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Mammalian brain development and our grandmothering life history

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ABSTRACT

Among mammals, including humans, adult brain size and the relative size of brain components depend precisely on the duration of a highly regular process of neural development. Much wider variation is seen in rates of body growth and the state of neural maturation at life history events like birth and weaning. Large brains result from slow maturation, which in humans is accompanied by weaning early with respect to both neural maturation and longevity. The grandmother hypothesis proposes this distinctive combination of life history features evolved as ancestral populations began to depend on foods that just weaned juveniles couldn't handle. Here we trace possible reciprocal connections between brain development and life history, highlighting the resulting extended neural plasticity in a wider cognitive ecology of allomaternal care that distinguishes human ontogeny with consequences for other peculiarities of our lineage.

1. Introduction

Evolutionary anthropologists, developmental psychologists and neurobiologists all have something to say about human development and evolution, but differ profoundly in what they hope to explain. Academic fields exist in part for the pragmatic reason that no scientist can routinely consider the advanced details of all realms of knowledge. Idiosyncratic x- and y-axes generate each field's data representations, their customary "independent" versus "dependent" variables profoundly influencing the questions they focus on and each field's views of how causal arrows point. Periodic attempts at alignment and integration are therefore essential. Here we propose to align evolutionary anthropology, psychology and neurobiology on the general subject of human development and demographic life history. In particular, we will describe the regularities in brain development across placental mammals including humans. We will connect those regularities to life history associations between duration of development and longevity, privileging the hypothesis that human post-menopausal longevity evolved as a consequence of allomaternal subsidies from ancestral grandmothers. Subsidies allowed mothers to wean infants still very dependent and so widened the cast of interacting characters at an early point in their neural development. Each field has produced its insights and narratives on this central subject in relative independence. As coauthors from different fields, we are not always in agreement, except on the importance of integrating these different lines of evidence about human development and evolution.

1.1. Anthropology versus psychology

Two contemporary misalignments, observed informally, will illustrate the divergence independent lines of explanation have produced. If an evolutionary anthropologist asks a group of psychologists how early development might differ between humans and non-human primates, the psychologists will typically center on the importance of the mother-infant bond for social and cognitive adjustment. If they are asked then to guess how early or late humans are weaned compared to other great apes, they will map "importance" onto duration, and hazard that human infants are dependent longer and so must require greater maternal effort, spend a longer time with their mothers and therefore nurse longer. Yet, evolutionary anthropologists comparing humans to our closest living relatives, the great apes have recognized for more that two decades that humans wean earlier, and space births more closely than they do [134].

For a converse example, if cognitive scientists ask anthropologists how large human brains have evolved, anthropologists will most often invoke selection on the size or properties of individual cortical areas,

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which highlights the critical role of selection over evolutionary time on particular behavioral competencies. Understanding improvement in a particular behavioral competency as selection on the size of a particular brain region can trace its lineage to Jerison's idea of "proper mass" (1973) [74] coupled with the earliest examples of lesion-symptom mapping in neurology.

Therefore, for example, increase in the size of "Broca's area" and immediately associated tracts in the cerebral cortex might be used as an index of selection on language competency [10,122]. The distributed, overlapping and network-based views of neural mechanisms arising in current neurology, cognitive and computational neuroscience, however, are increasingly at odds with this modular view [5,114,135].

1.2. Evolution of life histories, developmental neurobiology and evo-devo

The general accumulation and systematization of knowledge about the progress of life from conception, birth, development, sexual maturity and death across vertebrates will be found across multiple academic departments, principally different aspects of biology. As we will be discussing features of human life history in the context of primates, and great apes, and more generally mammals, we will be considering general control of growth and behavioral development, as well as specific events like birth, weaning, duration of childhood, sexual maturity, menopause and death, as they are typically studied in anthropology departments. This research concerns itself with description of basic commonality and diversity in strategies; interaction of life histories with particular ecologies; and costs, benefits, and contests in resource allocation. Theorizing in this area typically involves discovering the ecological and social determinates of developmental trajectories, where energetics have long been of special interest for human evolution (e.g., [1,69,70,93,94]) and are increasingly described more precisely [89,115,116]. But the mechanisms of development, except as realized, for example, in the energetic requirements of producing a brain of a certain size over a certain time, are not considered.

Developmental neurobiology, for the most of its history, did not focus on the evolution and the production of diversity among organisms. The generation and placement of neurons, wiring them up, placing them in environments and specifying initial parameters typically take place in a generic fruit fly-mouse-rhesus nervous system.

In the last twenty years, this gap in integration has been bridged by the joint study of evolution and development, "evo-devo". Many insights have been generated, but the central one we address here is what Kirschner and Gerhart [84] have called the "third pillar of Darwinism."

"Darwin's all-encompassing theory of evolution was based on three major supports: a theory of natural selection, a theory of heredity, and a theory of the generation of variation in the organism".

The first two pillars have been extensively studied, ever since Darwin. The last pillar, examination of and theorizing about the variation offered to selection by the organism, became structurally significant to our understanding when rapid cataloguing of the genome across organisms became possible. The first rudimentary steps in decoding the mechanistic path from genome to phenotype produced major surprises. The variation offered for selection by the genomes of existing creatures was anything but random. Random, point-by-point mutations of single DNA base-pairs, first guessed to be the main cause of variation, from "genetic drift" to "hopeful monsters" did occur, but their effects are typically made negligible by recurring replication of local sequences, large segments, and even entirety of the genome. Some fundamental components, notably the Hox and similar regulatory genes, which specify the major axes and components of the body and brain plans, have been conserved across invertebrates and vertebrates [110].

Moreover, evolution progressively filtered developmental mechanisms toward suites of control mechanisms that produced functional outcomes in the face of normal variation, either variation produced by the developing animal itself, or the environmental variation the animal will encounter. For example, in producing a limb, linking the generation of bone and muscle mass, vascularization and neural innervation to each other by molecular recognition and trophic interactions rather than separately generating and linking them post hoc is more efficient and less prone to end-stage failure [83]; in producing an eye, the length of the eye and the power of the lens and cornea are matched only roughly by genetics, but are brought to focus as the retina's activity (a measure of focus) slows eye growth as external light activates photoreceptors [143]. A single species may encounter resource-rich, stable environments or resource-poor unpredictable ones in its phylogenetic history, and come to be equipped with environment-appropriate multicomponent "game plans" executed after environmental quality is ascertained. This phenomenon has been much studied, but particularly as epigenetic responses to stress is another example [72].

"Evolvability" is the concept spanning these particular types of genomic variability and environmental interaction. The best way to describe the extreme conservation of basic body plan and organ structure, developmental programs coordinating functional systems, and prediction of environmental variability is under hot debate, as expected for a young research area [83]. Stable information may be distributed across genome and environment. It is not only the province of genes.

The overall goal of this article is to first, align basic facts about brains and life histories across the views of anthropology, psychology and neurobiology. Second, to the extent possible, we will try to integrate the views of anthropological examinations of life history and evo-devo approaches with each other. A review that spans G-proteins to grandmothers will present a different combination of challenge and boredom to each reader, but we will attempt to underline the relevance of the various necessary bodies of data to the eventual integration as we go.

Specifically, the features of human evolution we will consider in the frames of our disciplines are the following. First, human brains are very large, both absolutely and relatively compared to all mammals [74]. For primates overall, the best account of the evolutionary progression of brain size and body size is that body size was first reduced, producing relatively large brain sizes, and subsequently, in some radiations, larger brains evolved, placing those brains in the range of largest mammalian brains in both relative and absolute size [133]. Bodies are small for both age at maturity and longevity compared to non-primate mammals [20]. Since *relatively* larger brain size can arise from selection for smaller body size [133], widely used measures of brain size relative to body size, like encephalization indices, that incorporate both effects can obscure the separate roles of each. Our own hominid radiation, which includes great apes and humans, has the largest brains and bodies, longest offspring dependence, slowest maturations, and greatest longevities among all the primates [134]. Maturation is even slower in humans than other hominids, where it occurs in an intensely social context that varies across cultures [67]. Human females have unique and superficially contradictory life history features compared to other hominids: relatively earlier weaning with higher fertility, combined with longer duration of development and notably greater longevity even though latest last births occur at about the same age in women as in other great apes [59,134].

We will begin from developmental neurobiology, and present evidence about what mechanisms generate large brains, and what components these brains consist of. We will be interested in the interrelationships of the rate of brain growth, final brain mass, somatic growth, apparent maturational state both in morphology and behavior, and overall longevity. We will discuss what features like "extended maturation"

or "critical" or "sensitive" periods might correspond to structurally for complex abilities like language, or elaborated cognitive control. In the context of a changed social niche produced by changes in life history, we will highlight new evidence about comparative differences in brains other than size that could enable evolving humans to be maximally attentive to and able to learn from their changed social niche [28,45,141].

We aim to link this information from developmental neuroscience with theoretical and empirical work in demographic life history evolution. Life history theoreticians use stable population theory, which relies on the Euler-Lotka equation to explain the necessary interdependence of age- specific fertility and mortality rates throughout the lifespan [16,136]. Since the size of each cohort of newborns in age-structured populations depends on the number of females in the fertile ages and their rates of offspring production, and those females come from surviving cohorts of past newborns, the interdependencies result in a stable age distributions. In a stable population, the fraction in each age-class remains unchanged. A variant that alters the rate of fertility or mortality at any age can then affect the population growth rate. Comparing the growth rate of a variant to the growth rate of the background population indicates whether it would spread or decline against the common type (Hamilton, 1966, [16]). These consequences and the life histories of ancestral populations determine the range of life histories in immediate descendants.

Up to this point, however, the developmental mechanisms underpinning growth, fertility and longevity have usually been assumed to have multipotentiality by life history theorists. However, particular configurations of the genome (e.g. [6]) or developmental mechanisms filtered by survival through repeatedly encountered challenges [21,30], limit and channel the phenotypic space that adaptation and selection can explore.

