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Age-related decline in executive function as a hallmark of cognitive ageing in primates: an overview of cognitive and neurobiological studies

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Executive function (EF) is a complex construct that reflects multiple higher-order cognitive processes such as planning, updating, inhibiting and set-shifting. Decline in these functions is a hallmark of cognitive ageing in humans, and age differences and changes in EF correlate with age-related differences and changes in association cortices, particularly the prefrontal areas. Here, we review evidence for age-related decline in EF and associated neurobiological changes in prosimians, New World and Old World monkeys, apes and humans. While EF declines with age in all primate species studied, the relationship of this decline with age-related alterations in the prefrontal cortex remains unclear, owing to the scarcity of neurobiological studies focusing on the ageing brain in most primate species. In addition, the influence of sex, vascular and metabolic risk, and hormonal status has rarely been considered. We outline several methodological limitations and challenges with the goal of producing a comprehensive integration of cognitive and neurobiological data across species and elucidating how ageing shapes neurocognitive trajectories in primates with different life histories, lifespans and brain architectures. Such comparative investigations are critical for fostering translational research and understanding healthy and pathological ageing in our own species.

This article is part of the theme issue 'Evolution of the primate ageing process'.

1. Introduction

Living organisms adapt to their environment through processing information [1,2]. Nuanced control over information processing operations plays an important role in fostering efficient adaptation of individuals and species. In primates that must adapt to a wide variety of environments, such nuanced and precise control over cognition may be crucial for success. Executive function (EF), a construct that encompasses multiple aspects of cognitive control, exhibits significant age-related differences and may constitute a fundamental feature of human cognitive ageing. It remains unclear, however, whether other primates show age-related declines in EF. Comparative studies can illuminate features of cognitive and brain ageing that are unique to the human lineage versus those shared with other primates. Considering EF and its neural substrates, this comparative approach is particularly important. Primate species occupy distinct ecological niches and have diverse social structures. They may, therefore, have evolved qualitatively different EF

abilities [3], along with differential patterns of cortical organization. Yet, the patterns of brain and cognitive ageing may be conserved across primate orders, suggesting that general principles underlie the diversity of brains and cognitive abilities and their temporal dynamics. Below we describe patterns of age-related decline in EF and associated brain structures in humans and non-human primate (NHP) models of human cognitive ageing.

2. Humans

The importance of cognitive control for effective information processing is widely acknowledged [4,5]. The term 'executive functions' was introduced into the discussion of purposeful control of behaviour in the 1970s [6], with the prefrontal cortex (PFC) conceptualized as the prime brain substrate of EF. The idea of frontal cortex supremacy in cognition dates at least to the early nineteenth century, when Gratiolet crowned the frontal cortex as the most important region of the human brain signifying its supreme power (…*la plus importante région du cerveau humain le signe de la puissance souveraine*… [7, p. 64]).

EF is a multifactorial construct encompassing many cognitive skills and abilities, such as inhibition of irrelevant information, storing and simultaneously manipulating items in short-term memory, updating information during action, planning future actions, adjusting behaviour in response to changes in incentives, and shifting attention across tasks—to name a few [8]. Attempts at validating the EF construct via confirmatory factor analysis (CFA) revealed multiple EF sub-constructs—*shifting*, *monitoring and updating* of streaming information, and *inhibition* of prepotent responses—that were 'moderately [inter]correlated…but…clearly separable' [9, p. 49]. Such findings led to a proposed pattern of 'unity and diversity,' in which EF components are related but distinct [10].

Therefore, classic neuropsychological tests of EF, such as the Wisconsin Card Sorting Test (WCST)[1], Stroop task[2], Tower of Hanoi[3] or N-back task[4], assess inter-related cognitive skills and cannot be viewed as mere indicators of prefrontal integrity and functionality [11]. Nonetheless, EF indicators have been associated with PFC integrity in lesion studies [12,13] and with PFC volume in structural neuroimaging investigations [14], suggesting that 'bigger is better'. Meta-analyses of functional MRI studies also revealed reliable activation of this region during the WCST [15], Stroop [16] and working memory (WM) tasks [17].

