PHILOSOPHICAL TRANSACTIONS B

royalsocietypublishing.org/journal/rstb

Review

Cite this article: Campos FA *et al*. 2024 Wild capuchin monkeys as a model system for investigating the social and ecological determinants of ageing. *Phil. Trans. R. Soc. B* **379**: 20230482.
https://doi.org/10.1098/rstb.2023.0482

Received: 22 December 2023
Accepted: 10 May 2024

One contribution of 16 to a discussion meeting issue 'Understanding age and society using natural populations'.

Subject Areas:
behaviour, ecology, evolution, health and disease and epidemiology, physiology

Keywords:
Cebus, gerontology, healthspan, lifespan, nonhuman primate model, noninvasive sampling

Author for correspondence:
Fernando A. Campos
e-mail: fernando.campos@utsa.edu

Wild capuchin monkeys as a model system for investigating the social and ecological determinants of ageing

Fernando A. Campos[1], Eva C. Wikberg[1], Joseph D. Orkin[3,4], Yeonjoo Park[2], Noah Snyder-Mackler[5], Saul Cheves Hernandez[6], Ronald Lopez Navarro[6], Linda M. Fedigan[7], Michael Gurven[8], James P. Higham[9], Katharine M. Jack[10,†] and Amanda D. Melin[7,11,12,†]

[1]Department of Anthropology, and [2]Department of Management Science and Statistics, University of Texas at San Antonio, San Antonio, TX 78249, USA
[3]Département d'anthropologie, Université de Montréal, Montréal, Québec H3T 1N8, Canada
[4]Département de sciences biologiques, Université de Montréal, Montréal, Québec H2V 0B3, Canada
[5]Center for Evolution and Medicine, School of Life Sciences, School of Human Evolution and Social Change, Arizona State University, Tempe, AZ 85287, USA
[6]Área de Conservación Guanacaste, Guanacaste, Costa Rica
[7]Department of Anthropology and Archaeology, University of Calgary, Calgary, Alberta T2N 1N4, Canada
[8]Department of Anthropology, University of California, Santa Barbara, CA 93106, USA
[9]Department of Anthropology, New York University, NY 10003, USA
[10]Department of Anthropology, Tulane University, New Orleans, LA 70118, USA
[11]Department of Medical Genetics, and [12]Alberta Children's Hospital Research Institute, University of Calgary, Calgary, Alberta T2N 4N1, Canada

FAC, 0000-0001-9826-751X; JDO, 0000-0001-6922-2072; NS-M, 0000-0003-3026-6160; MG, 0000-0002-5661-527X; JPH, 0000-0002-1133-2030; ADM, 0000-0002-0612-2514

Studying biological ageing in animal models can circumvent some of the confounds exhibited by studies of human ageing. Ageing research in non-human primates has provided invaluable insights into human lifespan and healthspan. Yet data on patterns of ageing from wild primates remain relatively scarce, centred around a few populations of catarrhine species. Here, we introduce the white-faced capuchin, a long-lived platyrrhine primate, as a promising new model system for ageing research. Like humans, capuchins are highly social, omnivorous generalists, whose healthspan and lifespan relative to body size exceed that of other non-human primate model species. We review recent insights from capuchin ageing biology and outline our expanding, integrative research programme that combines metrics of the social and physical environments with physical, physiological and molecular hallmarks of ageing across the natural life courses of multiple longitudinally tracked individuals. By increasing the taxonomic breadth of well-studied primate ageing models, we generate new insights, increase the comparative value of existing datasets to geroscience and work towards the collective goal of developing accurate, non-invasive and reliable biomarkers with high potential for standardization across field sites and species, enhancing the translatability of primate studies.

This article is part of the discussion meeting issue 'Understanding age and society using natural populations'.

1. Introduction: individual variability in rates of ageing

One of the most enduring questions in public health is why some individuals remain healthy and high functioning well into old age, while others

[†]These authors contributed equally to the study.

THE ROYAL SOCIETY PUBLISHING

experience physical or cognitive deficits, chronic disease and premature mortality [1]. The profound social and economic consequences of rapidly ageing populations around the world [2,3] underscore the importance of efforts to extend human healthspan by reducing or delaying the burden of chronic diseases and disabilities associated with older ages [4,5]. Much variation in the progression of human ageing processes remains poorly explained, but a growing body of literature highlights the contributions of both the social and physical environment. The emerging field of translational geroscience seeks to understand the biological mechanisms of ageing and how they are connected to the emergence of chronic conditions associated with old age by studying parallel processes in animals [6,7].

In humans, biological ageing includes gradual declines in multiple body systems and functions, including loss of physical capability [8], impaired immune function [9], endocrine dysregulation [10] and molecular alterations [11,12]. These declines are accompanied by progressively increasing risks of a wide range of diseases—including cancer, cardiovascular disease and neurocognitive declines—and accelerating risk of death [13]. There are also a number of known differences in the propensity of males and females to develop age-related diseases. For example, men are more likely to develop certain cancers [14], while women are more likely to suffer from autoimmune diseases such as rheumatoid arthritis [15,16], and stress-associated conditions such as depression [17].

Slowing the rate of biological ageing—the age-dependent functional deterioration experienced by most organisms [6]—would delay the onset of age-related diseases and conditions, extending the human healthspan [18]. This goal, known as the geroscience hypothesis, is rooted in the idea that the pace of biological ageing is malleable [19]. Some of the clearest evidence that rates of ageing are strongly modulated by socio-environmental factors comes from captive or laboratory animal models, in which specific interventions (e.g. caloric restriction) or pharmacological treatments have been shown to extend lifespan and reduce the prevalence of diseases associated with old age [20]. However, key knowledge gaps remain, including: (i) which socio-environmental factors contribute most strongly to accelerated or decelerated biological ageing, (ii) the degree to which different body systems show these effects, and (iii) which individuals are most strongly affected and why.

Here, we highlight the value of a long-studied wild population of white-faced capuchin monkeys (*Cebus imitator*) that contributes to filling these knowledge gaps and advancing the understanding of the social and ecological determinants of ageing. We begin by highlighting the value of field studies of natural populations, then focus on why capuchins are an exceptionally interesting species for ageing research, detailing some of the insights provided by decades of research on white-faced capuchins in Costa Rica. We end by briefly describing our integrative study that combines metrics of the social and physical environment with physiological and molecular hallmarks of ageing across the life course in multiple social groups of white-faced capuchin monkeys studied by the Santa Rosa Primate Project.

2. Field studies of natural populations

Studies of human health and the pace of ageing are limited by the difficulty of collecting detailed within-subject longitudinal data on lived experience and health outcomes. Such studies also have numerous confounds, including individual variation in lifestyle, early-life experience, socioeconomic status and access to health care. Ageing research on animal models has been invaluable because it can circumvent many of these challenges. The logistical and analytical advantages of ageing research on captive animal models, in which a high degree of experimental control can be achieved, are extensive and widely acknowledged [21]. But research in captive settings cannot encompass the full scope and complexity of natural stressors and modifiers affecting natural populations, including humans, highlighting limitations associated with the loss of ecological validity in captive animal models compared to those living in their natural environments [22,23]. Only by studying wild animals in natural environments can we gain insights into selection pressures and evolved life history, with ageing (and its potential for plasticity) as a quintessential part. The study of ageing in animal models can be improved by bringing together complementary approaches in both wild and captive settings [24]. For instance, assessing the generalizability and validity of findings is essential for enhancing the translational value of ageing research in animal models, and these targets can be realized most effectively by integrating research from the wild and captivity. On one hand, validating correlational findings from the field may require conditions that are typically achievable only in captivity, such as the ability to apply experimental manipulations to isolated individuals, behaviours or responses to eliminate possible confounders and make strong causal inferences. On the other hand, observations from the wild are needed to confirm the ecological validity of observations from captivity, including the realism of environmental contexts and stimuli. In addition, studies that directly compare ageing in wild and captive individuals can reveal a wider range of biological variation than in either setting alone, helping to delimit the boundaries of possible trait values and understand their flexibility across environmental contexts [25–27]. For instance, by studying how processes such as domestication and urbanization impact ageing in animals, we can gain insights into the role of analogous processes throughout the course of human evolution.

Accumulating evidence has revealed that longitudinal studies of wild nonhuman primates can be particularly informative for generating insights into how social and physical environmental factors influence natural ageing processes and the human healthspan [27–31]. As the closest phylogenetic relatives of humans, non-human primate models are particularly valuable in ageing research because of the behavioural, functional and physiological characteristics that they share with humans [32–34]. Wild populations of primates living in natural environments hold underutilized potential for biogerontology because the mechanisms of ageing that they share with humans are likely to be universal across environmental contexts, making those mechanisms promising targets for effective, widely applicable interventions [35,36]. For example, as in humans, sex differences in mortality risk and disease presentation are also found in some non-human animal models, suggesting that such models can be useful for parsing out the contributions of extrinsic and intrinsic factors. The social construction of gender across human

cultures has health implications beyond that of biological sex, but influences of sex and gender on health are often interlinked, making potential biological and social influences of sex versus gender difficult to disassociate in human health and ageing research [37]. Because human gender socialization does not have a close parallel in nonhuman primates, sex differences that primates share with humans are likely to reflect biological commonalities beyond cultural factors [38]. And in contrast to studies of health and ageing in captive primates, which typically focus on the effects of isolated sources of severely adverse social or physical exposure [39,40], long-term studies of wild primates can shed light on the longitudinal and intergenerational consequences of multiple co-occurring and interrelated socio-environmental factors that are more closely matched to the conditions under which the biological responses evolved.

Although several non-human primate species living in captivity are established as models for ageing research [34], very few natural populations have been developed as comprehensive ageing models despite their significance for understanding human biology and behaviour. This scarcity reflects the challenges of studying ageing in non-human primates living in wild or naturalistic settings. For example, because they have relatively long lives compared to many other animals, prospective longitudinal studies on the life-course outcomes of non-human primates require decades of continuous data collection. The most well-established wild or naturalistic non-human primate models of ageing—in which multiple measures of ageing have been characterized in behavioural, physiological, functional, molecular and demographic domains—include populations of baboons (*Papio* sp.) [41], rhesus macaques (*Macaca mulatta*) [28] and chimpanzees (*Troglodytes schweinfurthii*) [42,43]. The current concentration of ageing research on natural populations of great apes and cercopithecines has some clear advantages, as these close relatives of humans show physiological changes across the life course that recapitulate human biology. However, understanding the *generality* of ageing mechanisms as well as potential interventions is a central goal in translational gerontology, and this goal may be best served by expanding ageing research on natural populations to taxa that are more distantly related to humans [44].