As findings from different research agendas accumulate and more is known about the proximate mechanisms, ontogeny, phylogeny, and likely adaptive effects of any collection of features [140], the more findings in one line of inquiry establish the range of possibilities for the others. The emerging evo-devo field has challenged the idea that evolutionary history might ever be usefully understood separated from development. Particularly, multiple instances of extreme conservation and covariation of developmental mechanisms, sequences and their mature outcomes eliminate the idea that any aspect of mature phenotypes are the result of "random walks" through developmental mechanisms [84,144]. Our task here is to consider convergence among findings in the neurobiology of mammalian brain development with human life history evolution including the timing and character of allomaternal care. The close covariation of duration and rate of brain development with eventual brain size and organization, timing of release from full dependence on the mother coupled with the effects that both extended duration of maturation and relatively early weaning may offer neuroplasticity will be the linkages of concern here [22,35].

2. Generation and growth of mammalian brains

We will first discuss the problem of understanding relative volumes (or number of neurons) of brain parts in different mammals, as a very well-worked-out problem, to introduce results from comparing developmental duration across species. If the volume of neocortex is the focus, the fact that the human brain has a disproportionately large cortex is obvious (Fig. 1). This kind of comparison makes cortex volume appear to be an object of special evolutionary selection in primates, and in humans particularly, and it is often described this way.

2.1. Describing and representing allometric differences fairly

However, studying variation across species of different sizes and developmental durations requires care. Even with "all else equal" the laws of space and time, of physics and chemistry, impose changes in both form and process as size and time change, making allometries a subject of long interest [37]. Gould [46] underlined the pervasiveness of allometry as "…perhaps the major principle regulating basic differences in form among related animals. It explains, among many other things, why large animals have relatively thick legs and small brains, why dachshunds can't be as large as elephants, why flies can walk up walls, and why large homeotherms metabolize so much more slowly than small ones." Moreover, in addition to the intrinsic geometry that results in allometric relationships (e.g., doubling the volume of a sphere lengthens its radius only 1.26 times), datasets we develop and explore have an underlying geometry that can mislead comparisons if represented "unfairly." Consider the Mercator projection of the earth's landmasses, where the continents of Africa and Greenland appear approximately equal in size, but when measured in its correct spherical coordinates, Africa is more than 10 times larger than Greenland.

Allometries are conventionally represented as scaling relationships. If the relationship between two features that correlate with each other in size, say 'x' and 'y,' is represented as $y = kx^a$ where 'k' is some constant, the exponent 'a' represents the rate at which 'y' changes with respect to a change in 'x.' If 'k' and 'a' both equal 1, then x = y. If 'k' is more or less than 1 but 'a' equals 1, they differ in size but their relationship is isometric. If 'a' is more or less than 1 then a change in 'y' is associated with an exponential change in 'x.' Exponential relationships can be plotted and visualized as linear ones by logarithmic transformation: $\log y = a\log x + \log k$. The exponent 'a' is the slope of the change in 'y' with respect to a change in 'x.'

Returning to brains, if any measure of cortex mass relative to other brain components, or to whole brain is represented on a logarithmic scale, it is clear that the human isocortex is exactly the size it should be following the size allometries among these units (Fig. 1). The entire human brain is large compared to other primates, and large with respect to body size, but given this large brain size, each part falls onto its expected position. The rate of increase of each brain component differs with respect to whole brain volume (or any index of brain volume or neuron number) as $y = kx^a$. Then, when the logarithms of x and y are plotted, the exponent 'a' is the slope of the change in y with respect to a change in x. The vertebrate brain itself has negative allometry with respect to body mass. Brains are larger in larger bodied mammals, but the scaling exponent, the slope of the increase in brain size, is less than one. As the body enlarges, brains become a progressively smaller component of whole body mass [74]. Considering only brain mass, the cortex has positive allometry with respect to the rest of the brain, a slope greater than one. Thus larger mammalian brains become progressively more composed of cortex, ranging from under 20% in relative volume in small shrews and rodents to over 80% in humans [34,65].

2.2. Why do researchers in evolution care so much, and write so much about allometric predictability?

Much energy, for example, has gone into a debate about whether a specific region of cortex, the prefrontal cortex, is "allometrically unexpected" in humans [132]. Every cortical area has its own exponent (slope in the log-transformed equation) for its change in relative volume compared to overall cortex volume. In mammals, both the prefrontal and parietal cortex regions have an exponent that is larger than the cortex's overall exponent, a positive allometry, while somatosensory and auditory cortices have negative allometry [75].

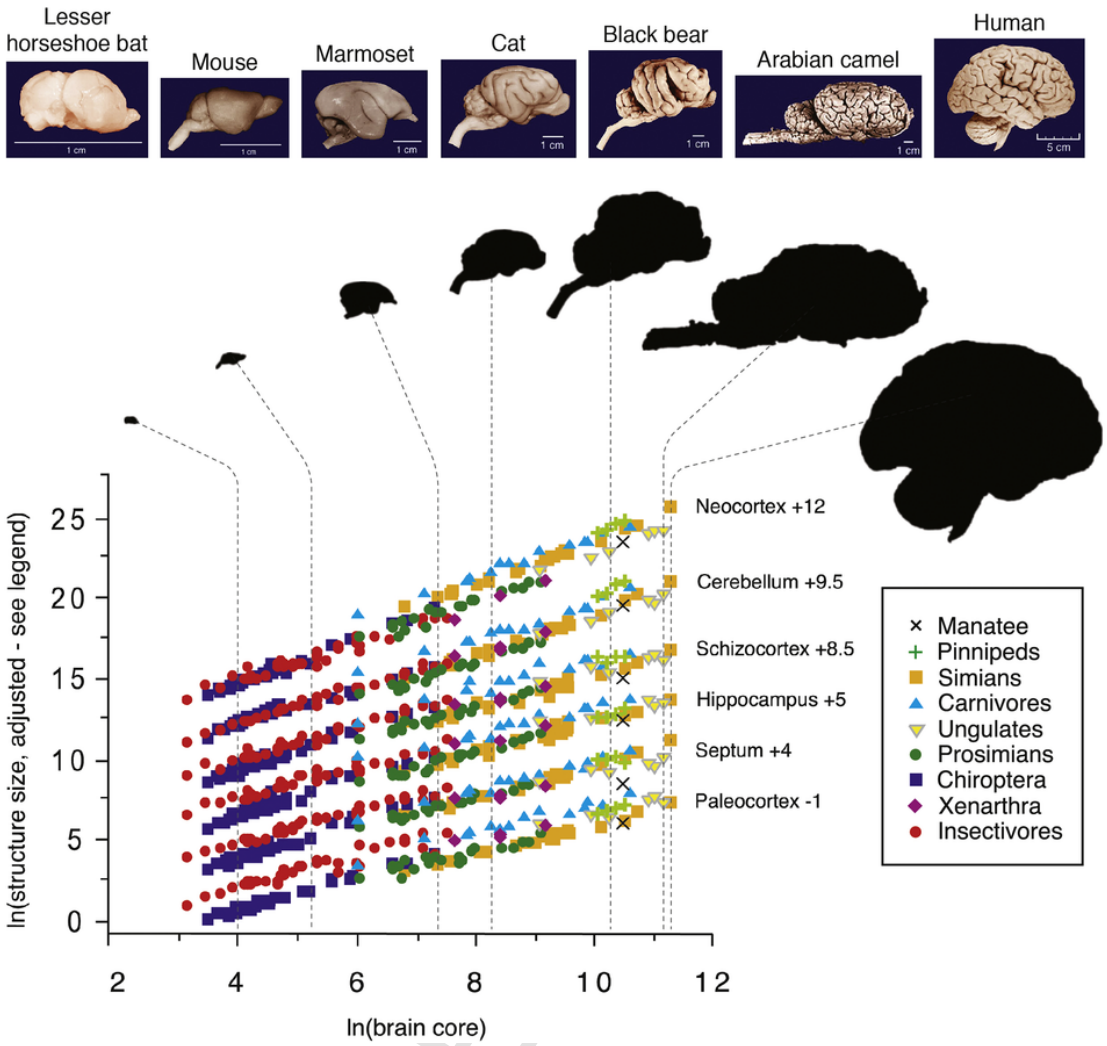

Fig. 1. Comparisons of brain volume representations.Top: Brain images, normalized to show varying sizes of components. Species: Lesser horseshoe bat, (*Rhinolophus hipposideros*); Mouse (*Mus musculus*); Common marmoset (*Callithrix jacchus*); Domestic cat (*Felis catusi*); American black bear (*Ursus americanus*); One- humped camel (*Camelus dromedaries*); Human (*Homo sapiens*) Images courtesy of University of Wisconsin and Michigan State Comparative Mammalian Brain Collections (http://neurosciencelibrary.org/). This site supported by the National Institutes of Health and the National Science foundation).Middle: Same images, vignetted midsagittal view above, to show absolute size. Image sources as above.Bottom: Brain component sizes from 160 mammalian species including the individual species shown above, both natural log scales, to demonstrate predictable scaling of brain components with respect to a brain volume index. The sizes of the 6 brain structures in the 160 species from the 9 keyed taxonomic groups are plotted relative to "brain core volume" (medulla, mesencephalon, diencephalon and striatum). The 6 individual structure volumes have been adjusted by the indicated arbitrary constants to the right of each structure's name to separate the 6 scatterplots visually. Data replotted from Reep, Finlay & Darlington [121].

The reason for concern about predictable allometries is that deviations could produce insights into the nature of past selection on brain and behavior. If researchers claim a region's volume is "allometrically unexpected" in humans, they are claiming that it must have been the target of special selection, typically because of special importance of the function ascribed to that brain region in that species. In the case of human frontal cortex, unexpectedly high allometric slopes might selectively enhance the cognitive abilities associated with frontal cortex, such as "cognitive control," the weighing of competing behavioral possibilities, or planning for the distant future [98]. By contrast, structures that change their volume according to their allometric rules, even if they look disproportionate on a linear scale, require no special explanation. If the entire brain has been under special selection for larger size in any species, every single change in the proportionality of its parts is generated by its change in overall size. Whether the volume of human frontal cortex is allometrically expected or not is still under debate, though the deviation, if it exists, is small enough to make is susceptible to relatively minor differences in methodology between research groups [4,14,109,131].