Negative associations between age and EF in healthy adults are well established. Older adults perform worse than their younger counterparts on EF tasks such as WM, WCST, Stroop and dual-task management [18,19]. Age-related declines in EF are also evident in longitudinal studies [20,21], and have been related to changes in the integrity of prefrontal areas. The lateral PFC shrinks at a rate of about 5% per decade [22]; the adjacent prefrontal white matter loses volume too [23,24]. Many studies found moderate associations between regional volumes of the PFC and EF performance [25]. Functional neuroimaging studies have also linked poor performance on EF indicators (e.g. WCST) and reduced activation within the PFC in older people [26,27].

Some authors have argued that EF dysfunction is a fundamental feature of human cognitive ageing (e.g. [28]). However, it is important to determine the extent to which other primates also exhibit age-related declines in EF and associated neural substrates. Cognitive ageing investigations have used several NHP species, based on close phylogenetic proximity to humans (chimpanzees, macaques), unique characteristics such as a short lifespan (grey mouse lemurs, marmosets), or likelihood of specific brain pathologies (chimpanzees, grey mouse lemurs). A comparative perspective, which contrasts patterns of neurocognitive ageing in primates with different brain organizations, life histories and lifespans, may foster identifying biological factors associated with cognitive ageing and revealing potential targets for therapeutic intervention against age-related cognitive decline in our own species.

3. Non-human primate studies: rationale and methods

NHPs share many neuroanatomical, physiological and behavioural characteristics with humans that make them invaluable for translational research [29,30]. NHPs provide further advantages for cognitive research because they perform in settings that closely match human testing conditions. Various test batteries adapted to NHPs capture the cognitive domains traditionally assessed in human neuropsychological studies, such as WM, set-shifting, and other EFs. These tests can be administered by a human experimenter who manually presents physical stimuli that the monkey may displace in order to receive a reward, most often in the context of a Wisconsin general testing apparatus (WGTA, [31]). More controlled automated systems, in which monkeys obtain a reward by responding via joystick or touch to stimuli displayed on a computer screen have replaced the WGTA over time. Studies of humans with focal brain damage [32] and NHPs with brain lesions [33] have allowed the mapping of cognitive processes onto specific neural structures and systems. As early as 1936, Jacobsen [33] reported that rhesus monkeys with bilateral prefrontal lesions exhibited severe impairment in delayed-response (DR) performance, a task in which they had to remember the position of a reward after various delays. The neurophysiological studies that followed demonstrated that neurons in the dorsolateral PFC (dlPFC) were active during the delays and encoded the location of the cue or the direction of the response, establishing the DR as a benchmark task for assessing WM in NHPs [34]. These seminal studies greatly influenced the field of cognitive ageing, which adopted the framework that age-related decline in a particular cognitive domain reflects compromised integrity of the underlying brain tissues [35–37]. Today, multiple neuroscience tools, such as *in vivo* neuroimaging, electrophysiology and post-mortem histological studies can be combined with cognitive assessments to uncover the neural bases of age-related cognitive decline. Most recent technological breakthroughs, which allow the monitoring and manipulation of neural activity in behaving animals, have considerable potential for advancing cognitive ageing research, but are still at various stages of development in NHPs [30].

4. The rhesus macaque

The majority of studies conducted in ageing NHPs have relied on the genus *Macaca*, primarily rhesus (*M. mulatta)*, with some studies conducted in cynomolgus (*M. fascicularis*) or bonnet macaques (*M. radiata*). Another useful Old World primate model for ageing and Alzheimer's disease (AD) [38] is the

vervet monkey (*Chlorocebus aethiops*). However, since data on age-related cognitive decline are not yet available in this species, the following section focuses on macaque data.