The small taxonomic breadth of current studies of ageing in natural primate populations also underscores a basic limitation: to date, animal research on ageing has been limited to populations where invasive sampling of blood and tissues has provided the source of much of the available molecular and physiological data. Invasive sampling methods and assessment tools that rely on such methods are not possible in most studies of wild primate populations that are highly relevant to human health and ageing. This may be due to ethical considerations, logistical challenges or laws and regulations that govern the scope of research protocols on natural populations that are often of the highest conservation concern. Advances in non-invasive sampling are, therefore, essential for creating new opportunities for ageing research on natural populations.

For example, DNA methylation is a well-established molecular hallmark of ageing in humans, non-human primates and other biomedical models [11,45], however, molecular sampling for studies of epigenetic alterations of wild and free-ranging primates to date relies on darting or trapping to collect invasive blood and tissue samples. Recent advances in the use of the intestinal epithelium to study epigenetic ageing in humans [46] indicate the feasibility of using epithelial cells sorted from faecal samples of non-human primates to quantify epigenetic ageing non-invasively from wild animals (figure 1). Indeed, whole genome sequencing and analysis can be undertaken by sorting cells found in faeces [47]. Such samples hold promise for examining the epigenome, as well as DNA sequence data.

Methods for measuring mammalian hormone metabolite concentrations were first developed in the late 1970s, and enabled a new era of field studies that were able to link the social and physical environment to the variation in animal conditions and underlying physiology [48]. Recent studies have validated several non-invasive biomarkers of immune activation and inflammation, such as urinary neopterin as a marker of cellular immune activation, demonstrating that they are robust to field collection and storage conditions for use in studies of wild primates [49,50]. Collectively, these non-invasive techniques have great potential for new studies of the links between the social and physical environment and the variation in the pace of ageing. But for many of these biomarkers, the relationship between naturally occurring variation and age needs to be established.

3. Capuchins as a model species for the study of ageing

White-faced capuchins are a promising model system for the study of biological ageing, with high potential to yield new knowledge about ageing processes. Natural populations of white-faced capuchins are among the most comprehensively studied wild primates in the world, with multiple long-term, individual-based, longitudinal field research projects offering a rich but until now underdeveloped foundation for ageing research [51–53]. Among these projects, the Santa Rosa capuchin research project is the longest running, most continuous, and least altered by anthropogenic activities [52,54], but other white-faced capuchin research projects also have high potential for ageing research, including those at the sites Lomas Barbudal [53] and Taboga [55]. The wild and unprovisioned Santa Rosa population lives in a highly accessible forest and has been studied in depth—including behavioural, life history, pedigree and environmental data—nearly continuously since 1983 [52,54]. These primates reside in groups of multiple related adult females, multiple immigrant males, and their immature offspring. Capuchin social groups in this population range in size from 5 to 35 individuals with a sex ratio skewed towards females [56]. We have documented relevant variation in the social and ecological environments of these capuchins, their social interactions, diets and foraging behaviours, as well as extensive variation in how long individuals live, their reproductive output and their later-life health [52,54]. In addition, the exceptional depth and quality of more than four decades of life-history data from the Santa Rosa capuchin population have enabled and contributed to ground-breaking comparative insights about natural patterns of age-specific mortality [27,57–61] and reproduction [60–63] in wild primates, helping to contextualize patterns of ageing in humans.

These characteristics of the Santa Rosa capuchins establish this population as an excellent model system in which to study both age-related variation cross sectionally, and the longitudinal progression of 'ageing in the wild' in multiple body systems,

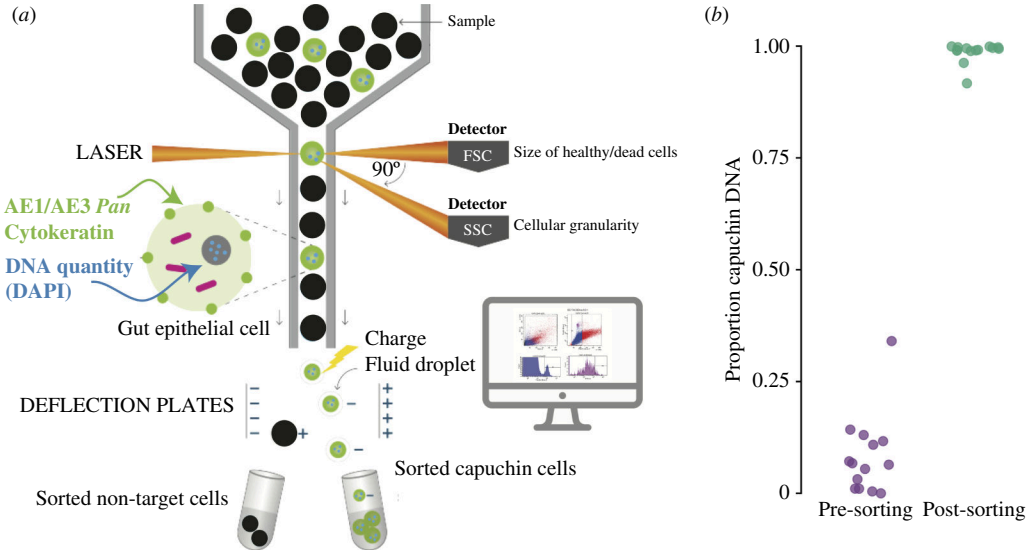

Figure 1. (*a*) Schematic representation of the faecal fluorescence-activated cell sorting (FACS) method for isolating epithelial cells from faecal samples to purify primate DNA, generating a source of tissue-grade DNA material for molecular analyses. (*b*) Proportion of capuchin DNA pre- and post-sorting based on droplet digital PCR using markers for bacteria and for primate DNA.

including social, environmental and demographic sources of variation in these patterns. Cross-sectional, population-level study designs are more common in ageing research, including in natural animal models, and they offer several advantages: they require less time and effort to implement and are less vulnerable to confounding effects of social or environmental factors that change over time. These characteristics make cross-sectional study designs ideal for discovering biomarkers that exhibit age-related variation. But cross-sectional study designs also have important shortcomings in ageing research that can only be overcome by employing longitudinal approaches. First, longitudinal sampling is necessary for characterizing individual trajectories of ageing in different domains (functional, physiological and molecular), which is a prerequisite for studies that seek to understand sources of variation in the pace of ageing. Second, cross-sectional approaches alone cannot establish links between factors of interest (e.g. biomarker values, socio-environmental conditions and metrics of illness and disease) and life-course outcomes, including morbidity and mortality. Finally, longitudinal study designs are less vulnerable to cohort and selective disappearance effects compared to cross-sectional study designs [64].

A distinct combination of life-history, behavioural, ecological and phylogenetic traits distinguishes capuchins from more established wild or naturalistic non-human primate models and demonstrates their potential to complement ageing studies in humans and in other animal models by expanding cross-species comparisons of parallel measures of ageing. In the remainder of this section, we detail why capuchins are particularly promising for answering questions about ageing, with an emphasis on traits that distinguish them from other widely used non-human primate models for ageing.

First, capuchins share several key life-history-related traits with humans, while standing out from other non-human primates in important ways. Compared to most other mammals, all primates have relatively large brains, a slow pace of development and extended longevity for their body sizes [65]. But capuchins are extraordinary even among the primates: they are roughly the size of a house cat, yet they have the largest ratio of brain size to body mass of any non-human primate [66], a slower pace of development in relation to their body size [67], and exceptional cognitive capabilities in multiple domains that are of particular relevance for ageing research [68–71]. We have recently made progress in identifying the genetic basis of some of these traits, including genes under positive selection that are associated with longevity and large brains [47].

Capuchins are not only well suited based on their suite of human-like traits but they also offer advantages as an animal model system over species that also share these traits. Other non-human primate species that exhibit a slow pace of development, large brains and extended longevity, such as chimpanzees, have substantially larger bodies, longer lifespans, slower generation times and tend to be more difficult to study both in the wild and in captivity [34]. These contrasts speak to conflicting goals for the establishment of animal models of human ageing. From a logistical perspective, the most practicable natural animal models are species with short lifespans and short generation times that can be studied easily in a variety of settings, all of which facilitate the accumulation of large sample sizes. But from a theoretical perspective, the most informative natural animal models of human ageing are likely to be those that have a more human-like ageing course, including prolonged development and long lifespans. The unique combination of life-history traits in capuchins presents a compelling balance between these divergent goals.

Second, there is high variance in longevity both within populations of wild capuchins and compared to those in captivity, suggesting high potential for environment-associated effects on health and longevity, and thus on the pace of biological ageing (figure 2). Because of high early-life mortality, wild white-faced capuchins have life expectancies at birth of only about 10 years, but if they survive to adulthood, wild capuchins of both sexes can expect to live into their mid-20s [27] and some individuals live more than 30 years [73] (figure 2). In captivity, capuchins have life expectancies at birth in the mid-20s, and long-lived individuals in captivity can reach over 50 years of age [27,74] (figure 2). Such within- and between-population variability is an essential quality of a good animal model of ageing and is crucial for identifying factors that influence rates of ageing

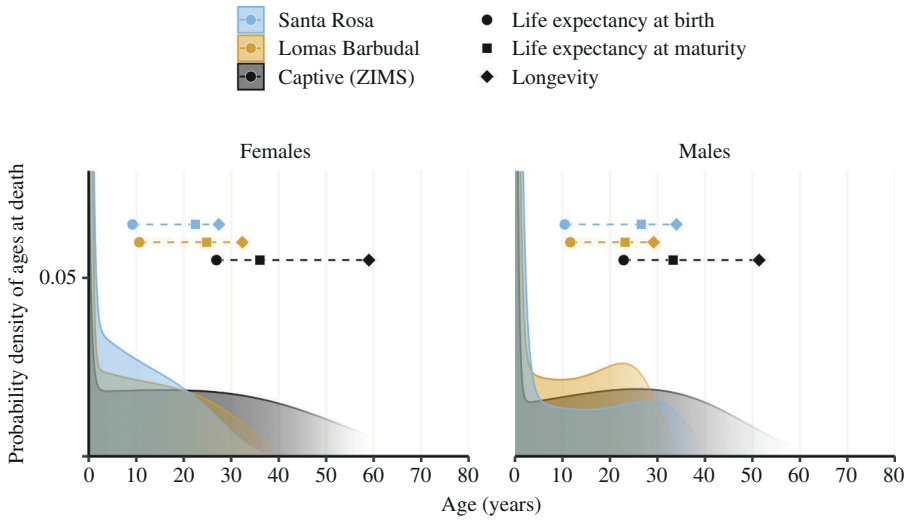

Figure 2. Age-specific and sex-specific mortality in white-faced capuchins from two well-studied natural populations (Lomas Barbudal and Santa Rosa) as well as captive animals in zoos (ZIMS, [72]). Transparency at older ages represents decreasing certainty of estimates. The points connected by dashed lines above the curves show three sex-specific point estimates for each population: life expectancy at birth, life expectancy at maturity and longevity (the age that only 5% of newborns are expected to reach). All mortality curves and length-of-life point estimates are calculated from the Siler model parameter values published in the supplementary materials of Colchero *et al.* [27].

by enabling quasi-experimental study designs in which putative socio-environmental determinants of ageing can be linked statistically to different life-course outcomes [44].