2.3. The allometry of developmental duration

The idea of "allometrically expected" changes in mass also applies to translations of developmental time from one species to another. The appropriate coordinate system to represent time translations will depend on the data components represented, and the representation desired, not presumed to be a linear scale. Just as each part of the brain has its own relative rate of enlargement with respect to total brain size, each brain part has its own relative duration or rate of development with respect to the overall duration of that species' maturation [149]. In order to compare developmental schedules between animals, enough data must be collected to reveal these allometric relationships from a number of relevant species. For example, if you wish to show that the mass of Broca's area has been the object of special selection in humans compared to a rhesus monkey, it is necessary to show that the size of Broca's area in humans exceeds its expected allometric position com-

pared to Broca's area in other primates [128]. You cannot compare the relative size of a "control structure" such as primary visual cortex in the two species to "normalize" the comparison, see that the ratio of relative sizes of Broca's area is greater than that of the two primary visual cortices, and conclude that the increased volume of Broca's area has been specially selected for in humans. If Broca's area has positive allometry compared to visual cortex, every contrast of a large and small mammalian brain will show disproportionate volume increase in Broca's area in the larger brain.

2.3.1. Norming and zeroing developmental allometries

Inappropriate norming procedures applied to developmental timing questions will produce the identical errors seen in comparing brain volumes. You cannot, for example, compare the time from birth to adolescence in chimpanzees versus humans, see that the duration is longer in humans, and conclude that human have been specially selected for a longer childhood. The duration may be entirely predictable from longevity. In addition to determination of the exponent, the slope in the allometric equation, a further important issue in the accuracy of such comparisons is what point in development represents 'zero,' the intercept or constant 'k' in the allometric equation. Although birth is often chosen as a natural zero, this choice can be very misleading. The range of maturational states at birth in mammals, including primates, is wide. Time from conception, not birth, proves to best explain variation in brain maturational state [34,149].

2.4. Developing methodologies for translating time across species

Over the past 20 years, a database and methodology to compare the progress of neural development across species has been elaborated, called "translating time" (www.translatingtime.net). The purpose of this work has been to describe a mammalian "Bauplan" for neural development, and thus be able to identify deviations from this plan that might mark taxon- or species-specific alterations corresponding to evolutionary adaptations, as we described earlier for the case of human frontal cortex. "Heterochrony" is the term used for this kind of developmental alteration [47]. The present model includes 18 species, and 271 "events" of mixed type. Here we include some detail to emphasize that virtually every measurable feature of brain production is included, not only the volume-generating kind. These include neurogenesis in particular structures or cell classes (e.g., Layer 4 of striate cortex; Purkinje cells in the cerebellum; onset of synaptogenesis in a thalamic nucleus; emergence of some minimal behavioral reactivity, and also milestones in continuous processes like increase in brain volume) extending to a maturational stage equal to approximately 3 years postnatal in humans [149]. Of equal importance is that development of non-brain aspects of the organism are not included, this decision is dictated by the empirical fact that brain "events" covary exceptionally strongly with each other while generation of organs and the body overall shows much more species variation in pattern, interruptibility, and responsiveness to earlier environmental stressors (e.g., [100])

Thus, only events in brain and some early behavioral capacities are included in the translating time model – no measures of body or organ maturation or volume, or interactional, life history events like birth or weaning are part of the dataset. A single "event scale" is fit iteratively to all the data, the best order and interval relationship of the 271 distinct neurodevelopmental events (x-axis, Fig. 2). The speed of progress of each individual species through these events is given as a regression equation, in days (y-axis). The differences in each species' slope show differences in maturational rate, with steeper slopes meaning slower progress through maturational stages in absolute time: the mouse takes only about 30 days to execute its neurodevelopmental 271 events, while the human takes 1000 days for the same 271, humans generating greater numbers of neurons and volumes of connectivity per event. The fit of model results to empirically-measured results is astonishingly close, 0.9929, including only two interaction terms, a delay in corticogenesis in primates, marsupials and carnivores associated with a larger isocortex in these species, and a delay in neurogenesis in the retina of

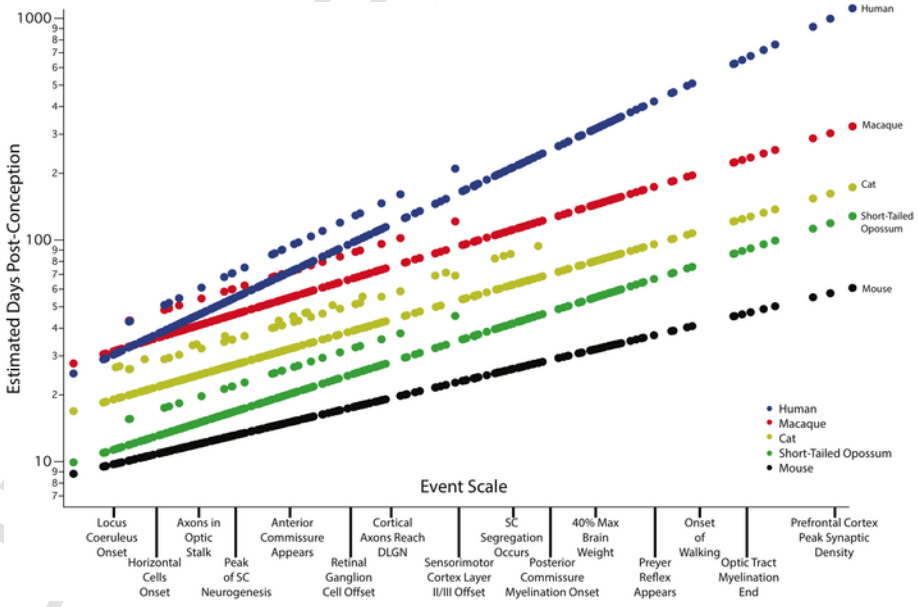

Fig. 2. Translating time model.Predicted developmental schedules for human (blue), macaque (red), cat (yellow), short-tailed opossum (green), and mouse (black), selected from the modeled 18 species to illustrate the full range of developmental durations. The x-axis, the "Event Scale," is a common ordering of developmental events across all species and shows a subset of the 271 observed events. This scale ranges from 0 to 1, but in this case, event scale numerical values are replaced by these example events. The y-axis, log scale, is the estimated date of occurrence of each event in each species, measured from conception. Also represented on this graph are interaction terms for corticogenesis and retinogenesis, with interaction terms always associated with individual species. The parallel lines for a subset of events in four of the species (black bordered circles for human, macaque, cat, and possum) represent delays in cortical neurogenesis with respect to their time of occurrence in the rodent and rabbit. In the cat, a second parallel line can be seen representing the delay of retinal neurogenesis, (yellow circle with a black dot). SC, superior colliculus; DLGN, dorsal lateral geniculate nucleus.Reproduced with permission, Figure 4 in [149]. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the nocturnal cat and ferret, associated with greater numbers of rods and rod-associated neurons (also owl monkey, [30]).

Empirical support for the surprising claim of an extremely conserved neurodevelopmental schedule can be found in several independent sources. The brains of all mammals continue to grow after birth, and Passingham [108] first noted that if the volume of the brain at birth is plotted against gestation length for an eclectic set of eutherian mammals (log transformed), a straight line results, suggesting brain mass is produced at the same rates in all species, smaller brains simply ceasing growth earlier. Halley [51], in a much larger and more closely measured data set of changes in brain volume post-conception, recently confirmed the same. The conservation of the order and sequence of the events of brain development has unexpectedly given us a new research tool, a ruler by which to describe how much somatic development and life history (age-specific fertility and mortality rates) vary across species.

2.4.1. Changes in rate or interruptions in continuity of neurodevelopment have not yet been observed in eutherian mammals, even when they might appear desirable

For example, some early behavioral capabilities follow the brain schedule. The time of the first unsupported step is highly predictable from a developmental allometric equation derived from adult brain mass, with one interaction term slightly accelerating the time of first step for those species with a plantigrade standing position [41]. That is, precocial ungulates like sheep and elk, who must be ready to run just after birth, have accomplished this evolutionarily by extending gestation and delaying birth to match conserved parameters of brain development, not by selectively advancing the rate of maturation in brain regions associated with running. A related peculiarity can be seen in precocial species with relatively small brains like the guinea pig and spiny mouse, who are born looking and moving quite maturely, furred, and with all sensory systems functional. While it might seem a reasonable strategy to make the most of every possible second for brain maturation in utero in such precocial species, to allow fine tuning of the coordinated behavior required immediately after birth, the conserved pace of brain maturation seems to rule this out. Since these animals must also produce large, mature bodies, which appears to require more time than the brain, the onset of neural development is actually substantially *delayed*. The apparently fixed amount of time required to generate a brain of guinea pig size begins later in these precocial animals, allowing somatic maturation a head start [149].

2.4.2. The timing of birth, a negotiation between mother and offspring, is quite variable

Interestingly, birth may occur at a wide range of stages in neural development in different species. Some rodents (mice and rats) are born at a stage of maturation equivalent to a human at 4–5 months gestation, while others like the guinea pig correspond to a human of approximately three postnatal years. Primates, in general, are born at a middle stage of neural maturation, neither so mature as the precocial ungulates nor as immature as a number of altricial rodents, and many carnivores. Nevertheless primates do show a range of (neural) maturational states at birth, rhesus macaques relatively mature at birth, chimpanzees intermediate, and humans least mature (Fig. 3, bottom graph). No trace of any inflection, halt or acceleration near birth can be found in basic central nervous system construction, with the important exception of a whole-brain surge of synaptogenesis which appears to predict the onset of *ex utero* or burrow experience in the four mammals studied to date (reviewed in [35]).

2.5. Consequences and causes of a fixed rate of neural development in eutherian mammals

A fixed neurodevelopmental program, duration predictable from adult brain mass, has wide-ranging consequences for understanding human evolution. First, recalling the idea of "allometrically expected", nothing as yet appears unexpected about the duration, rate or deviations from loglinearity in brain development for primates in general or for humans in particular. For example, Fig. 3 shows the rate of production of brain mass for rhesus macaque, chimpanzee and human plotted as in Fig. 2, calculated from conception until well past birth ([36] replotting [126,127]). Humans have the duration and rate of neural development exactly appropriate to produce a brain of typical human size. Therefore, though it is entirely accurate to say that humans have the longest period of brain development of any primate, as this parameter is virtually perfectly correlated with brain size [149], it is not "unexpected." Furthermore, either brain size, or developmental duration, or both might have been the object of selection. A certain developmental duration appears necessarily specified if a particular brain size is under selection [2,22]; and conversely, if selection favors a given duration of development, that carries with it a particular brain size. Decoupling of brain size and neurodevelopmental duration does not appear to occur in primates. We have found absolutely no evidence for "human exceptionalism" in any feature of brain maturation we have measured.