Rhesus monkeys can live up to 40 years, but have a median lifespan of 27 years and are considered old by the age of 20. The extant literature reveals close parallels between macaques and humans in cognitive and brain ageing [39–43]. Compared with younger animals, aged rhesus monkeys show reliable delay-dependent decrements in WM assessed by performance on the DR task [44,45]. Aged rhesus monkeys also show impaired cognitive flexibility, traditionally assessed via reversal learning or set-shifting tasks (e.g. WCST). In reversal learning, monkeys learn which of two stimuli is associated with a reward (discrimination), followed by a reversal, in which the reward becomes associated with the previously incorrect stimulus. Adjustment to reversals takes more trials in aged monkeys than in younger animals [39,44,46,47]. The former perseverate, i.e. continue following an established prepotent response despite changing task requirements. Perseverative errors are also observed in a modified version of the WCST [48], in which old rhesus monkeys (24–30 years) require more time than young adults (5–10 years) to learn the shifts. Importantly, these age differences are already observed in middle-age animals (12–19 years), suggesting that decline in EF is one of the earliest cognitive changes in normal primate ageing [49].

Area 46 of the dlPFC plays a critical role in EF and has been extensively studied in the rhesus monkey. As the dlPFC shows no neuronal loss [50], the thinning of grey matter (13%) in this area [51] may reflect loss of neuropil—dendrites and dendritic spines [52]. In addition, the dlPFC exhibits substantial age-related alterations in myelin [53] and white matter [54]. Alterations of the gross PFC structure in aged rhesus monkeys do not correlate with age-related declines in WM, as assessed by the DR [55]. Rather, impaired DR performance with age has been associated with reduced persistent firing of delay neurons in area 46 of the dlPFC [56] and with spine loss in the dlPFC [57]. Although one study reported that WCST scores in aged rhesus monkeys correlated with decrements in grey matter volume, including the PFC [58], more subtle neurobiological changes are likely to contribute to EF declines with age. For example, WCST performance correlated with decreases in α-1 and α-2 adrenergic receptor binding in the PFC in another study [59]. Notably, factors such as sex [60], hormonal status [61] and arterial hypertension [62] all influence EF in rhesus monkeys but are often overlooked in neurobiological studies.

In sum, macaques display age-related declines in EF starting at middle age. Alterations in the composition of the PFC neuropil rather than loss of neurons probably drive these age-related declines.

5. The grey mouse lemur

The grey mouse lemur (*Microcebus murinus*), endemic to Madagascar, is one of the smallest primates. Its average lifespan is around 3 years in the wild and 5–7 years in captivity, with maximum age of up to 14 years [63]. Following discoveries of age-related proteopathies, including β-amyloid [64] and tau [64,65], as well as cerebral atrophy [64] and behavioural and cognitive changes [66] in the mouse lemur, the histochemical, structural and cognitive/behavioural aspects of brain ageing in this species received great attention [67–69].

Using a test battery and a corridor apparatus designed for mouse lemurs, studies of age-related changes in EF demonstrated that old individuals (7–11 years) performed worse than young animals (2–4 years) in extra-dimensional set-shifting and in pairwise spatial discrimination reversal learning, with no age differences in a go/no go successive visual discrimination task [70]. The age-related deficits in extradimensional set-shifting and spatial reversal learning were later replicated [71], along with an impairment of pairwise visual discrimination reversal learning [71] also found in a touchscreen-based version of the task [72]. Age-related WM deficits are also apparent in this species. A small study comparing young (2–4 years) and old (9–10 years) animals on a DR task, showed more drastic performance decrements with increasing delay in aged individuals [73]. A linear age-related deficit in spontaneous alternation behaviour during exploration of a four-armed maze was interpreted as an age-related decline in spatial WM [74]. Notably, mouse lemurs show substantial individual differences in cognitive ageing. The aged cohorts, similar to those of humans, include individuals that age healthily, along with animals with age-related cerebral and cognitive pathologies [71,72].

While a possible linkage of EF decline and structural/cytochemical alterations of prefrontal cerebral structures in aged mouse lemurs has been postulated [70–72], there is little empirical evidence for such a link. Lesion studies or pharmacological intervention experiments targeting prefrontal function and cognitive performance are absent. *In vivo* MRI studies revealed differential patterns of structural brain ageing in lemurs. These investigations showed age-related ventricular expansions and atrophy in temporo-parietal areas in individuals older than 3.5 years [75]. As in humans, the pathological process progresses gradually, often starting in frontal brain regions of middle-aged individuals (3–6 years) and spreading to temporo-parietal and finally occipital areas [76]. Preliminary analysis from the same study also suggests concomitant intracellular amyloid accumulation [76]. As in humans, regional volumes and cortical thickness of the temporal and hippocampal regions showed the strongest association with age. However, in contrast to humans, significantly reduced volume and thickness were noted in occipital and cingulate regions, whereas the fronto-parietal areas appeared age-invariant (1–11 years, $n = 34$; [71]). The age differences in the cingulate, temporal and occipital regions were replicated in subsequent studies using manual delineation of brain areas [77], and automated voxel-based [78], or 3D-MRI atlas-based morphometry [79]. In addition, in line with human findings, marked age-related atrophy of the caudate nucleus was consistently reported [71,77–79]. However, age-related alterations of the premotor cortex and the superior parietal lobule were also described [77,79]. Finally, automated methods revealed age-related atrophy in an area that corresponds to Brodmann areas 13–16, believed to be functionally equivalent to prefrontal regions in humans [78,79]. Interestingly, the study by Picq and collaborators [71] also provided EF data for a subset of individuals (2–8 years, $n = 16$). A composite measurement of set-shifting and reversal learning correlated with the volume of the septal region, the caudate nucleus, and the splenium of the corpus callosum, as well as with the thickness of the anterior and posterior cingulate cortices and the associative visual cortical areas, suggesting overlap in EF circuitry between mouse lemurs and humans.

In summary, extradimensional set-shifting and reversal learning are consistently impaired with age in the mouse lemur, with moderate evidence for age-related deficits in WM. Additional studies are needed to clarify the neural basis of these changes.

6. The common marmoset

The common marmoset (*Callithrix jacchus*) is a small (300–500 g) New World monkey from the Callitrichinae subfamily and is endemic to Brazil. Its average lifespan in captivity is about 10 years, with a maximum of 21 years [80]. Marmosets are considered old by the age of 8, when first signs of ageing such as cortical β-amyloid deposition [81], reduced neurogenesis in the dentate gyrus [82], and presbycusis [83] are observed [84]. Owing to their small size and short lifespan, marmosets make an excellent model for ageing research [84–86]. Yet, marmosets have only recently been included in cognitive ageing investigations. A small study comparing reversal learning performance of four aged marmosets (10–14 years) with historical records from a younger cohort (1.7–3.5 years) reported more errors in aged animal for the initial discrimination and reversal but noted that this difference disappeared with further testing [87]. A larger study of 35 marmosets (2–14 years) found that aged monkeys exhibited deficits in reversal learning and delayed matching-to-position [88], with two critical periods identified for the emergence of deficits: a reduced ability to update WM information across trials was observed around 4–4.5 years of age, while difficulty with inhibitory control emerged around the 6–7th year.

To date, studies of ageing in the marmoset, like most investigations of ageing in all species, have been cross-sectional and therefore unable to determine whether neural or physiological changes precede or follow the decline in cognitive function. However, the short lifespan of the marmoset makes feasible longitudinal investigations of changes in individual animals undergoing the ageing process. In an ongoing study [89,90], 28 marmosets were followed longitudinally as they aged from 4–5 to 8–9 years. The animals performed reversal learning for 4 years using different stimuli each year. Reliable deficits emerged by year 4 in both the discrimination and the reversal portions of the task. In addition, sex differences in ageing trajectories were revealed, with females showing greater decline than males. Finally, prominent individual differences indicated pathological ageing in some individuals. These preliminary data suggest EF declines with age in marmosets and add longitudinal findings to the extant literature. In addition, they highlight a major influence of sex on age-related decline in EF that was overlooked in most NHP studies.

Being amenable to gene editing [91] bolsters marmosets' importance as a model for neuroscience research [92,93], and their use in neuroimaging, neurophysiological and neurobiological studies has increased in recent years. Yet, studies focusing on brain ageing in this species are still in their infancy. The marmoset brain exhibits increased β-amyloid deposition [81,94] and abnormally phosphorylated tau [94] with age. Decreased neurogenesis is also observed in the dentate gyrus of older animals [82]. Liu *et al*. [95] reported age-dependent reductions in whole brain and cerebral white and grey matter volumes in the marmoset brain, without providing region-specific analyses. A small study [96] investigating myelin composition in brain tissues of four aged and two young marmosets reported reduced myelin thickness, density and fraction in older age. Age-related myelin changes in the genu of the corpus callosum, which contains fibres connecting homologous PFC regions of the hemispheres, correlated with performance on the detour reaching task, a task of PFC-dependent inhibitory control.

Collectively, the extant data suggest age-related EF impairment in the marmoset, with more prominent declines observed in females. More research is needed to characterize brain ageing in this species and fully grasp the relationships between age-related neural changes and declining EF performance in each sex.

7. The chimpanzee

In captivity, the chimpanzee (*Pan troglodytes*) has a life expectancy of 28.3 years at birth, with an estimated maximum lifespan of 74 years [97]. Among NHPs, the chimpanzee has the longest lifespan, the largest brain and PFC [98], and cognitive abilities closest to those of humans [99]. Very little is known about cognitive ageing in chimpanzees. An early study in eight young (11–19 years) and eight old (28–40 years) chimpanzees of unspecified sex found no age differences in a series of object discriminations and a wheel-rotating task [100]. Later investigation in 19 chimpanzees (7–41 years) of unspecified sex revealed no age-related deficits in discrimination tasks but identified an age-related impairment at the shortest retention delays (less than 5 s) in the DR and in an oddity task [101]. After a pause of nearly half a century, research on chimpanzee cognitive ageing resumed with three studies providing evidence for age-related decline in the chimpanzee, especially in the EF domain. In the first study, 38 female chimpanzees (10–52 years) were tested over a period of 3 years on the primate cognition test battery (PCTB) [102], a battery originally designed to compare social and physical cognition in apes and humans [103]. Age-related performance decrements were observed in two tasks of social cognition (attention-getting behaviour and gaze following), with minimal age effects found for tasks of physical cognition (tasks of causality, quantity and spatial processing). However, the task of spatial WM (DR with minimal delay) showed performance decline in the older females (greater than 50 years) over the course of the study, suggesting that such deficits may develop at oldest ages. More recently, the PCTB was administered to a large sample of chimpanzees at the National Center for Chimpanzee Care (MD Anderson Cancer Center). When these data were combined with previously published data from the Yerkes Primate Center [104], DR scores were available in 223 individuals (9–54 years). No significant linear association was found between age and DR performance, but a significant quadratic trend was observed, with younger (9–20 years) and older individuals (≥40 years) performing worse than middle-aged chimpanzees (21–39 years). Similar findings were reported in a study that used a motor task to assess EF in five species of apes, including chimpanzees [105]. The participants slid a horizontal door to the left or to the right in order to obtain a reward. Following acquisition of the task, the response requirement was reversed. Both young and old apes made more errors in the reversal phase than the middle-aged ones, suggesting a developmental trajectory and an age-related decline in reversal learning performance. Finally, a recent study used the WCST to evaluate cognitive flexibility in 30 female chimpanzees (12–56 years old; [106]). The number of trials required for

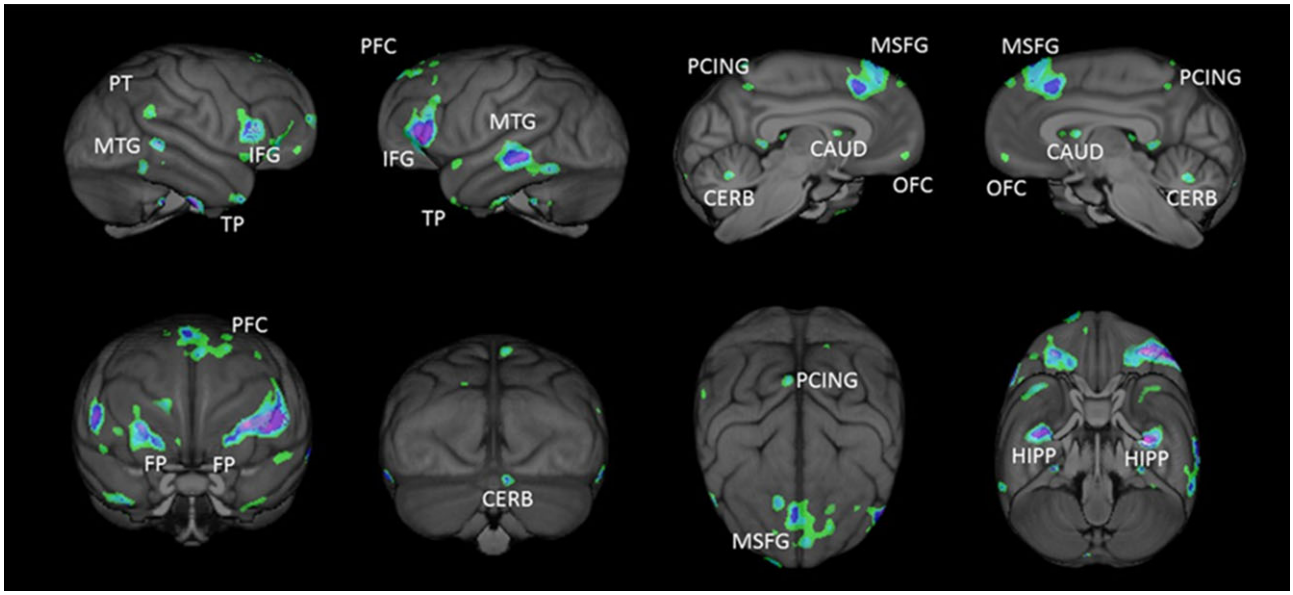

Figure 1. VBM analysis of age-related decline in grey matter volume ($p < 0.001$, uncorrected) in the chimpanzee brain ($n = 216$). The coloured regions reflect those brain areas that have reductions in grey matter volume with increasing age. PFC, prefrontal cortex; PT, planum temporale; FP, frontal pole; MSFG, medial superior frontal gyrus; MTG, middle temporal gyrus; TP, temporal lobe; IFG, inferior frontal gyrus; HIPP, hippocampus; PCING posterior cingulate; CAUD, caudate; CERB, cerebellum; OFC, orbitofrontal cortex. W. D. Hopkins, unpublished data.

each dimension increased with age and the number of perseverative errors was significantly higher in middle-aged and older chimpanzees than in younger ones.

In contrast to the behavioural results, *in vivo* and *ex vivo* MRI scans of chimpanzee brains reveal modest age-related changes. Sherwood and colleagues [107] measured total grey and white matter volume, as well as frontal lobe grey and white matter volume in a sample of 99 chimpanzees and 87 humans. Linear and cubic associations between age and grey and white matter volume for the entire neocortex and the frontal lobe were observed in humans. By contrast, no significant associations were found between age and any of the measures of total neocortical or frontal lobe grey and white matter volume in the chimpanzee. In a subsequent study of female humans ($n = 178$), chimpanzees ($n = 32$) and rhesus monkeys ($n = 20$), age was associated with smaller total grey matter volume in all three species [108], but no decreases were observed in white matter volume of the chimpanzees or macaques. In these studies, the chimpanzee samples included relatively few old animals (age ≥40 years). A later MRI study on 219 chimpanzees that included 38 older individuals [109] found little evidence for age-related differences in total grey and white matter volume, overall gyrification, and grey matter thickness. However, the brains of old chimpanzees were less gyrified than those of sub-adults and young adults, and white matter volume increased earlier in life but decreased in later years.

Voxel-based morphometry (VBM) of age-related changes in grey matter volume of 216 chimpanzees (9–54 years) revealed negative associations with age in some regions, with the strongest correlations found in the inferior frontal gyrus, left dlPFC, bilateral superior medial cortex, middle temporal gyrus, orbitofrontal cortex, right frontal pole and right inferior parietal cortex (figure 1). In another study, cortical thickness, surface area and local gyrification in 34 brain regions were estimated using Freesurfer [110] after the chimpanzee brain scans ($n = 77$) were warped in the human template. Only few associations between age and surface area were found; however, a number of negative associations between age and cortical thickness and gyrification were revealed, the most prominent within the frontal lobe. Thus, VBM analysis may be more likely to reveal age-related differences in the chimpanzee brain than gross volumetric measures.

In summary, three recent studies have provided evidence for age-related decline in EF in the chimpanzee, with some of the deficits emerging at middle age. However, the neural substrates underlying these age differences remain unclear, as imaging studies have revealed only modest age-related differences in the chimpanzee brain.

8. Conclusion

Prominent EF declines that characterize ageing in humans have been documented in all other primates studied so far. The ubiquity of these changes across primate orders, in species with widely different life histories, lifespans and brain architectures, suggests that age-related declines in EF are a fundamental feature of primate cognitive ageing. Furthermore, several observations suggest that EF decline represents one of the earliest age-related cognitive changes in humans, rhesus macaques and chimpanzees. Thus, EF is a useful construct and a potential early marker of age-related cognitive decline across primate species. More research is needed to precisely identify the neural bases of such cognitive changes. Age-related atrophy of the PFC, which is associated with age-related EF decrements in humans, appears to be absent in other NHPs, including chimpanzees. Instead, age-related changes in white matter and more subtle changes at the level of neurotransmitters, synaptic density and neuronal firing may underlie age-related EF declines in NHPs. As the majority of these neurobiological data come from macaques, it remains unclear whether species differences in primate brain ageing reflect differences in longevity, ecological niche or other factors.

Limitations that characterize NHP studies suggest caution in interpretation of the findings. Mitigating these limitations is crucial for advancing the understanding of primate cognitive ageing. First, there are few studies integrating cognitive and brain ageing assessments in NHPs. Second, small sample sizes, lack of sex comparisons and study of selective age ranges may lead to inaccurate or incomplete results. Low statistical power, typical in NHP research, precludes discovery of moderate or small changes and differences, which may be theoretically meaningful. Therefore, larger datasets are needed for a better understanding of the mechanisms of neurocognitive ageing in NHPs and in humans. Study of primate species with a shorter natural life expectancy (e.g. marmosets or mouse lemurs) may promote the creation of larger datasets with longitudinal measurements in both sexes. Third, noninvasive neuroimaging studies of NHPs, particularly great apes, are necessary for interpreting human studies in an evolutionary context. Such studies should employ methodologies similar to those used in humans. Fourth, given the prominent role of vascular and metabolic risk factors in human brain and cognitive ageing [111], more attention should be paid to these factors in NHP investigations. Specifically, publications should include detailed descriptions of diet, food restriction, activity opportunities and adiposity. Fifth, studies of NHPs should rely less upon single tasks to evaluate EF, and include multi-indicator, construct-based approaches. Such strategy would capitalize upon the rich findings that have emerged from studies of human cognitive and neural ageing.

Ethics. This article provides data collected in chimpanzees. The animals were cared for in accordance with the guidelines of the US National Research Council's Guide for the Care and Use of Laboratory Animals, the US Public Health Service's Policy on Humane Care and Use of Laboratory Animals, and the Guide for the Care and Use of Laboratory Animals (2011), 8th edition. All procedures used with the chimpanzees were approved by the local institutional animal care and use committees.

Data accessibility. The VBM analysis revealing the age-related changes in grey matter volume were performed on magnetic resonance images (MRI) available in the National Chimpanzee Brain Resource (www.chimpanzeebrain.org).

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Competing interests. We declare we have no competing interests.

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Endnotes

[1]Wisconsin Card Sorting Test: a test of set-shifting and inhibition of prepotent responses where participants have to sort cards according to changing rules according to colour, number or shape.
[2]Stroop task: a task in which participants view colour names written in congruent or incongruent colour and report the colour of the word, not the word itself. The task is considered a test of inhibition and response selection.
[3]Tower of Hanoi: a task of planning. Participants have to solve a puzzle in the shortest time while using a minimal number of moves. The planning of moves involves thinking ahead and using recursive approach.
[4]N-back task: a task of working memory. Participants view a sequence of stimuli, one-by-one. For each stimulus, they have to determine if the current stimulus is the same as the one presented *N* trials beforehand.

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