Importantly, some of the natural variation in health and longevity in the Santa Rosa capuchins can be attributed to both social and ecological factors. Like humans, the Santa Rosa capuchins experience environmental extremes, which include dramatic seasonal shifts and recurrent droughts [75], and like humans, they show resilience and differential susceptibility to those extremes [52,76]. High rainfall seasonality at the site is a dominant driver of intra-annual variation in capuchin behaviour, diet and physiology through its sweeping effects on dry-forest ecology, including effects on food availability [75,77,78]. For example, periods of fruit and water scarcity induce physiological signatures of metabolic distress in females, particularly those that are low ranking or pregnant [79]. Such periods are also associated with elevated glucocorticoid secretion [80] and shifts in the composition and function of the gut microbiome [78], both of which are key elements in the biology of human ageing. Environmental variability can also affect health by varying the risk of exposure to helminths and other parasites, both directly (e.g. through contact with parasites in standing water sources that accumulate parasites during dry conditions) and indirectly (e.g. through shifting social cohesion and the presence or density of intermediate vectors such as arthropod prey) [81,82]. In the Santa Rosa capuchins, the prevalence of some parasites varies seasonally [83,84], and although individuals differ in their infection status, older individuals are generally infected at higher rates and show greater parasite species richness [83]. Bacterial and viral pathogens, including some with the potential for zoonotic transmission to humans, are also found in platyrrhine primates, including some wild capuchin populations [85]. Individual-based longitudinal studies of the cumulative effects of infectious disease and illness on ageing in human hunter-gatherers [86] and in some wild primates [87] have led to important translational insights, but in wild capuchins this remains an underdeveloped area that should be prioritized.

In addition, inter-annual variation in drought severity strongly affects the demographic structure of the capuchin population through its effects on pre-adult mortality [60,88,89]. Apart from Santa Rosa, there are two other intensively studied populations of white-faced capuchins in this highly seasonal region of northwestern Costa Rica [51,73]. Ecological differences among the three sites appear to link diverse physiological responses to environmental seasonality [55], and severe El Niño-mediated droughts that occur in this region are associated with waves of elevated mortality in at least some of these populations [89]. In an analogous way, infrequent but severely adverse events experienced by human populations, such as the Dutch Hunger Winter, have been especially instructive for understanding how early-life environmental experiences can modify the progression of ageing and have long-lasting effects on health and longevity [90].

Third, capuchins show a varied repertoire of complex extractive foraging techniques [91], preferred foraging locations [92] and peculiar social rituals characterized by innovations that emerge and diffuse among individuals in age-dependent patterns [91,93]. Conspecifics and congeners at other sites are among the only natural populations of non-human primates, aside from chimpanzees, that habitually use stone tools to forage on well-protected, energy-dense food resources [71,94]. These complex behaviours are transmitted largely through social learning in which inexperienced individuals observe and emulate the techniques of more experienced, typically older individuals [71,91]. Capuchin societies thus offer rare opportunities to study age-dependent patterns of sociality that strongly echo those of human societies, in which age, foraging proficiency, ecological challenges and life-course outcomes are closely interlinked.

Fourth, white-faced capuchins experience frequent social turmoil, violence and adversity, factors which have been robustly linked to elevated risk of premature mortality in humans [95]. In the Santa Rosa capuchin population, these experiences arise primarily from predation and predation attempts, aggressive intergroup encounters and alpha male replacements, the latter often resulting in the injury, death or disappearance of multiple group members [73,96–98]. Males, particularly alpha males, are the most active participants in these types of events and thus are likely to experience more frequent and direct exposure to

high-stress situations in comparison with other age-sex classes. Additionally, infanticide is the leading cause of infant mortality in wild capuchins, which imposes significant costs on females and is a likely source of intense early-life stress for infant capuchins who are at risk of being targeted [99]. In the Santa Rosa population, the risk of early-life mortality is modulated by aspects of the infant's social environment: during socially stable periods, infant survival is highest among the offspring of highly sociable mothers who occupy a central position in the group, but during periods of high infanticide risk, these same infants appear to be targeted at higher rates, leading to elevated mortality [100]. Wild capuchins, therefore, offer a possibility to investigate how resilience to different types of social adversity varies across age, sex, social status and social connectivity—all of which are hypothesized to play important roles in the convergent pathways that link social environments with life expectancy and disease risk in humans and other social animals [31].

Fifth, white-faced capuchins exhibit social patterns that mirror those found in many human societies [101,102], but that are unlike those found in existing non-human primate models of ageing. For instance, in most non-human primate species long-lasting same-sex alliances either occur in only one sex or do not occur at all, but in capuchins, both sexes frequently form coalitions and maintain long-lasting alliances with same-sex social partners [100,103]. These coalitions are not invariably structured by kinship, as they often are in female cercopithecines. As in many other well-studied primates, female capuchins remain in their birth group for life, enabling the formation of life-long intergenerational social bonds that have the potential to influence fitness and survival. For example, the long-term study of capuchins in the Lomas Barbudal population found evidence linking social integration (determined by affiliative interactions and proximity to others in foraging contexts) to increased female survivorship, an effect that was independent of female rank status [104]. But unusually, male capuchins, which disperse from their birth group around the age of sexual maturity (approx. 6 years) and reside in multiple different social groups throughout their lives, are able to maintain strong ties with close male kin by dispersing together with other males or selectively joining groups containing familiar, previously dispersed males [105]. Co-resident male capuchins cooperate to compete against male coalitions in other groups, leading to frequent coalitionary group takeovers; this distinct mode of male–male coalition formation requires a different explanation than other modes that are more common across primates [106]. Additionally, our work in Santa Rosa has shown that dominance relationships in female capuchins are relatively relaxed compared with well-studied cercopithecines: while female dominance rank is primarily (but not solely) influenced by maternal rank, maintenance of high rank also depends on the presence and cooperation of close female kin (i.e. sisters and adult daughters) [107]. Male dominance rank strongly predicts reproductive success in white-faced capuchins, and attaining and maintaining high rank (alpha status) is largely dependent upon a male's ability to form successful coalitions with co-resident adult males with whom he may or may not be related [108,109]. However, some males that remain subordinate are nonetheless able to achieve high reproductive success by cooperating with and supporting the alpha male [110]. Thus, rank status for both sexes, and the preferential access to both social and ecological resources that it affords, is strongly shaped by an individual's social influence—the ability to affect the behaviour of others.

Human studies examining the impact of ageing on social influence have found both generalized declines in social cognition at older ages and increased 'wisdom' or skill in navigating social conflict [111–113], and some of these patterns are strongly recapitulated in ageing non-human primates [114]. For example, capuchins also show variability in the impacts of ageing on social influence, given that some of the oldest adult females and males retain alpha positions until their deaths, while others are deposed and become subordinate at similarly old ages.

4. Leveraging the long-term Santa Rosa study to assess the socio-environmental determinants of the pace of ageing

Building on our rich foundation of individual-based life histories and long-term monitoring of important social and ecological modifiers, we have recently expanded the scope of our work in the Santa Rosa capuchins to encompass a wider range of behavioural observations, non-invasive biospecimen collection and laboratory assays that are relevant to human ageing (figure 3). The overarching goal is to generate insights into how social and physical environmental factors contribute to heterogeneity in rates of ageing and health disparities. To achieve this, we are combining cross-sectional population-level sampling to identify and study hallmarks of ageing across behavioural, physiological and molecular domains with longitudinal sampling to characterize individual biomarker trajectories and link them to individual experiences and life-course outcomes. Below, we describe renewed efforts to leverage the long-term Santa Rosa project to make impactful contributions to our understanding of the social and ecological modifiers of ageing and healthspan.

(a) Broadening biomarkers of ageing and health from non-invasive samples

We are currently broadening the range of biomarkers under long-term study at Santa Rosa to provide a more complete representation of the 'hallmarks' [115] or 'pillars' [116] of ageing analogous to those of clinical relevance to humans [33]. To this end, we are validating putative biomarkers of physical condition, physiological function and molecular profiles cross-sectionally across dozens of animals of different ages, and sampling them longitudinally in the same animals throughout their adult lives (defined as 6 years of age or older) (table 1, figure 3). The biomarker validation process has involved establishing non-invasive sample collection methods, refining measurements of each biomarker, quantifying the accuracy and repeatability of biomarker values and determining the degree to which the biomarkers that show age-related differences in humans show similar patterns in capuchins. We are also using 'gold standard' sources (blood and tissue biopsies) from a cross section of