2.5.1. The reason for the close conservation of eutherian brain development is not yet known

While variation in the rate of development of other organs is marked, the rate of neurogenesis, including the secondary steps of neural construction is remarkably fixed and very closely related to adult brain size in eutherian mammals [52]. There is no hidden physical law about cell division per se that dictates the observed fixed rate – non-eutherian mammals, marsupials, generate their brains considerably more slowly, and with more variability [27]. Precocial birds, like chickens and ducks, can accelerate neural maturation selectively in their midbrains, anticipating hatching [24]. The answer may depend on particular advantageous outcomes embedded in the developmental mechanisms when their duration is extended. For example, when large cortices evolve, they do not change by simply enlarging in area, retaining a fixed internal structure. Rather, the increased number of neurons supporting initial sensory analysis and environmental description in posterior cortex allows faithful transcription of high-acuity sensory information and separation of channels for its analysis. Simultaneously, compression, or abstraction of information along the anterior-to-posterior axis (sensory, to motor, to cognitive control) also increases. This organizational change depends upon the precise relationship of an anterior-to-posterior gradient of neurogenesis and its subsequent interaction with neuron-type specification. Such a progressive, predictable reorganization would appear to facilitate the extraction, and ability to remember environmental features and behavioral strategies extended over greater space and time [35]. This progressive compression and abstraction of information in larger brains may be the basic structural feature of the computations allowing improved "cognitive control" (weighing the best behavioral response over competing possibilities) [98], better maintenance of behavioral choices over extended time intervals (Stevens, 2014) [xx], and more range in the hierarchical abstraction of concepts in the frontal lobe (Badre et al., 2009 [xx]). This duration-dependent computational structure and others not yet known could thus usefully predict the loose linkage of brain size, body size, range and longevity, and be selected in the way multiple coordinating developmental programs are stabilized. Whatever the reason, however,

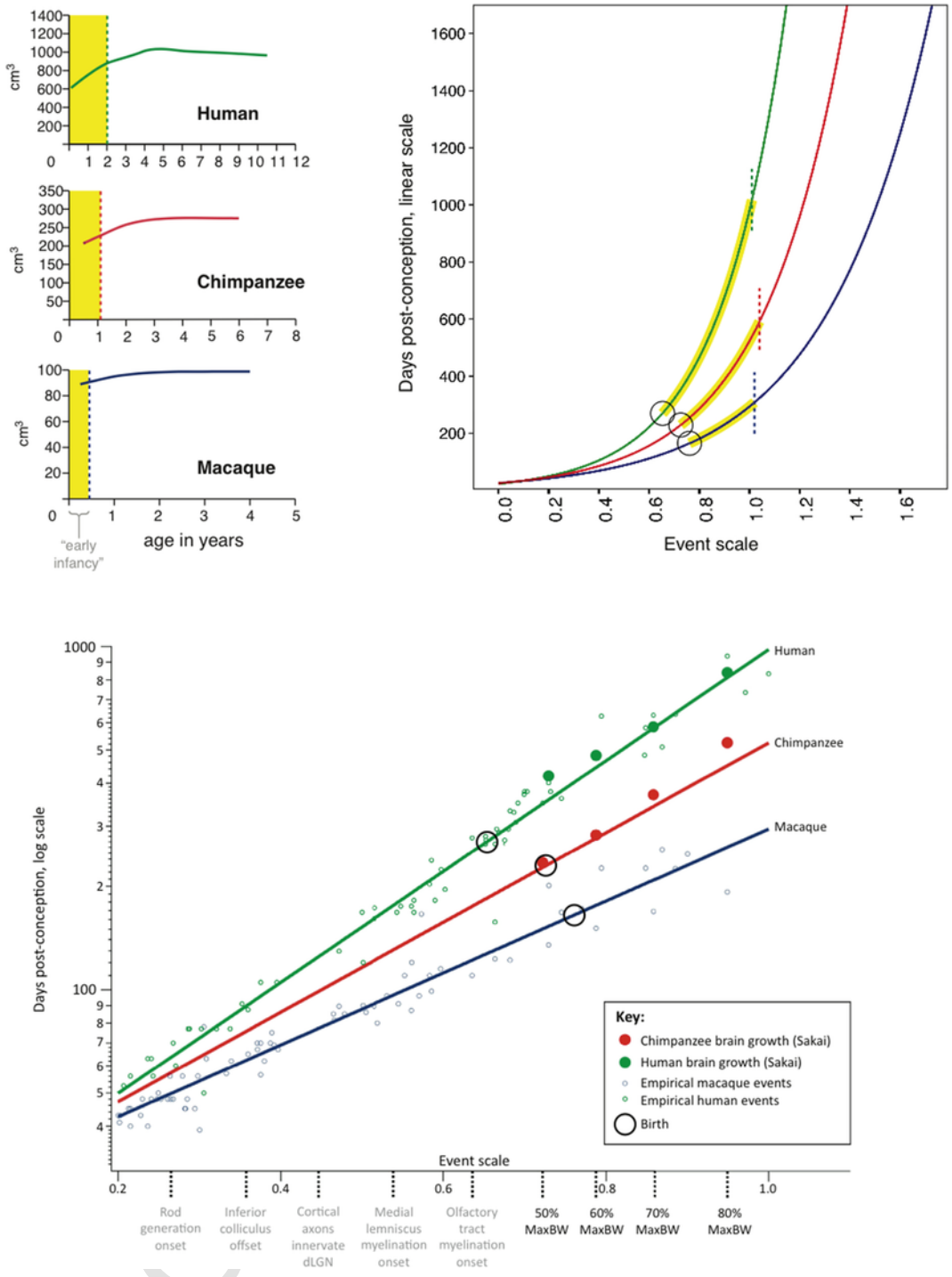

Fig. 3. Comparison of postnatal and prenatal brain growth in the rhesus macaque, *Macaca mulatta*; chimpanzee (*Pan troglodytes*) and human (*Homo sapiens*).Top left: Data replotted from Sakai et al. [127] to show postnatal volumetric growth of the monkey, chimpanzee and human brains on linear scales of volume (y axis) and years (x axis).Top right: Volume change and birth date position (open circle) plotted against a model overall of neural maturation in the same species from Workman et al. [149]. The "Event Scale", the x-axis, is a set of 271 normalized neural maturational events drawn from 18 species, including primates, and consists of such items as neurogenesis in multiple structures, myelination, and brain growth. The Y-axis, days post-conception, allows the day each species reaches each neural maturational milestone to be represented. The yellow overlay and dotted lines on the top left and right graphs represent identical points in the "Event Scale" in the two representations.Bottom graph: Growth in brain volumes for human, chimpanzee and macaque (solid points) embedded in other neurodevelopmental events (small open points) before and after birth (large open circles) plotted on the "Event Scale" showing the different relative slopes and absolute durations for the three primates. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the fixed rate of generation of neural tissue in eutherian mammals, including primates in general and ourselves in particular has large consequences for our theorizing. To select on brain size is to select on a particular neurodevelopmental duration. Selection on "duration" would also affect brain size. If so, are the various durations of life span components, time to adulthood, length of adulthood, rate of senescence and so forth, linked to the duration of neurodevelopment, how strongly, and through what underlying mechanisms?

2.6. What varies between brains? Some necessary warnings that duration of development is not the only source of variation between brains

The subject of evolutionary changes in brains is obviously complex, and cannot be reviewed completely here, but before turning to longevity and grandmothering, a few caveats are in order. The emphasis on allometric predictability of the relative sizes of brain parts and developmental durations should not be taken to indicate that "mosaic" changes, structure-specific volume changes, never occur, they certainly do. Birds show substantially more mosaicism by brain part than mammals do, and marsupials vary more in the rate of brain developmental than eutherian mammals do [29,71]. The interaction terms described earlier for the limbic system versus the cortex, or generation of the nocturnal or diurnal eyes are examples of mosaicism [30,121], and mosaicism may also be found in the residual variation, discussed at length in Finlay, Darlington and Nicastro (2001) [xx]. Due to the ease of measuring the volume of a brain part compared to understanding the details of its intrinsic circuitry, or varying environments or species-specific learning strategies, relative volumes were the focus of study for many years, accumulating the evidence of allometric regularities reviewed above. But, if relative volumes do not account for all or even much of the wide range of behavioral variation across taxa, something must generate the profound differences in behavioral competencies displayed by vertebrate species. Here we underline a few of particular interest.

2.6.1. What generates the wide range of species differences in behavior?

First, while the central nervous system remains relatively stable in organization prior to innervation, the sensory and motor periphery varies widely, including animals variably employing vision, whiskers, echolocation or electroreception to explore their immediate-to-distal environment [77]. The associated motor periphery ranges from limbless, to quadrupedal to prehensile tails and trunks, to dexterous hands. In every case studied closely, it is the details of these systems that impress and alter the nervous system to their particular forms in development and throughout individual lifetimes [87]. Similarly, the environment those sensory systems encounter can be a stable source of information for directing brain organization. For example, the regular statistical structure of the information provided by the visual environment in combination with a generic learning mechanism produce the receptive field structure that compresses and renders "sparse" (energetically compact) visual representations [105]. Initial human preference for faces begins as a preference for an ovoid form with more contrast-y elements in the top half. It requires 10–13 years of experience before adult discrimination of, and neural organization responding to individuals, expressions, and social markers stabilizes [76].

2.6.2. Connectivity of motivational and reward systems varies a great deal between mammals, and will prove to be essential in understanding the significance of life history alterations

Perhaps one of the most interesting features to come to the fore in current research is the profound malleability of circuitry associated with motivation and reward. Contrary to the expectations generated by the apparent volumetric and morphological conservatism of subcortical circuitry, connectivity between motivational, motor, and sensory systems therein can change profoundly over the lifespan of individuals, between individuals, and between species. The first observation to energize this field of research was demonstration of the relative simplicity of linkage between the presence of a particular individual and motivational systems. Monogamous voles possessing increases in vasopressin receptors will work hard to be in the presence of their preferred mate, clearly distinguishing males in one species from their promiscuous cousins. Profound differences in social behavior can thus be generated with rather minimal neural change, that is without changes in neuronal numbers or structure volumes, and without de novo generation of neuromodulators or their receptors [28]. Similar subcortical mechanisms for differing inter-species preferences for such varied things as numbers of conspecifics, territory size, vigilance or aggression rates are presently being described [45].

