3.66**
Precuneus	*31*	*L*	*-7*	*-74*	*29*	*48*	*3.89*	*3.67*
Precuneus	*7*	*L*	*-3*	*-57*	*50*	*40*	*3.82*	*3.64*
Inferior Temporal Gyrus	**20**	**R**	**50**	**-29**	**-14**	**57**	**3.88**	**3.63**
Inferior Parietal Lobule	**40**	**R**	**50**	**-47**	**24**	**55**	**3.81**	**3.59**
Middle Frontal Gyrus	**46**	**L**	**-50**	**46**	**8**	**68**	**3.79**	**3.57**
Middle Frontal Gyrus	*46*	*L*	*-50*	*46*	*8*	*33*	*3.79*	*3.56*
Inferior Frontal Gyrus	*46*	*L*	*-47*	*35*	*11*	*34*	*3.64*	*3.57*
Inferior Frontal Gyrus	**9**	**L**	**-46**	**2**	**23**	**86**	**3.71**	**3.56**
Shared mediated pathway for all modalities (Conjunction)								
Middle Temporal Gyrus	21	R	59	-19	-4	25	N/A	N/A
Inferior Frontal Gyrus	45	R	57	24	6	37	N/A	N/A
Inferior Frontal Gyrus	47	R	42	17	-11	8	N/A	N/A
Caudate	Body	R	19	4	17	38	N/A	N/A
Medial Frontal Gyrus	9	R	12	44	19	6	N/A	N/A
Superior Frontal Gyrus	8	R	7	17	54	5	N/A	N/A
Superior Frontal Gyrus	8	R	4	37	44	6	N/A	N/A
Cingulate Gyrus	24	R	3	-8	35	33	N/A	N/A
Anterior Cingulate	24	R	3	24	21	7	N/A	N/A
Superior Frontal Gyrus	8	L	-4	28	51	15	N/A	N/A
Caudate	Body	L	-6	11	9	24	N/A	N/A
Superior Frontal Gyrus	6	L	-12	11	57	6	N/A	N/A
Inferior Frontal Gyrus	47	L	-36	24	-12	28	N/A	N/A
Inferior Frontal Gyrus	47	L	-44	29	1	10	N/A	N/A

Table A3.1.7: Results from the mediation analysis, showing regions where the relationship between their Maintain>Neutral contrast value and reported modality usage was mediated by the contrast values in the modality-specific regions identified above, identifying the extended networks supporting modality-specific usage. To identify regions unique to each modality, the statistical map for each modality was thresholded at Z>3.5, p>.00005 and k>40 and exclusively masked with the maps for the other modalities. To identify shared regions, the thresholded maps were conjoined. Cluster maxima are reported in bold for main contrasts, while conjunction results report coordinates of the cluster centroid. Italicised entries denote local maxima of subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function).

A3.2. Supplemental tables: Chapter 6

Table A3.2.1. Reappraisal results

Region	Side	Extent (voxel)	*t* (max)	*t* (avg)	MNI x y z	Label/BA
Reappraisal> Watch-Negative						
Preparation phase						
Cerebellum	**R**	**376**	**7.88**	**3.75**	**44 -64 -20**	**Declive**
Cerebellum	*R*	*65*	*7.88*	*4.35*	*44 -64 -20*	*Declive*
Cerebellum	*R*	*49*	*6.52*	*4.12*	*9 -80 -21*	*Declive*
Cerebellum	*R*	*31*	*5.75*	*3.64*	*33 -47 -24*	*Declive*
Cerebellum	*R*	*94*	*5.14*	*3.67*	*9 -67 -14*	*Declive*
Cerebellum	*R*	*49*	*4.49*	*3.15*	*21 -64 -20*	*Declive*
Cerebellum	*L*	*245*	*6.88*	*3.81*	*-45 -73 -20*	*Declive*
Cerebellum	*L*	*49*	*6.88*	*4.14*	*-45 -73 -20*	*Declive*
Cerebellum	*L*	*47*	*6.31*	*4.01*	*-47 -61 -19*	*Declive*
Cerebellum	*L*	*57*	*5.72*	*3.72*	*-26 -79 -17*	*Declive*
Posterior Cingulate	**R**	**150**	**6.85**	**3.72**	**0 -41 15**	**29**
Posterior Cingulate	*R*	*88*	*6.85*	*4.19*	*0 -41 15*	*29*
Lingual Gyrus	**L**	**35**	**4.44**	**3.01**	**-9 -61 6**	**18**
Precentral Gyrus	**R**	**66**	**6.46**	**3.62**	**37 -17 61**	**4**
Precentral Gyrus	*R*	*34*	*6.46*	*3.64*	*37 -17 61*	*4*
Postcentral Gyrus	*R*	*32*	*6.37*	*3.60*	*45 -19 54*	*3*
Middle Temporal Gyrus	**L**	**104**	**6.17**	**3.82**	**-58 -32 3**	**22**
Middle Temporal Gyrus	*L*	*66*	*6.17*	*4.13*	*-58 -32 3*	*22*
Inferior Frontal Gyrus	**L**	**79**	**6.11**	**3.58**	**-55 34 -6**	**47**
Inferior Frontal Gyrus	*L*	*45*	*6.11*	*3.62*	*-55 34 -6*	*47*
Inferior Frontal Gyrus	*L*	*34*	*5.31*	*3.53*	*-50 46 3*	*46*
Middle Frontal Gyrus	**L**	**74**	**5.91**	**3.63**	**-53 13 30**	**9**
Middle Frontal Gyrus	*L*	*50*	*5.91*	*3.87*	*-53 13 30*	*9*
Middle Frontal Gyrus	**L**	**66**	**5.76**	**3.76**	**-47 5 43**	**6**
Inferior Parietal Lobule	**L**	**77**	**5.26**	**3.55**	**-33 -61 38**	**39**
Implementation phase						
Cerebellum	**L**	**137**	**11.02**	**4.74**	**-43 -76 -22**	**Declive**
Cerebellum	*L*	*146*	*11.02*	*6.02*	*-43 -76 -22*	*Declive*
Cerebellum	*R*	*112*	*9.87*	*6.05*	*41 -64 -20*	*Declive*
Cerebellum	*R*	*70*	*9.44*	*5.34*	*13 -82 -26*	*Uvula*
Cerebellum	*R*	*92*	*9.05*	*5.09*	*36 -50 -25*	*Culmen*
Cerebellum	*R*	*65*	*8.03*	*4.69*	*36 -81 -25*	*Uvula*
Cerebellum	*R*	*192*	*7.48*	*4.88*	*19 -70 -20*	*Declive*
Cerebellum	*L*	*111*	*7.03*	*4.76*	*-40 -57 -22*	*Declive*

Cerebellum	*L*	*116*	*6.84*	*4.65*	*-25 -64 -23*	*Uvula*
Cerebellum	*L*	*34*	*6.14*	*4.04*	*-20 -83 -29*	*Tuber*
Cerebellum	*L*	*53*	*6.05*	*4.03*	*-27 -82 -19*	*Declive*
Cerebellum	*R*	*35*	*5.81*	*4.24*	*46 -67 -31*	*Pyramis*
Cerebellum	*R*	*37*	*5.41*	*3.90*	*19 -77 -35*	*Inferior Semi-Lunar Lobule*
Cerebellum	*R*	*90*	*5.21*	*3.75*	*3 -60 -14*	*Declive*
Cerebellum	*L*	*53*	*5.20*	*3.73*	*-42 -61 -36*	*Tonsil*
Cerebellum	*R*	*36*	*4.26*	*3.29*	*9 -52 -11*	*Culmen*
Inferior Frontal Gyrus	**L**	**524**	**10.73**	**4.95**	**-48 46 3**	**46**
Inferior Frontal Gyrus	*L*	*159*	*10.73*	*5.98*	*-48 46 3*	*46*
Inferior Frontal Gyrus	*L*	*47*	*8.26*	*4.70*	*-55 31 -6*	*47*
Superior Frontal Gyrus	**L**	**88**	**7.80**	**5.12**	**-32 57 13**	**10**
Inferior Frontal Gyrus	*L*	*55*	*7.76*	*4.38*	*-46 29 -9*	*47*
Inferior Frontal Gyrus	*L*	*33*	*7.57*	*4.57*	*-53 17 1*	*47*
Superior Frontal Gyrus	**L**	**65**	**6.85**	**3.99**	**-29 46 1**	**10**
Middle Frontal Gyrus	*L*	*77*	*6.18*	*4.18*	*-29 41 20*	*10*
Middle Frontal Gyrus	**L**	**478**	**9.44**	**4.57**	**-47 5 43**	**6**
Middle Frontal Gyrus	*L*	*139*	*9.44*	*5.49*	*-47 5 43*	*6*
Middle Frontal Gyrus	*L*	*63*	*7.71*	*4.01*	*-35 1 55*	*6*
Inferior Frontal Gyrus	**L**	**98**	**6.64**	**4.43**	**-45 11 28**	**9**
Middle Frontal Gyrus	*L*	*73*	*6.43*	*4.11*	*-49 29 27*	*46*
Middle Frontal Gyrus	*L*	*68*	*5.99*	*4.51*	*-50 13 35*	*9*
Middle Temporal Gyrus	**L**	**149**	**8.55**	**4.79**	**-61 -34 0**	**21**
Middle Temporal Gyrus	*L*	*75*	*8.55*	*5.19*	*-61 -34 0*	*21*
Middle Temporal Gyrus	*L*	*59*	*7.26*	*4.72*	*-50 -37 0*	*22*
Inferior Frontal Gyrus	**R**	**139**	**7.12**	**4.08**	**42 50 -1**	**10**
Supramarginal Gyrus	**L**	**470**	**7.11**	**4.34**	**-52 -57 35**	**40**
Supramarginal Gyrus	*L*	*187*	*7.11*	*4.75*	*-52 -57 35*	*40*
Inferior Parietal Lobule	*L*	*149*	*6.69*	*4.45*	*-49 -49 41*	*40*
Precuneus	*L*	*42*	*5.37*	*3.41*	*-35 -73 42*	*19*
Supramarginal Gyrus	**L**	**92**	**4.86**	**3.73**	**-58 -49 25**	**40**
Middle Frontal Gyrus	**R**	**159**	**6.19**	**3.72**	**49 25 25**	**46**
Middle Frontal Gyrus	*R*	*47*	*6.19*	*3.94*	*49 25 25*	*46*
Middle Frontal Gyrus	**R**	**72**	**5.56**	**3.60**	**43 16 33**	**9**
Inferior Parietal Lobule	**R**	**171**	**6.03**	**4.15**	**53 -52 39**	**40**
Inferior Parietal Lobule	*R*	*89*	*6.03*	*4.30*	*53 -52 39*	*40*
Supramarginal Gyrus	*R*	*55*	*5.25*	*4.02*	*48 -49 29*	*40*
Superior Frontal Gyrus	***L***	***169***	***5.66***	***3.64***	***-1 28 54***	***6***
Superior Frontal Gyrus	*L*	*39*	*5.66*	*3.90*	*-1 28 54*	*6*
Medial Frontal Gyrus	*L*	*45*	*5.18*	*3.57*	*-4 26 39*	*8*
Superior Frontal Gyrus	*L*	*50*	*4.95*	*3.69*	*-4 17 49*	*8*
Superior Temporal Gyrus	**R**	**71**	**5.51**	**3.48**	**61 -23 -2**	**21**
Middle Frontal Gyrus	**R**	**58**	**4.81**	**3.08**	**36 42 13**	**10**
Watch-Negative> Reappraisal						

Preparation phase						
Medial Frontal Gyrus	**L**	**798**	**-13.92**	**-5.29**	**-10 30 -15**	**25**
Medial Frontal Gyrus	*L*	*172*	*-13.92*	*-6.61*	*-10 30 -15*	*25*
Medial Frontal Gyrus	*R*	*162*	*-13.14*	*-6.38*	*11 30 -15*	*25*
Superior Frontal Gyrus	*R*	*42*	*-10.00*	*-5.32*	*21 39 -14*	*11*
Medial Frontal Gyrus	*L*	*59*	*-9.17*	*-4.80*	*-14 42 -12*	*10*
Inferior Frontal Gyrus	*R*	*48*	*-8.59*	*-3.92*	*41 31 -10*	*47*
Anterior Cingulate	**R**	**102**	**-7.27**	**-4.01**	**0 44 5**	**32**
Medial Frontal Gyrus	*R*	*84*	*-7.10*	*-4.66*	*15 55 4*	*10*
Superior Frontal Gyrus	*R*	*36*	*-6.95*	*-4.29*	*22 57 7*	*10*
Medial Frontal Gyrus	*L*	*43*	*-6.67*	*-4.44*	*-8 57 3*	*10*
Medial Frontal Gyrus	**L**	**50**	**-6.25**	**-4.31**	**-11 47 7**	**10**
Inferior Frontal Gyrus	**L**	**50**	**-8.17**	**-4.00**	**-39 18 -17**	**47**
Thalamus	**L**	**89**	**-7.20**	**-4.43**	**-3 -8 2**	**0**
Superior Frontal Gyrus	**R**	**283**	**-6.08**	**-3.44**	**23 43 31**	**9**
Superior Frontal Gyrus	*R*	*71*	*-6.08*	*-3.80*	*23 43 31*	*9*
Medial Frontal Gyrus	*L*	*55*	*-5.84*	*-3.62*	*-4 44 33*	*9*
Middle Frontal Gyrus	*R*	*48*	*-4.48*	*-3.11*	*38 30 37*	*9*
Middle Frontal Gyrus	*R*	*76*	*-4.46*	*-3.30*	*22 34 42*	*8*
Precuneus	**R**	**67**	**-5.51**	**-3.56**	**10 -54 58**	**7**
Middle Frontal Gyrus	**L**	**224**	**-5.49**	**-3.74**	**-29 34 34**	**9**
Middle Frontal Gyrus	*L*	*111*	*-5.49*	*-3.77*	*-29 34 34*	*9*
Middle Frontal Gyrus	*L*	*68*	*-5.48*	*-3.75*	*-25 31 47*	*8*
Middle Frontal Gyrus	*L*	*45*	*-5.32*	*-3.64*	*-32 25 32*	*9*
Lentiform Nucleus	**R**	**104**	**-5.31**	**-3.52**	**19 -9 -3**	**Medial Globus Pallidus**
Lentiform Nucleus	*R*	*22*	*-5.31*	*-3.70*	*19 -9 -3*	*Medial Globus Pallidus*
Amygdala	*R*	*35*	*-5.02*	*-3.42*	*25 -9 -11*	*Superficial*
Parahippocampal Gyrus	*R*	*41*	*-4.93*	*-3.64*	*24 -22 -13*	*35*
Superior Frontal Gyrus	**R**	**50**	**-4.81**	**-3.37**	**15 19 52**	**6**
Implentation phase						
Medial Frontal Gyrus	**L**	**642**	**-16.55**	**-5.88**	**-10 28 -15**	**25**
Medial Frontal Gyrus	*L*	*184*	*-16.55*	*-7.26*	*-10 28 -15*	*25*
Medial Frontal Gyrus	*R*	*173*	*-15.40*	*-7.31*	*11 33 -16*	*25*
Anterior Cingulate	*R*	*67*	*-7.20*	*-4.36*	*0 44 5*	*32*
Medial Frontal Gyrus	**R**	**90**	**-6.04**	**-4.25**	**15 52 4**	**10**
Inferior Frontal Gyrus	*R*	*63*	*-5.39*	*-3.46*	*30 30 -6*	*47*
Medial Frontal Gyrus	*L*	*37*	*-4.79*	*-3.33*	*-4 55 1*	*10*
Superior Temporal Gyrus	**L**	**55**	**-8.58**	**-4.69**	**-35 13 -19**	**38**
Precuneus	**R**	**997**	**-8.50**	**-4.12**	**9 -60 54**	**7**
Precuneus	*R*	*127*	*-8.50*	*-4.51*	*9 -60 54*	*7*
Precuneus	*R*	*45*	*-7.68*	*-4.56*	*12 -71 50*	*7*
Precuneus	*L*	*155*	*-7.54*	*-4.33*	*-12 -53 56*	*7*
Precuneus	*R*	*159*	*-6.58*	*-4.66*	*19 -52 53*	*7*
Precuneus	*R*	*88*	*-6.57*	*-4.08*	*24 -76 43*	*7*
Middle Temporal Gyrus	**R**	**100**	**-5.98**	**-3.98**	**39 -80 17**	**19**
Inferior Parietal Lobule	*R*	*88*	*-5.78*	*-4.02*	*34 -44 51*	*40*

Precuneus	*R*	*47*	*-5.57*	*-3.70*	*9 -80 40*	*19*
Cingulate Gyrus	*R*	*38*	*-5.00*	*-3.41*	*6 -43 38*	*31*
Superior Occipital Gyrus	**R**	**68**	**-4.99**	**-3.69**	**33 -82 25**	**19**
Superior Parietal Lobule	*L*	*33*	*-4.60*	*-3.14*	*-24 -47 59*	*7*
Hypothalamus	**R**	**86**	**-8.21**	**-4.89**	**0 -8 4**	**Hypothalamus**
Parahippocampal Gyrus	**L**	**149**	**-7.66**	**-4.50**	**-30 -32 -11**	**36**
Parahippocampal Gyrus	*L*	*84*	*-7.66*	*-4.71*	*-30 -32 -11*	*36*
Parahippocampal Gyrus	*L*	*65*	*-6.18*	*-4.23*	*-24 -46 -9*	*37*
Superior Occipital Gyrus	**L**	**90**	**-6.36**	**-3.77**	**-33 -82 31**	**19**
Parahippocampal Gyrus	**R**	**196**	**-6.33**	**-3.98**	**30 -33 -10**	**36**
Parahippocampal Gyrus	*R*	*123*	*-6.33*	*-4.10*	*30 -33 -10*	*36*
Hypothalamus	*R*	*31*	*-6.11*	*-3.86*	*11 -28 0*	*Hypothalamus*
Parahippocampal Gyrus	*R*	*42*	*-5.68*	*-3.70*	*23 -24 -13*	*35*
Amygdala	**R**	**271**	**-6.30**	**-3.84**	**25 -9 -11**	**Central**
Amygdala	*R*	*129*	*-6.30*	*-4.10*	*25 -9 -11*	*Central*
Insula	*R*	*100*	*-6.01*	*-3.74*	*44 1 1*	*13*
Superior Temporal Gyrus	*R*	*42*	*-5.11*	*-3.31*	*51 -8 -1*	*22*
Posterior Cingulate	**R**	**74**	**-6.12**	**-3.43**	**16 -57 17**	**30**
Superior Temporal Gyrus	**L**	**75**	**-6.02**	**-3.65**	**-58 -32 21**	**42**
Superior Temporal Gyrus	*L*	*40*	*-6.02*	*-3.92*	*-58 -32 21*	*42*
Inferior Parietal Lobule	*L*	*35*	*-5.12*	*-3.33*	*-52 -27 31*	*40*
Superior Temporal Gyrus	**L**	**139**	**-5.47**	**-3.66**	**-46 1 -5**	**22**
Superior Temporal Gyrus	*L*	*52*	*-5.47*	*-4.03*	*-46 1 -5*	*22*
Superior Temporal Gyrus	*L*	*40*	*-4.87*	*-3.30*	*-52 0 3*	*22*

Table A3.2.1. Reappraisal results. Brain regions activated and deactivated by Reappraisal relative to the Watch Negative condition. Cluster maxima are reported in bold. Local maxima (*italicised*) denote subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha < .05$ ($T > 3.36$, $p < .001$, $k > 30$) as determined by AFNI's *Alphasim* Monte Carlo simulation method. For consistency, subclusters smaller than the respective cluster thresholds are not reported.

Table A3.2.2. Emotion induction validation contrasts

Region	Side	Extent (voxel)	*t* (max)	*t* (avg)	MNI x	MNI y	MNI z	Label/BA
Watch-Negative> Watch-Neutral								
Thalamus	**L**	**1217**	**12.32**	**5.21**	**-3**	**-5**	**4**	**Ventral Anterior**
Thalamus	*L*	*169*	*12.32*	*5.92*	*-3*	*-5*	*4*	*Ventral Anterior*
Midbrain	*R*	*120*	*11.68*	*5.99*	*12*	*-9*	*-3*	*Subthalamic Nucleus*
Midbrain	*L*	*132*	*9.15*	*5.28*	*-8*	*-25*	*-8*	*Substania Nigra*
Midbrain	*R*	*68*	*8.84*	*5.63*	*9*	*-16*	*-4*	*Subthalamic Nucleus*
Thalamus	*R*	*60*	*8.67*	*5.38*	*12*	*-23*	*-1*	*Mammillary Body*
Cerebellum	*R*	*112*	*8.50*	*5.21*	*14*	*-37*	*-12*	*Culmen*
Thalamus	*L*	*102*	*8.48*	*5.07*	*0*	*-19*	*11*	*Pulvinar*
Thalamus	*R*	*76*	*7.94*	*4.99*	*21*	*-22*	*-4*	*Lateral Geniculum Body*
Cerebellum	*L*	*69*	*7.16*	*4.89*	*-2*	*-45*	*-12*	*Cerebellar Lingual*
Superior Temporal Gyrus	*R*	*58*	*5.24*	*4.20*	*50*	*13*	*-2*	*22*
Superior Temporal Gyrus	**L**	**153**	**11.80**	**5.29**	**-55**	**9**	**4**	**22**
Superior Temporal Gyrus	*L*	*116*	*11.80*	*5.48*	*-55*	*9*	*4*	*22*
Precuneus	**R**	**448**	**10.53**	**5.78**	**22**	**-52**	**53**	**7**
Precuneus	*R*	*194*	*10.53*	*5.98*	*22*	*-52*	*53*	*7*
Superior Parietal Lobule	*R*	*96*	*9.10*	*5.83*	*34*	*-47*	*53*	*7*
Inferior Parietal Lobule	*R*	*81*	*8.41*	*5.57*	*31*	*-38*	*46*	*40*
Postcentral Gyrus	*R*	*77*	*8.37*	*5.44*	*7*	*-47*	*64*	*7*
Middle Occipital Gyrus	**L**	**1740**	**10.36**	**4.79**	**-49**	**-74**	**-6**	**19**
Middle Occipital Gyrus	*L*	*75*	*10.36*	*5.44*	*-49*	*-74*	*-6*	*19*
Cuneus	*R*	*64*	*8.48*	*5.52*	*21*	*-84*	*32*	*19*
Middle Occipital Gyrus	*L*	*77*	*8.37*	*5.62*	*-48*	*-76*	*2*	*19*
Middle Occipital Gyrus	*L*	*174*	*8.24*	*5.26*	*-40*	*-77*	*11*	*19*
Cuneus	*R*	*77*	*7.72*	*5.12*	*12*	*-81*	*35*	*19*
Posterior Cingulate	*L*	*226*	*7.59*	*4.95*	*0*	*-49*	*20*	*30*
Middle Temporal Gyrus	*L*	*175*	*7.47*	*5.02*	*-50*	*-60*	*12*	*39*
Cuneus	*L*	*178*	*7.08*	*4.82*	*-3*	*-68*	*32*	*7*
Precuneus	*R*	*91*	*7.03*	*4.69*	*24*	*-80*	*23*	*31*
Middle Occipital Gyrus	*L*	*145*	*6.33*	*4.47*	*-22*	*-88*	*15*	*18*
Posterior Cingulate	*L*	*59*	*5.44*	*4.21*	*-3*	*-28*	*22*	*23*
Cuneus	*L*	*59*	*5.39*	*4.06*	*-19*	*-80*	*29*	*18*
Lingual Gyrus	*R*	*86*	*5.27*	*4.16*	*9*	*-74*	*4*	*18*
Middle Occipital Gyrus	*R*	*53*	*4.36*	*3.86*	*24*	*-87*	*10*	*18*
Middle Temporal Gyrus	**R**	**542**	**10.13**	**5.78**	**45**	**-62**	**-1**	**37**
Middle Temporal Gyrus	*R*	*240*	*10.13*	*6.47*	*45*	*-62*	*-1*	*37*
Middle Occipital Gyrus	*R*	*81*	*9.71*	*6.91*	*53*	*-68*	*-4*	*19*
Middle Temporal Gyrus	*R*	*86*	*6.62*	*4.75*	*51*	*-76*	*8*	*39*
Middle Temporal Gyrus	*R*	*63*	*5.83*	*4.71*	*58*	*-60*	*7*	*21*
Inferior Occipital Gyrus	*R*	*57*	*5.83*	*4.47*	*43*	*-84*	*-7*	*18*
Medial Frontal Gyrus	**R**	**70**	**10.11**	**5.21**	**15**	**2**	**67**	**6**
Medial Frontal Gyrus	**R**	**172**	**10.08**	**5.95**	**7**	**45**	**-16**	**11**

Insula	**R**	**271**	**9.66**	**5.14**	**53**	**-33**	**20**	**13**
Superior Parietal Lobule	**L**	**331**	**9.26**	**4.47**	**-27**	**-53**	**56**	**7**
Superior Parietal Lobule	*L*	*102*	*9.26*	*5.19*	*-27*	*-53*	*56*	*7*
Inferior Parietal Lobule	*L*	*66*	*5.49*	*4.25*	*-41*	*-59*	*45*	*40*
Inferior Parietal Lobule	*L*	*53*	*4.89*	*4.07*	*-34*	*-52*	*36*	*40*
Sub-Gyral	*L*	*51*	*4.55*	*3.95*	*-30*	*-42*	*52*	*40*
Parahippocampal Gyrus	**R**	**85**	**7.89**	**4.68**	**42**	**-35**	**-10**	**36**
Cerebellum	**L**	**159**	**7.89**	**4.74**	**0**	**-64**	**-29**	**Uvula**
Cerebellum	*L*	*71*	*7.89*	*5.23*	*0*	*-64*	*-29*	*Uvula*
Postcentral Gyrus	**L**	**72**	**5.73**	**4.17**	**-58**	**-27**	**23**	**40**
Watch-Neutral>								
Watch-Negative								
Middle Frontal Gyrus	**R**	**9880**	**15.10**	**5.26**	**35**	**53**	**-5**	**10**
Middle Frontal Gyrus	*R*	*234*	*15.10*	*8.16*	*35*	*53*	*-5*	*10*
Cerebellum	*L*	*158*	*14.11*	*8.85*	*-46*	*-68*	*-30*	*Tuber*
Postcentral Gyrus	*R*	*151*	*12.95*	*6.66*	*45*	*-16*	*54*	*3*
Inferior Frontal Gyrus	*R*	*153*	*12.93*	*7.84*	*45*	*52*	*2*	*46*
Inferior Frontal Gyrus	*L*	*118*	*12.80*	*6.36*	*-48*	*46*	*0*	*46*
Precentral Gyrus	*R*	*154*	*12.23*	*6.48*	*29*	*-22*	*67*	*4*
Precentral Gyrus	*L*	*120*	*11.46*	*6.20*	*-28*	*-24*	*67*	*4*
Anterior Cingulate	*L*	*102*	*11.17*	*5.76*	*-3*	*7*	*-4*	*25*
Cerebellum	*L*	*82*	*11.16*	*7.19*	*-43*	*-73*	*-23*	*Tuber*
Precentral Gyrus	*R*	*222*	*10.53*	*6.04*	*17*	*-24*	*67*	*4*
Fusiform Gyrus	*L*	*70*	*10.36*	*5.47*	*-23*	*-84*	*-14*	*19*
Inferior Frontal Gyrus	*L*	*92*	*10.18*	*5.50*	*-17*	*31*	*-12*	*47*
Superior Frontal Gyrus	*R*	*270*	*10.00*	*6.02*	*23*	*52*	*13*	*10*
Posterior Cingulate	*R*	*207*	*9.97*	*5.14*	*22*	*-57*	*15*	*30*
Cerebellum	*L*	*158*	*9.80*	*5.90*	*-4*	*-43*	*-43*	*Cerebellar Tonsil*
Middle Frontal Gyrus	*L*	*142*	*9.61*	*6.01*	*-32*	*48*	*-5*	*10*
Postcentral Gyrus	*L*	*56*	*9.56*	*5.18*	*-44*	*-22*	*55*	*3*
Medial Frontal Gyrus	*R*	*175*	*9.41*	*5.71*	*12*	*44*	*19*	*9*
Cerebellum	*L*	*165*	*9.38*	*5.92*	*-25*	*-64*	*-23*	*Uvula*
Anterior Cingulate	*L*	*58*	*9.25*	*5.29*	*-13*	*16*	*-9*	*25*
Inferior Frontal Gyrus	*R*	*100*	*9.21*	*5.44*	*50*	*12*	*19*	*44*
Middle Frontal Gyrus	*L*	*231*	*9.17*	*5.76*	*-28*	*40*	*1*	*10*
Cerebellum	*R*	*132*	*9.09*	*5.51*	*3*	*-43*	*-46*	*Cerebellar Tonsil*
Cerebellum	*R*	*49*	*9.04*	*5.55*	*46*	*-67*	*-28*	*Tuber*
Middle Frontal Gyrus	*R*	*57*	*8.87*	*6.33*	*51*	*42*	*-8*	*47*
Cerebellum	*R*	*85*	*8.86*	*4.83*	*36*	*-47*	*-29*	*Culmen*
Inferior Parietal Lobule	*R*	*83*	*8.82*	*5.16*	*50*	*-45*	*47*	*40*
Middle Frontal Gyrus	*R*	*167*	*8.73*	*5.10*	*46*	*16*	*30*	*9*
Superior Frontal Gyrus	*R*	*51*	*8.60*	*5.51*	*7*	*34*	*49*	*8*
Cerebellum	*R*	*48*	*8.30*	*5.62*	*41*	*-64*	*-20*	*Declive*
Superior Frontal Gyrus	*R*	*122*	*8.28*	*5.30*	*11*	*25*	*54*	*6*
Medial Frontal Gyrus	*L*	*145*	*8.14*	*5.18*	*-10*	*-20*	*56*	*6*
Inferior Frontal Gyrus	*R*	*174*	*8.08*	*5.36*	*46*	*25*	*15*	*46*

Middle Frontal Gyrus	*R*	*179*	*8.02*	*5.43*	*41*	*33*	*22*	*46*
Medial Frontal Gyrus	*L*	*162*	*7.93*	*5.39*	*0*	*42*	*25*	*9*
Middle Frontal Gyrus	*R*	*208*	*7.89*	*4.86*	*31*	*18*	*35*	*8*
Middle Frontal Gyrus	*R*	*155*	*7.82*	*5.19*	*34*	*9*	*47*	*6*
Inferior Parietal Lobule	*R*	*97*	*7.67*	*5.20*	*52*	*-34*	*48*	*40*
Middle Frontal Gyrus	*L*	*195*	*7.61*	*4.68*	*-26*	*2*	*37*	*6*
Medial Frontal Gyrus	*L*	*169*	*7.52*	*5.17*	*-15*	*41*	*20*	*9*
Middle Frontal Gyrus	*L*	*116*	*7.40*	*4.83*	*-28*	*17*	*35*	*8*
Anterior Cingulate	*R*	*114*	*7.35*	*4.55*	*14*	*17*	*21*	*32*
Parahippocampal Gyrus	*L*	*96*	*7.32*	*4.91*	*-27*	*-44*	*-9*	*37*
Parahippocampal Gyrus	*R*	*72*	*7.28*	*4.62*	*30*	*-43*	*-6*	*19*
Insula	*L*	*74*	*7.27*	*4.77*	*-34*	*22*	*17*	*13*
Cerebellum	*L*	*69*	*7.19*	*4.82*	*-32*	*-47*	*-27*	*Anterior Lobe*
Postcentral Gyrus	*L*	*115*	*7.17*	*4.56*	*-28*	*-24*	*40*	*3*
Medial Frontal Gyrus	*R*	*153*	*7.13*	*4.84*	*7*	*35*	*41*	*8*
Inferior Parietal Lobule	*R*	*74*	*7.13*	*4.86*	*53*	*-41*	*37*	*40*
Parahippocampal Gyrus	*R*	*84*	*7.12*	*4.89*	*21*	*-43*	*6*	*30*
Middle Frontal Gyrus	*L*	*91*	*7.11*	*4.72*	*-30*	*13*	*25*	*9*
Anterior Cingulate	*L*	*114*	*7.05*	*5.03*	*-22*	*29*	*19*	*32*
Supramarginal Gyrus	*R*	*111*	*7.03*	*4.97*	*40*	*-46*	*30*	*40*
Middle Frontal Gyrus	*R*	*122*	*6.95*	*4.89*	*29*	*39*	*39*	*8*
Medial Frontal Gyrus	*L*	*152*	*6.94*	*4.61*	*-15*	*32*	*37*	*8*
Middle Frontal Gyrus	*R*	*144*	*6.91*	*4.85*	*24*	*7*	*42*	*6*
Cerebellum	*L*	*63*	*6.84*	*4.84*	*-23*	*-76*	*-38*	*Inferior Semi-Lunar Lobule*
Cingulate Gyrus	*R*	*68*	*6.67*	*4.64*	*20*	*-4*	*44*	*24*
Middle Frontal Gyrus	*R*	*119*	*6.64*	*4.43*	*29*	*32*	*24*	*9*
Paracentral Lobule	*L*	*99*	*6.54*	*4.75*	*-10*	*-32*	*52*	*5*
Cerebellum	*R*	*57*	*6.21*	*4.51*	*18*	*-66*	*-25*	*Anterior Lobe*
Insula	*L*	*53*	*6.16*	*4.39*	*-42*	*-18*	*17*	*13*
Precentral Gyrus	*L*	*88*	*6.14*	*4.78*	*-16*	*-23*	*65*	*4*
Insula	*L*	*52*	*6.11*	*4.40*	*-34*	*-25*	*19*	*13*
Cerebellum	*L*	*63*	*6.02*	*4.48*	*-23*	*-40*	*-18*	*Culmen*
Superior Temporal Gyrus	*R*	*105*	*6.01*	*4.40*	*42*	*-49*	*15*	*22*
Paracentral Lobule	*R*	*122*	*5.98*	*4.63*	*10*	*-33*	*55*	*5*
Precentral Gyrus	*R*	*48*	*5.94*	*4.21*	*59*	*-13*	*36*	*4*
Caudate	*R*	*98*	*5.86*	*4.39*	*20*	*-4*	*31*	*Caudate Body*
Paracentral Lobule	*L*	*85*	*5.86*	*4.29*	*-4*	*-11*	*47*	*31*
Posterior Cingulate	*L*	*75*	*5.80*	*4.38*	*-19*	*-58*	*11*	*30*
Anterior Cingulate	*R*	*51*	*5.76*	*4.36*	*7*	*52*	*-2*	*10*
Cerebellum	*L*	*62*	*5.73*	*4.34*	*-21*	*-55*	*-12*	*Declive*
Supramarginal Gyrus	*R*	*78*	*5.67*	*4.51*	*45*	*-46*	*37*	*40*
Cerebellum	*L*	*48*	*5.65*	*4.25*	*-27*	*-59*	*-42*	*Cerebellar Tonsil*
Caudate	*L*	*67*	*5.63*	*4.28*	*-14*	*21*	*16*	*Caudate Body*
Anterior Cingulate	*R*	*67*	*5.27*	*4.12*	*7*	*31*	*-7*	*24*
Insula	*L*	*74*	*5.20*	*4.04*	*-34*	*-34*	*24*	*13*
Anterior Cingulate	*R*	*135*	*4.94*	*4.05*	*17*	*32*	*7*	*32*
Middle Frontal Gyrus	*L*	*63*	*4.85*	*4.00*	*-35*	*34*	*27*	*9*

Postcentral Gyrus	*R*	*67*	*4.61*	*3.90*	*31*	*-23*	*42*	*3*
Inferior Frontal Gyrus	*R*	*49*	*4.47*	*3.95*	*50*	*3*	*33*	*9*
Postcentral Gyrus	*L*	*58*	*4.38*	*3.86*	*-33*	*-30*	*47*	*3*
Middle Temporal Gyrus	**R**	**75**	**10.31**	**5.34**	**62**	**-17**	**-4**	**21**
Middle Temporal Gyrus	**L**	**72**	**9.65**	**4.60**	**-59**	**-5**	**-4**	**21**
Superior Occipital Gyrus	**R**	**79**	**7.32**	**4.29**	**42**	**-77**	**33**	**19**

Table A3.2.2. Emotion induction validation contrasts. Brain regions activated by negative stimuli (watch-negative > watch- neutral contrast) Cluster maxima are reported in bold. Local maxima (*italicised*) denote subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha < .05$ ($T > 3.36$, $p < .001$, $k > 30$) as determined by AFNI's *Alphasim* Monte Carlo simulation method. For consistency, subclusters smaller than the respective cluster thresholds are not reported.

Table A3.2.3. Compassion-related deactivations.

Region	Side	Extent (voxel)	*t* (max)	*t* (avg)	MNI x	y	z	Label/BA
Watch-Negative> Compassion								
Preparation phase								
Cerebellum	**L**	**242**	**9.55**	**4.18**	**-2**	**-54**	**-51**	**Cerebellar Tonsil**
Cerebellum	*L*	*71*	*9.55*	*5.40*	*-2*	*-54*	*-51*	*Cerebellar Tonsil*
Cerebellum	*R*	*87*	*5.75*	*3.56*	*20*	*-48*	*-36*	*Cerebellar Tonsil*
Brainstem	*L*	*50*	*5.10*	*3.43*	*3*	*-30*	*-33*	*Pons*
Mid/Inferior Frontal Gyrus	**R**	**539**	**7.09**	**3.82**	**51**	**12**	**38**	**6/44**
Mid/Inferior Frontal Gyrus	R	57	7.09	4.16	51	12	38	**6/44**
Inferior Frontal Gyrus	*R*	*84*	*7.04*	*4.41*	*52*	*24*	*25*	*45/44*
Inferior Frontal Gyrus	*R*	*53*	*6.44*	*3.87*	*53*	*33*	*17*	*45*
Inferior Frontal Gyrus	*R*	*105*	*5.72*	*3.97*	*44*	*0*	*18*	*9*
Inferior Frontal Gyrus	*R*	*69*	*4.99*	*3.74*	*50*	*12*	*16*	*44*
Inferior Frontal Gyrus	*R*	*73*	*4.79*	*3.41*	*40*	*20*	*13*	*45*
Cerebellum	**R**	**231**	**6.57**	**3.55**	**8**	**-33**	**-9**	**Culmen**
Parahippocampal Gyrus	*R*	*55*	*5.44*	*3.62*	*20*	*-24*	*-15*	*Subicular*
Supramarginal gyrus	**R**	**168**	**5.94**	**3.99**	**48**	**-44**	**29**	**40**
Calcarine gyrus	**R**	**173**	**5.56**	**3.43**	**19**	**-57**	**17**	**30**
Implementation phase								
Precuneus	**R**	**784**	**9.85**	**4.18**	**12**	**-44**	**6**	**29**
Precuneus	*R*	*102*	*9.85*	*5.28*	*12*	*-44*	*6*	*29*
Hippocampus	*R*	*116*	*8.02*	*4.34*	*11*	*-37*	*-7*	*Subicular*
Lingual Gyrus	*L*	*126*	*7.75*	*4.61*	*-9*	*-46*	*1*	*18/Hipp*
Lingual Gyrus	*L*	*55*	*7.16*	*4.29*	*-14*	*-26*	*-11*	*18/Hipp*
Hippocampus	*L*	*52*	*6.63*	*3.87*	*-8*	*-33*	*-9*	*Subicular*
Fusiform Gyrus	*R*	*61*	*6.39*	*3.92*	*23*	*-41*	*-15*	*37*
Parahippocampal Gyrus	*R*	*57*	*5.83*	*3.90*	*17*	*-22*	*-14*	*Subicular*
Lingual Gyrus	*R*	*67*	*5.72*	*3.73*	*15*	*-54*	*-1*	*17/18*

Posterior Cingulate	*R*	*47*	*4.76*	*3.34*	*28*	*-63*	*6*	*30*
Inferior Frontal Gyrus	**R**	**375**	**8.10**	**4.03**	**53**	**33**	**17**	**45**
Inferior Frontal Gyrus	*R*	*79*	*8.10*	*4.45*	*53*	*33*	*17*	*45*
Inferior Frontal Gyrus	*R*	*61*	*6.98*	*4.13*	*53*	*33*	*-9*	*45*
Inferior Frontal Gyrus	*R*	*61*	*6.17*	*4.14*	*49*	*28*	*27*	*45*
Inferior Frontal Gyrus	*R*	*92*	*5.18*	*3.82*	*37*	*29*	*-5*	*47*
Inferior Frontal Gyrus	*R*	*52*	*4.72*	*3.45*	*59*	*15*	*14*	*44*
Superior Temporal Gyrus	**R**	**256**	**7.77**	**4.02**	**44**	**11**	**-16**	**38**
Superior Temporal Gyrus	*R*	*133*	*7.77*	*4.21*	*44*	*11*	*-16*	*38*
Insula	*R*	*65*	*5.56*	*3.49*	*43*	*-10*	*3*	*13*
Insula	*R*	*58*	*5.43*	*4.18*	*43*	*-1*	*-4*	*13*

Table A3.2.3. Compassion-related deactivation. . Brain regions deactivated during Compassion (Watch-Negative > Compassion) Cluster maxima are reported in bold. Local maxima (*italicised*) denote subclusters within a cluster found to be not connected to the already considered (central) mass in a higher-values-first watershed searching algorithm implemented in NeuroElf (i.e. the splitclustercoords function). Multiple comparisons were controlled for using cluster-level FWE correction at $\alpha < .05$ ($T > 3.36$, $p < .001$, $k > 30$) as determined by AFNI's *Alphasim* Monte Carlo simulation method. For consistency, subclusters smaller than the respective cluster thresholds are not reported.

A3.2. Supplemental tables: Chapter 7

Table A3.3.1. Dimension of trait affect. PCA results

Scale	Loading
PANAS_NEG	0.646
PANAS_POS	-0.702
TTPAS_Active	-0.418
TTPAS_Relaxed	-0.393
TTPAS_Warmth	-0.667
STAI_ta	0.867
bdi_total	0.577
atq_negative_affect	0.636
atq_extraversion	-0.531
NEO_NEG_AFF	0.742
NEO_POS_AFF	-0.635

Table A3.3.1. Dimension of trait affect. PCA results. Loadings of scales on the single trait affect component revealed by PCA analysis (see *Appendix*: Figure A2.3.1A). PANAS = Positive and Negative Affect Scale, TTPAS = Types of Positive Affect Scale, STAI_ta = State Trait Anxiety Inventory – Trait affect version, bdi_total = Beck's Depression Inventory II – full scale score, atq= Adult Temperament Questionnaire, NEO_NEG_AFF/ NEO_POS_AFF = NEO Five Factor Inventory, Positive and Negative affect subscales.

Table A3.3.2. Dimensions of emotion management: PCA results.

Scale	Emotion-focused	Maladaptive	Instrumental
CERQ.SelfBlame	**0.254**	**0.337**	0.11
CERQ.Acceptance	**0.64**	0.043	-0.106
CERQ.Rumination	0.101	**0.492**	**0.319**
CERQ.PosRefocus	**0.584**	0.024	0.044
CERQ.PlanningRefocus	**0.437**	**-0.209**	**0.52**
CERQ.PosReappraisal	**0.707**	-0.161	**0.266**
CERQ.Perspective	**0.665**	-0.121	0.089
CERQ.Catastrophizing	-0.153	**0.647**	0.146
CERQ.OtherBlame	0.186	**0.492**	0.016
COPE_Distraction	**0.355**	0.15	0.003
COPE_Denial	-0.032	**0.393**	-0.067
COPE_EmotionalSup	-0.068	0.1	**0.697**
COPE_Withdrawal	**0.28**	**0.279**	**-0.353**
COPE_PosReinterp	**0.68**	**-0.211**	0.197
COPE_Humor	**0.556**	0.088	-0.089
COPE_Active	**0.266**	**-0.283**	**0.634**
COPE_Drugs	0.013	0.47	0.027
COPE_InstrumentalSup	-0.134	0.094	**0.718**
COPE_ActOut	-0.005	**0.311**	**0.426**
COPE_Plan	**0.259**	-0.104	**0.597**
COPE_Accept	**0.581**	-0.17	0.037
COPE_SelfBlame	0.043	**0.596**	-0.115
EROS.ExtImprov	**0.314**	0.098	**0.393**
EROS.ExtWorse	-0.078	**0.551**	0.053
EROS.IntWorse	-0.136	**0.684**	0.001
EROS.IntImprov	**0.493**	0.206	**0.347**
atq_effortful_control	0.092	**-0.539**	**0.221**

Table A3.3.2. Dimensions of emotion management: PCA results. Loadings on the three components revealed by PCA analysis of scales measuring different emotion management styles (see *Appendix*: Figure A2.3.1B). Varimax rotation applied. Bolded items denote items loading more than .2. CERQ = Cognitive Emotion Regulation Questionnaire, COPE = Cope inventory, EROS = Emotion Regulation of Others and Self, ATQ = Adult Temperament Questionnaire.

Table A3.3.3. Assignment of non-described generation modalities.

Description	**Assigned**
Recalling past or future situations	Episodic Imagery
Optically imagining situations	Episodic Imagery
Memories	Episodic Imagery
Flying dream- Imagination is a mix of visual and bodily, and mostly mixed together	Episodic Imagery/ Bodily Interoception
Tactile memories	Bodily Interoception
Breathing	Bodily Interoception
Good and bad memories	Episodic Imagery
Memories of good and bad feelings	Episodic Imagery
Rekindling memories	Episodic Imagery
Going into the feeling	Not assignable
Concentrated on generating the feeling (feeling happy, positive, excited). Tried to amplify feelings, memories and images that were evoked.	Episodic Imagery
Recalling memories, mentally playing guitar	Episodic Imagery/ Auditory Imagery
I thought of stories or events in my own past.	Episodic Imagery
Memories of feelings	Episodic Imagery

Table A3.3.3. Assignment of non-described generation modalities. Translated descriptions for each of the 14 participants reporting using "Other" modalities is reported, together with their assignment

A4. List of abbreviations

µS	microsiemens
AC/PC	Anterior Commissure/Posterior Commissure
AI	Anterior insula
ALFF	Amplitude of low frequency fluctuations
AMY	Amygdala
AUC	Area-under-the-curve
BA	Broca's area
BDI-2	Beck's depression inventory
BOLD	Blood-oxygen level dependent
CM	Compassion meditation
CPCA	constrained principle component analysis
CSF	Cerebrospinal fluid
dACC	dorsal anterior cingulate cortex
dlPFC	Dorsolateral prefrontal cortex
DMN	Default mode network
dmPFC	Dorsomedial prefrontal cortex
EEG	Electroencephalogram
EnGE	Endogenous generation of emotion
EPN	Early posterior negativity
ERP	Event-related potential
ExGE	Exogenous generation of emotion
FIR	Finite impulse response
fMRI	Functional magnetic resonance imaging
FOV	Field of view
FPCN	The Frontoparietal Control Network
FWEc	Family wise error rate
GLM	General Linear Model
pgACC	Perigenual anterior cingulate cortex
GSR	Galvanic skin response
HC	Hippocampus
HRF	hemodynamic response function
Hz	Hertz
ICN	Intrinsic Connectivity Network
IFG	Inferior frontal gyrus
iPat	Integrated parallel acquisition
IQ	Intelligence quotient
ITG	Inferior temporal gyrus
LKM	Loving-kindness meditation
LMM	Linear mixed modelling
LPP	Late positive potential
LTM	Long-term meditation practitioners
MCC	Mid cingulate cortex
MEPM	Mediation effect parametric mapping
MFG	Middle frontal gyrus
mm	Millimeter
mOFC	Medial orbitofrontal cortex
ms	millisecond

MTG	Middle temporal gyrus
NACC	Nucleus accumbens
p	*p*-value
PAG	Periaqueductal gray
PCA	Principal component analysis
PCC	Posterior Cingulate Cortex
PCC	Posterior cingulate cortex
PET	Positron emission tomography
PFC	Prefrontal cortex
PMC	Premotor cortex
Pre-SMA	Presupplemental motor area
RAGE	The Regulation and Generation of Emotion
RE	Reappraisal
ROI	Region of interest
rs-fMRI	Resting state fMRI
s	second
SCID	Structured Clinical Interview for DSM-IV
SCL	Skin conductance level
SD	Standard deviation
SFG	Superior frontal gyrus
sgACC	Subgenual anterior cingulate cortex
SGT	Self-generated thought
SMG	Supramarginal gyrus
SN	The Salience Network
SN/VTA	Substantia nigra/ventral tegmental area
SoVT	Socio-affective Video Task
STAI-T	Spielberger trait anxiety scale
STG	Superior temporal gyrus
TRANS	Transversal Gyrus
TE	Echo time
TI	Inversion time
TPJ	Temporoparietal junction
TR	Repetition time
VAS	Visual analogue scale
VLPFC	Ventrolateral prefrontal cortex
VMPFC	Ventromedial prefrontal cortex
VS	Ventral striatum
WM	White matter

List of Figures

List of Tables

Bibliography

Addis, D. R., Pan, L., Vu, M.-A., Laiser, N., & Schacter, D. L. (2009). Constructive episodic simulation of the future and the past: Distinct subsystems of a core brain network mediate imagining and remembering. *Neuropsychologia*, *47*, 2222–2238.

Addis, D. R., Wong, A. T., & Schacter, D. L. (2007). Remembering the past and imagining the future: Common and distinct neural substrates during event construction and elaboration. *Neuropsychologia*, *45*, 1363–1377.

Adriaenssens, J., De Gucht, V., & Maes, S. (2014). Determinants and prevalence of burnout in emergency nurses: A systematic review of 25 years of research. *International Journal of Nursing Studies*, *52*, 649–661.

Albertin, S. V., Mulder, A. B., Tabuchi, E., Zugaro, M. B., & Wiener, S. I. (2000). Lesions of the medial shell of the nucleus accumbens impair rats in finding larger rewards, but spare reward-seeking behavior. *Behavioural Brain Research*, *117*, 173–183.

Alcaro, A., Huber, R., & Panksepp, J. (2007). Behavioral functions of the mesolimbic dopaminergic system: An affective neuroethological perspective. *Brain Research Reviews*, *56*, 283–321.

Aldao, A., Jazaieri, H., Goldin, P. R., & Gross, J. J. (2014). Adaptive and maladaptive emotion regulation strategies: Interactive effects during CBT for social anxiety disorder. *Journal of Anxiety Disorders*, *28*, 382–389.

Ally, B. A., Hussey, E. P., & Donahue, M. J. (2013). A case of hyperthymesia: rethinking the role of the amygdala in autobiographical memory. *Neurocase*, *19*, 166–181.

Andrews-Hanna, J. R. (2012). The brain's default network and its adaptive role in internal mentation. *The Neuroscientist*, *18*, 251–270.

Andrews-Hanna, J. R., Reidler, J. S., Sepulcre, J., Poulin, R., & Buckner, R. L. (2010). Functional-anatomic fractionation of the brain's default network. *Neuron*, *65*, 550–562.

Andrews-Hanna, J. R., Smallwood, J., & Spreng, R. N. (2014). The default network and self-generated thought: component processes, dynamic control, and clinical relevance. *Annals of the New York Academy of Sciences*, *1316*, 29–52.

Arbuthnott, K., & Frank, J. (2000). Trail making test, part B as a measure of executive control: validation using a set-switching paradigm. *Journal of Clinical and Experimental Psychology*, *22*, 518–528.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex: one decade on. *Trends in Cognitive Sciences*, *18*, 177–185.

Ashburner, J. (2007). A fast diffeomorphic image registration algorithm. *NeuroImage*, *38*, 95–113.

Badre, D., & Wagner, A. D. (2007). Left ventrolateral prefrontal cortex and the cognitive control of memory. *Neuropsychologia*, *45*, 2883–2901.

Balleine, B. W., & Killcross, S. (2006). Parallel incentive processing: an integrated view of amygdala function. *Trends in Neurosciences*, *29*, 272–279.

Banich, M. T., Mackiewicz, K. L., Depue, B. E., Whitmer, A. J., Miller, G. A., & Heller, W. (2009). Cognitive control mechanisms, emotion and memory: a neural perspective with implications for psychopathology. *Neuroscience & Biobehavioral Reviews*, *33*, 613–630.

Barrett, L. F. (2011). Constructing Emotion. *Psychological Topics*, *20*, 359–380.

Barrett, L. F. (2014). The Conceptual Act Theory: A Precis. *Emotion Review*, *6*, 292–297.

Barrett, L. F., & Bar, M. (2009). See it with feeling: affective predictions during object perception. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *364*, 1325–1334.

Barrett, L. F., & Wilson-Mendenhall, C. D. (2014). The Conceptual Act Theory: A road map.

In L. F. Barrett & J. A. Russell, *The Psychological Construction of Emotion*. New York, London: Guilford Publications.

Barrett, L. F., Mesquita, B., Ochsner, K. N., & Gross, J. J. (2007a). The Experience of Emotion. *Annual Review of Psychology*, *58*, 373–403.

Barrett, L. F., Ochsner, K. N., & Gross, J. J. (2007b). On The Automaticity of Emotion. In J. A. Bargh, *Social Psychology and the Unconscious*. Psychology Press.

Barsalou, L. W. (2008). Grounded Cognition. *Annual Review of Psychology*, *59*, 617–645.

Baumgartner, H., Pieters, R., & Bagozzi, R. P. (2008). Future-oriented emotions: conceptualization and behavioral effects. *European Journal of Social Psychology*, *38*, 685–696.

Beaty, R. E., Benedek, M., Silvia, P. J., & Schacter, D. L. (2016). Creative Cognition and Brain Network Dynamics. *Trends in Cognitive Sciences*, *20*, 87–95.

Beaty, R. E., Benedek, M., Wilkins, R. W., Jauk, E., Fink, A., Silvia, P. J., et al. (2014). Creativity and the default network: A functional connectivity analysis of the creative brain at rest. *Neuropsychologia*, *64*, 92–98.

Beaty, R. E., Burgin, C. J., Nusbaum, E. C., Kwapil, T. R., Hodges, D. A., & Silvia, P. J. (2013). Music to the inner ears: exploring individual differences in musical imagery. *Consciousness and Cognition*, *22*, 1163–1173.

Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. F. (1996). Comparison of Beck Depression Inventories-IA and-II in Psychiatric Outpatients. *Journal of Personality Assessment*, *67*, 588–597.

Beckmann, M., Johansen-Berg, H., & Rushworth, M. F. S. (2009). Connectivity-based parcellation of human cingulate cortex and its relation to functional specialization. *The Journal of Neuroscience*, *29*, 1175–1190.

Behbehani, M. M. (1995). Functional characteristics of the midbrain periaqueductal gray. *Progress in Neurobiology*, *46*, 575–605.

Benedek, M., & Kaernbach, C. (2010a). A continuous measure of phasic electrodermal activity. *Journal of Neuroscience Methods*, *190*, 80–91.

Benedek, M., & Kaernbach, C. (2010b). Decomposition of skin conductance data by means of nonnegative deconvolution. *Psychophysiology*, *47*, 647–658.

Benoit, R. G., Szpunar, K. K., & Schacter, D. L. (2014). Ventromedial prefrontal cortex supports affective future simulation by integrating distributed knowledge. *Proceedings of the National Academy of Sciences of the United States of America*, *111*, 16550–16555.

Bergman, R. L., & Craske, M. G. (2000). Verbalization and imagery during worry activity. *Depression and Anxiety*, *11*, 169–174.

Bernhardt, B. C., & Singer, T. (2012). The neural basis of empathy. *Annual Review of Neuroscience*, *35*, 1–23.

Bernhardt, B. C., Klimecki, O. M., Leiberg, S., & Singer, T. (2014). Structural covariance networks of the dorsal anterior insula predict females' individual differences in empathic responding. *Cerebral Cortex*, *24*, 2189–2198.

Berridge, K. C., & Kringelbach, M. L. (2013). Neuroscience of affect: brain mechanisms of pleasure and displeasure. *Current Opinion in Neurobiology*, *23*, 294–303.

Berridge, K. C., & Kringelbach, M. L. (2015). Pleasure Systems in the Brain. *Neuron*, *86*, 646–664.

Binder, J. R., Desai, R. H., Graves, W. W., & Conant, L. L. (2009). Where is the semantic system? A critical review and meta-analysis of 120 functional neuroimaging studies. *Cerebral Cortex*, *19*, 2767–2796.

Blackwell, S. E., & Holmes, E. A. (2010). Modifying interpretation and imagination in clinical depression: A single case series using cognitive bias modification. *Applied Cognitive Psychology*, *24*, 338–350.

Blechert, J., Wilhelm, F. H., Williams, H., Braams, B. R., Jou, J., & Gross, J. J. (2015).

Reappraisal facilitates extinction in healthy and socially anxious individuals. *Journal of Behavior Therapy and Experimental Psychiatry*, *46*, 141–150.
Bonanno, G. A., & Burton, C. L. (2013). Regulatory Flexibility: An Individual Differences Perspective on Coping and Emotion Regulation. *Perspectives on Psychological Science : a Journal of the Association for Psychological Science*, *8*, 591–612.
Borkenau, P., & Ostendorf, F. (2008). NEO-Fünf-Faktoren-Inventar nach Costa und McCrae (NEO-FFI) (Handweisung). Göttingen: Hogrefe.
Brass, M. (2002). The Role of the Frontal Cortex in Task Preparation. *Cerebral Cortex*, *12*, 908–914.
Brass, M., & Eickhoff, S. B. (2016). Co-Activation-Based Parcellation of the Lateral Prefrontal Cortex Delineates the Inferior Frontal Junction Area. *Cerebral Cortex*, *26*, 2225–2241.
Brewin, C. R., Gregory, J. D., Lipton, M., & Burgess, N. (2010). Intrusive images in psychological disorders: characteristics, neural mechanisms, and treatment implications. *Psychological Review*, *117*, 210–232.
Buchanan, T. W. (2007). Retrieval of emotional memories. *Psychological Bulletin*, *133*, 761–779.
Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The Brain's Default Network. *Annals of the New York Academy of Sciences*, *1124*, 1–38.
Buckner, R. L., Krienen, F. M., & Yeo, B. T. T. (2013). Opportunities and limitations of intrinsic functional connectivity MRI. *Nature Neuroscience*, *16*, 832–837.
Buhle, J. T., Kober, H., Ochsner, K. N., Mende-Siedlecki, P., Weber, J., Hughes, B. L., et al. (2013). Common representation of pain and negative emotion in the midbrain periaqueductal gray. *Social Cognitive and Affective Neuroscience*, *8*, 609–616.
Buhle, J. T., Silvers, J. A., Wager, T. D., Lopez, R., Onyemekwu, C., Kober, H., et al. (2014). Cognitive reappraisal of emotion: a meta-analysis of human neuroimaging studies. *Cerebral Cortex*, *24*, 2981–2990.
Buzsáki, G., & Moser, E. I. (2013). Memory, navigation and theta rhythm in the hippocampal-entorhinal system. *Nature Neuroscience*, *16*, 130–138.
Cameron, C. D., & Payne, B. K. (2011). Escaping affect: How motivated emotion regulation creates insensitivity to mass suffering. *Journal of Personality and Social Psychology*, *100*, 15.
Cameron, C. D., & Payne, B. K. (2012). The cost of callousness: regulating compassion influences the moral self-concept. *Psychological Science*, *23*, 225–229.
Canli, T., Cooney, R. E., Cooney, R. E., Goldin, P., Goldin, P., Shah, M., et al. (2005). Amygdala reactivity to emotional faces predicts improvement in major depression., *16*, 1267–1270.
Carver, C. S., & Scheier, M. (1998). On the self-regulation of behavior. Cambridge, UK ; New York, NY, USA : Cambridge University Press.
Catalino, L. I., & Fredrickson, B. L. (2011). A Tuesday in the life of a flourisher: The role of positive emotional reactivity in optimal mental health. *Emotion*, *11*, 938–950.
Cauda, F., D'Agata, F., Sacco, K., Duca, S., Geminiani, G., & Vercelli, A. (2011). Functional connectivity of the insula in the resting brain. *NeuroImage*, *55*, 8–23.
Cha, J., DeDora, D., Nedic, S., Ide, J., Greenberg, T., Hajcak, G., & Mujica-Parodi, L. R. (2016). Clinically Anxious Individuals Show Disrupted Feedback between Inferior Frontal Gyrus and Prefrontal-Limbic Control Circuit. *The Journal of Neuroscience*, *36*, 4708–4718.
Chang, L. J., Gianaros, P. J., Manuck, S. B., Krishnan, A., & Wager, T. D. (2015). A Sensitive and Specific Neural Signature for Picture-Induced Negative Affect. *PLoS Biology*, *13*, e1002180.
Chao-Gan, Y., & Yu-Feng, Z. (2010). DPARSF: A MATLAB Toolbox for “Pipeline” Data

Analysis of Resting-State fMRI. *Frontiers in Systems Neuroscience*, *4*, 13.
Christoff, K., Gordon, A. M., Smallwood, J., Smith, R., & Schooler, J. W. (2009). Experience sampling during fMRI reveals default network and executive system contributions to mind wandering. *Proceedings of the National Academy of Sciences of the United States of America*, *106*, 8719–8724.
Citron, F. M. M., Weekes, B. S., & Ferstl, E. C. (2013). Effects of valence and arousal on written word recognition: Time course and ERP correlates. *Neuroscience Letters*, *533*, 90–95.
Clark, D. A., & Beck, A. T. (2010). Cognitive theory and therapy of anxiety and depression: convergence with neurobiological findings. *Trends in Cognitive Sciences*, *14*, 418–424.
Cohn, M. A., & Fredrickson, B. L. (2010). In search of durable positive psychology interventions: Predictors and consequences of long-term positive behavior change. *The Journal of Positive Psychology*, *5*, 355–366.
Cole, M. W., Reynolds, J. R., Power, J. D., Repovs, G., Anticevic, A., & Braver, T. S. (2013). Multi-task connectivity reveals flexible hubs for adaptive task control. *Nature Neuroscience*, *16*, 1348–1355.
Condon, P., & Barrett, L. F. (2013). Conceptualizing and experiencing compassion. *Emotion*, *13*, 817–821.
Cooney, R. E., Joormann, J., Eugène, F., Dennis, E. L., & Gotlib, I. H. (2010). Neural correlates of rumination in depression. *Cognitive, Affective & Behavioral Neuroscience*, *10*, 470–478.
Corbit, L. H., Corbit, L. H., & Balleine, B. W. (2005). Double dissociation of basolateral and central amygdala lesions on the general and outcome-specific forms of pavlovian-instrumental transfer. *The Journal of Neuroscience*, *25*, 962–970.
Corbit, L. H., Corbit, L. H., Muir, J. L., Muir, J. L., Balleine, B. W., & Balleine, B. W. (2001). The role of the nucleus accumbens in instrumental conditioning: Evidence of a functional dissociation between accumbens core and shell. *The Journal of Neuroscience*, *21*, 3251–3260.
Corradi-Dell'Acqua, C., Tusche, A., Vuilleumier, P., & Singer, T. (2016). Cross-modal representations of first-hand and vicarious pain, disgust and fairness in insular and cingulate cortex. *Nature Communications*, *7*, 10904.
Costafreda, S. G., Fu, C. H. Y., Lee, L., Everitt, B., Brammer, M. J., & David, A. S. (2006). A systematic review and quantitative appraisal of fMRI studies of verbal fluency: role of the left inferior frontal gyrus. *Human Brain Mapping*, *27*, 799–810.
Cotman, C. W., & Berchtold, N. C. (2002). Exercise: a behavioral intervention to enhance brain health and plasticity. *Trends in Neurosciences*, *25*, 295–301.
Coulombe, M.-A., Erpelding, N., Kucyi, A., & Davis, K. D. (2016). Intrinsic functional connectivity of periaqueductal gray subregions in humans. *Human Brain Mapping*, *37*, 1514–1530.
Craig, A. D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Reviews Neuroscience*, *3*, 655–666.
Craig, A. D. B. (2009). How do you feel--now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, *10*, 59–70.
Craig, A. D. B. (2011). Significance of the insula for the evolution of human awareness of feelings from the body. *Annals of the New York Academy of Sciences*, *1225*, 72–82.
Dahl, C. J., Lutz, A., & Davidson, R. J. (2015). Reconstructing and deconstructing the self: cognitive mechanisms in meditation practice. *Trends in Cognitive Sciences*, *19*, 515–523.
Dale, A. M., Fischl, B., & Sereno, M. I. (1999). Cortical Surface-Based Analysis. *NeuroImage*, *9*, 179–194.
Damasio, A. (1994). Descartes' Error. New York: Putnam Publishing.
Damasio, A. R., Grabowski, T. J., Bechara, A., Damasio, H., Ponto, L. L. B., Parvizi, J., &

Hichwa, R. D. (2000). Subcortical and cortical brain activity during the feeling of self-generated emotions. *Nature Neuroscience*, *3*, 1049–1056.

Daselaar, S. M., Rice, H. J., Greenberg, D. L., Cabeza, R., LaBar, K. S., & Rubin, D. C. (2008). The spatiotemporal dynamics of autobiographical memory: neural correlates of recall, emotional intensity, and reliving. *Cerebral Cortex (New York, N.Y. : 1991)*, *18*, 217–229.

Decety, J., & Lamm, C. (2007). The Role of the Right Temporoparietal Junction in Social Interaction: How Low-Level Computational Processes Contribute to Meta-Cognition. *The Neuroscientist*, *13*, 580–593.

Delamillieure, P., Doucet, G., Mazoyer, B., Turbelin, M.-R., Delcroix, N., Mellet, E., et al. (2010). The resting state questionnaire: An introspective questionnaire for evaluation of inner experience during the conscious resting state. *Brain Research Bulletin*, *81*, 565–573.

Denkova, E., Dolcos, S., & Dolcos, F. (2015). Neural correlates of "distracting"from emotion during autobiographical recollection. *Social Cognitive and Affective Neuroscience*, *10*, 219–230.

Denny, B. T., Ochsner, K. N., Weber, J., & Wager, T. D. (2014). Anticipatory brain activity predicts the success or failure of subsequent emotion regulation. *Social Cognitive and Affective Neuroscience*, *9*, 403–411.

Diedrich, A., Hofmann, S. G., Cuijpers, P., & Berking, M. (2016). Self-compassion enhances the efficacy of explicit cognitive reappraisal as an emotion regulation strategy in individuals with major depressive disorder. *Behaviour Research and Therapy*, *82*, 1–10.

Dolan, R. J., & Vuilleuimier, P. (2006). Amygdala Automaticity in Emotional Processing. *Annals of the New York Academy of Sciences*, *985*, 348–355.

Dosenbach, N. U. F., Fair, D. A., Cohen, A. L., Schlaggar, B. L., & Petersen, S. E. (2008). A dual-networks architecture of top-down control. *Trends in Cognitive Sciences*, *12*, 99–105.

Dosenbach, N. U. F., Visscher, K. M., Palmer, E. D., Miezin, F. M., Wenger, K. K., Kang, H. C., et al. (2006). A Core System for the Implementation of Task Sets. *Neuron*, *50*, 799–812.

Dörfel, D., Lamke, J.-P., Hummel, F., Wagner, U., Erk, S., & Walter, H. (2014). Common and differential neural networks of emotion regulation by Detachment, Reinterpretation, Distraction, and Expressive Suppression: a comparative fMRI investigation. *NeuroImage*, *101*, 298–309.

Draganski, B., & May, A. (2008). Training-induced structural changes in the adult human brain. *Behavioural Brain Research*, *192*, 137–142.

Draganski, B., Gaser, C., Busch, V., Schuierer, G., Bogdahn, U., & May, A. (2004). Neuroplasticity: Changes in grey matter induced by training. *Nature*, *427*, 311–312.

Duvernoy, H. M. (1999). The Human Brain (2nd ed.). Wien: Springer.

Duvernoy, H. M., Cattin, F., & Risold, P.-Y. (2013). The Human Hippocampus. Heidelberg: Springer.

Dyrbye, L. N., West, C. P., Satele, D., Boone, S., Tan, L., Sloan, J., & Shanafelt, T. D. (2014). Burnout Among U.S. Medical Students, Residents, and Early Career Physicians Relative to the General U.S. Population. *Academic Medicine*, *89*, 443–451.

Eerola, T., & Vuoskoski, J. K. (2010). A comparison of the discrete and dimensional models of emotion in music. *Psychology of Music*, *39*, 18–49.

Eickhoff, S. B., Bzdok, D., Laird, A. R., Roski, C., Caspers, S., Zilles, K., & Fox, P. T. (2011). Co-activation patterns distinguish cortical modules, their connectivity and functional differentiation. *NeuroImage*, *57*, 938–949.

Eickhoff, S. B., Laird, A. R., Grefkes, C., Wang, L. E., Zilles, K., & Fox, P. T. (2009). Coordinate-based activation likelihood estimation meta-analysis of neuroimaging data: a

random-effects approach based on empirical estimates of spatial uncertainty. *Human Brain Mapping*, *30*, 2907–2926.
Eickhoff, S. B., Stephan, K. E., Mohlberg, H., Grefkes, C., Fink, G. R., Amunts, K., & Zilles, K. (2005). A new SPM toolbox for combining probabilistic cytoarchitectonic maps and functional imaging data. *NeuroImage*, *25*, 1325–1335.
El-Sheikh, M. (2007). Children's skin conductance level and reactivity: Are these measures stable over time and across tasks? *Developmental Psychobiology*, *49*, 180–186.
Engen, H. G., & Singer, T. (2015). Compassion-based emotion regulation up-regulates experienced positive affect and associated neural networks. *Social Cognitive and Affective Neuroscience*, *10*, 1291–1301.
Engen, H. G., & Singer, T. (2016). Affect and Motivation Are Critical in Constructive Meditation. *Trends in Cognitive Sciences*, *20*, 159–160.
Engen, H. G., Smallwood, J., & Singer, T. (2015). Differential impact of emotional task relevance on three indices of prioritised processing for fearful and angry facial expressions. *Cognition and Emotion*, in press.
Engen, H., & Kanske, P. (2013). How working memory training improves emotion regulation: neural efficiency, effort, and transfer effects. *The Journal of Neuroscience*, *33*, 12152–12153.
Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: a role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron*, *51*, 871–882.
Farb, N. A. S., Segal, Z. V., & Anderson, A. K. (2013). Mindfulness meditation training alters cortical representations of interoceptive attention. *Social Cognitive and Affective Neuroscience*, *8*, 15–26.
Fawver, B., Hass, C. J., Park, K. D., & Janelle, C. M. (2014). Autobiographically recalled emotional states impact forward gait initiation as a function of motivational direction. *Emotion*, *14*, 1125–1136.
Feindler, E. L., Marriott, S. A., & Iwata, M. (1984). Group Anger Control Training for junior high school delinquents. *Cognitive Therapy and Research*, *8*, 299–311.
Fields, H. L., Hjelmstad, G. O., Margolis, E. B., & Nicola, S. M. (2007). Ventral Tegmental Area Neurons in Learned Appetitive Behavior and Positive Reinforcement. *Annual Review of Neuroscience*, *30*, 289–316.
Fischl, B., & Dale, A. M. (2000). Measuring the thickness of the human cerebral cortex from magnetic resonance images. *Proceedings of the National Academy of Sciences*, *97*, 11050–11055.
Fischl, B., Sereno, M. I., & Dale, A. M. (1999). Cortical Surface-Based Analysis. *NeuroImage*, *9*, 195–207.
Fitzgerald, D. A., Posse, S., Moore, G. J., Tancer, M. E., Nathan, P. J., & Phan, K. L. (2004). Neural correlates of internally-generated disgust via autobiographical recall: a functional magnetic resonance imaging investigation. *Neuroscience Letters*, *370*, 91–96.
Folkman, S., & Moskowitz, J. T. (2000). Stress, Positive Emotion, and Coping. *Current Directions in Psychological Science*, *9*, 115–118.
Ford, B. Q., & Tamir, M. (2012). When getting angry is smart: Emotional preferences and emotional intelligence. *Emotion*, *12*, 685–689.
Forman, S. D., Cohen, J. D., Fitzgerald, M., Eddy, W. F., Mintun, M. A., & Noll, D. C. (1995). Improved Assessment of Significant Activation in Functional Magnetic Resonance Imaging (fMRI): Use of a Cluster-Size Threshold. *Magnetic Resonance in Medicine*, *33*, 636–647.
Fox, K. C. R., Nijeboer, S., Dixon, M. L., Floman, J. L., Ellamil, M., Rumak, S. P., et al. (2014). Is meditation associated with altered brain structure? A systematic review and meta-analysis of morphometric neuroimaging in meditation practitioners. *Neuroscience &*

Biobehavioral Reviews, *43*, 48–73.
Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist*.
Fredrickson, B. L. (2013). Learning to self-generate positive emotion. In D. Hermans, B. Rimé, & B. Mesquita, *Changing Emotions* (pp. 151–157). New York, NY: Psychology Press.
Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought-action repertoires. *Cognition and Emotion*, *19*, 313–332.
Fredrickson, B. L., Cohn, M. A., Coffey, K. A., Pek, J., & Finkel, S. M. (2008). Open hearts build lives: Positive emotions, induced through loving-kindness meditation, build consequential personal resources. *Journal of Personality and Social Psychology*, *95*, 1045–1062.
Freeston, M. H., Dugas, M. J., & Ladouceur, R. (1996). Thoughts, images, worry, and anxiety. *Cognitive Therapy and Research*, *20*, 265–273.
Frijda, N. H. (2007). The Laws of Emotion. Cambridge: Cambridge University Press.
Friston, K. (1995). Analysis of fMRI Time-Series Revisited. *NeuroImage*, *2*, 45–53.
Friston, K. J. (2004). Functional and effective connectivity in neuroimaging: A synthesis. *Human Brain Mapping*, *2*, 56–78.
Friston, K. J., Frith, C. D., Liddle, P. F., & Frackowiak, R. S. (1993). Functional connectivity: the principal-component analysis of large (PET) data sets. *Journal of Cerebral Blood Flow & Metabolism*, *13*, 5–14.
Gallese, V., & Caruana, F. (2016). Embodied Simulation: Beyond the Expression/Experience Dualism of Emotions. *Trends in Cognitive Sciences*, *20*, 397–398.
Garland, E. L., Fredrickson, B., Kring, A. M., Johnson, D. P., Meyer, P. S., & Penn, D. L. (2010). Upward spirals of positive emotions counter downward spirals of negativity: Insights from the broaden-and-build theory and affective neuroscience on the treatment of emotion dysfunctions and deficits in psychopathology. *Clinical Psychology Review*, *30*, 849–864.
Garnefski, N., & Kraaij, V. (2006). Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. *Personality and Individual Differences*, *40*, 1659–1669.
Gemar, M. C., Kapur, S., Segal, Z. V., Brown, G. M., & Houle, S. (1996). Effects of self-generated sad mood on regional cerebral activity: a PET study in normal subjects. *Depression*, *4*, 81–88.
George, M. S., Ketter, T. A., Parekh, P. I., & Herscovitch, P. (1996). Gender differences in regional cerebral blood flow during transient self-induced sadness or happiness. *Biological Psychiatry*, *40*, 859–871.
Gerlach, K. D., Spreng, R. N., Madore, K. P., & Schacter, D. L. (2014). Future planning: default network activity couples with frontoparietal control network and reward-processing regions during process and outcome simulations. *Social Cognitive and Affective Neuroscience*, *9*, 1942–1951.
Ghashghaei, H. T., & Barbas, H. (2002). Pathways for emotion: interactions of prefrontal and anterior temporal pathways in the amygdala of the rhesus monkey. *Neuroscience*, *115*, 1261–1279.
Gilbert, C. D., & Sigman, M. (2007). Brain States: Top-Down Influences in Sensory Processing. *Neuron*, *54*, 677–696.
Gilbert, P., McEwan, K., Mitra, R., Franks, L., Richter, A., & Rockliff, H. (2008). Feeling safe and content: A specific affect regulation system? Relationship to depression, anxiety, stress, and self-criticism. *The Journal of Positive Psychology*, *3*, 182–191.
Goetz, J. L., Keltner, D., & Simon-Thomas, E. (2010). Compassion: An evolutionary analysis and empirical review. *Psychological Bulletin*, *136*, 351–374.

Goldin, P. R., Hutcherson, C. A. C., Ochsner, K. N., Glover, G. H., Gabrieli, J. D. E., & Gross, J. J. (2005). The neural bases of amusement and sadness: a comparison of block contrast and subject-specific emotion intensity regression approaches. *NeuroImage*, *27*, 26–36.

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The Neural Bases of Emotion Regulation: Reappraisal and Suppression of Negative Emotion. *Biological Psychiatry*, *63*, 577–586.

Gollnisch, G., & Averill, J. R. (1993). Emotional imagery: Strategies and correlates. *Cognition and Emotion*, *7*, 407–429.

Gorgolewski, K. J., Lurie, D., Urchs, S., Kipping, J. A., Craddock, R. C., Milham, M. P., et al. (2014). A Correspondence between Individual Differences in the Brain's Intrinsic Functional Architecture and the Content and Form of Self-Generated Thoughts. *PloS One*, *9*, e97176.

Graver, M. (2009). Stoicism and Emotion. Chicago: University of Chicago Press.

Greve, D. N., & Fischl, B. (2009). Accurate and robust brain image alignment using boundary-based registration. *NeuroImage*, *48*, 63–72.

Gross, D. M. (2007a). The Secret History of Emotion. Chicago: University of Chicago Press.

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, *74*, 224–237.

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, *2*, 271–299.

Gross, J. J. (2007b). Emotion regulation: Conceptual foundations. In J. J. Gross, *Handbook of emotion regulation*. New York: Guilford Press.

Gross, J. J. (2015a). Emotion Regulation: Current Status and Future Prospects. *Psychological Inquiry*, *26*, 1–26.

Gross, J. J. (2015b). The Extended Process Model of Emotion Regulation: Elaborations, Applications, and Future Directions. *Psychological Inquiry*, *26*, 130–137.

Gross, J. J., & Barrett, L. F. (2011). Emotion Generation and Emotion Regulation: One or Two Depends on Your Point of View. *Emotion Review*, *3*, 8–16.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011). Emotion generation and emotion regulation: A distinction we should make (carefully). *Cognition and Emotion*, *25*, 765–781.

Gundersen, L. (2001). Physician Burnout. *Annals of Internal Medicine*, *135*, 145–148.

Gyurak, A., Goodkind, M. S., Kramer, J. H., Miller, B. L., & Levenson, R. W. (2012). Executive functions and the down-regulation and up-regulation of emotion. *Cognition and Emotion*, *26*, 103–118.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion*, *25*, 400–412.

Hajcak, G., & Olvet, D. M. (2008). The persistence of attention to emotion: Brain potentials during and after picture presentation. *Emotion*, *8*, 250–255.

Hamann, S. (2004). Individual differences in emotion processing. *Current Opinion in Neurobiology*, *14*, 233–238.

Han, X., Jovicich, J., Salat, D., van der Kouwe, A., Quinn, B., Czanner, S., et al. (2006). Reliability of MRI-derived measurements of human cerebral cortical thickness: The effects of field strength, scanner upgrade and manufacturer. *NeuroImage*, *32*, 180–194.

Handwerker, D. A., Ollinger, J. M., & D'Esposito, M. (2004). Variation of BOLD hemodynamic responses across subjects and brain regions and their effects on statistical analyses. *NeuroImage*, *21*, 1639–1651.

Harrison, B. J., Pujol, J., Ortiz, H., Fornito, A., Pantelis, C., & Yücel, M. (2008). Modulation of brain resting-state networks by sad mood induction. *PloS One*, *3*, e1794.

Hassabis, D., & Maguire, E. A. (2007). Deconstructing episodic memory with construction.

Trends in Cognitive Sciences, *11*, 299–306.
Hassabis, D., & Maguire, E. A. (2009). The construction system of the brain. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *364*, 1263–1271.
Hautzinger, M., Keller, F., Kühner, C., & Beck, A. T. (2009). Beck Depressions-Inventar-II (BDI II) Manual. Berlin: Pearson Assessment.
Heinzel, A., Bermpohl, F., Niese, R., Pfennig, A., Pascual-Leone, A., Schlaug, G., & Northoff, G. (2005). How do we modulate our emotions? Parametric fMRI reveals cortical midline structures as regions specifically involved in the processing of emotional valences. *Cognitive Brain Research*, *25*, 348–358.
Holmes, E. A., & Mathews, A. (2005). Mental Imagery and Emotion: A Special Relationship? *Emotion*, *5*, 489–497.
Holmes, E. A., & Mathews, A. (2010). Mental imagery in emotion and emotional disorders. *Clinical Psychology Review*, *30*, 349–362.
Holmes, E. A., & Pictet, A. (2013). The powerful impact of mental imagery in changing emotion. In *Changing Emotions*. New York, NY: Psychology Press.
Holmes, E. A., Blackwell, S. E., Burnett Heyes, S., Renner, F., & Raes, F. (2016). Mental Imagery in Depression: Phenomenology, Potential Mechanisms, and Treatment Implications. *Annual Review of Clinical Psychology*, *12*, 249–280.
Holmes, E. A., Lang, T. J., & Shah, D. M. (2009). Developing interpretation bias modification as a "cognitive vaccine" for depressed mood: Imagining positive events makes you feel better than thinking about them verbally. *Journal of Abnormal Psychology*, *118*, 76–88.
Holmes, E. A., Mathews, A., Dalgleish, T., & Mackintosh, B. (2006). Positive interpretation training: effects of mental imagery versus verbal training on positive mood. *Behavior Therapy*, *37*, 237–247.
Hutton, C., Draganski, B., Ashburner, J., & Weiskopf, N. (2009). A comparison between voxel-based cortical thickness and voxel-based morphometry in normal aging. *NeuroImage*, *48*, 371–380.
Ito, R., Ito, R., Robbins, T. W., Robbins, T. W., Pennartz, C. M., Pennartz, C. M., et al. (2008). Functional interaction between the hippocampus and nucleus accumbens shell is necessary for the acquisition of appetitive spatial context conditioning. *The Journal of Neuroscience*, *28*, 6950–6959.
Jabbi, M., Bastiaansen, J., & Keysers, C. (2008). A common anterior insula representation of disgust observation, experience and imagination shows divergent functional connectivity pathways. *PloS One*, *3*, e2939.
Jao, T., Vértes, P. E., Alexander-Bloch, A. F., Tang, I.-N., Yu, Y.-C., Chen, J.-H., & Bullmore, E. T. (2013). Volitional eyes opening perturbs brain dynamics and functional connectivity regardless of light input. *NeuroImage*, *69*, 21–34.
Jazaieri, H., Jinpa, G. T., McGonigal, K., Rosenberg, E. L., Finkelstein, J., Simon-Thomas, E., et al. (2012). Enhancing Compassion: A Randomized Controlled Trial of a Compassion Cultivation Training Program. *Journal of Happiness Studies*, *14*, 1113–1126.
Jensen, J., Smith, A. J., Willeit, M., Crawley, A. P., Mikulis, D. J., Vitcu, I., & Kapur, S. (2007). Separate brain regions code for salience vs. valence during reward prediction in humans. *Human Brain Mapping*, *28*, 294–302.
Kahneman, D., & Tversky, A. (1982). The Simulation Heuristic. In *Judgment Under Uncertainty*. Cambridge University Press.
Kanske, P., Heissler, J., Schönfelder, S., Bongers, A., & Wessa, M. (2011). How to regulate emotion? Neural networks for reappraisal and distraction. *Cerebral Cortex*, *21*, 1379–1388.
Kappes, A., Singmann, H., & Oettingen, G. (2012). Mental contrasting instigates goal pursuit by linking obstacles of reality with instrumental behavior. *Journal of Experimental Social*

Psychology, *48*, 811–818.
Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review*, *30*, 865–878.
Kashdan, T. B., Barrett, L. F., & McKnight, P. E. (2015). Unpacking Emotion Differentiation Transforming Unpleasant Experience by Perceiving Distinctions in Negativity. *Current Directions in Psychological Science*, *24*, 10–16.
Keller, J., Young, C. B., Kelley, E., Prater, K., Levitin, D. J., & Menon, V. (2013). Trait anhedonia is associated with reduced reactivity and connectivity of mesolimbic and paralimbic reward pathways. *Journal of Psychiatric Research*, *47*, 1319–1328.
Killingsworth, M. A., & Gilbert, D. T. (2010). A wandering mind is an unhappy mind. *Science*, *330*, 932–932.
Kim, M. Y., Ford, B. Q., Mauss, I., & Tamir, M. (2015). Knowing when to seek anger: Psychological health and context-sensitive emotional preferences. *Cognition and Emotion*, *29*, 1126–1136.
Kim, S. (2015). ppcor: An R Package for a Fast Calculation to Semi-partial Correlation Coefficients. *Communications for Statistical Applications and Methods*, *22*, 665–674.
Kimbrell, T. A., George, M. S., Parekh, P. I., Ketter, T. A., Podell, D. M., Danielson, A. L., et al. (1999). Regional brain activity during transient self-induced anxiety and anger in healthy adults. *Biological Psychiatry*, *46*, 454–465.
Kleinke, C. L., Peterson, T. R., & Rutledge, T. R. (1998). Effects of self-generated facial expressions on mood. *Journal of Personality and Social Psychology*, *74*, 272–279.
Klimecki, O. M., Leiberg, S., Lamm, C., & Singer, T. (2013). Functional neural plasticity and associated changes in positive affect after compassion training. *Cerebral Cortex*, *23*, 1552–1561.
Klimecki, O. M., Leiberg, S., Ricard, M., & Singer, T. (2014). Differential pattern of functional brain plasticity after compassion and empathy training. *Social Cognitive and Affective Neuroscience*, *9*, 873–879.
Knoll, N., Rieckmann, N., & Schwarzer, R. (2005). Coping as a mediator between personality and stress outcomes: a longitudinal study with cataract surgery patients. *European Journal of Personality*, *19*, 229–247.
Kober, H., Barrett, L. F., Joseph, J., Bliss-Moreau, E., Lindquist, K. A., & Wager, T. D. (2008). Functional grouping and cortical–subcortical interactions in emotion: A meta-analysis of neuroimaging studies. *NeuroImage*, *42*, 998–1031.
Koelsch, S., Skouras, S., Fritz, T., Herrera, P., Bonhage, C., Küssner, M. B., & Jacobs, A. M. (2013). The roles of superficial amygdala and auditory cortex in music-evoked fear and joy. *NeuroImage*, *81*, 49–60.
Kok, B. E., Coffey, K. A., Cohn, M. A., Catalino, L. I., Vacharkulksemsuk, T., Algoe, S. B., et al. (2013). How positive emotions build physical health: perceived positive social connections account for the upward spiral between positive emotions and vagal tone. *Psychological Science*, *24*, 1123–1132.
Kolb, B., Gibb, R., & Robinson, T. E. (2003). Brain plasticity and behavior. *Current Directions in Psychological Science*, *12*, 1–5.
Kortte, K. B., Horner, M. D., & Windham, W. K. (2002). The Trail Making Test, Part B: Cognitive Flexibility or Ability to Maintain Set? *Applied Neuropsychology*, *9*, 106–109.
Koster, E. H. W., De Lissnyder, E., Derakshan, N., & De Raedt, R. (2011). Understanding depressive rumination from a cognitive science perspective: The impaired disengagement hypothesis. *Clinical Psychology Review*, *31*, 138–145.
Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology*, *84*, 394–421.
Kringelbach, M. L., & Berridge, K. C. (2009). Towards a functional neuroanatomy of pleasure and happiness. *Trends in Cognitive Sciences*, *13*, 479–487.

Krohne, H. W., Egloff, B., & Kohlmann, C. W. (1996). Untersuchungen mit einer deutschen Version der“ Positive and Negative Affect Schedule”(PANAS). *Diagnostica*, 139–156.

Kross, E., & Ayduk, O. (2008). Facilitating Adaptive Emotional Analysis: Distinguishing Distanced-Analysis of Depressive Experiences From Immersed-Analysis and Distraction. *Personality and Social Psychology Bulletin*, *34*, 924–938.

Kross, E., Ayduk, O., & Mischel, W. (2005). When asking “why” does not hurt. Distinguishing rumination from reflective processing of negative emotions. *Psychological Science*, *16*, 709–715.

Kross, E., Davidson, M., Weber, J., & Ochsner, K. (2009). Coping with emotions past: the neural bases of regulating affect associated with negative autobiographical memories. *Biological Psychiatry*, *65*, 361–366.

Kuperberg, G. R., Broome, M. R., McGuire, P. K., David, A. S., Eddy, M., Ozawa, F., et al. (2003). Regionally Localized Thinning of the Cerebral Cortex in Schizophrenia. *Archives of General Psychiatry*, *60*, 878–888.

Kupfer, J., Brosig, B., & Brähler, E. (2000). [Testing and validation of the 26-Item Toronto Alexithymia Scale in a representative population sample]. *Zeitschrift Fur Psychosomatische Medizin Und Psychotherapie*, *46*, 368–384.

Kühn, S., Fernyhough, C., Alderson-Day, B., & Hurlburt, R. T. (2014a). Inner experience in the scanner: can high fidelity apprehensions of inner experience be integrated with fMRI? *Frontiers in Psychology*, *5*, 1393.

Kühn, S., Vanderhasselt, M.-A., De Raedt, R., & Gallinat, J. (2012). Why ruminators won't stop: The structural and resting state correlates of rumination and its relation to depression. *Journal of Affective Disorders*, *141*, 352–360.

Kühn, S., Vanderhasselt, M.-A., De Raedt, R., & Gallinat, J. (2014b). The neural basis of unwanted thoughts during resting state. *Social Cognitive and Affective Neuroscience*, *9*, 1320–1324.

Laird, A. R., Fox, P. M., Eickhoff, S. B., Turner, J. A., Ray, K. L., McKay, D. R., et al. (2011). Behavioral interpretations of intrinsic connectivity networks. *Journal of Cognitive Neuroscience*, *23*, 4022–4037.

LaLumiere, R. T., LaLumiere, R. T., Nawar, E. M., & McGaugh, J. L. (2005). Modulation of memory consolidation by the basolateral amygdala or nucleus accumbens shell requires concurrent dopamine receptor activation in both brain regions. *Learning & Memory*, *12*, 296–301.

Lama, D., & Paul Ekman, P. D. (2008). Emotional Awareness. New York: Macmillan.

Lamm, C., & Decety, J. (2008). Is the Extrastriate Body Area (EBA) Sensitive to the Perception of Pain in Others? *Cerebral Cortex*, *18*, 2369–2373.

Lamm, C., Decety, J., & Singer, T. (2011). Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *NeuroImage*, *54*, 2492–2502.

Lang, T. J., Blackwell, S. E., Harmer, C. J., Davison, P., & Holmes, E. A. (2011). Cognitive Bias Modification Using Mental Imagery for Depression: Developing a Novel Computerized Intervention to Change Negative Thinking Styles. *European Journal of Personality*, *26*, 145–157.

Larsen, J. T., McGraw, A. P., & Cacioppo, J. T. (2001). Can people feel happy and sad at the same time? *Journal of Personality and Social Psychology*, *81*, 684–696.

Larsen, R. J. (2000). Toward a Science of Mood Regulation. *Psychological Inquiry*, *11*, 129–141.

Laux, L., Glanzmann, P. S., Schaffner, P., & Spielberger, C. D. (1981). Das State-Trait-Angstinventar [The State-Trait Anxiety Inventory]. Beltz: Weinheim.

Lavigne, K. M., Metzak, P. D., & Woodward, T. S. (2015). Functional brain networks underlying detection and integration of disconfirmatory evidence. *NeuroImage*, *112*, 138–

151.
Lazar, S. W., Kerr, C. E., Wasserman, R. H., Gray, J. R., Greve, D. N., Treadway, M. T., et al. (2005). Meditation experience is associated with increased cortical thickness. *Neuroreport*, *16*, 1893.
Lazarus, R. S., & Folkman, S. (1984). Stress, Appraisal, and Coping. New York, NY: Springer.
Ledoux, J. (1998). The Emotional Brain (1st ed.). New York: Simon and Schuster.
Leech, R., & Sharp, D. J. (2014). The role of the posterior cingulate cortex in cognition and disease. *Brain*, *137*, 12–32.
Leiberg, S., Klimecki, O., & Singer, T. (2011). Short-term compassion training increases prosocial behavior in a newly developed prosocial game. *PloS One*, *6*, e17798.
Lerch, J. P. (2004). Focal Decline of Cortical Thickness in Alzheimer's Disease Identified by Computational Neuroanatomy. *Cerebral Cortex*, *15*, 995–1001.
Levenson, R. W. (2014). The Autonomic Nervous System and Emotion. *Emotion Review*, *6*, 100–112.
Lieblich, S. M., Castle, D. J., Pantelis, C., Hopwood, M., Young, A. H., & Everall, I. P. (2015). High heterogeneity and low reliability in the diagnosis of major depression will impair the development of new drugs. *British Journal of …*, *1*, e5–e7.
Lindquist, K. A., & Barrett, L. F. (2008). Constructing Emotion The Experience of Fear as a Conceptual Act. *Psychological Science*, *19*, 898–903.
Lindquist, K. A., & Barrett, L. F. (2012). A functional architecture of the human brain: emerging insights from the science of emotion. *Trends in Cognitive Sciences*, *16*, 533–540.
Lindquist, K. A., Satpute, A. B., Wager, T. D., Weber, J., & Barrett, L. F. (2016). The Brain Basis of Positive and Negative Affect: Evidence from a Meta-Analysis of the Human Neuroimaging Literature. *Cerebral Cortex*, *26*, 1910–1922.
Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences*, *35*, 121–143.
Liotti, M., Mayberg, H. S., Brannan, S. K., McGinnis, S., Jerabek, P., & Fox, P. T. (2000). Differential limbic--cortical correlates of sadness and anxiety in healthy subjects: implications for affective disorders. *Biological Psychiatry*, *48*, 30–42.
Liu, Y., Huang, H., McGinnis-Deweese, M., Keil, A., & Ding, M. (2012). Neural substrate of the late positive potential in emotional processing. *The Journal of Neuroscience*, *32*, 14563–14572.
Loch, N., Hiller, W., & Witthöft, M. (2011). Der Cognitive Emotion Regulation Questionnaire (CERQ). *Zeitschrift Für Klinische Psychologie Und Psychotherapie*, *40*, 94–106.
Loftus, G. R., & Masson, M. E. J. (1994). Using confidence intervals in within-subject designs. *Psychonomic Bulletin & Review*, *1*, 476–490.
Logothetis, N. K., Pauls, J., Augath, M., Trinath, T., & Oeltermann, A. (2001). Neurophysiological investigation of the basis of the fMRI signal. *Nature*, *412*, 150–157.
Luders, E., Kurth, F., Mayer, E. A., Toga, A. W., Narr, K. L., & Gaser, C. (2012). The Unique Brain Anatomy of Meditation Practitioners: Alterations in Cortical Gyrification. *Frontiers in Human Neuroscience*, *6*, 34.
Lutz, A., Brefczynski-Lewis, J., Johnstone, T., & Davidson, R. J. (2008). Regulation of the Neural Circuitry of Emotion by Compassion Meditation: Effects of Meditative Expertise. *PloS One*, *3*, e1897.
Lutz, A., Greischar, L. L., Perlman, D. M., & Davidson, R. J. (2009). BOLD signal in insula is differentially related to cardiac function during compassion meditation in experts vs. novices. *NeuroImage*, *47*, 1038–1046.

Macey, P. M., Macey, K. E., Kumar, R., & Harper, R. M. (2004). A method for removal of global effects from fMRI time series. *NeuroImage*, *22*, 360–366.

Magen, E., & Gross, J. J. (2010). Getting our act together: Toward a general model of self-control. In R. Hassin, K. Ochsner, & Y. Trope, *Self control in society, mind, and brain* (pp. 335–353). Oxford: Oxford University Press.

Manzano García, G., & Ayala Calvo, J. C. (2012). Emotional exhaustion of nursing staff: influence of emotional annoyance and resilience. *International Nursing Review*, *59*, 101–107.

Martinelli, P., Sperduti, M., & Piolino, P. (2013). Neural substrates of the self-memory system: New insights from a meta-analysis. *Human Brain Mapping*, *34*, 1515–1529.

Mascaro, J. S., Rilling, J. K., Negi, L. T., & Raison, C. L. (2013). Compassion meditation enhances empathic accuracy and related neural activity. *Social Cognitive and Affective Neuroscience*, *8*, 48–55.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The Tie That Binds? Coherence Among Emotion Experience, Behavior, and Physiology. *Emotion*, *5*, 175–190.

Mayberg, H. S., Liotti, M., Brannan, S. K., McGinnis, S., Mahurin, R. K., Jerabek, P. A., et al. (1999). Reciprocal limbic-cortical function and negative mood: converging PET findings in depression and normal sadness. *The American Journal of Psychiatry*, *156*, 675–682.

Mayberg, H. S., Lozano, A. M., Voon, V., McNeely, H. E., Seminowicz, D., Hamani, C., et al. (2005). Deep Brain Stimulation for Treatment-Resistant Depression. *Neuron*, *45*, 651–660.

Mazaika, P. K., Whitfield, S., & Cooper, J. C. (2005). Detection and repair of transient artifacts in fMRI data. *NeuroImage*, *27*, 624–634.

McCrae, R. R., & Costa, P. T. (1992). Discriminant Validity of NEO-PIR Facet Scales. *Educational and Psychological Measurement*, *52*, 229–237.

McLaughlin, K. A., Mennin, D. S., & Farach, F. J. (2007). The contributory role of worry in emotion generation and dysregulation in generalized anxiety disorder. *Behaviour Research and Therapy*, *45*, 1735–1752.

McRae, K., Ciesielski, B., & Gross, J. J. (2012a). Unpacking cognitive reappraisal: Goals, tactics, and outcomes. *Emotion*, *12*, 250–255.

McRae, K., Hughes, B., Chopra, S., Gabrieli, J. D. E., Gross, J. J., & Ochsner, K. N. (2010). The Neural Bases of Distraction and Reappraisal. *Journal of Cognitive Neuroscience*, *22*, 248–262.

McRae, K., Jacobs, S. E., Ray, R. D., John, O. P., & Gross, J. J. (2012b). Individual differences in reappraisal ability: Links to reappraisal frequency, well-being, and cognitive control. *Journal of Research in Personality*, *46*, 2–7.

McRae, K., Misra, S., Prasad, A. K., Pereira, S. C., & Gross, J. J. (2012c). Bottom-up and top-down emotion generation: implications for emotion regulation. *Social Cognitive and Affective Neuroscience*, *7*, 253–262.

Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: a network model of insula function. *Brain Structure and Function*, *214*, 655–667.

Milazzo, A.-C., Ng, B., Jiang, H., Shirer, W., Varoquaux, G., Poline, J.-B., et al. (2016). Identification of Mood-Relevant Brain Connections Using a Continuous, Subject-Driven Rumination Paradigm. *Cerebral Cortex*, *26*, 933–942.

Miller, G. A., Levin, D. N., Kozak, M. J., Cook, E. W., III, McLean, A., Jr, & Lang, P. J. (1987). Individual differences in imagery and the psychophysiology of emotion. *Cognition and Emotion*, *1*, 367–390.

Millgram, Y., Joormann, J., Huppert, J. D., & Tamir, M. (2015). Sad as a Matter of Choice? Emotion-Regulation Goals in Depression. *Psychological Science*, *26*, 1216–1228.

Min, J.-A., Yu, J. J., Lee, C.-U., & Chae, J.-H. (2013). Cognitive emotion regulation strategies contributing to resilience in patients with depression and/or anxiety disorders. *Comprehensive Psychiatry*, *54*, 1190–1197.

Mitchell, J. P. (2009). Inferences about mental states. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *364*, 1309–1316.

Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Weiskopf, N., Seymour, B., et al. (2007). When Fear Is Near: Threat Imminence Elicits Prefrontal-Periaqueductal Gray Shifts in Humans. *Science*, *317*, 1079–1083.

Morina, N., Deeprose, C., Pusowski, C., Schmid, M., & Holmes, E. A. (2011). Prospective mental imagery in patients with major depressive disorder or anxiety disorders. *Journal of Anxiety Disorders*, *25*, 1032–1037.

Mrazek, M. D., Franklin, M. S., Phillips, D. T., Baird, B., & Schooler, J. W. (2013). Mindfulness Training Improves Working Memory Capacity and GRE Performance While Reducing Mind Wandering. *Psychological Science*, *24*, 776–781.

Murphy, S. E., Clare O'Donoghue, M., Drazich, E. H. S., Blackwell, S. E., Christina Nobre, A., & Holmes, E. A. (2015). Imagining a brighter future: The effect of positive imagery training on mood, prospective mental imagery and emotional bias in older adults. *Psychiatry Research*, *230*, 36–43.

Murray, E. A. (2007). The amygdala, reward and emotion. *Trends in Cognitive Sciences*, *11*, 489–497.

Naidich, T. P., Duvernoy, H. M., Delman, B. N., Sorensen, A. G., Kollias, S. S., & Haacke, E. M. (2009). Duvernoy's Atlas of the Human Brain Stem and Cerebellum. Wien: Springer.

Nelson, S. M., Dosenbach, N. U. F., Cohen, A. L., Wheeler, M. E., Schlaggar, B. L., & Petersen, S. E. (2010). Role of the anterior insula in task-level control and focal attention. *Brain Structure and Function*, *214*, 669–680.

Nichols, T., Brett, M., Andersson, J., Wager, T., & Poline, J.-B. (2006). Optimal EPI parameters for reduction of susceptibility-induced BOLD sensitivity losses: A whole-brain analysis at 3 T and 1.5 T. *NeuroImage*, *33*, 493–504.

Niedenthal, P. M., Winkielman, P., Mondillon, L., & Vermeulen, N. (2009). Embodiment of emotion concepts. *Journal of Personality and Social Psychology*, *96*, 1120–1136.

Nili, U., Goldberg, H., Weizman, A., & Dudai, Y. (2010). Fear Thou Not: Activity of Frontal and Temporal Circuits in Moments of Real-Life Courage. *Neuron*, *66*, 949–962.

Nitschke, J. B., & Heller, W. (2005). Distinguishing neural substrates of heterogeneity among anxiety disorders. *International Review of Neurobiology*, *67*, 1–42.

Niven, K., Totterdell, P., Stride, C. B., & Holman, D. (2011). Emotion Regulation of Others and Self (EROS): The Development and Validation of a New Individual Difference Measure. *Current Psychology*, *30*, 53–73.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking Rumination. *Perspectives on Psychological Science*, *3*, 400–424.

Noonan, K. A., Jefferies, E., Visser, M., & Lambon Ralph, M. A. (2013). Going beyond inferior prefrontal involvement in semantic control: evidence for the additional contribution of dorsal angular gyrus and posterior middle temporal cortex. *Journal of Cognitive Neuroscience*, *25*, 1824–1850.

Nussbaum, M. C. (2008). Upheavals of Thought. Cambridge, UK: Cambridge University Press.

Ochsner, K. N., & Gross, J. J. (2008). Cognitive Emotion Regulation Insights From Social Cognitive and Affective Neuroscience. *Current Directions in Psychological Science*, *17*, 153–158.

Ochsner, K. N., Hughes, B., Robertson, E. R., Cooper, J. C., & Gabrieli, J. D. E. (2009a). Neural Systems Supporting the Control of Affective and Cognitive Conflicts. *Journal of Cognitive Neuroscience*, *21*, 1841–1854.

Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., et al. (2009b). Bottom-up and top-down processes in emotion generation: common and distinct neural mechanisms. *Psychological Science*, *20*, 1322–1331.

Ochsner, K. N., Silvers, J. A., & Buhle, J. T. (2012). Functional imaging studies of emotion regulation: a synthetic review and evolving model of the cognitive control of emotion. *Annals of the New York Academy of Sciences*, *1251*, E1–24.

Oettingen, G., & Mayer, D. (2002). The motivating function of thinking about the future: Expectations versus fantasies. *Journal of Personality and Social Psychology*, *83*, 1198–1212.

Oettingen, G., Mayer, D., & Portnow, S. (2016). Pleasure Now, Pain Later: Positive Fantasies About the Future Predict Symptoms of Depression. *Psychological Science*, *27*, 345–353.

Oettingen, G., Mayer, D., Sevincer, A. T., Stephens, E. J., Pak, H.-J., & Hagenah, M. (2009). Mental Contrasting and Goal Commitment: The Mediating Role of Energization. *Personality and Social Psychology Bulletin*, *35*, 608–622.

Ohman, A. (2002). Automaticity and the Amygdala: Nonconscious Responses to Emotional Faces. *Current Directions in Psychological Science*, *11*, 62–66.

Ohman, A., Carlsson, K., Lundqvist, D., & Ingvar, M. (2007). On the unconscious subcortical origin of human fear. *Physiology & Behavior*, *92*, 180–185.

Ohman, A., Soares, S. C., Juth, P., Lindström, B., & Esteves, F. (2012). Evolutionary derived modulations of attention to two common fear stimuli: Serpents and hostile humans. *Journal of Cognitive Psychology*, *24*, 17–32.

Ollinger, J. M., Shulman, G. L., & Corbetta, M. (2001). Separating Processes within a Trial in Event-Related Functional MRI. *NeuroImage*, *13*, 210–217.

Oosterwijk, S., Lindquist, K. A., Anderson, E., Dautoff, R., Moriguchi, Y., & Barrett, L. F. (2012). States of mind: Emotions, body feelings, and thoughts share distributed neural networks. *NeuroImage*, *62*, 2110–2128.

Otto, B., Misra, S., Prasad, A., & McRae, K. (2014). Functional overlap of top-down emotion regulation and generation: An fMRI study identifying common neural substrates between cognitive reappraisal and cognitively generated emotions. *Cognitive, Affective & Behavioral Neuroscience*, *14*, 923–938.

Panksepp, J. (2004). Affective Neuroscience: The Foundations of Human and Animal Emotions. Oxford: Oxford University Press.

Papez, J. W. (1937). A proposed mechanism of emotion. *Archives of Neurology and Psychiatry*, *38*, 725–743.

Pardo, J. V., & Raichle, M. E. (1993). Neural correlates of self-induced dysphoria. *The American Journal of Psychiatry*, *150*, 713–719.

Pessoa, L., & Adolphs, R. (2010). Emotion processing and the amygdala: from a 'low road' to “many roads” of evaluating biological significance. *Nature Reviews Neuroscience*, *11*, 773–783.

Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional Neuroanatomy of Emotion: A Meta-Analysis of Emotion Activation Studies in PET and fMRI. *NeuroImage*, *16*, 331–348.

Philippe, F. L., Lecours, S., & Beaulieu-Pelletier, G. (2009). Resilience and positive emotions: examining the role of emotional memories. *Journal of Personality*, *77*, 139–175.

Philippot, P., Chapelle, G., & Blairy, S. (2002). Respiratory feedback in the generation of emotion. *Cognition and Emotion*, *16*, 605–627.

Phillips, M. L., Ladouceur, C. D., & Drevets, W. C. (2008). A neural model of voluntary and automatic emotion regulation: implications for understanding the pathophysiology and neurodevelopment of bipolar disorder. *Molecular Psychiatry*, *13*, 833–857.

Pictet, A., Coughtrey, A. E., Mathews, A., & Holmes, E. A. (2011). Fishing for happiness:

The effects of generating positive imagery on mood and behaviour. *Behaviour Research and Therapy*, *49*, 885–891.

Plamper, J. (2015). The History of Emotions. Oxford: Oxford University Press.

Poerio, G. L., Totterdell, P., Emerson, L.-M., & Miles, E. (2015a). Helping the heart grow fonder during absence: Daydreaming about significant others replenishes connectedness after induced loneliness. *Cognition and Emotion*, 1–11.

Poerio, G. L., Totterdell, P., Emerson, L.-M., & Miles, E. (2015b). Love is the triumph of the imagination: Daydreams about significant others are associated with increased happiness, love and connection. *Consciousness and Cognition*, *33*, 135–144.

Poldrack, R. A. (2000). Imaging Brain Plasticity: Conceptual and Methodological Issues— A Theoretical Review. *NeuroImage*, *12*, 1–13.

Power, J. D., Barnes, K. A., Snyder, A. Z., Schlaggar, B. L., & Petersen, S. E. (2012). Spurious but systematic correlations in functional connectivity MRI networks arise from subject motion. *NeuroImage*, *59*, 2142–2154.

Preuschoff, K., Preuschoff, K., Quartz, S. R., Quartz, S. R., Bossaerts, P., & Bossaerts, P. (2008). Human insula activation reflects risk prediction errors as well as risk. *The Journal of Neuroscience*, *28*, 2745–2752.

Raichle, M. E., & Snyder, A. Z. (2007). A default mode of brain function: a brief history of an evolving idea. *NeuroImage*, *37*, 1083–1090.

Rangel, A., & Hare, T. (2010). Neural computations associated with goal-directed choice. *Current Opinion in Neurobiology*, *20*, 262–270.

Rathschlag, M., & Memmert, D. (2014). Self-Generated Emotions and Their Influence on Sprint Performance: An Investigation of Happiness and Anxiety. *Journal of Applied Sport Psychology*, *27*, 186–199.

Raune, D., MacLeod, A., & Holmes, E. A. (2005). The simulation heuristic and visual imagery in pessimism for future negative events in anxiety. *Clinical Psychology & Psychotherapy*, *12*, 313–325.

Raven, J. (2000). The Raven's progressive matrices: change and stability over culture and time. *Cognitive Psychology*, *41*, 1–48.

Ray, K. L., McKay, D. R., Fox, P. M., Riedel, M. C., Uecker, A. M., Beckmann, C. F., et al. (2013). ICA model order selection of task co-activation networks. *Frontiers in Neuroscience*, *7*, 237.

Reiman, E. M., Lane, R. D., Ahern, G. L., Schwartz, G. E., Davidson, R. J., Friston, K. J., et al. (1997). Neuroanatomical correlates of externally and internally generated human emotion. *The American Journal of Psychiatry*, *154*, 918–925.

Reitan, R. M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual and Motor Skills*, *8*, 271.

Ricard, M. (2006). Happiness: A guide to developing life's most important skill. New York: Little, Brown.

Ridgway, G. R., Omar, R., Ourselin, S., Hill, D. L. G., Warren, J. D., & Fox, N. C. (2009). Issues with threshold masking in voxel-based morphometry of atrophied brains. *NeuroImage*, *44*, 99–111.

Ripke, S., Wray, N. R., Lewis, C. M., Hamilton, S. P., Weissman, M. M., Breen, G., et al. (2012). A mega-analysis of genome-wide association studies for major depressive disorder. *Molecular Psychiatry*, *18*, 497–511.

Riquelme, C. E. S., Radovic, D., Castro, O., & Turnbull, O. H. (2015). Internally and externally generated emotions in people with acquired brain injury: preservation of emotional experience after right hemisphere lesions. *Frontiers in Psychology*, *6*, 426.

Roozendaal, B., Roozendaal, B., McReynolds, J. R., McReynolds, J. R., Van der Zee, E. A., Van der Zee, E. A., et al. (2009). Glucocorticoid effects on memory consolidation depend on functional interactions between the medial prefrontal cortex and basolateral amygdala.

The Journal of Neuroscience, *29*, 14299–14308.
Rosas, H. D., Liu, A. K., Hersch, S., Glessner, M., Ferrante, R. J., Salat, D. H., et al. (2002). Regional and progressive thinning of the cortical ribbon in Huntington's disease. *Neurology*, *58*, 695–701.
Roy, M., Shohamy, D., & Wager, T. D. (2012). Ventromedial prefrontal-subcortical systems and the generation of affective meaning. *Trends in Cognitive Sciences*, *16*, 147–156.
Roy, M., Shohamy, D., Daw, N., Jepma, M., Wimmer, G. E., & Wager, T. D. (2014). Representation of aversive prediction errors in the human periaqueductal gray. *Nature Neuroscience*, *17*, 1607–1612.
Ruby, F. J. M., Smallwood, J., Engen, H., & Singer, T. (2013). How self-generated thought shapes mood--the relation between mind-wandering and mood depends on the socio-temporal content of thoughts. *PloS One*, *8*, e77554.
Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, *110*, 145–172.
Russell, J. A. (2009). Emotion, core affect, and psychological construction. *Cognition and Emotion*, *23*, 1259–1283.
Russell, J. A. (2014). My Psychological Constructionist Perspective, with a Focus on Conscious Affective Experience. In L. F. Barrett & J. A. Russell, *The Psychological Construction of Emotion* (pp. 183–208). Guilford Publications.
Salas, C. E., Radovic, D., & Turnbull, O. H. (2012). Inside-out: Comparing internally generated and externally generated basic emotions. *Emotion*, *12*, 568–578.
Salzberg, S. (2001). Loving-kindness - the revolutionary art of happiness. Boston: Shambhala.
Sanchez-Cubillo, I., Perianez, J. A., Adrover-Roig, D., Rodriguesz-Sanchez, J. M., Rios-Lago, M., Tirapu, J., & Barcelo, F. (2009). Construct validity of the Trail Making Test: role of task-switching, working memory, inhibition/interference control, and visuomotor abilities. *Journal of the International Neuropsychological Society : JINS*, *15*, 438–450.
Sartre, J.-P. (1939). Sketch for a Theory of the Emotions. (P. Mairet). Abingdon, UK: Taylor & Francis.
Satpute, A. B., Wager, T. D., Cohen-Adad, J., Bianciardi, M., Choi, J. K., Buhle, J. T., et al. (2013). Identification of discrete functional subregions of the human periaqueductal gray. *Proceedings of the National Academy of Sciences*, *110*, 17101–17106.
Saxe, R., & Powell, L. J. (2006). It's the Thought That Counts: Specific Brain Regions for One Component of Theory of Mind. *Psychological Science*, *17*, 692–699.
Schacter, D. L., & Addis, D. R. (2007). The cognitive neuroscience of constructive memory: remembering the past and imagining the future. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *362*, 773–786.
Schacter, D. L., Addis, D. R., Hassabis, D., Martin, V. C., Spreng, R. N., & Szpunar, K. K. (2012). The Future of Memory: Remembering, Imagining, and the Brain. *Neuron*, *76*, 677–694.
Schiller, D., Levy, I., Niv, Y., LeDoux, J. E., & Phelps, E. A. (2008). From fear to safety and back: reversal of fear in the human brain. *The Journal of Neuroscience*, *28*, 11517–11525.
Schultz, W. (2006). Behavioral Theories and the Neurophysiology of Reward. *Annual Review of Psychology*, *57*, 87–115.
Schultz, W. (2010). Subjective neuronal coding of reward: temporal value discounting and risk. *European Journal of Neuroscience*, *31*, 2124–2135.
Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., et al. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *The Journal of Neuroscience*, *27*, 2349–2356.
Seth, A. K. (2013). Interoceptive inference, emotion, and the embodied self. *Trends in Cognitive Sciences*, *17*, 565–573.
Setlow, B., Roozendaal, B., & McGaugh, J. L. (2008). Involvement of a basolateral amygdala

complex-nucleus accumbens pathway in glucocorticoid-induced modulation of memory consolidation. *European Journal of Neuroscience*, *12*, 367–375.
Seymour, B., & Dolan, R. (2008). Emotion, Decision Making, and the Amygdala. *Neuron*, *58*, 662–671.
Shanafelt, T. D., Boone, S., Tan, L., Dyrbye, L. N., Sotile, W., Satele, D., et al. (2012). Burnout and satisfaction with work-life balance among US physicians relative to the general US population. *Archives of Internal Medicine*, *172*, 1377–1385.
Sheppes, G., & Gross, J. J. (2011). Is Timing Everything? Temporal Considerations in Emotion Regulation. *Personality and Social Psychology Review*, *15*, 319–331.
Singer, T., & Klimecki, O. M. (2014). Empathy and compassion. *Current Biology : CB*, *24*, R875–8.
Singer, T., Ben Seymour, O'Doherty, J., Kaube, H., Dolan, R. J., & Frith, C. D. (2004). Empathy for Pain Involves the Affective but not Sensory Components of Pain. *Science*, *303*, 1157–1162.
Singer, T., Critchley, H. D., & Preuschoff, K. (2009). A common role of insula in feelings, empathy and uncertainty. *Trends in Cognitive Sciences*, *13*, 334–340.
Singer, T., Kok, B. E., Bornemann, B., Zurborg, S., Bolz, M., & Bochow, C. (2016). The ReSource Project. Background, design, samples, and measurements (2nd ed.). Leipzig, Germany: Max Planck Institute for Human Cognitive and Brain Sciences.
Singer, T., Seymour, B., O'Doherty, J. P., Stephan, K. E., Dolan, R. J., & Frith, C. D. (2006). Empathic neural responses are modulated by the perceived fairness of others. *Nature*, *439*, 466–469.
Smallwood, J., & Schooler, J. W. (2015). The Science of Mind Wandering: Empirically Navigating the Stream of Consciousness. *Annual Review of Psychology*, *66*, 487–518.
Smallwood, J., Brown, K., Baird, B., & Schooler, J. W. (2012). Cooperation between the default mode network and the frontal-parietal network in the production of an internal train of thought. *Brain Research*, *1428*, 60–70.
Smallwood, J., Tipper, C., Brown, K., Baird, B., Engen, H., Michaels, J. R., et al. (2013). Escaping the here and now: evidence for a role of the default mode network in perceptually decoupled thought. *NeuroImage*, *69*, 120–125.
Solomon, R. C. (2003). On the Passivity of the Passions (2001). In *Not Passion's Slave* (pp. 195–232). Oxford: Oxford University Press.
Song, X.-W., Dong, Z.-Y., Long, X.-Y., Li, S.-F., Zuo, X.-N., Zhu, C.-Z., et al. (2011). REST: a toolkit for resting-state functional magnetic resonance imaging data processing. *PloS One*, *6*, e25031.
Spielberger, C. D., Gorsuch, R. L., Lushene, R., & Vagg, P. R. (1983). Manual for the state-trait anxiety scale. Palo Alto: Consulting Psychologists Press.
Spreng, R. N., & Grady, C. L. (2010). Patterns of Brain Activity Supporting Autobiographical Memory, Prospection, and Theory of Mind, and Their Relationship to the Default Mode Network. *Journal of Cognitive Neuroscience*, *22*, 1112–1123.
Spreng, R. N., Mar, R. A., & Kim, A. S. N. (2009). The common neural basis of autobiographical memory, prospection, navigation, theory of mind, and the default mode: a quantitative meta-analysis. *Journal of Cognitive Neuroscience*, *21*, 489–510.
Spreng, R. N., Sepulcre, J., Turner, G. R., Stevens, W. D., & Schacter, D. L. (2013). Intrinsic Architecture Underlying the Relations among the Default, Dorsal Attention, and Frontoparietal Control Networks of the Human Brain. *Journal of Cognitive Neuroscience*, *25*, 74–86.
Spreng, R. N., Stevens, W. D., Chamberlain, J. P., Gilmore, A. W., & Schacter, D. L. (2010). Default network activity, coupled with the frontoparietal control network, supports goal-directed cognition. *NeuroImage*, *53*, 303–317.
Sridharan, D., Levitin, D. J., & Menon, V. (2008). A critical role for the right fronto-insular

cortex in switching between central-executive and default-mode networks. *Proceedings of the National Academy of Sciences of the United States of America*, *105*, 12569–12574.
Stein, J. L., Wiedholz, L. M., Bassett, D. S., Weinberger, D. R., Zink, C. F., Mattay, V. S., & Meyer-Lindenberg, A. (2007). A validated network of effective amygdala connectivity. *NeuroImage*, *36*, 736–745.
Stöber, J. (1998). Worry, problem elaboration and suppression of imagery: the role of concreteness. *Behaviour Research and Therapy*, *36*, 751–756.
Strathearn, L., Fonagy, P., Amico, J., & Montague, P. R. (2009). Adult attachment predicts maternal brain and oxytocin response to infant cues. *Neuropsychopharmacology*, *34*, 2655–2666.
Suess, F., & Abdel Rahman, R. (2015). Mental imagery of emotions: Electrophysiological evidence. *NeuroImage*, *114*, 147–157.
Svoboda, E., McKinnon, M. C., & Levine, B. (2006). The functional neuroanatomy of autobiographical memory: A meta-analysis. *Neuropsychologia*, *44*, 2189–2208.
Szpunar, K. K. (2010). Episodic Future Thought An Emerging Concept. *Perspectives on Psychological Science*, *5*, 142–162.
Szpunar, K. K., & Schacter, D. L. (2013). Get real: Effects of repeated simulation and emotion on the perceived plausibility of future experiences. *Journal of Experimental Psychology. General*, *142*, 323.
Tamir, M., Mitchell, C., & Gross, J. J. (2008). Hedonic and Instrumental Motives in Anger Regulation. *Psychological Science*, *19*, 328.
Taubert, M., Lohmann, G., Margulies, D. S., Villringer, A., & Ragert, P. (2011). Long-term effects of motor training on resting-state networks and underlying brain structure. *NeuroImage*, *57*, 1492–1498.
Taylor, J. C., Wiggett, A. J., & Downing, P. E. (2007). Functional MRI analysis of body and body part representations in the extrastriate and fusiform body areas. *Journal of Neurophysiology*, *98*, 1626–1633.
Taylor, S. E., & Schneider, S. K. (1989). Coping and the Simulation of Events. *Social Cognition*, *7*, 174–194.
Taylor, S. E., Pham, L. B., Rivkin, I. D., & Armor, D. A. (1998). Harnessing the imagination: Mental simulation, self-regulation, and coping. *American Psychologist*, *53*, 429–439.
Thiruchselvam, R., Blechert, J., Sheppes, G., Rydstrom, A., & Gross, J. J. (2011). The temporal dynamics of emotion regulation: An EEG study of distraction and reappraisal. *Biological Psychology*, *87*, 84–92.
Thompson, H. E., Henshall, L., & Jefferies, E. (2016). The role of the right hemisphere in semantic control: A case-series comparison of right and left hemisphere stroke. *Neuropsychologia*, *85*, 44–61.
Tingley, D., Yamamoto, T., Hirose, K., Keele, L., & Imai, K. (2014). mediation: R Package for Causal Mediation Analysis. *Journal of Statistical Software*, *59*, 1–38.
Tomasi, D., & Volkow, N. D. (2014). Functional Connectivity of Substantia Nigra and Ventral Tegmental Area: Maturation During Adolescence and Effects of ADHD. *Cerebral Cortex*, *24*, 935–944.
Touroutoglou, A., Bliss-Moreau, E., Zhang, J., Mantini, D., Vanduffel, W., Dickerson, B. C., & Barrett, L. F. (2016). A ventral salience network in the macaque brain. *NeuroImage*, *132*, 190–197.
Touroutoglou, A., Hollenbeck, M., Dickerson, B. C., & Barrett, L. F. (2012). Dissociable large-scale networks anchored in the right anterior insula subserve affective experience and attention. *NeuroImage*, *60*, 1947–1958.
Tugade, M. M., & Fredrickson, B. L. (2004). Resilient Individuals Use Positive Emotions to Bounce Back From Negative Emotional Experiences. *Journal of Personality and Social Psychology*, *86*, 320–333.

Tusche, A., Smallwood, J., Bernhardt, B. C., & Singer, T. (2014). Classifying the wandering mind: revealing the affective content of thoughts during task-free rest periods. *NeuroImage*, *97*, 107–116.

Tziortzi, A. C., Searle, G. E., Tzimopoulou, S., Salinas, C., Beaver, J. D., Jenkinson, M., et al. (2011). Imaging dopamine receptors in humans with [11C]-(+)-PHNO: dissection of D3 signal and anatomy. *NeuroImage*, *54*, 264–277.

Verduyn, P., Delvaux, E., Van Coillie, H., Tuerlinckx, F., & Van Mechelen, I. (2009). Predicting the duration of emotional experience: Two experience sampling studies. *Emotion*, *9*, 83–91.

Vossel, S., Geng, J. J., & Fink, G. R. (2014). Dorsal and ventral attention systems: distinct neural circuits but collaborative roles. *The Neuroscientist*, *20*, 150–159.

Vrana, S. R., Cuthbert, B. N., & Lang, P. J. (1986). Fear Imagery and Text Processing. *Psychophysiology*, *23*, 247–253.

Vrtička, P., Andersson, F., Grandjean, D., Sander, D., & Vuilleumier, P. (2008). Individual attachment style modulates human amygdala and striatum activation during social appraisal. *PloS One*, *3*, e2868.

Vytal, K., & Hamann, S. (2010). Neuroimaging Support for Discrete Neural Correlates of Basic Emotions: A Voxel-based Meta-analysis. *Journal of Cognitive Neuroscience*, *22*, 2864–2885.

Wager, T. D., Atlas, L. Y., Lindquist, M. A., Roy, M., Woo, C.-W., & Kross, E. (2013). An fMRI-based neurologic signature of physical pain. *The New England Journal of Medicine*, *368*, 1388–1397.

Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008). Prefrontal-subcortical pathways mediating successful emotion regulation. *Neuron*, *59*, 1037–1050.

Wager, T. D., Keller, M. C., Lacey, S. C., & Jonides, J. (2005). Increased sensitivity in neuroimaging analyses using robust regression. *NeuroImage*, *26*, 99–113.

Wagner, S., Sebastian, A., Lieb, K., Tüscher, O., & Tadić, A. (2014). A coordinate-based ALE functional MRI meta-analysis of brain activation during verbal fluency tasks in healthy control subjects. *BMC Neuroscience*, *15*, 1.

Wang, X., Zhang, T., Chaim, T. M., Zanetti, M. V., & Davatzikos, C. (2015). Classification of MRI under the Presence of Disease Heterogeneity using Multi-Task Learning: Application to Bipolar Disorder. In N. Navab, J. Hornegger, W. M. Wells, & A. F. Frangi, *Medical Image Computing and Computer-Assisted Intervention -- MICCAI 2015* (Vol. 9349, pp. 125–132). Cham: Springer International Publishing.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063–1070.

Webb, T. L., Miles, E., & Sheeran, P. (2012). Dealing with feeling: A meta-analysis of the effectiveness of strategies derived from the process model of emotion regulation. *Psychological Bulletin*, *138*, 775–808.

Weng, H. Y., Fox, A. S., Shackman, A. J., Stodola, D. E., Caldwell, J. Z. K., Olson, M. C., et al. (2013). Compassion training alters altruism and neural responses to suffering. *Psychological Science*, *24*, 1171–1180.

Whalen, P. J. (1998). Fear, Vigilance, and Ambiguity: Initial Neuroimaging Studies of the Human Amygdala. *Current Directions in Psychological Science*, *7*, 177–188.

Whalen, P. J., Kagan, J., Cook, R. G., Davis, F. C., Kim, H., Polis, S., et al. (2004). Human amygdala responsivity to masked fearful eye whites. *Science*, *306*, 2061–2061.

Wheeler, M. E., Petersen, S. E., & Buckner, R. L. (2000). Memory's echo: vivid remembering reactivates sensory-specific cortex. *Proceedings of the National Academy of Sciences*, *97*, 11125–11129.

White, T. P., Engen, N. H., Sørensen, S., Overgaard, M., & Shergill, S. S. (2014). Uncertainty and confidence from the triple-network perspective: voxel-based meta-analyses. *Brain and Cognition*, *85*, 191–200.

Whitman, J. C., Metzak, P. D., Lavigne, K. M., & Woodward, T. S. (2013). Functional connectivity in a frontoparietal network involving the dorsal anterior cingulate cortex underlies decisions to accept a hypothesis. *Neuropsychologia*, *51*, 1132–1141.

Williamson, V. J., Jilka, S. R., Fry, J., Finkel, S., Mullensiefen, D., & Stewart, L. (2012). How do "earworms" start? Classifying the everyday circumstances of Involuntary Musical Imagery. *Psychology of Music*, *40*, 259–284.

Wilson-Mendenhall, C. D., Barrett, L. F., & Barsalou, L. W. (2013). Situating emotional experience. *Frontiers in Human Neuroscience*, *7*, 764.

Wilson-Mendenhall, C. D., Barrett, L. F., Simmons, W. K., & Barsalou, L. W. (2011). Grounding emotion in situated conceptualization. *Neuropsychologia*, *49*, 1105–1127.

Wiltink, J., Vogelsang, U., & Beutel, M. E. (2006). Temperament and personality: the German version of the Adult Temperament Questionnaire (ATQ). *Psycho-Social Medicine*, *3*, Doc10.

Woodward, T. S., Cairo, T. A., Ruff, C. C., Takane, Y., Hunter, M. A., & Ngan, E. T. C. (2006). Functional connectivity reveals load dependent neural systems underlying encoding and maintenance in verbal working memory. *Neuroscience*, *139*, 317–325.

Woodward, T. S., Feredoes, E., Metzak, P. D., Takane, Y., & Manoach, D. S. (2013). Epoch-specific functional networks involved in working memory. *NeuroImage*, *65*, 529–539.

Worsley, K. J., Taylor, J. E., Carbonell, F., Chung, M. K., Duerden, E., Bernhardt, B., et al. (2009). SurfStat: A Matlab toolbox for the statistical analysis of univariate and multivariate surface and volumetric data using linear mixed effects models and random field theory. *NeuroImage*, *47*, S102.

Yan, C., Liu, D., He, Y., Zou, Q., Zhu, C., Zuo, X., et al. (2009). Spontaneous Brain Activity in the Default Mode Network Is Sensitive to Different Resting-State Conditions with Limited Cognitive Load. *PloS One*, *4*, e5743.

Yang, H., Long, X.-Y., Yang, Y., Yan, H., Zhu, C.-Z., Zhou, X.-P., et al. (2007). Amplitude of low frequency fluctuation within visual areas revealed by resting-state functional MRI. *NeuroImage*, *36*, 144–152.

Yarkoni, T., Poldrack, R. A., Nichols, T. E., Van Essen, D. C., & Wager, T. D. (2011). Large-scale automated synthesis of human functional neuroimaging data. *Nature Methods*, *8*, 665–670.

Yeo, B. T. T., Krienen, F. M., Sepulcre, J., Sabuncu, M. R., Lashkari, D., Hollinshead, M., et al. (2011). The organization of the human cerebral cortex estimated by intrinsic functional connectivity. *Journal of Neurophysiology*, *106*, 1125–1165.

Yonelinas, A. P., & Ritchey, M. (2015). The slow forgetting of emotional episodic memories: an emotional binding account. *Trends in Cognitive Sciences*, *19*, 259–267.

Zaki, J., Davis, J. I., & Ochsner, K. N. (2012). Overlapping activity in anterior insula during interoception and emotional experience. *NeuroImage*, *62*, 493–499.

Zang, Y.-F., He, Y., Zhu, C.-Z., Cao, Q.-J., Sui, M.-Q., Liang, M., et al. (2007). Altered baseline brain activity in children with ADHD revealed by resting-state functional MRI. *Brain & Development*, *29*, 83–91.

Zeman, A., Dewar, M., & Sala, Della, S. (2015). Lives without imagery – Congenital aphantasia. *Cortex*, *73*, 378–380.

Zeng, X., Chiu, C. P. K., Wang, R., Oei, T. P. S., & Leung, F. Y. K. (2015). The effect of loving-kindness meditation on positive emotions: a meta-analytic review. *Frontiers in Psychology*, *6*, 187.

Zhang, Z., Lu, G., Zhong, Y., Tan, Q., Chen, H., Liao, W., et al. (2010). fMRI study of mesial temporal lobe epilepsy using amplitude of low-frequency fluctuation analysis. *Human*

Brain Mapping, *31*, 1851–1861.
Zou, Q.-H., Zhu, C.-Z., Yang, Y., Zuo, X.-N., Long, X.-Y., Cao, Q.-J., et al. (2008). An improved approach to detection of amplitude of low-frequency fluctuation (ALFF) for resting-state fMRI: Fractional ALFF. *Journal of Neuroscience Methods*, *172*, 137–141.
Zuo, X.-N., Di Martino, A., Kelly, C., Shehzad, Z. E., Gee, D. G., Klein, D. F., et al. (2010). The oscillating brain: Complex and reliable. *NeuroImage*, *49*, 1432–1445.

MPI Series in Human Cognitive and Brain Sciences:

1 Anja Hahne
Charakteristika syntaktischer und semantischer Prozesse bei der auditiven Sprachverarbeitung: Evidenz aus ereigniskorrelierten Potentialstudien

2 Ricarda Schubotz
Erinnern kurzer Zeitdauern: Behaviorale und neurophysiologische Korrelate einer Arbeitsgedächtnisfunktion

3 Volker Bosch
Das Halten von Information im Arbeitsgedächtnis: Dissoziationen langsamer corticaler Potentiale

4 Jorge Jovicich
An investigation of the use of Gradient- and Spin-Echo (GRASE) imaging for functional MRI of the human brain

5 Rosemary C. Dymond
Spatial Specificity and Temporal Accuracy in Functional Magnetic Resonance Investigations

6 Stefan Zysset
Eine experimentalpsychologische Studie zu Gedächtnisabrufprozessen unter Verwendung der funktionellen Magnetresonanztomographie

7 Ulrich Hartmann
Ein mechanisches Finite-Elemente-Modell des menschlichen Kopfes

8 Bertram Opitz
Funktionelle Neuroanatomie der Verarbeitung einfacher und komplexer akustischer Reize: Integration haemodynamischer und elektrophysiologischer Maße

9 Gisela Müller-Plath
Formale Modellierung visueller Suchstrategien mit Anwendungen bei der Lokalisation von Hirnfunktionen und in der Diagnostik von Aufmerksamkeitsstörungen

10 Thomas Jacobsen
Characteristics of processing morphological structural and inherent case in language comprehension

11 Stefan Kölsch
Brain and Music
A contribution to the investigation of central auditory processing with a new electrophysiological approach

12 Stefan Frisch
Verb-Argument-Struktur, Kasus und thematische Interpretation beim Sprachverstehen

13 Markus Ullsperger
The role of retrieval inhibition in directed forgetting – an event-related brain potential analysis

14 Martin Koch
Measurement of the Self-Diffusion Tensor of Water in the Human Brain

15 Axel Hutt
Methoden zur Untersuchung der Dynamik raumzeitlicher Signale

16 Frithjof Kruggel
Detektion und Quantifizierung von Hirnaktivität mit der funktionellen Magnetresonanztomographie

17 Anja Dove
Lokalisierung an internen Kontrollprozessen beteiligter Hirngebiete mithilfe des Aufgabenwechselparadigmas und der ereigniskorrelierten funktionellen Magnetresonanztomographie

18 Karsten Steinhauer
Hirnphysiologische Korrelate prosodischer Satzverarbeitung bei gesprochener und geschriebener Sprache

19 Silke Urban
Verbinformationen im Satzverstehen

20 Katja Werheid
Implizites Sequenzlernen bei Morbus Parkinson

21 Doreen Nessler
Is it Memory or Illusion? Electrophysiological Characteristics of True and False Recognition

22 Christoph Herrmann
Die Bedeutung von 40-Hz-Oszillationen für kognitive Prozesse

23 Christian Fiebach
Working Memory and Syntax during Sentence Processing.
A neurocognitive investigation with event-related brain potentials and functional magnetic resonance imaging

24 Grit Hein
Lokalisation von Doppelaufgabendefiziten bei gesunden älteren Personen und neurologischen Patienten

25 Monica de Filippis
Die visuelle Verarbeitung unbeachteter Wörter. Ein elektrophysiologischer Ansatz

26 Ulrich Müller
Die katecholaminerge Modulation präfrontaler kognitiver Funktionen beim Menschen

27 Kristina Uhl
Kontrollfunktion des Arbeitsgedächtnisses über interferierende Information

28 Ina Bornkessel
The Argument Dependency Model: A Neurocognitive Approach to Incremental Interpretation

29 Sonja Lattner
Neurophysiologische Untersuchungen zur auditorischen Verarbeitung von Stimminformationen

30 Christin Grünewald
Die Rolle motorischer Schemata bei der Objektrepräsentation: Untersuchungen mit funktioneller Magnetresonanztomographie

31 Annett Schirmer
Emotional Speech Perception: Electrophysiological Insights into the Processing of Emotional Prosody and Word Valence in Men and Women

32 André J. Szameitat
Die Funktionalität des lateral-präfrontalen Cortex für die Verarbeitung von Doppelaufgaben

33 Susanne Wagner
Verbales Arbeitsgedächtnis und die Verarbeitung ambiger Wörter in Wort- und Satzkontexten

34 Sophie Manthey
Hirn und Handlung: Untersuchung der Handlungsrepräsentation im ventralen prämotorischen Cortex mit Hilfe der funktionellen Magnet-Resonanz-Tomographie

35 Stefan Heim
Towards a Common Neural Network Model of Language Production and Comprehension: fMRI Evidence for the Processing of Phonological and Syntactic Information in Single Words

36 Claudia Friedrich
Prosody and spoken word recognition: Behavioral and ERP correlates

37 Ulrike Lex
Sprachlateralisierung bei Rechts- und Linkshändern mit funktioneller Magnetresonanztomographie

38 Thomas Arnold
Computergestützte Befundung klinischer Elektroenzephalogramme

39 Carsten H. Wolters
Influence of Tissue Conductivity Inhomogeneity and Anisotropy on EEG/MEG based Source Localization in the Human Brain

40 Ansgar Hantsch
Fisch oder Karpfen? Lexikale Aktivierung von Benennungsalternative bei der Objektbenennung

41 Peggy Bungert
Zentralnervöse Verarbeitung akustischer Informationen Signalidentifikation, Signallateralisation und zeitgebundene Informationsverarbeitung bei Patienten mit erworbenen Hirnschädigungen

42 Daniel Senkowski
Neuronal correlates of selective attention: An investigation of electrophysiological brain responses in the EEG and MEG

43 Gert Wollny
Analysis of Changes in Temporal Series of Medical Images

S 1 Markus Ullsperger & Michael Falkenstein
Errors, Conflicts, and the Brain Current Opinions on Performance Monitoring

44 Angelika Wolf
Sprachverstehen mit Cochlea-Implantat: EKP-Studien mit postlingual ertaubten erwachsenen CI-Trägern

45 Kirsten G. Volz
Brain correlates of uncertain decisions: Types and degrees of uncertainty

46 Hagen Huttner
Magnetresonanztomographische Untersuchungen über die anatomische Variabilität des Frontallappens des menschlichen Großhirns

47 Dirk Köster
Morphology and Spoken Word Comprehension: Electrophysiological Investigations of Internal Compound Structure

48 Claudia A. Hruska
Einflüsse kontextueller und prosodischer Informationen in der auditorischen Satzverarbeitung: Untersuchungen mit ereigniskorrelierten Hirnpotentialen

49 Hannes Ruge
Eine Analyse des raum-zeitlichen Musters neuronaler Aktivierung im Aufgabenwechselparadigma zur Untersuchung handlungssteuernder Prozesse

50 Ricarda I. Schubotz
Human premotor cortex: Beyond motor performance

51 Clemens von Zerssen
Bewusstes Erinnern und falsches Wiedererkennen: Eine funktionelle MRT Studie neuroanatomischer Gedächtniskorrelate

52 Christiane Weber
Rhythm is gonna get you.
Electrophysiological markers of rhythmic processing in infants with and without risk for Specific Language Impairment (SLI)

53 Marc Schönwiesner
Functional Mapping of Basic Acoustic Parameters in the Human Central Auditory System

54 Katja Fiehler
Temporospatial characteristics of error correction

55 Britta Stolterfoht
Processing Word Order Variations and Ellipses: The Interplay of Syntax and Information Structure during Sentence Comprehension

56 Claudia Danielmeier
Neuronale Grundlagen der Interferenz zwischen Handlung und visueller Wahrnehmung

57 Margret Hund-Georgiadis
Die Organisation von Sprache und ihre Reorganisation bei ausgewählten, neurologischen Erkrankungen gemessen mit funktioneller Magnetresonanztomographie – Einflüsse von Händigkeit, Läsion, Performanz und Perfusion

58 Jutta L. Mueller
Mechanisms of auditory sentence comprehension in first and second language: An electrophysiological miniature grammar study

59 Franziska Biedermann
Auditorische Diskriminationsleistungen nach unilateralen Läsionen im Di- und Telenzephalon

60 Shirley-Ann Rüschemeyer
The Processing of Lexical Semantic and Syntactic Information in Spoken Sentences: Neuroimaging and Behavioral Studies of Native and Non-Native Speakers

61 Kerstin Leuckefeld
The Development of Argument Processing Mechanisms in German. An Electrophysiological Investigation with School-Aged Children and Adults

62 Axel Christian Kühn
Bestimmung der Lateralisierung von Sprachprozessen unter besondere Berücksichtigung des temporalen Cortex, gemessen mit fMRT

63 Ann Pannekamp
Prosodische Informationsverarbeitung bei normalsprachlichem und deviantem Satzmaterial: Untersuchungen mit ereigniskorrelierten Hirnpotentialen

64 Jan Derrfuß
Functional specialization in the lateral frontal cortex: The role of the inferior frontal junction in cognitive control

65 Andrea Mona Philipp
The cognitive representation of tasks – Exploring the role of response modalities using the task-switching paradigm

66 Ulrike Toepel
Contrastive Topic and Focus Information in Discourse – Prosodic Realisation and Electrophysiological Brain Correlates

67 Karsten Müller
Die Anwendung von Spektral- und Waveletanalyse zur Untersuchung der Dynamik von BOLD-Zeitreihen verschiedener Hirnareale

68 Sonja A.Kotz
The role of the basal ganglia in auditory language processing: Evidence from ERP lesion studies and functional neuroimaging

69 Sonja Rossi
The role of proficiency in syntactic second language processing: Evidence from event-related brain potentials in German and Italian

70 Birte U. Forstmann
Behavioral and neural correlates of endogenous control processes in task switching

71 Silke Paulmann
Electrophysiological Evidence on the Processing of Emotional Prosody: Insights from Healthy and Patient Populations

72 Matthias L. Schroeter
Enlightening the Brain – Optical Imaging in Cognitive Neuroscience

73 Julia Reinholz
Interhemispheric interaction in object- and word-related visual areas

74 Evelyn C. Ferstl
The Functional Neuroanatomy of Text Comprehension

75 Miriam Gade
Aufgabeninhibition als Mechanismus der Konfliktreduktion zwischen Aufgabenrepräsentationen

76 Juliane Hofmann
Phonological, Morphological, and Semantic Aspects of Grammatical Gender Processing in German

77 Petra Augurzky
Attaching Relative Clauses in German – The Role of Implicit and Explicit Prosody in Sentence Processing

78 Uta Wolfensteller
Habituelle und arbiträre sensomotorische Verknüpfungen im lateralen prämotorischen Kortex des Menschen

79 Päivi Sivonen
Event-related brain activation in speech perception: From sensory to cognitive processes

80 Yun Nan
Music phrase structure perception: the neural basis, the effects of acculturation and musical training

81 Katrin Schulze
Neural Correlates of Working Memory for Verbal and Tonal Stimuli in Nonmusicians and Musicians With and Without Absolute Pitch

82 Korinna Eckstein
Interaktion von Syntax und Prosodie beim Sprachverstehen: Untersuchungen anhand ereigniskorrelierter Hirnpotentiale

83 Florian Th. Siebörger
Funktionelle Neuroanatomie des Textverstehens: Kohärenzbildung bei Witzen und anderen ungewöhnlichen Texten

84 Diana Böttger
Aktivität im Gamma-Frequenzbereich des EEG: Einfluss demographischer Faktoren und kognitiver Korrelate

85 Jörg Bahlmann
Neural correlates of the processing of linear and hierarchical artificial grammar rules: Electrophysiological and neuroimaging studies

86 Jan Zwickel
Specific Interference Effects Between Temporally Overlapping Action and Perception

87 Markus Ullsperger
Functional Neuroanatomy of Performance Monitoring: fMRI, ERP, and Patient Studies

88 Susanne Dietrich
Vom Brüllen zum Wort – MRT-Studien zur kognitiven Verarbeitung emotionaler Vokalisationen

89 Maren Schmidt-Kassow
What's Beat got to do with ist? The Influence of Meter on Syntactic Processing: ERP Evidence from Healthy and Patient populations

90 Monika Lück
Die Verarbeitung morphologisch komplexer Wörter bei Kindern im Schulalter: Neurophysiologische Korrelate der Entwicklung

91 Diana P. Szameitat
Perzeption und akustische Eigenschaften von Emotionen in menschlichem Lachen

92 Beate Sabisch
Mechanisms of auditory sentence comprehension in children with specific language impairment and children with developmental dyslexia: A neurophysiological investigation

93 Regine Oberecker
Grammatikverarbeitung im Kindesalter: EKP-Studien zum auditorischen Satzverstehen

94 Şükrü Barış Demiral
Incremental Argument Interpretation in Turkish Sentence Comprehension

95 Henning Holle
The Comprehension of Co-Speech Iconic Gestures: Behavioral, Electrophysiological and Neuroimaging Studies

96 Marcel Braß
Das inferior frontale Kreuzungsareal und seine Rolle bei der kognitiven Kontrolle unseres Verhaltens

97 Anna S. Hasting
Syntax in a blink: Early and automatic processing of syntactic rules as revealed by event-related brain potentials

98 Sebastian Jentschke
Neural Correlates of Processing Syntax in Music and Language – Influences of Development, Musical Training and Language Impairment

99 Amelie Mahlstedt
The Acquisition of Case marking Information as a Cue to Argument Interpretation in German
An Electrophysiological Investigation with Pre-school Children

100 Nikolaus Steinbeis
Investigating the meaning of music using EEG and fMRI

101 Tilmann A. Klein
Learning from errors: Genetic evidence for a central role of dopamine in human performance monitoring

102 Franziska Maria Korb
Die funktionelle Spezialisierung des lateralen präfrontalen Cortex: Untersuchungen mittels funktioneller Magnetresonanztomographie

103 Sonja Fleischhauer
Neuronale Verarbeitung emotionaler Prosodie und Syntax: die Rolle des verbalen Arbeitsgedächtnisses

104 Friederike Sophie Haupt
The component mapping problem: An investigation of grammatical function reanalysis in differing experimental contexts using eventrelated brain potentials

105 Jens Brauer
Functional development and structural maturation in the brain's neural network underlying language comprehension

106 Philipp Kanske
Exploring executive attention in emotion: ERP and fMRI evidence

107 Julia Grieser Painter
Music, meaning, and a semantic space for musical sounds

108 Daniela Sammler
The Neuroanatomical Overlap of Syntax Processing in Music and Language - Evidence from Lesion and Intracranial ERP Studies

109 Norbert Zmyj
Selective Imitation in One-Year-Olds: How a Model's Characteristics Influence Imitation

110 Thomas Fritz
Emotion investigated with music of variable valence – neurophysiology and cultural influence

111 Stefanie Regel
The comprehension of figurative language: Electrophysiological evidence on the processing of irony

112 Miriam Beisert
Transformation Rules in Tool Use

113 Veronika Krieghoff
Neural correlates of Intentional Actions

114 Andreja Bubić
Violation of expectations in sequence processing

115 Claudia Männel
Prosodic processing during language acquisition: Electrophysiological studies on intonational phrase processing

116 Konstanze Albrecht
Brain correlates of cognitive processes underlying intertemporal choice for self and other

117 Katrin Sakreida
Nicht-motorische Funktionen des prämotorischen Kortex: Patientenstudien und funktionelle Bildgebung

118 Susann Wolff
The interplay of free word order and pro-drop in incremental sentence processing: Neurophysiological evidence from Japanese

119 Tim Raettig
The Cortical Infrastructure of Language Processing: Evidence from Functional and Anatomical Neuroimaging

120 Maria Golde
Premotor cortex contributions to abstract and action-related relational processing

121 Daniel S. Margulies
Resting-State Functional Connectivity fMRI: A new approach for assessing functional neuroanatomy in humans with applications to neuroanatomical, developmental and clinical questions

122 Franziska Süß
The interplay between attention and syntactic processes in the adult and developing brain: ERP evidences

123 Stefan Bode
From stimuli to motor responses: Decoding rules and decision mechanisms in the human brain

124 Christiane Diefenbach
Interactions between sentence comprehension and concurrent action: The role of movement effects and timing

125 Moritz M. Daum
Mechanismen der frühkindlichen Entwicklung des Handlungsverständnisses

126 Jürgen Dukart
Contribution of FDG-PET and MRI to improve Understanding, Detection and Differentiation of Dementia

127 Kamal Kumar Choudhary
Incremental Argument Interpretation in a Split Ergative Language: Neurophysiological Evidence from Hindi

128 Peggy Sparenberg
Filling the Gap: Temporal and Motor Aspects of the Mental Simulation of Occluded Actions

129 Luming Wang
The Influence of Animacy and Context on Word Order Processing: Neurophysiological Evidence from Mandarin Chinese

130 Barbara Ettrich
Beeinträchtigung frontomedianer Funktionen bei Schädel-Hirn-Trauma

131 Sandra Dietrich
Coordination of Unimanual Continuous Movements with External Events

132 R. Muralikrishnan
An Electrophysiological Investigation Of Tamil Dative-Subject Constructions

133 Christian Obermeier
Exploring the significance of task, timing and background noise on gesture-speech integration

134 Björn Herrmann
Grammar and perception: Dissociation of early auditory processes in the brain

135 Eugenia Solano-Castiella
In vivo anatomical segmentation of the human amygdala and parcellation of emotional processing

136 Marco Taubert
Plastizität im sensomotorischen System – Lerninduzierte Veränderungen in der Struktur und Funktion des menschlichen Gehirns

137 Patricia Garrido Vásquez
Emotion Processing in Parkinson's Disease: The Role of Motor Symptom Asymmetry

138 Michael Schwartze
Adaptation to temporal structure

139 Christine S. Schipke
Processing Mechanisms of Argument Structure and Case-marking in Child Development: Neural Correlates and Behavioral Evidence

140 Sarah Jessen
Emotion Perception in the Multisensory Brain

141 Jane Neumann
Beyond activation detection: Advancing computational techniques for the analysis of functional MRI data

142 Franziska Knolle
Knowing what's next: The role of the cerebellum in generating predictions

143 Michael Skeide
Syntax and semantics networks in the developing brain

144 Sarah M. E. Gierhan
Brain networks for language
Anatomy and functional roles of neural pathways supporting language comprehension and repetition

145 Lars Meyer
The Working Memory of Argument-Verb Dependencies
Spatiotemporal Brain Dynamics during Sentence Processing

146 Benjamin Stahl
Treatment of Non-Fluent Aphasia through Melody, Rhythm and Formulaic Language

147 Kathrin Rothermich
The rhythm's gonna get you: ERP and fMRI evidence on the interaction of metric and semantic processing

148 Julia Merrill
Song and Speech Perception – Evidence from fMRI, Lesion Studies and Musical Disorder

149 Klaus-Martin Krönke
Learning by Doing?
Gesture-Based Word-Learning and its Neural Correlates in Healthy Volunteers and Patients with Residual Aphasia

150 Lisa Joana Knoll
When the hedgehog kisses the frog
A functional and structural investigation of syntactic processing in the developing brain

151 Nadine Diersch
Action prediction in the aging mind

152 Thomas Dolk
A Referential Coding Account for the Social Simon Effect

153 Mareike Bacha-Trams
Neurotransmitter receptor distribution in Broca's area and the posterior superior temporal gyrus

154 Andrea Michaela Walter
The role of goal representations in action control

155 Anne Keitel
Action perception in development: The role of experience

156 Iris Nikola Knierim
Rules don't come easy: Investigating feedback-based learning of phonotactic rules in language.

157 Jan Schreiber
Plausibility Tracking: A method to evaluate anatomical connectivity and microstructural properties along fiber pathways

158 Katja Macher
Die Beteiligung des Cerebellums am verbalen Arbeitsgedächtnis

159 Julia Erb
The neural dynamics of perceptual adaptation to degraded speech

160 Philipp Kanske
Neural bases of emotional processing in affective disorders

161 David Moreno-Dominguez
Whole-brain cortical parcellation: A hierarchical method based on dMRI tractography

162 Maria Christine van der Steen
Temporal adaptation and anticipation mechanisms in sensorimotor synchronization

163 Antje Strauß
Neural oscillatory dynamics of spoken word recognition

164 Jonas Obleser
The brain dynamics of comprehending degraded speech

165 Corinna E. Bonhage
Memory and Prediction in Sentence Processing

S 2 Tania Singer, Bethany E. Kok, Boris Bornemann, Matthias Bolz, and Christina A. Bochow
The Resource Project
Background, Design, Samples, and Measurements

166 Anna Wilsch
Neural oscillations in auditory working memory

167 Dominique Goltz
Sustained Spatial Attention in Touch: Underlying Brain Areas and Their Interaction

168 Juliane Dinse
A Model-Based Cortical Parcellation Scheme for High-Resolution 7 Tesla MRI Data

169 Gesa Schaadt
Visual, Auditory, and Visual-Auditory Speech Processing in School Children with Writing Difficulties

170 Laura Verga
Learning together or learning alone: Investigating the role of social interaction in second language word learning

171 Eva Maria Quinque
Brain, mood and cognition in hypothyroidism

172 Malte Wöstmann
Neural dynamics of selective attention to speech in noise

173 Charles-Étienne Benoit
Music-based gait rehabilitation in Parkinson's disease

174 Anja Fengler
How the Brain Attunes to Sentence Processing Relating Behavior, Structure, and Function

175 Emiliano Zaccarella
Breaking Down Complexity: The Neural Basis of the Syntactic Merge Mechanism in the Human Brain

S 2 Tania Singer, Bethany E. Kok, Boris Bornemann, Matthias Bolz, and Christina A. Bochow
2nd Edition The Resource Project
Background, Design, Samples, and Measurements

176 Manja Attig
Breaking Down Complexity: The Neural Basis of the Syntactic Merge Mechanism in the Human Brain

177 Andrea Reiter
Out of control behaviors?
Investigating mechanisms of behavioral control in alcohol addition, binge eating disorder, and associated risc factors

178 Anna Strotseva-Feinschmidt
The processing of complex syntax in early childhood

179 Smadar Ovadia-Caro
Plasticity following stroke: the recovery of functional networks as measured by resting-state functional connectivity

180 Indra Kraft
Predicting developmental dyslexia at a preliterate age by combining behavioral assessment with structural MRI

181 Sabine Frenzel
How actors become attractors
A neurocognitive investigation of linguistic actorhood

182 Anja Dietrich
Food craving regulation in the brain: the role of weight status and associated personality aspects