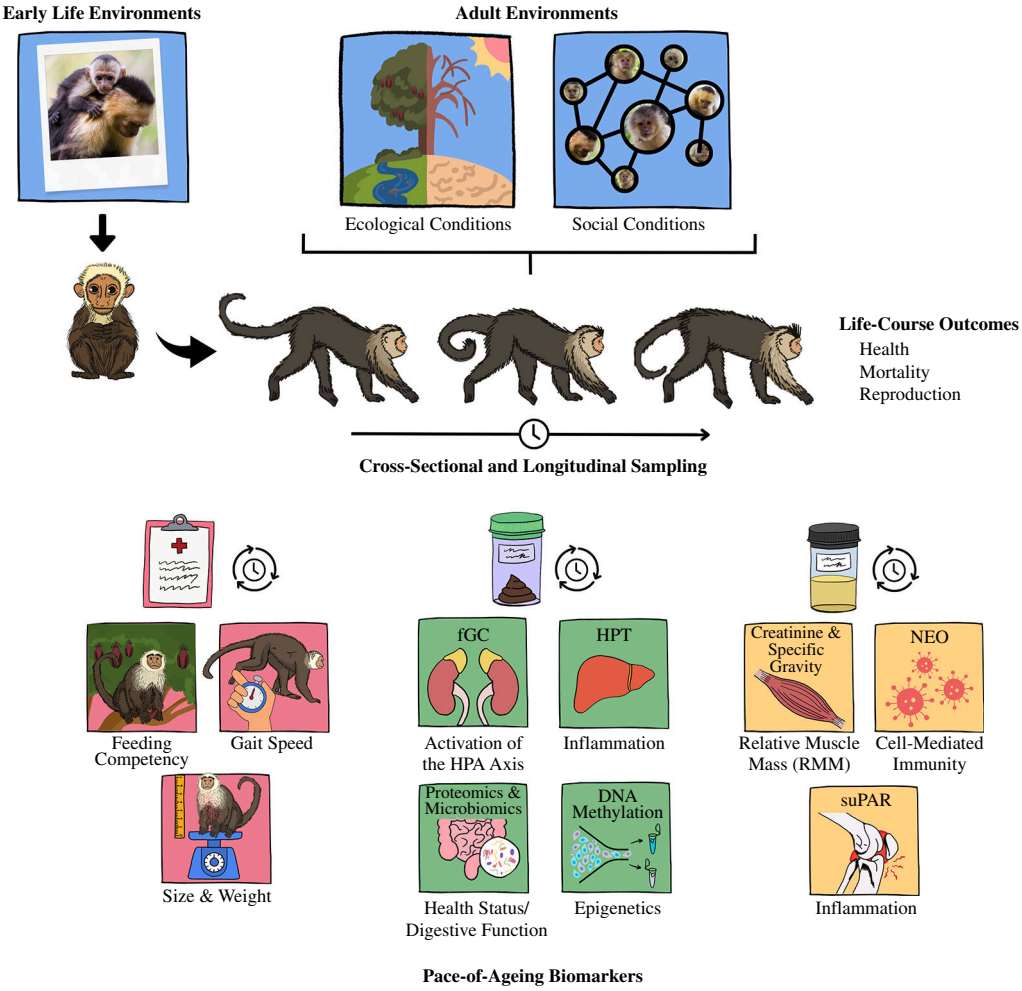

Figure 3. Conceptual overview of research on the social and ecological determinants of ageing in the Santa Rosa capuchin population. Artwork by Jordie Hoffman.

capuchin monkeys housed in captivity to validate FACS-derived DNA methylation analyses (figure 1) to ensure that similar methylation profiles are obtained from each source and that they accurately reflect chronological age.

The biomarkers that we are targeting include those that have been associated with elevated morbidity or mortality risk in humans, and which therefore may signal the divergence of individual frailties at older ages (table 1). For instance, age-related loss of skeletal muscle mass (sarcopenia) is a risk factor of disability and mortality in older humans [117] and develops along a similar trajectory in some non-human primates [118,119]. Likewise, changes in adrenal function and the development of a pathological proinflammatory phenotype in old age ('inflammaging') are widely recognized as hallmarks of ageing [115,116], with certain measures of immune activation and inflammation being associated with elevated risk of age-related morbidities, including cardiovascular disease [120] and Alzheimer's [121], as well as elevated all-cause mortality risk [122]. Furthermore, characterizing gut microbial and proteomic senescence from faecal samples has generated new data on age-related biological processes and maladies, provided insight into declining digestive function, and even allowed for the generation of microbial 'clocks' based on diversity, differential abundances and putative functions of common bacterial genera [123].

(b) Behavioural assessments of physical function and social environments

Quantitative measures of human healthspan have become a central focus in biogerontology and are primary outcomes targeted for clinical interventions [124]. Accordingly, there is growing interest in finding measures of healthspan in animal models that translate meaningfully to humans [33,125]. To date, measures of physical function have been among the most successful non-invasive approaches to quantifying healthspan in animals [126–130], and multiple measures of physical capability are useful for identifying older people with an elevated risk of morbidity and mortality. For example, grip strength, standing balance, chair rise time and walking speed all show characteristic age-related declines in older people, comprise key elements of 'frailty syndrome,' and correlate significantly with all-cause mortality [131,132]. Additionally, low masticatory ability in older people is associated with low nutritional status and other indicators of poor health [133]. We have developed behavioural assessments of physical function in the Santa Rosa capuchins that are designed to have clinical relevance to humans by employing non-invasive analogues of these geriatric assessments: we measure gait speed, posture and feeding selectivity and efficiency on foods that differ in their ease of access and processing.

Similarly, we have expanded and refined our assessments of capuchin social environments across the life course, focusing on measures with direct parallels to factors widely recognized by social scientists as being strongly associated with health and survival in humans. For example, older people who are socially isolated have significantly elevated risk of inflammation and

Table 1. Biomarkers of ageing sampled from adult white-faced capuchins in Santa Rosa.

ageing domain	biomarker	non-invasive method	relevance to human ageing
behavioural / functional	gait speed	terrestrial walking speed	slow gait speed in older people is correlated with higher mortality risk
behavioural / functional	feeding selectivity and competency	bite rates and food selectivity with respect to mechanical properties (hardness, toughness) and processing difficulty of food items	low masticatory ability and strength in older people is associated with low nutritional status, depression and other indicators of poor health
behavioural / functional	posture	body orientation and support postures during different behaviours with respect to relative grip and core strength required to maintain them	manual grip strength, standing balance and chair rise time all show age-related declines in older people and correlate significantly with all-cause mortality
physical	body mass	strategic placement of remotely triggered electronic scale at frequented waterholes	weight changes in older people are associated with higher mortality risk
physical	body size	paired-laser photogrammetry to measure shoulder-to-rump length	together with body mass, used for calculation of BMI, a strong predictor of mortality risk
physical	relative muscle mass	indexing urinary creatinine to urinary specific gravity	loss of skeletal muscle is a health risk factor
physiological	inflammation	haptoglobin (HPT) in faeces	HPT is a nonspecific biochemical marker of inflammation
physiological	inflammation	soluble form of the urokinase plasminogen activator receptor (suPAR) in urine	suPAR rises in response to inflammatory conditions
physiological	immune activation	neopterin (NEO) concentrations in urine	NEO is a marker of infection and disease
physiological	adrenal function	metabolites of glucocorticoids in faeces (fGC)	older people show overproduction of GCs and greater cortisol responsiveness to stressors
molecular	DNA methylation	low-input targeted bisulfite sequencing of DNA isolated from gut and urinary epithelial cells	DNA methylation-based age acceleration predicts poorer health and increased mortality in humans
molecular	gut microbial senescence	generating a 'microbiome clock' based on declining microbial diversity and differential abundances of common bacterial genera	age-related changes to the gut microbiome are associated with alterations to intestinal function, permeability of the intestinal lining, inflammation and frailty in humans
molecular	protein health markers	faecal proteomics using liquid chromatography–tandem mass spectrometry (LC–MS/MS) to generate age-related proteomic profiles	changes in the gut proteome associated with health status, diet and age-related functional decline in humans

hypertension [134] and face a 1.5-fold increase in all-cause mortality [135], and remarkably parallel links have been found in a range of social mammals, including in wild non-human primates of both sexes [31,136]. In the Santa Rosa capuchins, some of these key measures of individual social advantage and social adversity, including social status and social instability, have been tracked for decades as part of our long-term data collection [52,100]. Analogues of other structural and functional measures of social integration that are important in humans, such as affiliative network centrality and the strength and stability of social support, required new behavioural data collection approaches that we implemented in conjunction with our expanding focus on measures of ageing (figure 3).

(c) Contributions of social and physical environments to the pace of ageing

The long-term aim of the Santa Rosa ageing project is to undertake longitudinal characterization of validated health and ageing biomarkers across the natural life course of capuchins to infer socio-environmental influences on individual trajectories of ageing, as well as sex differences in these relationships. Current approaches to measuring the pace of ageing involve the calculation of composite indices of biological age that summarize an assortment of different biomarkers, most commonly in cross sections [137]. Although innovative longitudinal approaches are underway in human cohorts, they involve a comparison of the change in biological age from an initial reference point (e.g. young adulthood) to a single or at most a few follow-ups separated by multiple years [137–139]. These approaches can place an individual's pace of ageing on a spectrum from fast to slow, but they provide only a limited picture of the ageing trajectory's dynamic behaviour, its fundamental modes of variation and when and why ageing trajectories begin to diverge. A more complete picture of the determinants of individual variation in the pace of ageing can be achieved by combining cross-sectional biomarker identification with densely sampled longitudinal characterization of biomarker trajectories.

Furthermore, the discovery that the risk of age-related diseases that reduce adult healthspan and lifespan can be predicted in childhood has increased focus on the critical role of early-life adversity in determining the pace of ageing [140]. For example, children who experience more early-life adversity are more susceptible as adults to a wide range of health problems—including diabetes, obesity and cardiovascular disease [141]—as well as earlier mortality [95]. Adverse childhood experiences

can become biologically 'embedded' and are detectable before the onset of age-related disease in the form of dysregulated adrenal function, increased proinflammatory signalling and altered DNA methylation [142–146]. The cumulative effects of experiencing multiple adverse events in early life on later-life health and survival are particularly potent [95]. Recent findings indicate that the strong link between early-life adversity and adult health and survival is recapitulated in some social mammals [39,147–149], demonstrating their utility as models for studying parallel processes in humans [31]. Additionally, abundant evidence linking social and physical environments in adulthood to health and survival suggests that these conditions may modulate the long-lasting effects of early-life adversity [135]. For example, the 'social buffering' hypothesis posits that negative effects of early-life experience on later-life morbidity and mortality can be moderated by the protective influence of adult social integration or exacerbated by adult social isolation [150]. Cumulative risk models further posit that the effects of social advantage and disadvantage act cumulatively across the life course, their influence varying dynamically with changing later-life experiences [151–153]. Finally, some studies do not support a special role for early-life adversity but rather show additive effects of particular adverse conditions on health that remain similar in magnitude over the entire life course [154]. Longitudinal life-course studies that measure early-life conditions, adult socio-environmental factors and health outcomes in the same subjects are needed to make progress in this area [137,155]. While significant hurdles remain to testing these connections in human subjects, recent work has demonstrated that the hurdles can be surmounted in animal models [156]. Our work in this area with the Santa Rosa capuchins is designed to test whether specific sources of early-life adversity in capuchins (including loss of mother, absence of father during social instability and exposure to severe drought or food scarcity) accelerate the pace of ageing, and whether later-life sources of social and physical adversity (social isolation and drought) or advantage (high social integration and high social status) further moderate ageing trajectories.

5. Conclusion

Non-human primates are invaluable ageing models, and the comparative study of wild populations has generated many insights. At present, strong taxonomic bias towards primates in the parvorder Catarrhini (monkeys found in Africa and Asia, and apes) limits the generalizability of discoveries. We discuss the value of comprehensive study of capuchin monkeys as an outstanding non-human primate model. These primates are part of the other major taxonomic branch of monkeys, the Platyrrhini, but due to their similarities with humans, including exceptional longevity and brain size, as well as their complex social and ecological proclivities, they are well positioned to be of high translational potential. Our research on the Santa Rosa capuchins aims to capitalize on this potential, investigating patterns and processes of ageing that may generalize to many other species and systems, including humans. Our individual measures are designed to be analogues for understanding basic ageing processes in humans, and the biomarkers of ageing that we are measuring all have direct clinical relevance to measuring the pace of ageing in humans. Furthermore, the socio-environmental factors on which we focus as potential modulators of ageing (early-life conditions, adult social and physical environments) represent forms of adversity that can influence development and health, and thus have broader relevance for understanding how environmental factors can get under the skin at different points in the life course to influence the pace of ageing. And while the Santa Rosa capuchin population has many desirable qualities for a wild animal model, it shares many of these advantageous qualities with other long-running capuchin study sites, including the nearby Lomas Barbudal Monkey Project [53]. Importantly, by selecting biomolecular, social and physical metrics that are as close as possible to those used by other long-running studies of non-human primates and of humans [157,158], we maximize the translational potential of this research. Finally, we have spearheaded efforts to develop new non-invasive techniques and share protocols to measure physical, physiological and epigenetic ageing that will translate to a wide range of other systems.

Studies of wild primate ageing are the most valuable when sampling can be done densely, longitudinally and comprehensively. By developing and validating a comprehensive set of ageing biomarkers that are non-invasive but designed to mirror those established in humans and existing animal models, we improve the ability of field studies to track age-related functional changes in multiple domains and biological systems. Our aim is to determine how socio-environmental factors lead to distinct patterns in trajectories of ageing, when and in whom these patterns emerge and which ageing phenotypes are most strongly affected. Leveraging this intensively studied wild population of capuchins as a new model system for investigating the social and ecological determinants of ageing will provide critical comparative data, has created and will continue to create new opportunities for collaborations and training within Costa Rica, and globally, for interested researchers with synergistic questions, and promote a better understanding of primate health and ageing processes.

Ethics. All work involving primates was reviewed and approved by the IACUC committees at Tulane University, The University of Calgary and The University of Texas at San Antonio. The research was carried out with permission from the Área de Conservación Guanacaste, SINAC and CONAGEBIO in Costa Rica.

Data accessibility. This article has no additional data.

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. F.A.C.: conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, project administration, resources, supervision, visualization, writing—original draft, writing—review and editing; E.C.W.: data curation, funding acquisition, investigation, methodology, writing—review and editing; J.D.O.: data curation, formal analysis, funding acquisition, investigation, methodology, writing—review and editing; Y.P.: funding acquisition, methodology, writing—review and editing; N.S.-M.: investigation, methodology, resources, writing—review and editing; S.C.H.: data curation, investigation, methodology, project administration; R.L.N.: data curation, investigation, methodology, project administration; L.M.F.: data curation, funding acquisition, investigation, methodology, project administration, resources, supervision; M.G.: conceptualization, funding acquisition, investigation, writing—review and editing; J.P.H.: conceptualization, funding acquisition, investigation, methodology, writing—review and editing; K.M.J.: conceptualization, data curation, funding acquisition, investigation, methodology, project administration, resources, supervision, writing—original draft, writing—review

and editing; A.D.M.: conceptualization, data curation, funding acquisition, investigation, methodology, project administration, resources, supervision, visualization, writing—original draft, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. This work was supported by the National Institutes of Health (NIA R61-AG078529); the National Science Foundation (NSF BCS-2051573); The Natural Sciences and Engineering Research Council of Canada (RGPIN-2016-03623, RGPIN-2017-03782, RGPIN-2023-04399, DGECR-2023-00272), Canada Research Chairs programme (1000200962-2001, 1000207382-2008, 950-231257), The Leakey Foundation; the National Geographic Society and Tulane University's Stone Center for Latin American Studies, Newcomb Institute, Lurcy Fund, Committee on Research and Lavin Bernick Fund for Faculty Research.

Acknowledgements. We thank the field team for the Santa Rosa ageing project, which included Cielo De La Rosa Meza, Philippine Delga, Leah Orman, Anka Pohle, Danielka Rugama Taylor, Peyton Schmidli and Lindy Wolhuter. In addition, Megan Mah, Nelle Kulick, Evin Murillo Chacon, Jenna Owens and Nick Chapoy all made important contributions to protocol development for the Santa Rosa ageing project. We are grateful to the administrators of Sector Santa Rosa, especially Roger Blanco and Maria Martha Chavarria, for supporting our research and for assistance with permits and logistics. We also thank two anonymous reviewers for constructive comments that improved the manuscript.

References

1. Crimmins EM. 2015 Lifespan and healthspan: past, present, and promise. *Gerontologist* **55**, 901–911. (doi:10.1093/geront/gnv130)
2. Lee RD. 2007 *Global population aging and its economic consequences*. Washington, DC: A E I Press.
3. He W, Goodkind D, Kowal PR. 2016 *An aging world: 2015*. Washington, DC: US Government Publishing Office. (doi:10.13140/RG.2.1.1088.9362)
4. Beard JR, Bloom DE. 2015 Towards a comprehensive public health response to population ageing. *The Lancet* **385**, 658–661. (doi:10.1016/S0140-6736(14)61461-6)
5. Butler RN. 1997 Population aging and health. *BMJ* **315**, 1082–1084. (doi:10.1136/bmj.315.7115.1082)
6. Sierra F. 2019 Geroscience and the challenges of aging societies. *Aging Med.* **2**, 132–134. (doi:10.1002/agm2.12082)
7. Kaeberlein M. 2017 Translational geroscience: a new paradigm for 21st century medicine. *Transl. Med. Aging* **1**, 1–4. (doi:10.1016/j.tma.2017.09.004)
8. Studenski S *et al*. 2011 Gait speed and survival in older adults. *J. Am. Med. Assoc.* **305**, 50–58. (doi:10.1001/jama.2010.1923)
9. Franceschi C, Campisi J. 2014 Chronic inflammation (inflammaging) and its potential contribution to age-associated diseases. *J. Gerontol. A* **69**, S4–9. (doi:10.1093/gerona/glu057)
10. Wang PS, Lo MJ, Kau MM. 1997 Glucocorticoids and aging. *J. Formos. Med. Assoc.* **96**, 792–801.
11. Horvath S, Raj K. 2018 DNA methylation-based biomarkers and the epigenetic clock theory of ageing. *Nat. Rev. Genet.* **19**, 371–384. (doi:10.1038/s41576-018-0004-3)
12. Sanders JL, Newman AB. 2013 Telomere length in epidemiology: a biomarker of aging, age-related disease, both, or neither? *Epidemiol. Rev.* **35**, 112–131. (doi:10.1093/epirev/mxs008)
13. Kennedy BK *et al*. 2014 Geroscience: linking aging to chronic disease. *Cell* **159**, 709–713. (doi:10.1016/j.cell.2014.10.039)
14. Jemal A, Center MM, DeSantis C, Ward EM. 2010 Global patterns of cancer incidence and mortality rates and trends. *Cancer Epidemiol. Biomarkers Prev.* **19**, 1893–1907. (doi:10.1158/1055-9965.EPI-10-0437)
15. Voskuhl R. 2011 Sex differences in autoimmune diseases. *Biol. Sex Differ.* **2**, 1. (doi:10.1186/2042-6410-2-1)
16. Whitacre CC. 2001 Sex differences in autoimmune disease. *Nat. Immunol.* **2**, 777–780. (doi:10.1038/ni0901-777)
17. Reynolds K, Pietrzak RH, El-Gabalawy R, Mackenzie CS, Sareen J. 2015 Prevalence of psychiatric disorders in U.S. older adults: findings from a nationally representative survey. *World Psychiatry* **14**, 74–81. (doi:10.1002/wps.20193)
18. Kaeberlein M, Rabinovitch PS, Martin GM. 2015 Healthy aging: the ultimate preventative medicine. *Science* **350**, 1191–1193. (doi:10.1126/science.aad3267)
19. Austad SN. 2016 The geroscience hypothesis: is it possible to change the rate of aging? In *Advances in geroscience* (eds F Sierra, R Kohanski), pp. 1–36. Cham, Switzerland: Springer International Publishing.
20. Fontana L, Partridge L, Longo VD. 2010 Extending healthy life span—from yeast to humans. *Science* **328**, 321–326. (doi:10.1126/science.1172539)
21. Mitchell SJ, Scheibye-Knudsen M, Longo DL, de Cabo R. 2015 Animal models of aging research: implications for human aging and age-related diseases. *Annu. Rev. Anim. Biosci.* **3**, 283–303. (doi:10.1146/annurev-animal-022114-110829)
22. Sikes RS, Paul E. 2013 Fundamental differences between wildlife and biomedical research. *ILAR J.* **54**, 5–13. (doi:10.1093/ilar/ilt015)
23. Lahvis GP. 2017 Unbridle biomedical research from the laboratory cage. *eLife* **6**, e27438. (doi:10.7554/eLife.27438)
24. Spence-Aizenberg A, Evans S, Wolovich C, Larson S, Fernandez-Duque E, Williams L. 2023 Aotus: a model to synthesize studies of captive and wild primates. In *Developments in primatology: progress and prospects* (ed. L Barrett), pp. 731–755. Cham, Switzerland: Springer International Publishing.
25. Lemaître JF, Gaillard JM, Lackey LB, Clauss M, Müller DWH. 2013 Comparing free-ranging and captive populations reveals intra-specific variation in aging rates in large herbivores. *Exp. Gerontol.* **48**, 162–167. (doi:10.1016/j.exger.2012.12.004)
26. Tidière M, Gaillard JM, Berger V, Müller DWH, Bingaman Lackey L, Gimenez O, Clauss M, Lemaître JF. 2016 Comparative analyses of longevity and senescence reveal variable survival benefits of living in zoos across mammals. *Sci. Rep.* **6**, 36361. (doi:10.1038/srep36361)
27. Colchero F *et al*. 2021 The long lives of primates and the 'invariant rate of ageing' hypothesis. *Nat. Commun.* **12**, 3666. (doi:10.1038/s41467-021-23894-3)
28. Newman LE *et al*. 2023 The biology of aging in a social world: insights from free-ranging rhesus macaques. *Neurosci. Biobehav. Rev.* **154**, 105424. (doi:10.1016/j.neubiorev.2023.105424)
29. Tung J, Lange EC, Alberts SC, Archie EA. 2023 Social and early life determinants of survival from cradle to grave: a case study in wild baboons. *Neurosci. Biobehav. Rev.* **152**, 105282. (doi:10.1016/j.neubiorev.2023.105282)
30. Emery Thompson M *et al*. 2020 Wild chimpanzees exhibit humanlike aging of glucocorticoid regulation. *Proc. Natl Acad. Sci. USA* **117**, 8424–8430. (doi:10.1073/pnas.1920593117)
31. Snyder-Mackler N *et al*. 2020 Social determinants of health and survival in humans and other animals. *Science* **368**, eaax9553. (doi:10.1126/science.aax9553)
32. Phillips KA *et al*. 2014 Why primate models matter. *Am. J. Primatol.* **76**, 801–827. (doi:10.1002/ajp.22281)
33. Justice JN, Cesari M, Seals DR, Shively CA, Carter CS. 2016 Comparative approaches to understanding the relation between aging and physical function. *J. Gerontol. A***71**, 1243–1253. (doi:10.1093/gerona/glv035)
34. Colman RJ. 2018 Non-human primates as a model for aging. *Biochim. Biophys. Acta Mol. Basis Dis.* **1864**, 2733–2741. (doi:10.1016/j.bbadis.2017.07.008)

35. Nussey DH, Froy H, Lemaitre JF, Gaillard JM, Austad SN. 2013 Senescence in natural populations of animals: widespread evidence and its implications for bio-gerontology. *Ageing Res. Rev*. **12**, 214–225. (doi:10.1016/j.arr.2012.07.004)
36. Roach DA, Carey JR. 2014 Population biology of aging in the wild. *Annu. Rev. Ecol. Evol. Syst.* **45**, 421–443. (doi:10.1146/annurev-ecolsys-120213-091730)
37. Mauvais-Jarvis F *et al*. 2020 Sex and gender: modifiers of health, disease, and medicine. *Lancet* **396**, 565–582. (doi:10.1016/S0140-6736(20)31561-0)
38. De Petrillo F, Rosati AG. 2021 Variation in primate decision-making under uncertainty and the roots of human economic behaviour. *Phil. Trans. R. Soc. B* **376**, 20190671. (doi:10.1098/rstb.2019.0671)
39. Conti G, Hansman C, Heckman JJ, Novak MFX, Ruggiero A, Suomi SJ. 2012 Primate evidence on the late health effects of early-life adversity. *Proc. Natl Acad. Sci. USA* **109**, 8866–8871. (doi:10.1073/pnas.1205340109)
40. Shively CA, Clarkson TB. 2009 The unique value of primate models in translational research. *Am. J. Primatol.* **71**, 715–721. (doi:10.1002/ajp.20720)
41. Alberts SC, Altmann J. 2012 The Amboseli baboon research project: 40 years of continuity and change. In *Long-term field studies of primates* (eds PM Kappeler, DP Watts), pp. 261–287. Berlin, Germany: Springer. (doi:10.1007/978-3-642-22514-7_12)
42. Thompson ME, Muller MN, Machanda ZP, Otali E, Wrangham RW. 2020 The Kibale chimpanzee project: over thirty years of research, conservation, and change. *Biol. Conserv*. **252**, 108857. (doi:10.1016/j.biocon.2020.108857)
43. Wilson ML *et al*. 2020 Research and conservation in the Greater Gombe ecosystem: challenges and opportunities. *Biol. Conserv*. **252**, 108853. (doi:10.1016/j.biocon.2020.108853)
44. Austad SN. 1997 Comparative aging and life histories in mammals. *Exp. Gerontol*. **32**, 23–38. (doi:10.1016/s0531-5565(96)00059-9)
45. Lu AT *et al*. 2023 Universal DNA methylation age across mammalian tissues. *Nat. Aging* **3**, 1144–1166. (doi:10.1038/s43587-023-00462-6)
46. Horvath S. 2013 DNA methylation age of human tissues and cell types. *Genome Biol*. **14**, R115. (doi:10.1186/gb-2013-14-10-r115)
47. Orkin JD *et al*. 2021 The genomics of ecological flexibility, large brains, and long lives in capuchin monkeys revealed with fecalFACS. *Proc. Natl Acad. Sci. USA* **118**, e2010632118. (doi:10.1073/pnas.2010632118)
48. Higham JP. 2016 Field endocrinology of nonhuman primates: past, present, and future. *Horm. Behav*. **84**, 145–155. (doi:10.1016/j.yhbeh.2016.07.001)
49. Higham JP, Kraus C, Stahl-Hennig C, Engelhardt A, Fuchs D, Heistermann M. 2015 Evaluating noninvasive markers of nonhuman primate immune activation and inflammation. *Am. J. Phys. Anthropol*. **158**, 673–684. (doi:10.1002/ajpa.22821)
50. Heistermann M, Higham JP. 2015 Urinary neopterin, a non-invasive marker of mammalian cellular immune activation, is highly stable under field conditions. *Sci. Rep*. **5**, 16308. (doi:10.1038/srep16308)
51. Tinsley Johnson E, Benítez ME, Fuentes A, McLean CR, Norford AB, Ordoñez JC, Beehner JC, Bergman TJ. 2020 High density of white-faced capuchins (*Cebus capucinus*) and habitat quality in the Taboga Forest of Costa Rica. *Am. J. Primatol*. **82**, e23096. (doi:10.1002/ajp.23096)
52. Melin AD *et al*. 2020 Primate life history, social dynamics, ecology, and conservation: contributions from long-term research in área de conservación Guanacaste, Costa Rica. *Biotropica* **52**, 1041–1064. (doi:10.1111/btp.12867)
53. Perry S, Godoy I, Lammers W. 2012 The Lomas Barbudal monkey project: two decades of research on *Cebus capucinus*. In *Long-term field studies of primates* (eds PM Kappeler, DP Watts), pp. 141–163. Berlin, Germany: Springer.
54. Fedigan LM, Jack KM. 2012 Tracking neotropical monkeys in Santa Rosa: lessons from a regenerating Costa Rican dry forest. In *Long-term field studies of primates* (eds PM Kappeler, DP Watts), pp. 165–184. Berlin, Germany: Springer. (doi:10.1007/978-3-642-22514-7_8)
55. Beehner JC *et al*. 2022 Using an on-site laboratory for fecal steroid analysis in wild white-faced capuchins. *Gen. Comp. Endocrinol*. **329**, 114109. (doi:10.1016/j.ygcen.2022.114109)
56. Fedigan LM, Jack KM. 2011 Two girls for every boy: the effects of group size and composition on the reproductive success of male and female white-faced capuchins. *Am. J. Phys. Anthropol.* **144**, 317–326. (doi:10.1002/ajpa.21414)
57. Strier KB *et al*. 2010 The primate life history database: a unique shared ecological data resource. *Methods Ecol. Evol.* **1**, 199–211. (doi:10.1111/j.2041-210X.2010.00023.x)
58. Bronikowski AM *et al*. 2011 Aging in the natural world: comparative data reveal similar mortality patterns across primates. *Science* **331**, 1325–1328. (doi:10.1126/science.1201571)
59. Colchero F *et al*. 2016 The emergence of longevous populations. *Proc. Natl Acad. Sci. USA* **113**, E7681–E7690. (doi:10.1073/pnas.1612191113)
60. Campos FA *et al*. 2017 Does climate variability influence the demography of wild primates? Evidence from long-term life-history data in seven species. *Glob. Chang. Biol*. **23**, 4907–4921. (doi:10.1111/gcb.13754)
61. Zipple MN *et al*. 2021 Maternal death and offspring fitness in multiple wild primates. *Proc. Natl Acad. Sci. USA* **118**, e2015317118. (doi:10.1073/pnas.2015317118)
62. Alberts SC *et al*. 2013 Reproductive aging patterns in primates reveal that humans are distinct. *Proc. Natl Acad. Sci. USA* **110**, 13440–13445. (doi:10.1073/pnas.1311857110)
63. Campos FA *et al*. 2022 Female reproductive aging in seven primate species: patterns and consequences. *Proc. Natl Acad. Sci. USA* **119**, e2117669119. (doi:10.1073/pnas.2117669119)
64. Caruana EJ, Roman M, Hernández-Sánchez J, Solli P. 2015 Longitudinal studies. *J. Thorac. Dis*. **7**, E537–40. (doi:10.3978/j.issn.2072-1439.2015.10.63)
65. Jones JH. 2011 Primates and the evolution of long, slow life histories. *Curr. Biol*. **21**, R708–17. (doi:10.1016/j.cub.2011.08.025)
66. Dicke U, Roth G. 2016 Neuronal factors determining high intelligence. *Phil. Trans. R. Soc. B* **371**, 20150180. (doi:10.1098/rstb.2015.0180)
67. Eisenberg JF. 1979 Habitat, economy, and society: some correlations and hypotheses for the neotropical primates. In *Primate ecology and human origins: ecological influences on social organization* (eds IS Bernstein, E Smith), pp. 215–262. New York, NY: Garland Press.
68. de Waal FB, Berger ML. 2000 Payment for labour in monkeys. *Nature* **404**, 563. (doi:10.1038/35007138)
69. de Waal FBM, Davis JM. 2003 Capuchin cognitive ecology: cooperation based on projected returns. *Neuropsychologia* **41**, 221–228. (doi:10.1016/s0028-3932(02)00152-5)
70. Brosnan SF, Waal FBM, De. 2003 Monkeys reject unequal pay. *Nature* **425**, 297–299. (doi:10.1038/nature01963)
71. Ottoni EB, Izar P. 2008 Capuchin monkey tool use: overview and implications. *Evol. Anthropol.* **17**, 171–178. (doi:10.1002/evan.20185)
72. Conde DA *et al*. 2019 Data gaps and opportunities for comparative and conservation biology. *Proc. Natl Acad. Sci. USA* **116**, 9658–9664. (doi:10.1073/pnas.1816367116)
73. Perry S. 2012 The behavior of wild white-faced capuchins: demography, life history, social relationships, and communication. In *Advances in the study of behavior* (eds HJ Brockmann, TJ Roper, M Naguib, JC Mitani, LW Simmons), pp. 135–181. London, UK: Academic Press. (doi:10.1016/B978-0-12-394288-3.00004-6)
74. Weigl R. 2005 *Longevity of mammals in captivity: from the living collections of the world: a list of mammalian longevity in captivity*. Stuttgart, Germany: Schweizerbart Science Publishers.
75. Campos FA. 2018 A synthesis of long-term environmental change in Santa Rosa, Costa Rica. In *Primate life histories, sex roles, and adaptability* (eds U Kalbitzer, KM Jack), pp. 331–358. Berlin, Germany: Springer. (doi:10.1007/978-3-319-98285-4_16)
76. Robbins Schug G *et al*. 2023 Climate change, human health, and resilience in the Holocene. *Proc. Natl Acad. Sci. USA* **120**, e2209472120. (doi:10.1073/pnas.2209472120)

77. Melin AD, Young HC, Mosdossy KN, Fedigan LM. 2014 Seasonality, extractive foraging and the evolution of primate sensorimotor intelligence. *J. Hum. Evol*. **71**, 77–86. (doi:10.1016/j.jhevol.2014.02.009)
78. Orkin JD, Campos FA, Myers MS, Cheves Hernandez SE, Guadamuz A, Melin AD. 2019 Seasonality of the gut microbiota of free-ranging white-faced capuchins in a tropical dry forest. *ISME J*. **13**, 183–196. (doi:10.1038/s41396-018-0256-0)
79. Bergstrom ML, Kalbitzer U, Campos FA, Melin AD, Emery Thompson M, Fedigan LM. 2020 Non-invasive estimation of the costs of feeding competition in a neotropical primate. *Horm. Behav*. **118**, 104632. (doi:10.1016/j.yhbeh.2019.104632)
80. Schoof VAM, Bonnell TR, Jack KM, Ziegler TE, Melin AD, Fedigan LM. 2016 Male endocrine response to seasonally varying environmental and social factors in a neotropical primate, *Cebus capucinus*. *Am. J. Phys. Anthropol*. **159**, 671–682. (doi:10.1002/ajpa.22925)
81. Griffin RH, Nunn CL. 2012 Community structure and the spread of infectious disease in primate social networks. *Evol. Ecol*. **26**, 779–800. (doi:10.1007/s10682-011-9526-2)
82. Titcomb G, Mantas JN, Hulke J, Rodriguez I, Branch D, Young H. 2021 Water sources aggregate parasites with increasing effects in more arid conditions. *Nat. Commun*. **12**, 7066. (doi:10.1038/s41467-021-27352-y)
83. Parr NA, Fedigan LM, Kutz SJ. 2013 Predictors of parasitism in wild white-faced capuchins (*Cebus capucinus*). *Int. J. Primatol*. **34**, 1137–1152. (doi:10.1007/s10764-013-9728-2)
84. Pinto SL, Henriquez MC, Cheves Hernandez S, Duytschaever G, Wit J, Avramenko RW, Gilleard JS, Orkin JD, Melin AD. 2023 Promise and limitations of 18S genetic screening of extracted fecal DNA from wild capuchins. *Front. Ecol. Evol.* **11**, 1176681. (doi:10.3389/fevo.2023.1176681)
85. Oliveira A, Santos R. 2023 Infectious diseases of neotropical primates. *Braz. J. Vet. Pathol.* **16**, 1–34. (doi:10.24070/bjvp.1983-0246.v16i1p1-34)
86. Gurven M, Kaplan H. 2007 Longevity among hunter–gatherers: a cross-cultural examination. *Popul. Dev. Rev.* **33**, 321–365. (doi:10.1111/j.1728-4457.2007.00171.x)
87. Negrey JD *et al*. 2020 Demography, life-history trade-offs, and the gastrointestinal virome of wild chimpanzees. *Phil. Trans. R. Soc. B* **375**, 20190613. (doi:10.1098/rstb.2019.0613)
88. Campos FA, Jack KM, Fedigan LM. 2015 Climate oscillations and conservation measures regulate white-faced capuchin population growth and demography in a regenerating tropical dry forest in Costa Rica. *Biol. Conserv.* **186**, 204–213. (doi:10.1016/j.biocon.2015.03.017)
89. Campos FA *et al*. 2020 Differential impact of severe drought on infant mortality in two sympatric neotropical primates. *R. Soc. Open Sci.* **7**, 200302. (doi:10.1098/rsos.200302)
90. Roseboom TJ, van der Meulen JH, Ravelli AC, Osmond C, Barker DJ, Bleker OP. 2001 Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Mol. Cell. Endocrinol.* **185**, 93–98. (doi:10.1016/s0303-7207(01)00721-3)
91. Perry S. 2020 Behavioural variation and learning across the lifespan in wild white-faced capuchin monkeys. *Phil. Trans. R. Soc. B* **375**, 20190494. (doi:10.1098/rstb.2019.0494)
92. Williamson RE, Webb SE, Dubreuil C, Lopez R, Cheves Hernandez S, Fedigan LM, Melin AD. 2021 Sharing spaces: niche differentiation in diet and substrate use among wild capuchin monkeys. *Anim. Behav.* **179**, 317–338. (doi:10.1016/j.anbehav.2021.06.002)
93. Perry SE, Barrett BJ, Godoy I. 2017 Older, sociable capuchins (*Cebus capucinus*) invent more social behaviors, but younger monkeys innovate more in other contexts. *Proc. Natl Acad. Sci. USA* **114**, 7806–7813. (doi:10.1073/pnas.1620739114)
94. Barrett BJ, Monteza-Moreno CM, Dogandžić T, Zwyns N, Ibáñez A, Crofoot MC. 2018 Habitual stone-tool-aided extractive foraging in white-faced capuchins. *R. Soc. Open Sci.* **5**, 181002. (doi:10.1098/rsos.181002)
95. Brown DW, Anda RF, Tiemeier H, Felitti VJ, Edwards VJ, Croft JB, Giles WH. 2009 Adverse childhood experiences and the risk of premature mortality. *Am. J. Prev. Med*. **37**, 389–396. (doi:10.1016/j.amepre.2009.06.021)
96. Brasington LF, Wikberg EC, Kawamura S, Fedigan LM, Jack KM. 2017 Infant mortality in white-faced capuchins: the impact of alpha male replacements. *Am. J. Primatol*. **79**. (doi:10.1002/ajp.22725)
97. Jack K, Fedigan LM. 2004 The demographic and reproductive context of male replacements in *Cebus capucinus*. *Behaviour* **141**, 755–775. (doi:10.1163/1568539042245178)
98. Fedigan LM, Jack KM. 2013 Sexual conflict in white-faced capuchins. In *Evolution's empress* (eds ML Fisher, JR Garcia), pp. 281–303. New York, NY: Oxford University Press.
99. Fedigan LM, Hogan JD, Campos FA, Kalbitzer U, Jack KM. 2021 Costs of male infanticide for female capuchins: when does an adaptive male reproductive strategy become costly for females and detrimental to population viability? *Am. J. Phys. Anthropol*. **176**, 349–360. (doi:10.1002/ajpa.24354)
100. Kalbitzer U, Bergstrom ML, Carnegie SD, Wikberg EC, Kawamura S, Campos FA, Jack KM, Fedigan LM. 2017 Female sociality and sexual conflict shape offspring survival in a neotropical primate. *Proc. Natl Acad. Sci. USA* **114**, 1892–1897. (doi:10.1073/pnas.1608625114)
101. Hill KR *et al*. 2011 Co-residence patterns in hunter–gatherer societies show unique human social structure. *Science* **331**, 1286–1289. (doi:10.1126/science.1199071)
102. Rodseth L, Wrangham R. 2004 Human kinship: a continuation of politics by other means. In *Kinship and behavior in primates* (eds B Chapais, CM Berman), pp. 389–419. Oxford, UK: Oxford University Press.
103. Wikberg EC, Jack KM, Fedigan LM, Campos FA, Yashima AS, Bergstrom ML, Hiwatashi T, Kawamura S. 2017 Inbreeding avoidance and female mate choice shape reproductive skew in capuchin monkeys (Cebus capucinus imitator). *Mol. Ecol.* **26**, 653–667. (doi:10.1111/mec.13898)
104. Kajokaite K, Whalen A, Koster J, Perry S. 2022 Social integration predicts survival in female white-faced capuchin monkeys. *Behav. Ecol*. **33**, 807–815. (doi:10.1093/beheco/arac043)
105. Wikberg EC, Jack KM, Fedigan LM, Kawamura S. 2018 The effects of dispersal and reproductive patterns on the evolution of male sociality in white-faced capuchins. In *Primate life histories, sex roles, and adaptability: essays in honour of Linda M. Fedigan* (eds U Kalbitzer, KM Jack), pp. 117–132. Cham, Switzerland: Springer International Publishing.
106. van Schaik CP, Pandit SA, Vogel ER. 2006 Toward a general model for male–male coalitions in primate groups. In *Cooperation in primates and humans* (eds PM Kappeler, CP Schaik), pp. 151–171. Berlin, Germany: Springer.
107. Bergstrom ML, Fedigan LM. 2013 Dominance style of female white-faced capuchins. *Am. J. Phys. Anthropol*. **150**, 591–601. (doi:10.1002/ajpa.22231)
108. Schoof VAM, Jack KM. 2014 Male social bonds: strength and quality among co-resident white-faced capuchin monkeys (*Cebus capucinus*). *Behaviour* **151**, 963–992. (doi:10.1163/1568539X-00003179)
109. Perry S, Godoy I, Lammers W, Lin A. 2017 Impact of personality traits and early life experience on timing of emigration and rise to alpha male status for wild male white-faced capuchin monkeys (Cebus capucinus) at Lomas Barbudal biological reserve, Costa Rica. *Behaviour* **154**, 195–226. (doi:10.1163/1568539X-00003418)
110. Wikberg EC, Jack KM, Campos FA, Bergstrom ML, Kawamura S, Fedigan LM. 2022 Should I stay or should I go now: dispersal decisions and reproductive success in male white-faced capuchins (*Cebus imitator*). *Behav. Ecol. Sociobiol.* **76**, 88. (doi:10.1007/s00265-022-03197-3)
111. Grossmann I, Na J, Varnum MEW, Park DC, Kitayama S, Nisbett RE. 2010 Reasoning about social conflicts improves into old age. *Proc. Natl Acad. Sci. USA* **107**, 7246–7250. (doi:10.1073/pnas.1001715107)
112. Henry JD, Phillips LH, Ruffman T, Bailey PE. 2013 A meta-analytic review of age differences in theory of mind. *Psychol. Aging* **28**, 826–839. (doi:10.1037/a0030677)
113. Happé FGE, Winner E, Brownell H. 1998 The getting of wisdom: theory of mind in old age. *Dev. Psychol.* **34**, 358–362. (doi:10.1037//0012-1649.34.2.358)
114. Fischer J. 2017 On the social life and motivational changes of aging monkeys. *Gerontology* **63**, 572–579. (doi:10.1159/000477401)
115. López-Otín C, Blasco MA, Partridge L, Serrano M, Kroemer G. 2013 The hallmarks of aging. *Cell* **153**, 1194–1217. (doi:10.1016/j.cell.2013.05.039)

116. Sierra F. 2016 The emergence of geroscience as an interdisciplinary approach to the enhancement of health span and life span. *Cold Spring Harb. Perspect. Med*. **6**, a025163. (doi:10.1101/cshperspect.a025163)
117. Santilli V, Bernetti A, Mangone M, Paoloni M. 2014 Clinical definition of sarcopenia. *Clin. Cases Miner. Bone Metab*. **11**, 177–180. (doi:10.11138/ccmbm/2014.11.3.177)
118. Colman RJ, McKiernan SH, Aiken JM, Weindruch R. 2005 Muscle mass loss in Rhesus monkeys: age of onset. *Exp. Gerontol*. **40**, 573–581. (doi:10.1016/j.exger.2005.05.001)
119. Thompson ME, Machanda ZP, Fox SA, Sabbi KH, Otali E, González NT, Muller MN, Wrangham RW. 2020 Evaluating the impact of physical frailty during ageing in wild chimpanzees (*Pan troglodytes schweinfurthii*). *Phil. Trans. R. Soc. B* **375**, 20190607. (doi:10.1098/rstb.2019.0607)
120. Liberale L, Montecucco F, Tardif JC, Libby P, Camici GG. 2020 Inflamm-ageing: the role of inflammation in age-dependent cardiovascular disease. *Eur. Heart J.* **41**, 2974–2982. (doi:10.1093/eurheartj/ehz961)
121. Akiyama H *et al*. 2000 Inflammation and Alzheimer's disease. *Neurobiol. Aging* **21**, 383–421. (doi:10.1016/s0197-4580(00)00124-x)
122. Proctor MJ, McMillan DC, Horgan PG, Fletcher CD, Talwar D, Morrison DS. 2015 Systemic inflammation predicts all-cause mortality: a Glasgow inflammation outcome study. *PLoS One* **10**, e0116206. (doi:10.1371/journal.pone.0116206)
123. Galkin F, Mamoshina P, Aliper A, Putin E, Moskalev V, Gladyshev VN, Zhavoronkov A. 2020 Human gut microbiome aging clock based on taxonomic profiling and deep learning. *iScience* **23**, 101199. (doi:10.1016/j.isci.2020.101199)
124. Burch JB *et al*. 2014 Advances in geroscience: impact on healthspan and chronic disease. *J. Gerontol. A* **69**, S1–3. (doi:10.1093/gerona/glu041)
125. Kirkland JL. 2013 Translating advances from the basic biology of aging into clinical application. *Exp. Gerontol*. **48**, 1–5. (doi:10.1016/j.exger.2012.11.014)
126. Justice JN, Carter CS, Beck HJ, Gioscia-Ryan RA, McQueen M, Enoka RM, Seals DR. 2014 Battery of behavioral tests in mice that models age-associated changes in human motor function. *Age***36**, 583–592. (doi:10.1007/s11357-013-9589-9)
127. Shively CA *et al*. 2012 Aging and physical mobility in group-housed Old World monkeys. *Age* **34**, 1123–1131. (doi:10.1007/s11357-011-9350-1)
128. Carter CS, Sonntag WE, Onder G, Pahor M. 2002 Physical performance and longevity in aged rats. *J. Gerontol. A* **57**, B193–7. (doi:10.1093/gerona/57.5.b193)
129. Graber TG, Maroto R, Fry CS, Brightwell CR, Rasmussen BB. 2021 Measuring exercise capacity and physical function in adult and older mice. *J. Gerontol. A* **76**, 819–824. (doi:10.1093/gerona/glaa205)
130. Ross CN *et al*. 2019 Cross-sectional comparison of health-span phenotypes in young versus geriatric marmosets. *Am. J. Primatol*. **81**, e22952. (doi:10.1002/ajp.22952)
131. Cooper R, Kuh D, Hardy R, Mortality Review Group. 2010 Objectively measured physical capability levels and mortality: systematic review and meta-analysis. *BMJ* **341**, c4467. (doi:10.1136/bmj.c4467)
132. Fried LP, Cohen AA, Xue QL, Walston J, Bandeen-Roche K, Varadhan R. 2021 The physical frailty syndrome as a transition from homeostatic symphony to cacophony. *Nat. Aging* **1**, 36–46. (doi:10.1038/s43587-020-00017-z)
133. Okamoto N, Amano N, Nakamura T, Yanagi M. 2019 Relationship between tooth loss, low masticatory ability, and nutritional indices in the elderly: a cross-sectional study. *BMC Oral Health* **19**, 110. (doi:10.1186/s12903-019-0778-5)
134. Yang YC, Boen C, Gerken K, Li T, Schorpp K, Harris KM. 2016 Social relationships and physiological determinants of longevity across the human life span. *Proc. Natl Acad. Sci. USA* **113**, 578–583. (doi:10.1073/pnas.1511085112)
135. Holt-lunstad J, Smith T, Layton JB. 2010 Social relationships and mortality risk: a meta-analytic review. *PLoS Med.* **7**, e1000316.
136. Campos FA, Villavicencio F, Archie EA, Colchero F, Alberts SC. 2020 Social bonds, social status and survival in wild baboons: a tale of two sexes. *Phil. Trans. R. Soc. B* **375**, 20190621. (doi:10.1098/rstb.2019.0621)
137. Moffitt TE, Belsky DW, Danese A, Poulton R, Caspi A. 2017 The Longitudinal Study of Aging in Human Young Adults: knowledge gaps and research agenda. *J. Gerontol. A* **72**, 210–215. (doi:10.1093/gerona/glw191)
138. Belsky DW *et al*. 2015 Quantification of biological aging in young adults. *Proc. Natl Acad. Sci. USA* **112**, E4104–10. (doi:10.1073/pnas.1506264112)
139. Belsky DW *et al*. 2020 Quantification of the pace of biological aging in humans through a blood test, the DunedinPoAm DNA methylation algorithm. *eLife* **9**, e54870. (doi:10.7554/eLife.54870)
140. Power C, Kuh D, Morton S. 2013 From developmental origins of adult disease to life course research on adult disease and aging: insights from birth cohort studies. *Annu. Rev. Public Health* **34**, 7–28. (doi:10.1146/annurev-publhealth-031912-114423)
141. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, Koss MP, Marks JS. 1998 Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *Am. J. Prev. Med*. **14**, 245–258. (doi:10.1016/S0749-3797(98)00017-8)
142. Miller GE, Chen E, Fok AK, Walker H, Lim A, Nicholls EF, Cole S, Kobor MS. 2009 Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proc. Natl Acad. Sci. USA* **106**, 14716–14721. (doi:10.1073/pnas.0902971106)
143. Ehrlich KB, Ross KM, Chen E, Miller GE. 2016 Testing the biological embedding hypothesis: is early life adversity associated with a later proinflammatory phenotype? *Dev. Psychopathol*. **28**, 1273–1283. (doi:10.1017/S0954579416000845)
144. Danese A *et al*. 2011 Biological embedding of stress through inflammation processes in childhood. *Mol. Psychiatry* **16**, 244–246. (doi:10.1038/mp.2010.5)
145. Rasmussen LJH. 2019 Cumulative childhood risk is associated with a new measure of chronic inflammation in adulthood. *Child Psych. Psychiatry* **60**, 199–208. (doi:10.1111/jcpp.12928)
146. Hertzman C, Boyce T. 2010 How experience gets under the skin to create gradients in developmental health. *Annu. Rev. Public Health* **31**, 329–47 (doi:10.1146/annurev.publhealth.012809.103538)
147. Tung J, Archie EA, Altmann J, Alberts SC. 2016 Cumulative early life adversity predicts longevity in wild baboons. *Nat. Commun*. **7**, 11181. (doi:10.1038/ncomms11181)
148. Strauss ED, Shizuka D, Holekamp KE. 2020 Juvenile rank acquisition is associated with fitness independent of adult rank. *Proc. Biol. Sci.* **287**, 20192969. (doi:10.1098/rspb.2019.2969)
149. Patterson SK *et al*. 2024 Early life adversity has sex-dependent effects on survival across the lifespan in rhesus macaques. *Phil. Trans. R. Soc. B.* **379**, 20220456. (doi:10.1098/rtsb.2022.0456)
150. Cohen S, Wills TA. 1985 Stress, social support, and the buffering hypothesis. *Psychol. Bull*. **98**, 310–357. (doi:10.1037/0033-2909.98.2.310)
151. Hatch SL. 2005 Conceptualizing and identifying cumulative adversity and protective resources: implications for understanding health inequalities. *J. Gerontol. B* **60**, S130–S134. (doi:10.1093/geronb/60.Special_Issue_2.S130)
152. Ferraro KF, Shippee TP. 2009 Aging and cumulative inequality: how does inequality get under the skin? *Gerontologist* **49**, 333–343. (doi:10.1093/geront/gnp034)
153. Willson AE, Shuey KM, Elder Jr GH. 2007 Cumulative advantage processes as mechanisms of inequality in life course health. *Am. J. Sociol*. **112**, 1886–1924. (doi:10.1086/512712)
154. Lea AJ, Martins D, Kamau J, Gurven M, Ayroles JF. 2020 Urbanization and market integration have strong, nonlinear effects on cardiometabolic health in the Turkana. *Sci. Adv*. **6**. (doi:10.1126/sciadv.abb1430)

155. Ferrucci L, Gonzalez-Freire M, Fabbri E, Simonsick E, Tanaka T, Moore Z, Salimi S, Sierra F, de Cabo R. 2020 Measuring biological aging in humans: a quest. *Aging Cell* **19**, e13080. (doi:10.1111/acel.13080)
156. Rosenbaum S, Zeng S, Campos FA, Gesquiere LR, Altmann J, Alberts SC, Li F, Archie EA. 2020 Social bonds do not mediate the relationship between early adversity and adult glucocorticoids in wild baboons. *Proc. Natl Acad. Sci. USA* **117**, 20052–20062. (doi:10.1073/pnas.2004524117)
157. Gurven M, Stieglitz J, Trumble B, Blackwell AD, Beheim B, Davis H, Hooper P, Kaplan H. 2017 The Tsimane Health and Life History Project: integrating anthropology and biomedicine. *Evol. Anthropol*. **26**, 54–73. (doi:10.1002/evan.21515)
158. Kappeler PM, Watts DP (eds). 2012 *Long-term field studies of primates*. Berlin, Germany: Springer.