Considering human gesture and language learning, motivational reorganization rather than committed modules are increasingly reported. Human infants are unusually responsive to social reward compared to other primate infants, that responsiveness scaffolding early visual and language learning [42]. While human birth is slightly early in terms of neural maturation compared to chimpanzees and macaques (Fig. 3 bottom graph), more notable is the comparative neural immaturity of human infants at weaning (Fig. 4). No longer fully reliant on mothers while their developing brains are still early in the maturational sequence, infants extend their initial preference for voices and face-like forms [76] to any actions that elicit attention and action from parents or alloparents. This early responsiveness is coming to give a better account of language, gesture and expression learning than the invocation of a committed "language module" [3,38,106] Additionally, motivational preferences underlying human cognition can likely employ the

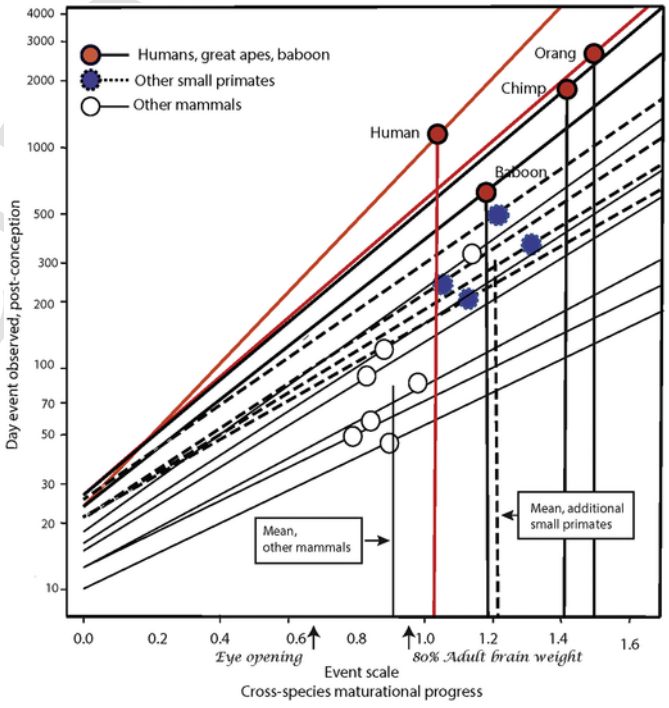

Fig. 4. Variability in weaning compared to neural maturational state.Predicted developmental schedules for eight primate and seven nonprimate species, with observed timing of weaning indicated for each. The event scale on the x-axis has been extended beyond its original range of 0–1, to allow for extrapolation of developmental trajectories into the range in which weaning occurs. Examples of events included in the model are displayed at their respective positions on the event scale on the x-axis. The y-axis indicates the estimated date of the occurrence of each event in each species. Plotted species include those whose developmental schedules are directly predicted by the model as well as those that are not. The latter consists of all of the primates except for the human and rhesus macaque, which are the two primates included in the current version of model. For the unmodeled primate species, we used adult brain weight to estimate slope and gestation length to estimate intercept, as these variables have been shown to predict the timing of events with reasonable accuracy. Species are as follows: baboon (Papio cynocephalus); cat (*Felis catus*); chimpanzee (*Pan troglodytes*); ferret (*Mustela putorius*); gerbil (*Meriones unguiculatus*); guinea pig (*Cavia porcellus*); hamster (*Mesocricetus auratus*); human (*Homo sapiens*);lemur (*Lemur catta*); macaque (*Macaca mulatta*); marmoset (*Callithrix jacchus*); Orangutan (*Pongo pygmaeus* pygmaeus); owl monkey (Aotus azarae); rabbit (*Oryctolagus cuniculus*); rat (*Rattus norvegicus*); sheep (*Ovis aries*). Source data for weaning are reviewed in Finlay and Uchiyama [35].

same subcortical mechanisms that direct species-typical preferences in other mammals, operating over extended domains [139,141].

3. Life history evolution

3.1. Regularities in mammalian life history variation

The pace of discovery in mammalian life history evolution cannot match that of neuroscience, in part because aspects of brains and bodies can be measured on single representatives of a species, but population vital rates cannot. Even when mortality risk is tightly correlated with age, each individual only dies once. Evolutionary life history theory developed in the 1980s only as (hard won) data on age-specific fertility and mortality in mammal populations began to accumulate [136]. Wide differences in the pace of life cycles, and the association between that variation and adult body size had long been of interest (Bonner, 1965; [12,111]). Yet as comparative analyses began, some of the apparent life history tradeoffs seemed initially puzzling. Instead of longer gestation length allowing shorter time to weaning, or later weaning shortening time required to reach maturity, correlations among these variables turned out to be positive. This vastly expands the range of potential population growth rates from species that mature quickly, produce many offspring, and die soon to those that take a long time to mature, and produce few offspring over long adult lives [117]. The smallest and largest primates differ a thousand-fold in adult body size. That difference translates from three into nine orders of magnitude in potential population growth rates: "A female mouse lemur (*Microcebus murinus*) born at the same time as a female gorilla (*Gorilla gorilla*) could leave 10 million descendants before the gorilla became sexually mature" ([55]: 14).

3.2. The importance of mortality risk

After some feasible threshold, the time spent growing larger instead of producing offspring is reproduction foregone, an opportunity cost with geometrically increasing consequences. There must be benefits that compensate that cost. Clearly mortality must play a major role. Mammals are determinant growers. Unlike most fish that continue to grow larger throughout life, mammals grow until reaching maturity (age of first reproduction), and then stop growing to reproduce. It takes longer to grow bigger, but perhaps larger size improves predator defense, or increases mobility over larger foraging ranges and tolerance of lower diet quality or local resource fluctuations, all of which would reduce adult mortality risk [26,86,107,145,146]. But the direction of causality could run the other way. Larger body sizes could be a *consequence* of lower adult mortality risk. If reproductive efficiency increased with age or size [55], then lower mortality risks would allow growing longer before maturing to reap more of those benefits [17,85]. Positive correlation between longevity and age at maturity is consistent with either direction of causality. As the empirical associations among the life history variables became irrefutable, so did their stronger correlation with each other than with body size. The timing variables remain correlated even when the effects of body size are statistically removed, suggesting that body size might not be as important as previously assumed [56,117,120,138].

3.3. Allometric regularities in life history variables

Charnov [17] drew attention to the fact that average adult lifespan and age at maturity each increase with body size at the same allometric rate. The same ¾ power scaling means that the product of the variables (e.g. the product of age at maturity times average adult mortality) remains the same across large changes in body size. He constructed a model of mammalian life history evolution aimed at recovering these allometries and explaining the invariance of these products across transformations of body size [18]. Building on his own previous work and that of many others, he used a simple growth model in which a juvenile's production goes into increasing her own size and then is redirected at maturity into producing offspring. Recognizing the importance of interspecific differences in rates of growth and of offspring production [13], Charnov identified a lineage specific production coefficient, 'A,' to capture that rate. Separating it can explain why correlations between adult mortality, age at maturity, and rate of offspring production are tighter than between any of them and body size. Species with low 'A' grow slowly; so are relatively small at a given age of maturity. With low 'A' they also produce offspring at a low rate for a given adult size. Because adult body size for a given age at maturity varies with 'A,' and 'A' varies among lineages, body size correlates less closely with age at maturity, reproductive rate, and lifespan than those variables do with each other. The difference between primate and non-primate mammals is quantified by different values of that one parameter, 'A.' Fitting data to the model, gives 'A' of about 0.4 for primates compared to about 1.0 for non-primate mammals generally, explaining why, for a given adult size, primates have greater longevity, later maturity, and slower rate of offspring production than do most non-primate mammals [20].

3.4. Body growth vs brain growth

Previous sections are strong foundation for skepticism that variation in rates of body growth could be usefully captured with such an extreme simplification. As noted above, variation in mammalian somatic growth rates contrast with tight cross-species regularities in brain growth. Charnov's mammal model highlighted the empirical pattern that primate bodies grow slowly compared to non-primate mammals but did not deal with brains [20]. Recent analyses [52] confirm previous findings [108] that fetal body growth is much more variable than fetal brain growth, extending the evidence that body growth in primates is notably slow compared to non-primate eutherians. Slow body growth, not faster brain growth results in exceptional encephalization in primates across gestation and beyond.

The evidence of extreme regularity in neurogenesis, in which developmental duration determines final brain size and organization, points to brain size as a potential index of features that propel life history evolution. Pursuing that possibility must await future work. For now we take advantage of Charnov's use of the simple body growth model because it reproduces relationships among adult mortality rates, age at maturity, and length of offspring dependence that characterize the broad mammalian variation, which provides the context for identifying distinctive life history features that evolved in humans. Those features will bring us back to the role of allomaternal care in extending the duration of development and propelling infants into a wider social context during early neural maturation in our lineage.

4. Brains & our grandmothering life history

4.1. Humans compared to great apes

To display an important regularity in mammalian life history variation Charnov [19] plotted average female adult lifespans by age at maturity for 15 primate subfamilies using data from Millar & Zammuto [101] and Harvey & Clutton-Brock [54]. The received taxonomy at the time classified humans as the only living hominids with the other great apes separated as pongids. The plot showed that great apes have the longest adult lifespans and oldest ages at maturity of the non-human primates, but their values are substantially exceeded by higher values for humans. Yet both fit the approximate "invariance" of an important

product in his model, αM, age at maturity times average adult mortality.

That humans fit this invariant went unremarked at the time, but is initially quite puzzling. Charnov's assembly rules for the life history of female mammals assume that adulthood is spent producing offspring. But since women cease childbearing at about the same age as great ape females (the pongids) do, a substantial fraction of female adult years in humans is post-fertile. Genetic evidence now puts humans and great apes in the same hominid family and shows that some of them are even more closely related to us than they are to each other. The age female fertility ends is similar in all living hominids, suggesting that is the ancestral condition; but great ape females become decrepit with age and – as in mammals generally - rarely outlive their fertile years. Women, in contrast, can remain strong and productive well past menopause. Our low adult mortality could account for distinctively late human maturity by pathways in Charnov's mammal model only if all of adulthood contributed to producing descendants [63].

4.2. Costs and benefits of increased somatic maintenance

In Charnov's initial model [18] adult mortality is entirely exogenous. Yet evolutionary life history theory recognizes that selection adjusts vulnerability to mortality risks by allocation to somatic maintenance and repair, an allocation that must compete with growth and reproduction throughout life [40,147,148]. Kirkwood's [80–82] disposable soma model focuses specifically on the question of optimal maintenance, directing attention to the tradeoff with current reproduction. If the risk of dying is inevitably high, then allocation to maintenance and repair nets little benefit because the chance of dying is high no matter the repair. Conversely, if the risk is low, selection can favor trading off some growth and/or current reproduction for more maintenance and repair. But, in addition, as noted by Williams ([147], and see [19], Chapter 7) the likely contribution to future generations gained with increasing adult age also affects this tradeoff. The more descendants that result from persistent competence, the more selection favors maintenance and repair. While we will follow this argument about life history trade-offs, remember how tightly constrained brain development is by developmental duration, constraints we will return to at the end.

With this framework of life history interactions as a guide, [19] plot of the relationship between age at maturity and average adult lifespan showing that humans (under a hunting and gathering mortality regime) obey Charnov's symmetry model invites a question. If slower maturation in humans results from lower adult mortality, and lower adult mortality comes with more allocated to maintenance and repair leaving less for current reproduction, what ancestral condition could have favored those tradeoffs in our lineage?

4.3. Lessons from the modern Hadza

Ethnographic observations of Hadza hunter-gatherers in northern Tanzania [8] supplied a promising clue. Hadza are modern people, not living ancestors, but as foragers they face ecological challenges and tradeoffs that reveal ancient problems our ancestors faced foraging for a living before the origins of agriculture. Moreover the Hadza occupy the best modern analog of the savannas that witnessed the evolution of genus *Homo*.

Hadza children are productive and energetic foragers [9]. But young children are not very effective at handling an important dietary staple, the deeply buried underground storage organ of a plant that requires some strength to excavate [61]. Women past menopause are especially active tuber diggers [60]. In this population, mothers' foraging effort has a measurable effect on their children's nutritional welfare except when those mothers bear another baby and reallocate effort to include nursing their new infants. Then it is the work of postmenopausal grandmothers that differentially affects the growth and survival of weaned children [8,62]. This association between subsidies from post-fertile women and the welfare of weaned grandchildren could have propelled evolution toward human life histories long before the appearance of *Homo sapiens* [104]. Subsequent work across the diverse subsistence and social systems of contemporary people has shown that grandmothers usually continue to play a measurable role in their descendants' welfare (e.g. [91,129,130,142], see [59]).

The tradeoffs observed among the modern Hadza offer a lesson about likely ancestral ones. In the other living hominids, as in mammals generally, infants are weaned when they can forage for themselves. In our lineage ancestral mothers shifted from forest to savanna foods, many of which their young offspring could not handle for themselves. Under these circumstances the few surviving elder females who had no newborns of their own could have novel effects on their own inclusive fitness. As elders continued to exploit those savanna foods, their economic productivity would allow their daughters to wean earlier, raising their own fitness by moving on to next babies sooner with less cost to the survival of previous offspring.

In this ancestral scenario, elders that were aging slightly more slowly could help more. Previously selection would not have favored more allocation to somatic maintenance because it left less for current reproduction. Now mothers allocating less to current reproduction themselves would still be more successful at offspring production due to subsidies from grandmothers. These interdependencies point to pathways for selection to increase longevity as longer-lived grandmothers left more descendants. This grandmother hypothesis proposes that our distinctive postmenopausal longevity evolved as a consequence of ancestral grandmother effects [57,63]. While explaining the greater longevity and later age at maturity that fit humans to Charnov's αM invariant (age at maturity times average adult mortality), it also explains the human departure from another of Charnov's invariants, the αb product (age at maturity times baby production). The rate of offspring production should be higher for a grandmothering mammal than predicted for a mammal without grandmothering as postmenopausal longevity results in more descendants by subsidizing (and so increasing) baby production during the fertile years. Thus higher human fertility, shorter birth spacing and weaning earlier than predicted for a primate with our age at maturity could be the consequence of ancestral grandmothering [63].

4.4. Mathematical models of life history evolution

Could grandmothering really be enough to explain these features? Absent a time machine, formal modeling allows some check on whether or not it might. Peter Kim's two-sex agent-based model allows simulations of the verbal grandmother hypothesis [78,79]. Starting with a great ape-like life history in which female fertility ends in the forties as it does in living hominids, and costs of increased longevity include later age at maturity and longer offspring dependence as assumed in Charnov's mammal model, variants that alter longevity away from the great ape equilibrium do not spread. At this model equilibrium, as with living great apes, very few females survive their fertility. But when those rare females subsidize still dependent juveniles, mothers can wean them earlier and have next babies sooner. Then increased longevity is favored, and the proportion of post-fertile females expands to result in an age structure like that observed among modern hunter-gatherers. Once grandmothering subsidies propel a population out of the great ape-like basin of attraction, selection on longevity moves agent populations steadily to the human equilibrium. Evaluating the hypothesis with partial differential equations rather than agent-based simulations, assuming a more realistic mortality schedule, and allowing both longevity and the end of female fertility to vary, found the same

two equilibria, one with and one without grandmothering [15]. This is consistent with the hypothesis that subsidies of dependent juveniles by grandmothers are foundation for the evolution genus *Homo*, long antedating the appearance of our species [104].

5. What is the structure of selectable variation in longevity? Evo-devo meets grandmothers.

The social and energetic factors considered to affect selection on life histories could not be more distinct from a neurodevelopmental account of why sequences and durations of neurogenesis and synaptic connectivity assemble in the way they do. Nevertheless, both are speaking of the same human evolution, and there is a tantalizing convergence in the two in the potential relationship of longevity, grandmothering, and brain size.

The first 20 years of work in evo-devo, demonstrating actively conserved processes of immense antiquity coexisting side-by side with fully variable processes suggests that conservation produces mammalian-general properties useful across multiple niches (Gerhardt & Kirschner, 2005). Covariation of the duration of life phases in mammals has both logical and empirical support. From the perspective of evolutionary life history, longer duration of development can only persist in the context of lower mortality. A consequence is then both cause and effect relationships among brain size, the developmental duration required to produce a certain size brain, and what the brain is used for. A large brain will require a long period of development that can be employed for extended environmental learning, integration over long times and spaces matched to the computing power such brains possess.

Relationships among brain size, behavioral/cognitive complexity, and ecological niche variables are evident from cross-taxa comparisons. For example, large brains in cetaceans are associated with wider breadth of social behaviors, prey diversity, and aspects of range [39]. Across multiple species including primates, larger brain size is associated with greater behavioral innovation, better success in niche invasion, tool use [92], and better cognitive control [98]. No claim is made that primates or cetaceans have a unique organization of their brains, simply that their radiations are enriched with large brained species. The work we have described here relates brain size to neurodevelopmental duration. A strong relationship between brain size and overall longevity independent of body size has been demonstrated [44,64,125]. It is not implausible that an overall developmental regulator or timekeeper sets the pace of early neural development and continues through the lifespan.

A new way to consider the evolution of longevity is to take an evo-devo stance and ask what structure, or components of variation in longevity could be offered to selection. Investigation of longevity is mostly of the medical variety, analyzing predispositions and risk factors for diseases. But there has been substantial application of a life history approach to varying mortality risk across human groups and associated variation in somatic maturation rates and age at first birth (e.g., Draper & Harpending, 1982; Belsky et al., 1991; Geronimus, 1996; Daly & Wilson, 1997; [32,33,50,97,102,103,112,113]). These life history adjustments are developmental plasticity of a particular sort, widely hypothesized to be adaptive. If so, they are the result of natural selection on ancestral populations that regularly faced similar environmental variation (e.g. [88,90]). Behavioral ecologists often use concepts like the phenotypic gambit, strategy set, and reaction norms to study such phenotypic variation [48,49]. As noted by West-Eberhard,"The concept of reaction norm bridges the gap between phenotypic plasticity and quantitative genetic studies of natural selection by connecting quantitative phenotypic plasticity and genotype…. Because reaction norms vary among individuals, the environment is not only an agent of selection but also a determinant of the range of phenotypes expressed to selection" ([144]: 26–7). Norms of reaction for variation in age at first birth mimic what appears to be the life history variation "game plan" across lineages. As duration of development is the single factor most clearly associated with major differences in brain mass and organization across mammalian species, it seems clear there must sometimes be heritable variation on this dimension, but it has never been empirically demonstrated. However, there are tantalizing hints – the Hox-gene-regulated rate of segment generation of the spinal cord can be more than doubled in snakes, multiplying this feature while others are unchanged [43]; cerebellar precursor cells from short-lived species inserted into the brains of long-lived species develop into mature neurons that share the greater longevity of their hosts [99]. In general, however, this question has been oddly neglected and the nature of control processes regulating developmental duration have been virtually unexplored, a perplexing absence in the research literature.

6. Special features of the progress of human infancy and childhood: a conserved neurodevelopmental program within an unusual life history

In the prior section, we concentrated on the hypothesis that distinctive human postmenopausal longevity is linked with our longer developmental period and earlier weaning, all potentially initiated from ancestral grandmothering subsidies. Preceding sections reviewed the mammalian evidence that later maturity and greater longevity are inevitably accompanied by larger brains. From the point of view of the infant and child this combination of features proves to have especially interesting consequences for important aspects of human sociality. The conserved neurodevelopmental program assures that it takes longer to reach larger final brain size. Yet weaning is unexpectedly early for a mammal with our age at maturity. In terms of neural maturation weaning is especially early (Fig. 4). As in other species, the highly predictable duration and stages of development produce a distinctive developmental ecology. Earlier we gave two examples of how precocial rodents and ungulates coordinate fixed brain growth patterns with gestation; here we describe some emergent, or possibly selected features of more altricial human somatic development.

Human infants are intermediate in the state of neural maturation at birth compared to the mammalian range. Their eyes are open and fully functional, hearing well established, but motorically they are somewhat delayed, unable to support their unusually heavy heads, or guide their hands in the first three months of life. Is this a bug or a feature? Exciting research is now showing that the maturational state of the infant at birth and thereafter may have an important role in guiding the trajectory of facial and vocal communication, and object recognition and manipulation. Unable to lift its heavy head, the infant spends a great deal of time observing a small number of faces in much detail (2–4 depending on culture), those faces conveniently in a relatively standard position and orientation with respect to the infant, an introductory seminar in facial recognition and expression [73]. Next, when the infant gains control of its head and begins to control its hands, the following three months move from concentration on faces to concentration on objects and manipulation [25]. The idea of an "object," a nameable entity recognizable over translations and rotations, is not nearly so central to vision across vertebrates as it is to us. For most mammals identifiable individuals or object types are often limited to a few conspecifics, food items, predators and prey species, whereas we routinely interact with multiple individuals, artifacts, signs and symbols. Early attention to hands and very basic manipulation of food, toys and attractive contraband, enforced by the immature absence of independent mobility, neatly links object, vision, manipulation and eventual tool use, and the substrate of naming and eventual language in an organic package.

Finally, when toddlers are becoming motorically independent, though still far from fully competent at locomotion and long before they can feed independently, they are displaced from the center of their mothers' attention. Not only is weaning much earlier than the age expected for a primate with our age at maturity [63,123], it is especially early in neural development compared to in other great apes (Fig. 4). Human infants, with their very immature brains, are released from complete dependence on mothers into a social milieu populated with a large number of alloparents and social partners. The social ecology they confront is variable among cultures and responses fitting those variations inevitable.

Subsidies that allow the early weaning and short birth intervals have evolutionary consequences for both mothers and infants that have been especially well elaborated [66–68]. When mothers can net higher fitness by overlapping dependents, their distributed attention dilutes focus on infants. Without the full maternal commitment that is the birthright of apes that rear one dependent at a time, infant features that attract more attention from mothers and allomothers are exposed to selection. Survival advantages for precocious responsiveness can help explain the evolution of a distinctly human social appetite for what Michael Tomasello (1999, Tomasello et al., 2005) labeled "shared intentionality." Reviewing descriptions of captive chimpanzee infants, some mother-reared and others raised by multiple attentive carers, Hrdy [68:29] identified a "virtual experiment," testing likely developmental consequences when a hominid used to independent mothering is exposed to regular allomothering. Although "none of these captive infants were reared under species typical conditions, … those exposed to multiple responsive others developed to be more-other regarding than their exclusively mother reared counterparts"[68:35]. Such effects of social context are directly consistent with the hypothesis that a shift to dependence on allomothering exposed ancestral infants to interactions that had profound effects. "New modes of child-rearing meant changing our minds" ([68]: 23).

Building on Hrdy's insights, Hawkes [58] added heterochrony inferences and linked differences in white matter development between infant human and infant chimpanzee brains [126] to exceptional social capacities and appetites in human infants. That missed Finlay and Workman's [36] reanalysis showing the chimpanzee-human white matter differences to be simply the consequence of the longer developmental duration to larger size of human brains. Regularities in mammalian brain development combined with comparative neural and somatic maturation in human infants indicate that features of our sociality may have arisen as necessary consequences of life history shifts on a developmental framework shared with other primates. The distributed attention of mothers and the importance of allomothers present human infants with a complex social landscape, even before weaning. Links among longevity, longer duration of development, early weaning, and distinctive dimensions of human sociality – perhaps even that most distinctive of all: language - may be a cognitive version of emerging universal relationships of growth and form, like the stouter legs and the lowered metabolic rate of larger bodies, links whose significance we are only just beginning to appreciate.

Uncited references

[7,11,23,31,53,95,96,118,119,124,137]

References

[1] L.C. Aiello, P. Wheeler, The expensive-tissue hypothesis: the brain and the digestive system in human and primate evolution, Curr. Anthropol. 36 (1995) 199–221.

[2] N.L. Barrickman, M.L. Bastian, K. Isler, C.P. van Schaik, Life history costs and benefits of encephalization: a comparative test using data from long-term studies of primates in the wild, J. Hum. Evol. 54 (2008) 568–590.

[3] R.A. Barton, P.H. Harvey, Mosaic evolution of brain structure in mammals, Nature 405 (6790) (2000) 1055–1058.

[4] R.A. Barton, C. Venditti, Human frontal lobes are not relatively large, Proc. Natl. Acad. Sci. USA, vol. 110 (2013) 9001–9006.

[5] E. Bates, S.M. Wilson, A.P. Saygin, F. Dick, M.I. Sereno, R.T. Knight, N.F. Dronkers, Voxel-based lesion-symptom mapping, Nat. Neurosci. 6 (5) (2003) 448–450.

[6] Z.D. Blount, C.Z. Borland, R.E. Lenski, Historical contingency and the evolution of a key innovation in an experimental population of Escherichia coli, Proc. Natl. Acad. Sci. USA 105 (23) (2008) 7899–7906, https://doi.org/10.1073/pnas.0803151105.

[7] L. Blueweiss, H. Fox, V. Kudzma, D. Nakashima, R. Peters, S. Sams, Relationships between body size and some life history parameters, Oecologia 37 (1978) 257–272.

[8] N.G. Blurton Jones, Demography and Evolutionary Ecology of Hadza Hunter-Gatherers, Cambridge University Press, Cambridge, 2016.

[9] N.G. Blurton Jones, K. Hawkes, J.F. O'Connell, Modeling and measuring the costs of children in two foraging societies, in: V. Standen, R. Foley (Eds.), Comparative Socioecology: The Behavioural Ecology of Humans and Other Mammals, Basil Blackwell, London, 1989, pp. 367–390.

[10] E. Bruner, Language, paleoneurology, and the fronto-parietal system, Front. Hum. Neurosci. 11 (2017) https://doi.org/10.3389/fnhum.2017.00349.

[11] D.J. Cahalane, C.J. Charvet, B.L. Finlay, Modeling local and cross-species neuron number variations in the cerebral cortex as arising from a common mechanism, Proc. Natl. Acad. Sci. USA 111 (2014) 17642–17647.

[12] W.A. Calder, Size, Function, and Life History, Harvard University Press, Cambridge, 1984.

[13] T.J. Case, On the evolution and adaptive significance of postnatal growth rates in terrestrial vertebrates, Q. Rev. Biol. 53 (1978) 243–282.

[14] T.A. Chaplin, H.-H. Yu, J.G.M. Soares, R. Gattass, M.G.P. Rosa, A conserved pattern of differential expansion of cortical areas in simian primates, J. Neurosci. 33 (2013) 15120–15125.

[15] M. Chan, K. Hawkes, P.S. Kim, Evolution of longevity, age at last birth and sexual conflict with grandmothering, J. Theor. Biol. 393 (2016) 145–157.

[16] B. Charlesworth, Evolution in Age Structure Populations, 2nd Ed, Cambridge University Press, Cambridge, 1984.

[17] E.L. Charnov, On the evolution of age of maturity and the adult lifespan, J. Evol. Biol. 3 (1990) 139–144.

[18] E.L. Charnov, Evolution of life history variation in female mammals, Proc. Natl. Acad. Sci. USA 88 (1991) 1134–1137.

[19] E.L. Charnov, Life History Invariants: Some Explorations of Symmetry In Evolutionary Ecology, Oxford University Press, Oxford, 1993.

[20] E.L. Charnov, D. Berrigan, Why do female primates have such long lifespans and so few babies? Or life in the slow lane, Evol. Anthropol. 1 (1993) 191–194.

[21] C.J. Charvet, R.B. Darlington, B.L. Finlay, Variation in human brains may facilitate evolutionary change toward a limited range of phenotypes, Brain Behav. Evol. 81 (2013) 74–85, https://doi.org/10.1159/000345940.

[22] C.J. Charvet, B.L. Finlay, Embracing covariation in brain evolution: large brains, extended development, and flexible primate social systems, in: A.H. Michel, D. Falk (Eds.), Progress in Brain Research, vol. 195, Elsevier, 2012, pp. 71–87.

[23] C.J. Charvet, B.L. Finlay, Evo-devo and the primate isocortex: the central organizing role of intrinsic gradiants of neurogenesis, Brain Behav. Evol. 84 (2014) 81–92.

[24] C.J. Charvet, G.F. Striedter, Bigger brains cycle faster before neurogenesis begins: a comparison of brain development between chickens and bobwhite quail, Proc. R. Soc. B 277 (1699) (2010) 3469–3475.

[25] E.M. Clerkin, E. Hart, J.M. Rehg, C. Yu, L.B. Smith, Real-world visual statistics and infants' first- learned object names, Phil. Trans. R. Soc. B (2017) 372, (PMCID: PMC5124080).

[26] T.H. Clutton-Brock, P.H. Harvey, J.F. Eisenberg, D.G. Kleiman, The functional significance of variation in body size among mammals, In: Recent Advances in the Study of Mammalian Behavior, no. 7, Special Publication of the American Society of Mammalogists, Shippensburg, PA, 1983, pp. 623–663.

[27] R.B. Darlington, S.A. Dunlop, B.L. Finlay, Neural development in metatherian and eutherian mammals: variation and constraint, J. Comp. Neurol. 411 (1999) 359–368.

[28] Z.R. Donaldson, L.J. Young, Oxytocin, vasopressin, and the neurogenetics of sociality, Science 322 (5903) (2008) 900–904.

[29] R.B. Darlington, S.A. Dunlop, B.L. Finlay, Neural development in metatherian and eutherian mammals: variation and constraint, J. Comp. Neurol. 411 (1999) 359–368.

[30] M.A. Dyer, R. Martins, M. de Silva Filho, J.A.P.C. Muniz, L.C.L. Silvera, C.L. Cepko, B.L. Finlay, Developmental sources of conservation and variation in the evolution of the primate eye, Proc. Natl. Acad. Sci. USA 106 (2009) 8963–8968.

[31] A.C. Economos, Brain-lifespan conjecture: a reevaluation of the evidence, Gerontology 26 (1980) 82–89.

[32] B.J. Ellis, J. Bianchi, V. Griskevicius, W.E. Frankenhuis, Beyond Risk and protective factors: an adaptation-based approach to resilience, Perspect. Psychol. Sci. 12 (4) (2017) 561–587.

[33] B.J. Ellis, A.J. Figueredo, B.H. Brumbach, G.L. Schlomer, Fundamental dimensions of environmental risk: the impact of harsh versus unpredictable environ-

ments on the evolution and development of life history strategies, Hum. Nat. 20 (2009) 204–268, https://doi.org/10.1007/s12110-009-9063-7.
[34] B.L. Finlay, R.B. Darlington, Linked regularities in the development and evolution of mammalian brains, Science 268 (1995) 1578–1584.
[35] B.L. Finlay, R. Uchiyama, The timing of brain maturation, early experience, and the human social niche, in: J. Kaas (Ed.), Evolution of Nervous Systems, 2 ed., vol. 3, Elsevier, Oxford, 2017, pp. 123–148.
[36] B.L. Finlay, A.D. Workman, Human exceptionalism, Trends Cogn. Sci. 17 (5) (2013) 199–201, https://doi.org/10.1016/j.tics.2013.03.001.
[37] J.G. Fleagle, Size and adaptation in primates, in: W.L. Jungers (Ed.), Size and Scaling in Primate Biology, Plenum Press, New York, 1985, pp. 1–19.
[38] J.A. Fodor, The Modularity of Mind, MIT Press, Cambridge, 1983.
[39] K.C.R. Fox, M. Muthukrishna, S. Shultz, The social and cultural roots of whale and dolphin brains, Nat. Ecol. Evol. 1 (11) (2017) 1699–1705, https://doi.org/10.1038/s41559-017-0336-y.
[40] M. Gadgil, W.H. Bossert, The life historical consequences of natural selection, Am. Nat. 104 (1974) 1–24.
[41] M. Garwicz, M. Christensson, E. Psouni, A unifying model for timing of walking onset in humans and other mammals, Proc. Natl. Acad. Sci. USA 106 (2009) 21889–21893.
[42] M.H. Goldstein, J.A. Schwade, Social feedback to infants' babbling facilitates rapid phonological learning, Psychol. Sci. 19 (5) (2008) 515–523.
[43] C. Gomez, E.M. Ozbudak, J. Wunderlich, D. Baumann, J. Lewis, O. Pourquie, Control of segment number in vertebrate embryos, Nature 454 (7202) (2008) 335–339 http://www.nature.com/nature/journal/v454/n7202.
[44] C. Gonzalez-Lagos, D. Sol, S.M. Reader, Large-brained mammals live longer, J. Evol. Biol. 23 (5) (2010) 1064–1074, https://doi.org/10.1111/j.1420-9101.2010.01976.x.
[45] J.L. Goodson, R.R. Thompson, Nonapeptide mechanisms of social cognition, behavior and species-specific social systems, Curr. Opin. Neurobiol. 20 (6) (2010) 784–794, https://doi.org/10.1016/j.conb.2010.08.020.
[46] S.J. Gould, Allometry in primates, with emphasis on scaling and the evolution of the brain, Contrib. Primatol. 5 (1975) 244–292.
[47] S.J. Gould, Ontogeny and Phylogeny, Belknap Press, Cambridge, 1977.
[48] A. Grafen, Natural selection, kin selection and group selection, in: J.R. Krebs, N.B. Davies (Eds.), Behavioral Ecology: An Evolutionary Approach, 2nd ed, Blackwell Scientific Publications, Oxford, 1984, pp. 62–84.
[49] A. Grafen, The formal Darwinism project: a mid-term report, J. Evol. Biol. 20 (2007) 1243–1254.
[50] V. Griskevicius, A.W. Delton, T.E. Robertson, J.M. Tybur, Environmental contingency in life history strategies: the influence of mortality and socioeconomic status on reproductive timing, J. Pers. Soc. Psychol. 100 (2) (2011) 241–254.
[51] A.C. Halley, Prenatal brain-body allometry in mammals, Brain Behav. Evol. 88 (1) (2016) 14–24.
[52] A.C. Halley, Minimal variation in eutherian brain growth rates during fetal neurogenesis, Proc. R. Soc. B 284 (1854) (2017) https://doi.org/10.1098/rspb.2017.0219.
[53] P.H. Harvey, J.R. Krebs, Comparing brains, Science 249 (1990) 140–146.
[54] P.H. Harvey, T.H. Clutton-Brock, Life history variation in primates, Evolution 39 (1985) 559–581.
[55] P.H. Harvey, A.F. Read, D.E.L. Promislow, Life history variation in placental mammals: unifying data with theory, Oxf. Surv. Evol. Biol. 6 (1989) 13–31.
[56] P.H. Harvey, R.M. Zammuto, Patterns of mortality and age at first reproduction in natural populations of mammals, Nature 315 (1985) 319–320.
[57] K. Hawkes, Grandmothers and the evolution of human longevity, Am. J. Hum. Biol. 15 (2003) 380–400.
[58] K. Hawkes, Primate sociality to human cooperation, why us & not them?, Hum. Nat. 25 (1) (2014) 28–48.
[59] K. Hawkes, J.E. Coxworth, Grandmothers and the evolution of human longevity: a Review of findings and future directions, Evol. Anthropol. 22 (6) (2013) 294–302.
[60] K. Hawkes, J.F. O'Connell, N.G. Blurton Jones, Hardworking hadza grandmothers, in: V. Standen, R.A. Foley (Eds.), Comparative Socioecology: The Behavioural Ecology of Humans and Other Mammals, Basil Blackwell, London, 1989, pp. 341–366.
[61] K. Hawkes, J.F. O'Connell, N.G. Blurton Jones, Hadza children's foraging: juvenile dependency, social arrangements and mobility among hunter-gatherers, Curr. Anthropol. 36 (4) (1995) 688–700.
[62] K. Hawkes, J.F. O'Connell, N.G. Blurton Jones, Hadza women's time allocation, offspring provisioning, and the evolution of post-menopausal lifespans, Curr. Anthropol. 38 (1997) 551–578.
[63] K. Hawkes, J.F. O'Connell, N.G. Blurton Jones, E.L. Charnov, H.P. Alvarez, Grandmothering, menopause, and the evolution of human life histories, Proc. Natl. Acad. Sci. USA 95 (1998) 1336–1339.
[64] M.A. Hofman, Energy metabolism, brain size and longevity in mammals, Q. Rev. Biol. 58 (1983) 495–512.
[65] M.A. Hofman, On the evolution and geometry of the brain in mammals, Prog. Neurobiol. 32 (1989) 137–158.
[66] S.B. Hrdy, Mother Nature: A History of Mothers, Infants, and Natural Selection, Pantheon Random House, New York, 1999.
[67] S.B. Hrdy, Mothers and Others: The Evolutionary Origins of Mutual Understanding, Harvard University Press, Cambridge, MA, 2009.
[68] S.B. Hrdy, Development plus social selection in the emergence of "emotionally modern" humans, in: C.L. Meehan, A.N. Crittenden (Eds.), Childhood: Origins, Evolution, and Implications, SAR Press, Santa Fe, NM, 2016, pp. 11–45.
[69] K. Isler, C.P. van Schaik, The expensive brain: a framework for explaining evolutionary changes in brain size, J. Hum. Evol. 57 (2009) 392–400.
[70] K. Isler, C.P. van Schaik, Allomaternal care, life history and brain size evolution in mammals, J. Hum. Evol. 63 (2012) 52–63.
[71] A.N. Iwaniuk, K.M. Dean, J.E. Nelson, A mosaic pattern characterizes the evolution of the avian brain, Proc. R. Soc. B 271 (Suppl. 4) (2004) S148–S151.
[72] E. Jablonka, G. Raz, Transgenerational epigenetic inheritance: prevalence, mechanisms, and implications for the study of heredity and evolution, Q. Rev. Biol. 84 (2) (2009) 131–176, https://doi.org/10.1086/598822.
[73] S. Jayaraman, C. Fausey, L.B. Smith, Why are faces denser in the visual experience of younger than older infants?, Dev. Psychol. 53 (1) (2017) 38–49.
[74] H. Jerison, Evolution of the Brain and Intelligence, Academic Press, New York, 1973.
[75] H.J. Jerison, Evolution of prefrontal cortex, in: N.A. Krasnegor, G.R. Lyon, P.S. Goldman–Rakic (Eds.), Development of the Prefrontal Cortex: Evolution, Neurobiolgy and Behavior, H. Brooks Publishing Co, Baltimore, 1997, pp. 9–26.
[76] M.H. Johnson, Subcortical face processing, Nat. Rev. Neurosci. 6 (10) (2005) 766–774.
[77] P. Kaskan, C. Franco, E. Yamada, L.C.L. Silveira, R. Darlington, B.L. Finlay, Peripheral variability and central constancy in mammalian visual system evolution, Proc. R. Soc. B 272 (2005) 91–100.
[78] P.S. Kim, J.E. Coxworth, K. Hawkes, Increased longevity evolves from grandmothering, Proc. R. Soc. B 279 (2012) 4880–4884.
[79] P.S. Kim, J.S. McQueen, J.E. Coxworth, K. Hawkes, Grandmothering drives the evolution of longevity in a probabilistic model, J. Theor. Biol. 353 (2014) 84–94.
[80] T.B.L. Kirkwood, Evolution of aging, Nature 270 (1977) 301–302.
[81] T.B.L. Kirkwood, Repair and its evolution: survival versus reproduction, in: C.R. Townsend, P. Calow (Eds.), Physiological Ecology: An Evolutionary Approach to Resource Use, Blackwell Scientific, Oxford, 1981, pp. 165–189.
[82] T.B.L. Kirkwood, M.R. Rose, Evolution of senescence: late survival sacrificed for reproduction, Phil. Trans. R. Soc. Lond. B 332 (1991) 15–24.
[83] M. Kirschner, Beyond Darwin: evolvability and the generation of novelty, BMC Biol. 11 (1) (2013) 110, https://doi.org/10.1186/1741-7007-11-110.
[84] M.W. Kirschner, J.C. