

1 **Surprise About Sensory Event Timing Drives** 2 **Cortical Transients in the Beta Frequency Band**

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20 21 **Author contributions:**

22 Conceptualization, T.M., N.A.K., E.J.W. and T.H.D.; Investigation, T.M.; Formal
23 analysis T.M.; Analytic tools, E.J.W. and N.A.K.; Writing - Original draft, T.M. and
24 T.H.D.; Writing - Review & Editing, T.M., N.A.K., A.K.E., E.J.W. and T.H.D.; Funding
25 Acquisition, A.K.E. and T.H.D.; Supervision, T.H.D.

26
27 **Number of pages: 21**

28 **Number of figures: 5**

29 **Number of tables/multimedia/3D models: 0**

30 **Number of words Abstract: 248**

31 **Number of words Significance Statement: 119**

32 **Number of words Introduction: 499**

33 **Number of words Discussion: 1287**

34
35 **Conflict of Interest:** The authors declare no competing financial interests.

36
37 **Acknowledgements:** This work was supported by the Netherlands Organization
38 for Scientific Research (NWO, dossiernummer 406-14-016, to T.H.D. and T.M.); the
39 Amsterdam Brain and Cognition priority program (ABC2014-01, to T.H.D.); the
40 European Union Seventh Framework Programme (FP7/2007-2013) under grant
41 agreement no. 604102 (Human Brain Project) (to T.H.D. and A.K.E.); and the
42 German Research Foundation (DFG): Heisenberg Professorship DO 1240/3-1 (to
43 T.H.D.), the Collaborative Research Centers SFB 936 (Projects A2/A3, A7, to A.K.E.,
44 T.H.D.) and TRR 169 (Project B1 to A.K.E.). We thank Udo Böhm for methodological
45 advice and all members of the Donner lab for helpful discussion.

46 **Abstract**

47 Learning the statistical structure of the environment is crucial for adaptive behavior.
48 Humans and non-human decision-makers seem to track such structure through a
49 process of probabilistic inference, which enables predictions about behaviorally
50 relevant events. Deviations from such predictions cause surprise, which in turn helps
51 improve inference. Surprise about the timing of behaviorally relevant sensory events
52 drives phasic responses of neuromodulatory brainstem systems, which project to the
53 cerebral cortex. Here, we developed a computational model-based
54 magnetoencephalography (MEG) approach for mapping the resulting cortical
55 transients across space, time, and frequency, in the human brain (N=28, 17 female).
56 We used a Bayesian ideal observer model to learn the statistics of the timing of
57 changes in a simple visual detection task. This model yielded quantitative trial-by-trial
58 estimates of temporal surprise. The model-based surprise variable predicted trial-by-
59 trial variations in reaction time more strongly than the externally observable interval
60 timings alone. Trial-by-trial variations in surprise were negatively correlated with the
61 power of cortical population activity measured with MEG. This surprise-related power
62 suppression occurred transiently around the behavioral response, specifically in the
63 beta frequency band. It peaked in parietal and prefrontal cortices, remote from the
64 motor cortical suppression of beta power related to overt report (button press) of
65 change detection. Our results indicate that surprise about sensory event timing
66 transiently suppresses ongoing beta-band oscillations in association cortex. This
67 transient suppression of frontal beta-band oscillations might reflect an active reset
68 triggered by surprise, and is in line with the idea that beta-oscillations help maintain
69 cognitive sets.

70

71

72 **Significance statement**

73 The brain continuously tracks the statistical structure of the environment to anticipate
74 behaviorally relevant events. Deviations from such predictions cause surprise, which
75 in turn drives neural activity in subcortical brain regions that project to the cerebral
76 cortex. We used magnetoencephalography in humans to map out surprise-related
77 modulations of cortical population activity across space, time, and frequency.
78 Surprise was elicited by variable timing of visual stimulus changes requiring a
79 behavioral response. Surprise was quantified by means of an ideal observer model.
80 Surprise predicted behavior as well as a transient suppression of beta frequency-
81 band oscillations in frontal cortical regions. Our results are in line with conceptual
82 accounts that have linked neural oscillations in the beta-band to the maintenance of
83 cognitive sets.

84 Introduction

85 Humans and other organisms continuously adapt their behavior to the statistical
86 structure of their environment. This suggests that the brain is equipped with neural
87 machinery for statistical learning, which can interact with the processes driving goal-
88 directed behavior. Of particular importance here is surprise (Dayan and Yu, 2006;
89 O'Reilly et al., 2013), a violation of one's expectation about the next event, which
90 might indicate a sudden change in the environmental structure, and can transiently
91 boost central arousal state, increasing the organism's sensitivity and learning rate
92 (Yu and Dayan, 2005; Nassar et al., 2012).

93 Expectation, uncertainty, and surprise are intricately related. The precision of
94 expectations scales with uncertainty, that is, the width of the distribution of observed
95 events: high uncertainty precludes forming precise expectations. Violations of
96 expectations cause surprise, the level of which depends on the difference between
97 the expected and actually observed event (often termed prediction error). These
98 intuitions can be formalized within the framework of Bayesian statistics and used to
99 search for neurophysiological correlates (see Materials and Methods: *Bayesian ideal*
100 *observer model: General approach and rationale*).

101 One important dimension of the environment is the timing of relevant sensory
102 events (Gibbon et al., 1997; Nobre et al., 2007). Two lines of work have studied the
103 neural basis of temporal expectation effects. One has shown that environments with
104 rhythmic (i.e., precise) temporal structure entrain neural oscillations in the cerebral
105 cortex, the phase of which then modulates sensory cortical responses, perception,
106 and cognition (Lakatos et al., 2008; Schroeder and Lakatos, 2009; Rohenkohl and
107 Nobre, 2011; Rohenkohl et al., 2012; Riecke et al., 2015; van Ede et al., 2017). In
108 these rhythmic changes of the environment, surprise is minimized (once the structure
109 is learned expectations match observations). Consequently, this first line of work has
110 identified neural correlates of temporal expectation, rather than of surprise.

111 The second line of work has studied neural responses of subcortical,
112 neuromodulatory centers, specifically, dopaminergic centers of the midbrain, to
113 sensory events entailing reward. Because event timing here varied non-periodically
114 from trial to trial as in many natural environments, this work could link phasic
115 neuromodulatory responses to temporal surprise (Hollerman and Schultz, 1998;
116 Fiorillo et al., 2008). Surprise-driven phasic responses might also occur in other
117 neuromodulatory brainstem systems, such the noradrenergic system (Dayan and Yu,
118 2006). Because brainstem neuromodulatory systems have widespread projections to
119 the cortical networks underlying goal-directed behavior, one would expect changes in
120 cortical population activity elicited by surprise (Bouret and Sara, 2005). However, this
121 second line of work on temporal expectation has focused on surprise-related activity
122 in subcortical systems.

123 Here, we studied responses to surprise about the timing of sensory events in
124 human cortex. A computational model-based magnetoencephalography (MEG)
125 approach enabled us to map surprise-related cortical transients across space, time,
126 and frequency. We used a Bayesian model that accumulated previously experienced
127 durations of the interval between visual changes into posterior beliefs about the next
128 interval duration. This ideal observer model provided trial-to-trial measures of
129 temporal surprise, which predicted modulations of prefrontal and parietal cortical
130 beta-band dynamics.

131

132 Materials & Methods

133 This paper reports a re-analysis of an MEG data set that has previously been used
134 for a study into decision-related feedback signals in visual cortex (Meindertsma et al.,
135 2017). Here, we focus on those aspects of the experimental design that are most

136 relevant for the issue addressed in the current paper: uncertainty and surprise about
137 the timing of the experimental events specified below. We refer to our previous paper
138 (Meindersma et al., 2017) for a more detailed description of the visual stimulus and
139 the behavioral task.

140

141 *Participants*

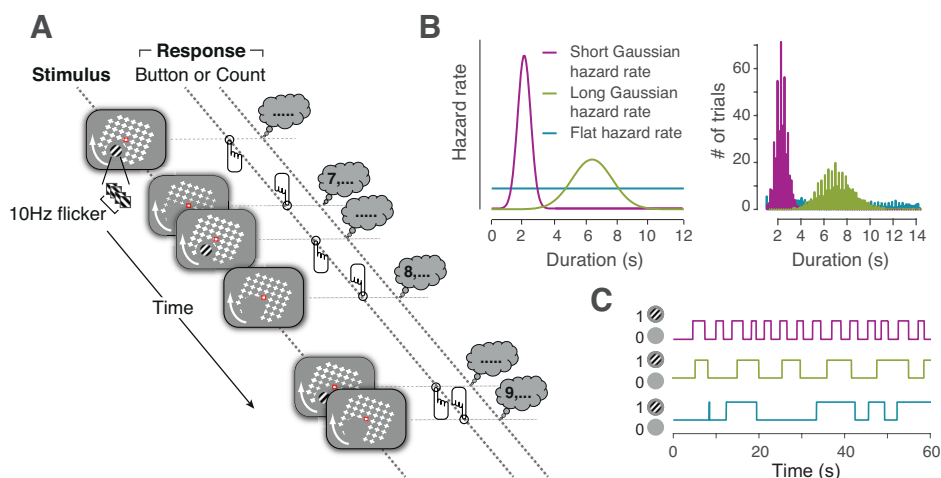
142 Thirty-one volunteers participated in the experiment. Two participants were excluded
143 due to incomplete data and one participant did not complete the experiment due to
144 poor quality of simultaneously acquired pupil data. Thus, 28 participants (17 female,
145 age range 20 - 54 years, mean age 28.3, SD 9.2) were included in the analysis. All
146 participants had normal or corrected-to-normal vision and no known history of
147 neurological disorders. The experiment was conducted in accordance with the
148 Declaration of Helsinki and approved by the local ethics committee of the Hamburg
149 Medical Association. Each participant gave written informed consent.

150

151 *Stimulus*

152 MEG was measured while subjects viewed the intermittent presentation of a target
153 (full contrast Gabor patch; diameter: 2°) and reported the on- and offset of the target
154 (Figure 1A). Gabor targets flickered at 10 Hz (counter-phase) through alternation of
155 two out-of phase Gabors every 50 ms. This caused steady-state evoked responses
156 over visual cortex at 10 and 20 Hz (data not shown), distinct in terms of spectral
157 profile, topography and functional characteristics from the surprise-related
158 modulations we focused on here. The target was located in either the lower left or
159 lower right visual field quadrant (eccentricity: 5°, counterbalanced between subjects),
160 surrounded by a rotating mask (17°x17° grid of black crosses), and superimposed on
161 a gray background. The mask rotated at a speed of 160°/s. The target was separated
162 from the mask by a gray “protection zone” subtending about 2° around the target
163 (Bonneh et al., 2001). Subjects fixated on a fixation mark (red outline, white inside,
164 0.8° width and length) centered on the mask in the middle of the screen. Stimuli
165 were presented using the Presentation Software (NeuroBehavioral Systems, Albany, CA,
166 USA, RRID:SCR_002521). Stimuli were back-projected on a transparent screen
167 using a Sanyo PLC-XP51 projector with a resolution of 1024x768 pixels at 60 Hz.
168 Subjects were seated 58 centimeters from the screen in a whole-head
169 magnetoencephalography (MEG) scanner setup in a dimly lit room.

170



171

172 **Figure 1: Behavioral task.** **A.** Schematic depiction of the stimulus and task. A salient, flickering
173 target (Gabor patch) temporarily appeared and disappeared on a rotating background. Subjects fixated
174 on the red fixation mark and reported stimulus changes either by direct button press or silently counting
175 the disappearances and reporting the total number at the end of the run. **B.** The interval duration
176 between stimulus changes was randomly drawn from one of three distributions that corresponded to

177 three hazard rates (left), resulting in distinct distributions of intervals (right, average histogram over
178 subjects). **C.** Example time courses of target presence (1 = present, 0 = absent) drawn from these
179 distributions.

180

181 *Behavioral task and experimental design*

182 The subjects' task was to maintain stable fixation and detect the physical offsets and
183 onsets of the target, the predictability of which fluctuated from trial to trial, and the
184 mean predictability of which varied systematically across blocks. To this end, the
185 interval durations between stimulus changes were sampled from three different
186 distributions in the different blocks. These distributions were computed so as to
187 produce three predetermined so-called hazard functions, which describe the
188 probability that an event will occur at a particular time, given that it has not occurred
189 yet. The hazard function formalizes the expectation of a change and affects human
190 reaction times in simple detection tasks (Luce, 1986). The hazard function can be
191 computed as follows:

192

$$193 \quad \lambda_t = \frac{f_t}{1-F_t}, \quad \text{Eq. 1}$$

194

195 where λ_t is the value of the hazard function at time point t , f_t is the value of
196 distribution f on time point t , and F_t is the area under the curve of distribution f from
197 $-\infty$ to time t .

198 We used the following procedure to construct three 'environments', referred to
199 as 'Short', 'Long', and 'Flat' below. We first selected three hazard functions that
200 systematically differed in their level of predictability (Figure 1B, C). We then
201 computed the actual distributions of intervals by re-arranging Eq. 1 as follows:

202

$$203 \quad f_t = \lambda_t * (1 - F_t), \quad \text{Eq. 2}$$

204

205 The interval durations were then randomly selected from f . Specifically, the temporal
206 environments were defined as follows:

207 *Short:* The hazard function was a narrow Gaussian distribution with a mean of
208 2 s and a standard deviation of 0.2 s. This resulted in nearly periodic and, thus,
209 largely predictable intervals between events.

210 *Long:* This condition used the same hazard function as the previous
211 condition, but with a larger mean and standard deviation (6 s and 0.6 s, respectively)
212 thus rendering event timings less predictable (Fiorillo et al., 2008).

213 *Flat:* The hazard function was flat with a mean of 6s, yielding the least
214 predictable interval durations. The resulting distribution of interval durations, f_t ,
215 therefore, approximated an exponential distribution; characterizing a memory-less
216 process (i.e. the timing of the next event could not be predicted from previously
217 encountered intervals, Feller, 1959).

218 Computational analysis with a Bayesian model (Fig. 2) described below
219 confirmed that the sampled intervals from these three environments gave rise to
220 different mean levels of uncertainty and surprise (Fig. 2G,H). The three environments
221 were presented in separate three-minute blocks.

222 Within each of the above temporal environments, there were two behavioral
223 tasks. Both tasks required subjects to monitor the changes of the small visual target.
224 In one task (called *Detection-button*), they were asked to report those changes
225 immediately. Specifically, subjects reported target offsets or onsets by pressing a
226 button with the right index or middle finger, respectively. In the other task (*Detection-*
227 *count*), they were asked to count and report the changes at the end of the block.
228 Subjects silently counted the number of target offsets and reported the total in

229 response to a 4-AFC question at the end to the block. The two tasks were randomly
230 selected before each block under the constraint that both would occur equally often.
231 We here analyzed both task conditions, but only found robust effect for Detection-
232 button.

233 All subjects completed a total of 6 blocks of the Short environment, and 16
234 blocks of the other Long and Flat environments, resulting in about the same number
235 of trials per environment. Additionally, subjects performed a motion-induced
236 blindness task and a functional localizer task, which were not relevant for the current
237 study, but are reported in our previous paper (Meindertsma et al., 2017). All blocks
238 within an environment were completed in succession; the order of environments was
239 counter-balanced across subjects.

240

241 *Bayesian ideal observer model: General approach and rationale*

242 We developed an ideal observer model to quantify surprise and uncertainty about the
243 timing of sensory events (i.e., the target on- and offsets). The model tracked the
244 evolving predictive distribution of upcoming interval durations; more specifically, it
245 computes the posterior predictive of unobserved interval durations, conditional on the
246 observed data, throughout each block of the experiment. We assumed that subjects
247 tracked the temporal statistics of the task in a similar way, and we used the posterior
248 predictive distribution as a proxy of the subjects' belief states (i.e., their prediction of
249 the timing of the next stimulus change).

250 While we used an ideal observer model that prescribed the optimal inference
251 for our task, we are agnostic to the precise inference process that was used by our
252 subjects and we do not claim that subjects used the exact computations used by the
253 model. Our central assumption was that subjects accumulated observations
254 throughout each block (i.e., over more than just one or two previous intervals). This
255 assumption was derived from a substantial body of work on other forms of learning
256 and evidence accumulation (Sutton and Barto, 1998; Gold and Shadlen, 2007; Glaze
257 et al., 2015), and it was supported by the findings described in Results. Our model
258 implemented the normative accumulation strategy by perfectly integrating across the
259 entire history of the observations (here: of interval durations) and updating internal
260 representations accordingly. A practical benefit of this approach was that it did not
261 require fitting of model parameters, for which our current data did not provide
262 sufficiently strong constraints.

263 The only free parameter in the model was the level of temporal estimation
264 noise, which we allowed to scale with the magnitude of the interval duration
265 according to Weber's law (Gibbon et al., 1997). To this end, we transformed the
266 discrete values of the observed intervals into Gaussian distributions that were used
267 to update the model (see next section). The mean of these distributions was equal to
268 the observed interval t and their standard deviation was equal to the observed
269 interval t times a Weber's fraction (coefficient of variation, Gibbon et al., 1997). We
270 simulated the model with 34 Weber's fraction values ranging from 0.001 to 0.5
271 (0.001, 0.05:0.01:0.35, 0.4, 0.5). We then computed the correlation between the
272 measured single-trial reaction times (pooled across all subjects) and surprise (see
273 *Bayesian ideal observer model: Implementation*), separately for each Weber fraction,
274 and selected the Weber fraction that maximized this correlation. To this end, we fitted
275 a second order polynomial to the correlation coefficients as a function of Weber's
276 fraction and extracted the maximum of the polynomial. This yielded a Weber's
277 fraction of 0.17 (Figure 2F), which was used for all analyses reported in this paper.
278 Using model-based surprise from a noise-free version of the model yielded
279 qualitatively identical results (data now shown).

280

281 *Bayesian ideal observer model: Implementation*

282 We assumed that the subjects used a model in which the observed intervals have
283 been generated from a gamma distribution with parameters alpha (shape) and beta
284 (scale). These parameters were given uninformative prior distributions (Lee and
285 Wagenmakers, 2013), which were updated by the data to posterior distributions.

286 Using the interval duration distributions as the observations, we could obtain
287 the expectations about to-be-observed intervals by generating posterior predictives
288 (i.e., drawing an alpha-beta pair from the joint posterior distribution and then drawing
289 a predicted interval from the associated gamma distribution; repeating this process
290 many times yields a posterior predictive distribution for the to-be-observed interval).
291 We assumed that the subjects updated their belief state after each observation of a
292 new interval duration. Likewise, the model was updated after every interval t by
293 computing a new posterior predictive distribution, based on the durations of intervals
294 $1:t$ and the prior.

295 We generated a posterior predictive distribution over the to-be-observed
296 intervals using Gibbs sampling (a Markov chain Monte Carlo, or MCMC, algorithm;
297 Andrieu et al., 2003) in the software JAGS (Plummer, 2003) and Matlab (version
298 R2013a, RRID:SCR_001622). We used two Markov chains with different starting
299 points comprised of 2500 samples per chain with 500 samples burn-in, for a
300 combined total of 4000 samples. The posterior predictive MCMC samples $Y_{1...4000}$
301 for the next interval, $t+1$, were then summarized by a gamma distribution using the
302 functions 'gamfit' and 'gampdf' in Matlab (Figure 2A,B):

303
304
$$Y_{1...4000}^{(t+1)} \sim \text{Gamma}(\kappa_{t+1}, \theta_{t+1}) \equiv f_{t+1}, \quad \text{Eq. 3}$$

305
306 where $Y_{1...4000}^{(t+1)}$ are the MCMC samples, and κ_{t+1} and θ_{t+1} are the parameters of the
307 gamma distribution f_{t+1} ; hence, f_{t+1} is the continuous posterior predictive distribution
308 for the upcoming interval after having observed the preceding intervals $1...t$.

309 To be able to relate trial-to-trial uncertainty and surprise to behavior and the
310 MEG data, we extracted two information theoretic metrics from the time-evolving
311 posterior predictive distribution f_{t+1} (i.e., belief).

312 *Uncertainty:* We quantified trial-to-trial uncertainty about the timing of the
313 upcoming interval $t+1$ as the entropy of the posterior predictive distribution f_{t+1} (i.e.,
314 the posterior predictive based on intervals $1...t$):

315
316
$$H_{t+1} = - \int_0^{\infty} (f_{t+1}(x) * \log f_{t+1}(x)) dx, \quad \text{Eq. 4}$$

317
318 where H_{t+1} is the entropy after intervals $1...t$, and the integral is over all possible
319 values x for the upcoming interval. Entropy depended on the width of f_{t+1} , and thus
320 uncertainty was higher when predictions of interval durations were less precise
321 (Figure 2A,C,D). For clarity, in what follows we will use the term entropy when
322 referring to this uncertainty.

323 *Surprise:* For every upcoming interval $t+1$, we computed the surprise about
324 the corresponding interval duration in terms of the Shannon information conveyed by
325 the interval duration x_{t+1} , given the posterior predictive distribution f_{t+1} :

326
327
$$I_{t+1} = -\log f_{t+1}(x_{t+1}), \quad \text{Eq. 5}$$

328
329 where I_{t+1} is the information gained by interval $t+1$, given f_{t+1} . Thus, surprise was
330 defined as the negative log-probability of the upcoming interval $t+1$, given the
331 intervals that had been presented so far.

332 We added one further transformation in the computation of surprise. The
333 surprise measure defined in Eq. 5 quantified the surprise about the upcoming event
334 timing based on the posterior predictive distribution f_{t+1} , but disregarding the time
335 elapsed in the current interval. It is unlikely that exactly this distribution translated into
336 subjects' level of surprise: as time passed and no event occurred in a given interval,
337 all interval durations shorter than the elapsed time become impossible. Subjects
338 likely discounted these impossible intervals in their expectation of the timing of the
339 upcoming event, which should have also affected their level of surprise. In other
340 words, their internal representation of the posterior predictive distribution changed
341 dynamically throughout each trial, as a function of elapsed time. We constructed a
342 time-varying version of the posterior predictive distribution f_{t+1} , which was also
343 conditioned on the elapsed time on interval t . This version was equal to f_{t+1} for
344 elapsed time equal to 0 and then increasingly deviated from f_{t+1} as elapsed time grew.
345 We approximated this time-varying distribution, denoted as f'_{t+1} in the following, by
346 setting all probabilities in f_{t+1} up to the current time point to zero and renormalizing the
347 remaining distribution to integrate to 1 (Figure 2B). We then computed surprise
348 based on this new distribution f'_{t+1} using Eq. 5. The time-variant prior f'_{t+1} converged
349 to 1 as time passed, and thus surprise approached zero for longer intervals.

350

351 *Regressing computational variables against behavior*

352 We used reaction time (RT) during Detection-button as behavioral readout of the
353 impact of uncertainty and surprise. Accuracy approached ceiling for all subjects, due
354 to the high saliency of the target. We computed and compared mean RTs per
355 environment and stimulus event (target off- and onset).

356 We also correlated RT to our trial-to-trial estimates of surprise and entropy.
357 RT was log-transformed so as to normalize the skewed RT distributions. To test if the
358 model-based surprise fitted the behavioral (RT) data better than a linear combination
359 of just the two previous interval durations (i.e., a leaky accumulation with strong
360 leak), we used multiple linear regressions to compare the following two nested
361 models:

362

$$363 \text{ M1: } \log(\text{RT}) \sim \text{Interval}_t * \text{Env} * \text{Event} + \text{Interval}_{t-1} * \text{Env} * \text{Event}$$

$$364 \text{ M2: } \log(\text{RT}) \sim \text{Interval}_t * \text{Env} * \text{Event} + \text{Interval}_{t-1} * \text{Env} * \text{Event} + \text{Surprise} * \text{Con} * \text{Event},$$

365

366 where Interval_t and Interval_{t-1} corresponded to the durations of the two intervals
367 preceding the visual change (i.e., interval on trial t and $t-1$), and Surprise was the
368 computational model-derived metric. Predictors were multiplied by categorical
369 variables environment (Env , the three different temporal environments) and Event
370 (target offset or onset). Both variables strongly affected RT (Figure 3A). We fitted
371 both M1 and M2 and compared the fits per subject using adjusted R^2 .

372

373 *MEG data collection*

374 Magnetoencephalography (MEG) data were acquired on a CTF 275 MEG system
375 (VSM/CTF Systems, Port Coquitlam, British Columbia, Canada) with a sample rate of
376 1200 Hz. The location of the subjects' head was measured in real-time using three
377 fiducial markers placed in the both ears and on the nasal bridge to control for
378 excessive movement. Furthermore, electrooculogram (EOG) and electrocardiogram
379 (ECG) were recorded to aid artifact rejection. All data were recorded in sets of four
380 blocks of three minutes duration (or two blocks at the end of an environment set).

381

382

383

384 *MEG data analysis*

385 *Preprocessing.* The data were analyzed in Matlab (version R2013a, The Mathworks,
386 Natick, MA, USA, RRID:SCR_001622) using the Fieldtrip (Oostenveld et al., 2011,
387 RRID:SCR_004849) toolbox and custom-made software.

388 *Trial extraction.* In blocks involving subjects' reports, we extracted trials of
389 variable duration, centered on subjects' button presses, from the 3 min blocks of
390 continuous stimulation. We call this method for trial extraction "response-locked". The
391 following constraints were used to avoid mixing data segments from different
392 percepts when averaging across trials: (i) The maximum trial duration ranged from
393 -1.5 s to 1.5 s relative to report; (ii) when another report occurred within this interval,
394 the trial was terminated 0.5 s from this report; (iii) when two reports succeeded one
395 another within 0.5 s, no trial was defined; (iv) for the analysis of Detection-button
396 blocks, we included only those reports that were preceded by a physical change of
397 the target stimulus within 0.2 to 1 s, thus discarding reports not related to stimulus
398 changes. We used this method for the analyses related to surprise. In an alternative
399 analysis of all Detection blocks, trials were defined in the same way as described
400 above, but now aligned to physical target on- and offsets ("stimulus-locked"). In the
401 Detection-count task, no button responses were given during the block, so stimulus-
402 locked trial extraction was the only option. We used this method for the analysis
403 related to entropy (see Kloosterman et al., 2015b & Meindertsma et al., 2017 for a
404 similar procedure).

405 *Artifact rejection.* All epochs that contained artifacts caused by environmental
406 noise, eye-blinks, muscle activity or squid jumps were excluded from further analysis
407 using standard automatic methods included in the Fieldtrip toolbox. Epochs that were
408 marked as containing an artifact were discarded after every artifact detection step.
409 For all artifact detection steps the artifact thresholds were set individually for all
410 subjects. Both of these choices aimed at optimization of artifact exclusion. Line-noise
411 was filtered out by subtracting the 50 , 100 , 150 and 200 Hz frequency components
412 from the signal.

413 *Time-frequency decomposition.* We used a sliding window Fourier transform
414 to compute the time-frequency representation for each sensor and each trial of the
415 MEG data. The sliding window had a length of 200 ms and a time step size of 50 ms,
416 with one Hanning taper (frequency range 5 - 35 Hz, frequency resolution 5 Hz and
417 frequency step size 1 Hz). The data was baseline corrected for every frequency bin
418 and MEG sensor separately. The baseline was computed by averaging single-trial
419 power over a baseline time window. The baseline time windows ranged from -1.25 to
420 -0.75 s for response-locked and -1 to -0.5 s for stimulus-locked analyses,
421 respectively. The time course of every frequency bin and sensor combination was
422 baseline corrected by subtracting the single-trial baseline power at that frequency
423 and dividing by the mean baseline power across trials within an experimental
424 environment. We used the single-trial baseline power for subtraction to eliminate the
425 effect of slow power fluctuations, because any surprise-related power modulations
426 could only have occurred after the sensory event that elicited surprise. We used the
427 mean baseline for division in order to minimize noise in the single-trial estimates of
428 the single-trial power modulation values. This division was used to compensate for
429 the common decay of power with frequency, which hinder identification of effects at
430 higher frequencies, and to normalize the single-trial modulation values (Siegel and
431 Donner, 2010). It did not systematically alter the association of power modulation
432 values with other variables.

433 *Source reconstruction.* We used an adaptive linear spatial filtering method
434 called linear beamforming (Van Veen et al., 1997; Gross et al., 2001) to estimate
435 single-trial modulations of MEG power at the source level. We computed a common

436 filter for a baseline time window (1 to 0.5 s before response), a ‘transient’ time
437 window, and a frequency band of interest (0 to 0.5 s after response, 20 Hz +/- 4 Hz
438 spectral smoothing, see dashed box in Figure 4A). The transient time window and
439 frequency band of interest were selected based on cluster-based statistics at the
440 sensor level (see next section). We used the measured head positions and individual
441 single-shell volume conductor models, based on individual images from T1-weighted
442 structural MRI. We computed the power values, in both baseline and transient time
443 windows, for each trial and source grid point (i.e., voxel) as follows. First, we
444 projected the sensor-level MEG power values from the time window of interest as
445 well as from a baseline time window through the common spatial filter. Second, we
446 converted the estimated power values during the time window of interest into units of
447 power modulation, again by subtracting and dividing by the corresponding baseline
448 power values.

449

450 *Correlating single-trial computational variables to MEG power*

451 We correlated the MEG power modulation to our measures of entropy and surprise,
452 as derived using our model (see *Bayesian ideal observer model: Implementation*)
453 across trials. Although intricately related (see Introduction), uncertainty and surprise
454 entailed different computations (see above). A key difference was *when* during the
455 course of a trial these two quantities were computed. So, we reasoned that neural
456 correlates of these computational quantities should also differ in their dynamics:
457 uncertainty about event timing should be reflected in the neural baseline state before
458 occurrence of the sensory event, whereas surprise should be reflected in a transient
459 response elicited by that event. Thus, we used different components of the single-
460 trial MEG power estimates for the analyses of entropy and surprise.

461 *Entropy:* We correlated entropy to the MEG power modulation separately in
462 every MEG sensor and frequency bin. This was done within subject and separately
463 for the three environments. There are structural differences in entropy and surprise
464 between these environments (Figure 2G,H), thus pooling over these conditions might
465 result in inflated correlations that reflect session differences instead of the true
466 correlation between entropy and MEG power. We reasoned that entropy should
467 affect baseline or tonic arousal, where high entropy should cause higher arousal. As
468 our task was continuous, we considered the time window right before the stimulus
469 change the best reflection of a baseline state. For this reason we averaged the MEG
470 power over the time period right before a stimulus change (-0.5 to -0.25s with respect
471 to the target offset or onset) before correlating to entropy.

472 The results were then averaged over the three environments and transformed
473 with the Fisher z transformation (Fisher, 1915):

474

$$475 \quad z = 0.5 \cdot \ln \left(\frac{1+r}{1-r} \right) \quad \text{Eq. 6}$$

476

477 We used two-tailed permutation tests with a cluster-based correction for
478 multiple corrections to test the correlation coefficients against zero (Efron and
479 Tibshirani, 1998; Maris and Oostenveld, 2007).

480 *Surprise:* Correlations between surprise and MEG power modulation were
481 performed using the same method, with the following exceptions. First, we attuned
482 the analysis in two ways to account for the correlation between surprise and RT
483 (Figure 2F, 3). Because of this correlation, any post-stimulus correlations between
484 surprise and MEG power modulation might reflect differences in the timing of the
485 button press. We performed this analysis response-locked, because these RT
486 differences are difficult to disentangle from genuine effects of surprise when the
487 power modulations are time-locked to the stimulus change. Additionally, to account

488 for confounding effects of RT and the duration of the previous interval, we also
489 performed a partial correlation analysis between surprise and MEG power
490 modulation with the interval duration preceding the stimulus change or RT as
491 covariate. Second, for the correlation between surprise and MEG power modulation
492 we did not average over a specific time window, but instead performed correlations
493 separately for every time point, resulting in a 3-dimensional matrix of correlations
494 (sensor * frequency bin * time point). Consequently, we also performed cluster-based
495 permutation statistics over these three dimensions. The correlations that survived
496 cluster correction were visualized by integrating (i.e. computing the area under the
497 curve) over sensors and frequency bins (for the time course), sensors and time
498 points (for the frequency spectrum), frequency bins and time points (for the
499 topography) or just over sensors for the time frequency representation (see Hipp et
500 al., 2012 for a similar approach).

501 To assess the robustness of the emerging clusters we performed a cross-
502 validation analysis using a leave-one-out procedure. To this end, we repeated the
503 cluster-based permutation statistics on all possible iterations of N-1 subjects, each
504 time using the resulting cluster as a mask to calculate the average correlation in the
505 left-out subject, separately for target offset and onset trials. These values were tested
506 against zero and against each other across subjects using permutation tests (10.000
507 permutations).

508 We also computed the correlation between trial-to-trial power modulation
509 averaged over the whole cluster and log(RT). The resulting correlations were tested
510 against zero across subjects using a permutation test (10.000 permutations).

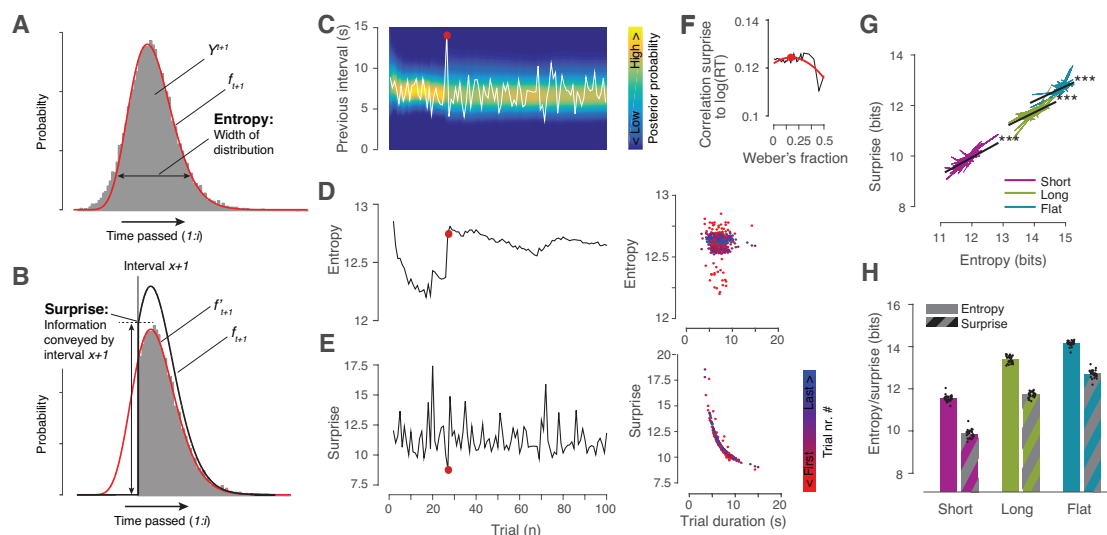
511 The transient modulations of MEG power estimated for each voxel in the
512 source grid, derived by means of source reconstruction (see *MEG data analysis:
513 Source reconstruction*), were correlated to the trial-to-trial measure of surprise. This
514 was done separately within each subject and the resulting correlations averaged over
515 subjects after Fischer's z-transformation (Eq. 6). For comparison, we also computed
516 the average modulations of MEG power in the same time window and frequency
517 band. The resulting maps of correlation or average power modulation were
518 nonlinearly aligned to a template brain (Montreal Neurological Institute) using the
519 individual images from structural MRI. To test the similarity of the spatial topography
520 of the correlation to the average modulation of power, we correlated the two
521 corresponding source maps per subject and tested the correlation coefficients again
522 zero on the group level by means of a permutation test (10.000 permutations).

523

524 **Results**

525 Subjects (N=28) performed a simple visual detection task reporting on- and offsets of
526 a small, but salient target stimulus (Figure 1A). In different blocks, target events were
527 administered using three different temporal environments (Figure 1B,C) translating
528 into different overall levels of uncertainty and surprise about the timing of target
529 events (Figure 2G,H). In order to quantify these two computational variables not only
530 across conditions, but also across individual trials, we developed a Bayesian belief-
531 updating model. The model incorporated the evolving beliefs (i.e. the posterior
532 predictive distributions) of an ideal observer about the temporal intervals between the
533 sensory events. Beliefs were dynamically updated across trials and even within trials
534 (for surprise, see *Materials and Methods*). From these time-evolving probability
535 distributions, we extracted trial-by-trial measures of information-theoretic entropy
536 (quantifying uncertainty) and surprise, which we related to the behavior and neural
537 dynamics of our participants.

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Figure 2: Bayesian updating model of belief about temporal structure. **A-F.** The model estimated the posterior predictive distribution over timings of stimulus changes for each upcoming interval $t+1$. This distribution is denoted as f_{t+1} . The gray histogram shows the distribution of MCMC-samples (Y^{t+1}) from the posterior predictive distribution for interval $t+1$. f_t was estimated by fitting a gamma probability density function (red line) to Y^{t+1} ; it was then used to extract two different information-theoretic computational variables for each trial: entropy and surprise. **A.** Entropy, a measure of the uncertainty about the timing of the interval duration of the current interval, computed from the complete distribution f_{t+1} using Eq. 4 (see main text). The wider the distribution, the higher entropy. **B.** Surprise, a measure of information provided by each new interval duration, was also computed from the posterior predictive distribution, but with one extra step (see main text): the part of the distribution up to the current interval duration was truncated, and the remainder of the distribution re-normalized to integrate to 1 (f'_{t+1} , black line). Surprise was defined based on this truncated function using Eq. 5 (see main text). **C.** Example sequence of interval durations (white line, from the long Gaussian condition) with posterior predictive distribution f (color coded). **D.** Entropy corresponding to interval durations in panel C (left); relationship between interval duration and entropy (right). **E.** Surprise, analogous to panel D. Red dot: example of exceptionally long interval (see duration in panel C). Surprise on this trial was low (panel E) because time dependent surprise decreased over time. After observing this interval entropy increased (panel D) because the observed interval was longer than the expected duration, given previous intervals. **F.** Correlation between $\log(\text{RT})$ and surprise as a function of different Weber fractions (black line, see Materials and Methods). Second-order polynomial fit over these correlations used to select the Weber fraction yielding peak correlation. (red line; red dot depicts peak = 0.17). **G.** Regression of surprise on entropy. Thin colored lines, regression lines of single subjects; black lines, group average regression. **H.** Trial-averaged surprise and entropy for the three experimental environments defined in Figure 1. Bars, group average; black dots, single subjects. *** $p = 0$ for all tests, permutation tests across subjects.

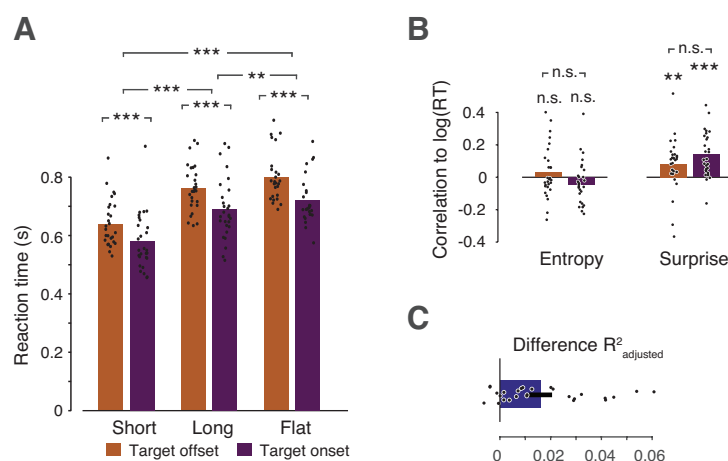
Estimates of entropy and surprise fluctuated across trials, especially in the early part of each block (Figure 2C-E). The trial averages of both measures within each block also varied lawfully between the different experimental conditions, scaling with the predictability of the stimulus changes (Figure 2G,H). Estimates were smallest for the Short condition, intermediate for the Long condition, and largest for the Flat condition. As expected, variations in entropy and surprise were weakly correlated across trials ($r=0.13$ for Short and Flat, $r=0.19$ for Long condition, Figure 2G), since both measures were computed from the same probability distribution (Materials and Methods). Even so, these two variables entailed distinct computations, possibly by distinct neural circuits. Critically, both computational variables could be computed at different times during a trial, thus possibly leading to different dynamical modulations cortical population activity.

581 *Surprise predicts reaction time*

582 The model-derived computational variable surprise predicted subjects' reaction time
 583 (RT) in the detection task. Mean RT scaled with the different temporal environments
 584 in the same way as surprise and entropy, with the fastest RTs for Short and slowest
 585 RTs for the Flat condition (Figure 3A, compare with Figure 2E). RT correlated with
 586 surprise also at the single trial level (Figure 3B). We did not find robust correlations to
 587 RT for entropy.

588 We also tested whether model-derived surprise (entailing accumulation of
 589 intervals across the entire experimental block) predicted RT over and above a linear
 590 combination of only the two previous intervals (entailing, e.g. a leaky accumulation
 591 with strong leak). To this end, we used a nested regression model, which quantified
 592 the predictive power of a combination of surprise and the previous two intervals in
 593 accounting for the influence of temporal environments and target on- or offset, on RT
 594 (Materials and Methods). We compared this against a simpler model with only the
 595 two previous intervals. Because model-based surprise depended on all previous
 596 intervals, the comparison between the above two nested models assessed the
 597 impact on reaction time of intervals beyond the second one. We used adjusted R^2 for
 598 comparison, which penalized model complexity. This comparison yielded higher
 599 adjusted R^2 values for the model including surprise in 22 of 28 subjects (Figure 3C),
 600 indicating that surprise predicted RT over and above the duration of the previous two
 601 intervals.

602 Taken together, these results indicate that subjects tracked the temporal
 603 structure of the task by accumulating interval distributions at least over more than two
 604 intervals, akin to what was prescribed by the ideal observer model. We next
 605 searched the whole-brain MEG data for a dynamical neurophysiological signature of
 606 this process. To this end, we focused on the trial-to-trial fluctuations of surprise within
 607 each of the environments (Short, Long, Flat), which were more pronounced than the
 608 differences in mean surprise between environments.
 609



610 **Figure 3: Link between computational variables and behavior.** **A.** Average reaction time (RT)
 611 per interval distribution, separate for reports of target offsets and onsets. Bars show average over
 612 subjects; black dots depict average per subject. *** $p < 0.001$, ** $p < 0.01$, permutation tests across
 613 subjects, 10,000 permutations (differences between target off- and onsets, p-values were $p=0$, $p=0$ and
 614 $p=0.003$, for Short, Long and Flat, respectively; between conditions, $p=0$, $p=0$ and $p=0.001$, for Short-
 615 Long, Short-Flat and Long-flat, respectively). **B.** Correlation between trial-to-trial fluctuations in Entropy
 616 (left)/Surprise (right) and log-transformed RT. Mean correlation coefficient Entropy: mean $r=0.03$
 617 (s.d.=0.02), $p=0.17$ and $r=-0.04$ (s.d.=0.03), $p=0.07$ for off- and onset, respectively, difference off-on
 618 $p=0.47$; Surprise: mean $r=0.09$ (s.d.=0.02), $p=0.007$ and $r=0.15$ (s.d.=0.03), $p=0$ for off and onset,
 619 respectively, difference off-on $p=0.48$. **C.** Model comparisons between partial (including only 2 previous
 620 intervals) and full regression models (further including surprise; see main text). Difference in adjusted
 621 R^2 . Positive values indicate support for full model. Dots depict individual subjects; bars depict the mean,
 622 error bars depict SEM.
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624 *Widespread cortical beta-band transient driven by surprise*

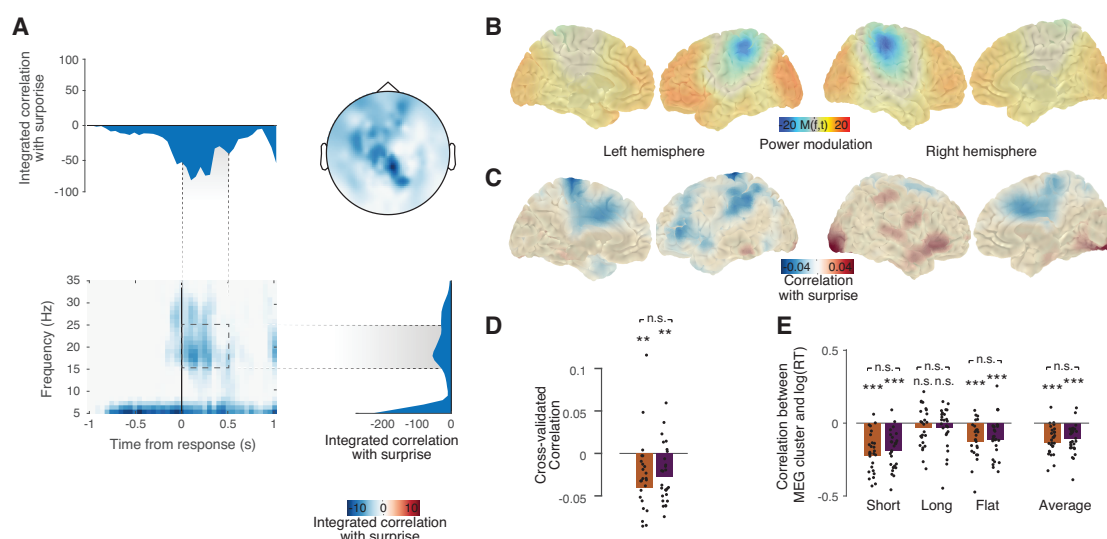
625 We mapped out the cortical responses to trial-to-trial fluctuations in surprise by
626 correlating the model-based surprise measures to modulations of MEG power,
627 around the time of subjects' behavioral responses to sensory events. We did this in
628 an exhaustive fashion across every time and frequency bin and MEG sensor and
629 tested for clusters of significant correlations across these three dimensions, while
630 applying cluster-based multiple comparison correction (*Materials and Methods*). This
631 approach revealed negative correlations in the beta (~20 Hz) frequency range, as
632 well as in the lowest frequency bin resolved (5 Hz), indicating that higher surprise
633 was associated with lower power in these frequency ranges. The peak in this
634 negative correlation cluster started about 0.2 s before and reached its maximum
635 about 0.25 s after subjects' report of the stimulus change. This cluster exhibited
636 several peaks over central, left frontal, and to a lesser extent left parietal cortex
637 (Figure 4A,C).

638 For all analyses shown in Figures 4 and 5, we used partial correlations,
639 controlling for reaction time, and we focused on the Detection-button task that
640 entailed immediate behavioral report of the change of the visual target (*Materials and*
641 *Methods*). We controlled for reaction time because (i) the data showed that the latter
642 was affected by surprise (Figure 3), and (ii) motor responses are known to modulate
643 beta-power around the time of response (Donner et al., 2009). Thus, button-presses
644 could have potentially influenced the modulation by surprise. We focused on
645 Detection-button because (i) we could only establish links between surprise and
646 behavior for this task and (ii) it allowed us to lock neural dynamics more closely to the
647 conscious registration of the visual change. When performing the correlation analysis
648 for the Detection-count task (then locked to the physical stimulus change), we did not
649 obtain any in significant correlation clusters. In two control analyses, we confirmed
650 that the above results were robust to (i) using the 'raw' correlation between surprise
651 and MEG power and (ii) controlling for the preceding interval duration. Both analyses
652 resulted in highly similar clusters of negative correlations (data not shown).

653 The surprise-related cluster was robust and not driven by outliers, and the
654 effect was not specific to the type of stimulus event (target on- or offset). We used a
655 leave-one-out cross-validation procedure to test the robustness of the correlations on
656 both target on- and offsets (*Materials and Methods*). We found robust negative
657 correlations in the left-out subjects (Figure 4D). Furthermore, the correlation was
658 found for both target offsets and onsets (Figure 4D, mean -0.036, -0.025, SEM 0.010,
659 0.006, $p = 0.006$, 0.002, for target offsets and onsets, respectively; difference:
660 $p=0.26$, permutation tests, 10,000 permutations).

661 As expected from previous work on modulations of MEG power around motor
662 responses (Donner et al., 2009), the overall modulation of MEG power in the time-
663 frequency window of the surprise-correlation cluster (16-24 Hz, 0–0.5 s from
664 response, normalized by the baseline 1-0.5 s before response) peaked in bilateral
665 motor cortex (Figure 4B). But the component of beta-power modulations that
666 correlated with trial-by-trial surprise showed a different cortical distribution, with
667 negative correlations that peaked in the central sulcus, extending from motor- to
668 more frontal cortex, and in left frontal and parietal cortex (compare Figure 4B and
669 4C). Indeed, there was no similarity between the individual topographies of the
670 surprise-linked and the overall power modulations (mean correlation across subjects:
671 $r=-0.02$, $p=0.42$). These observations indicate that the report-locked modulation
672 linked to surprise and of overall power were located in distinct cortical networks.

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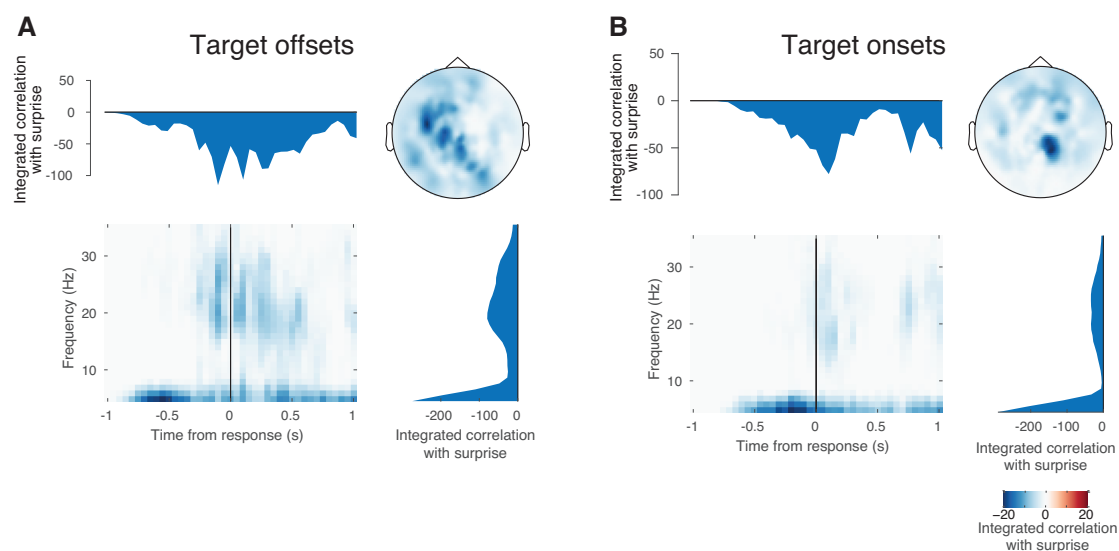
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Figure 4: Widespread cortical beta-band transient driven by surprise. **A.** Exhaustive partial correlation (controlling for RT) between trial-to-trial measures of surprise and MEG power modulation in all sensors, time and frequency bins results in one cluster (cluster-based correction for multiple comparison, $p = 0.001$, two-sided) of negative correlation. Different panels show different dimensions of the cluster by integrating over the other dimensions; top left: time course, top right: spatial topography, bottom left: time-frequency representation; bottom right: frequency spectrum. **B.** Source reconstruction of the power modulation in the time window in which surprise-MEG correlation was strongest (dashed box in panel A). **C.** Source-reconstructed illustration of the correlation between transient modulation and trial-to-trial surprise depicted in panel B. These source maps are not statistically thresholded, but instead serve for comparing the correlation's spatial distribution with the transient power modulation in panel B (average correlation between surprise and power modulation across subjects = 0.02, $p=0.42$). **D.** Leave-one-out cross-validation of the cluster found in panel A, separately for target offsets and onsets. Cluster-based permutation was performed on N-1 subjects and the average correlation in the resulting cluster was computed for the remaining subject (black dots); bars show averages over subjects. Correlation values were tested against 0 (permutation test; ** $p<0.01$; $p=0.005$ and $p=0.002$ for target offsets and onsets, respectively, $p=0.19$ for offsets-onsets difference). **E.** Correlation between MEG power in the cluster and log(RT) for separate distributions and average RT; permutation tests. All *** $p<0.001$, all offset-onset differences $p>0.05$ (lowest was $p=0.21$).

694 The surprise-related cluster for target offsets and onsets both exhibited a
695 bimodal in the frequency domain, similar to the pooled analysis (Figure 5; compare to
696 Figure 4A): next to the peak around 20 Hz just after response, an additional peak
697 was evident in the lowest frequency bin resolved (5 Hz). For offsets, the effect was
698 quite sustained in time (-0.25 to 0.5s around response); the topography showed
699 peaks over parietal and occipital cortex and over left frontal cortex (Figure 5A). By
700 contrast, the cluster for target onsets was more confined in time (with a sharp peak
701 ~ 0.1 s after report) and a different topography that peaked over central parietal cortex
702 (Figure 5B). Taken together, our results suggest that perceptual surprise about both
703 target on- and offsets elicited cortical transients in the beta-band. We consider them
704 general dynamical correlates of temporal surprise monitoring. In addition, stimulus
705 changes seem to have recruited additional processes expressed in the very low (\leq
706 5 Hz) frequency range.

707 Finally, we asked whether the trial-to-trial fluctuations in beta-power
708 modulations also predicted trial-to-trial variations in subjects' (log-transformed) RTs.
709 Here, we used the Pearson correlation values (i.e., without regressing out RT;
710 *Materials and Methods*). Just as surprise, beta-power in the cluster also robustly
711 predicted RT (Figure 4E). These correlations were negative, as expected based on
712 the negative correlation between surprise and MEG-power (Figure 4A). We also
713 compared the strength of this correlation between MEG-power and RT to the
714 strength of the correlation between surprise and RT across, this correlation between
715 correlations was positive, but not significant ($r=0.19$, $p=0.33$).

716 *No robust correlations between MEG baseline power and entropy*
717 We did not find any evidence for a correlation of the raw baseline (-0.5 to 0 s with
718 respect to stimulus change) MEG power with uncertainty, as measured in entropy.
719 Correlations between entropy and MEG power spectra in the time window before
720 stimulus change did not result in any significant (sensor-frequency) clusters that
721 survived multiple-comparison correction (data not shown). It is likely that this lack of
722 robust correlation reflected the continuous reduction in trial-to-trial variations of
723 entropy over the course of each block (Figure 2C).
724



725 **Figure 5: Separate correlations for offsets and onsets.** Correlation analysis and cluster-based
726 statistics performed separately for target offsets (A) and target onsets (B); $p=0.001$ for both analyses.
727
728

729 Discussion

730 In this study, we comprehensively mapped cortical transients elicited by surprise
731 about the timing of sensory events. We used a Bayesian updating model to estimate
732 trial-to-trial variations of surprise and correlated these to subjects' behavior as well as
733 to neural dynamics, across the cortical surface. The model-derived surprise
734 estimates predicted across-trial and environment variations in RT. The surprise
735 estimates also predicted transient suppressions of low-frequency and beta-band
736 power in a widespread network comprising motor-, prefrontal and parietal cortical
737 regions, predominantly in the left hemisphere. The model-derived surprise estimates
738 were more closely related to both behavior and cortical dynamics than the mere trial-
739 to-trial variations in externally observable interval timings.

740 The signatures of surprise we uncovered in the beta frequency band were
741 quite similar around target on- and offset (Figure 5). This stands in sharp contrast to
742 the opposite beta-band modulation during (illusory or veridical) target
743 disappearances and reappearances, proposed to reflect a decision-related feedback
744 signal to in visual cortex (Meindertsma et al., 2017). The beta-band transients
745 identified here likely reflected a distinct process that did not encode the content of the
746 perceptual change, but rather the level of surprise about it.

747 One possibility is that surprise is computed in those fronto-parietal cortical
748 networks exhibiting the surprise-related modulation of beta-oscillations observed
749 here. Another possibility is that the surprise-related modulations are inherited from
750 other regions projecting to those fronto-parietal networks. Indeed, neuromodulatory
751 brainstem systems are a prominent candidate source. In particular the dopaminergic
752 and noradrenergic systems are driven by temporal expectation and surprise (Aston-
753 Jones and Cohen, 2005; Dayan and Yu, 2006; Fiorillo et al., 2008). Further, there is

754 mounting evidence for a link between neuromodulation and beta-band power in
755 visual cortex (Belitski et al., 2008; Donner and Siegel, 2011; Safaai et al., 2015;
756 Zaldivar et al., 2018).

757 Specifically, phasic responses in dopaminergic nuclei, encode not only
758 reward, but also the expected timing of reward arrival. The strength of these phasic
759 neuronal responses inversely scales with predictability of the timing of reward, in line
760 with encoding surprise about reward arrival, and it also predicted behavioral
761 anticipation of reward (i.e. licking behavior) in monkeys (Fiorillo et al., 2008). Our
762 current study complements this previous work, by unraveling the cortex-wide
763 dynamics elicited by surprising events. Our design did not involve rewards but rather
764 neutral, yet behaviorally relevant sensory events.

765 In a previous report based on the same data set as the current one
766 (Kloosterman et al., 2015a), we showed that mean pupil dilation responses during
767 the perceptual changes scaled in amplitude across the three environments in line
768 with mean surprise as shown in the current Figure 2H. Pupil dilation is closely linked
769 to phasic responses in neuromodulatory brain systems, in particular the
770 noradrenergic locus coeruleus (Joshi et al., 2016; Reimer et al., 2016; de Gee et al.,
771 2017). Thus, if the surprise-related modulations of cortical activity observed here
772 were driven by phasic neuromodulation, one would expect to find correlations
773 between single-trial pupil responses and surprise (Preuschoff et al., 2011; Nassar et
774 al., 2012). Due to the sluggish dynamics of the peripheral pupil apparatus (Hoeks
775 and Levelt, 1993; De Gee et al., 2014), testing for trial-by-trial correlations between
776 pupil dilations and surprise (or, likewise, between baseline pupil diameter and
777 uncertainty) in our experiment requires dedicated analysis approaches that tease
778 apart fluctuating baseline levels and responses evoked by individual events. Using a
779 general linear model (Hoeks and Levelt, 1993; De Gee et al., 2014), we failed to
780 obtain reliable single-trial pupil responses and correlations to single-trial surprise
781 (data not shown). This failure was likely, at least in part, due to the rapid nature of the
782 current experimental design. Future work should use more widely spaced intervals to
783 test whether pupil dilations reflect trial-to-trial variations of surprise.

784 Our current study provides a comprehensive picture of the cortical transients
785 elicited by surprise, by systematically mapping these transients across the cortical
786 surface and time-frequency plane. Previous work in humans has also studied neural
787 correlates of model-derived measures of surprise, although this entailed surprise
788 about stimulus identity, and not timing. Electrophysiological work found surprise
789 about cue identity to modulate the P3 component of the EEG event-related potential
790 as well as motor cortical excitability (Bestmann et al., 2008; Mars et al., 2008).
791 Functional magnetic resonance imaging work linked surprise about the spatial
792 location of stimuli to transients in posterior parietal cortex (O'Reilly et al., 2013). An
793 EEG study dissociated oscillatory neural signatures of surprise and evidence
794 accumulation (Gould et al., 2012). This latter study also found surprise-related
795 modulation of beta-band power primarily at frontal and parieto-occipital electrodes,
796 but the underlying cortical distribution was not estimated. Future studies of surprise in
797 other domains (e.g. about cue identity) should use a similar approach to assess if
798 surprise-related cortical transients are domain-general or -specific. Further,
799 simultaneous EEG and MEG recordings (Schurger et al., 2015) are necessary to
800 unravel the relationship between surprise-linked modulations of fronto-parietal beta-
801 band oscillations and of the P3-component.

802 Another line of work has investigated the functional role of externally
803 entrained low-frequency oscillations in temporal expectation. For fixed intervals,
804 alpha phase in sensory cortices was found to be predictive of expected time of target
805 arrival and lowered the threshold for sensory detection (Lakatos et al., 2008; Cravo et

806 al., 2011, 2013; Rohenkohl and Nobre, 2011). Alpha oscillations might reflect
807 rhythmic fluctuations in cortical excitability, entrained by rhythmic sensory input,
808 which aids stimulus processing and perceptual performance (Schroeder and Lakatos,
809 2009). The high variability in interval durations (see Figure 1B,C inset) might explain
810 the lack of alpha-band effects in our study. First, the range of possible durations was
811 too broad to form predictions that fall within a specific phase of an alpha cycle.
812 Second, even when oscillatory phase was modulated by temporal expectation in our
813 task, the trial-to-trial variability would make it difficult to align trials and make these
814 modulations visible.

815 It is tempting to relate our results to conceptual accounts of the functional role
816 of beta-band oscillations in the brain (Engel and Fries, 2010; Spitzer and Haegens,
817 2017). One account (Engel and Fries, 2010) holds that beta-band oscillations help
818 maintain the current sensorimotor or cognitive state (termed the ‘status quo’).
819 Another account (Spitzer and Haegens, 2017) holds that beta-band oscillations help
820 activate the currently relevant task sets. In both frameworks, the need for maintaining
821 the current status quo, or task set, is low when surprise (the violation of expectation,
822 or probability of change in the environment) is high, in line with our observation of a
823 suppression of beta-band oscillations under high surprise.

824 While our current work presents an important first step towards unraveling the
825 modulation of cortical dynamics by surprise, it is limited in that we only studied
826 environments with constant statistical structure within each block. Once a posterior
827 distribution has been learned, there remains no unexpected uncertainty, only
828 expected uncertainty (Yu and Dayan, 2005). By contrast, the statistical structure of
829 natural environments is often volatile. Richer experimental designs, that are volatile
830 and include unmarked changes, allow for probing into richer, presumably hierarchical
831 dynamics (Sugrue et al., 2004; Nassar et al., 2012; Meyniel et al., 2015). A more
832 volatile task-environment would also lead to an increase in trial-to-trial variability of
833 our entropy measure, providing a better-suited context to study the effects of this
834 type of uncertainty on cortical processing. Our ongoing work aims to push beyond
835 these limits by using richer environmental statistics that require more complex
836 inference processes.

837 To conclude, we here uncovered a novel signature of temporal surprise that
838 affected an elementary perceptual decision (target detection) and was characterized
839 by a temporally focal, but spatially widespread, modulation of cortical population
840 activity. This modulation might be instrumental in translating inferences about the
841 behaviorally-relevant temporal structure into its consequences for action.

842

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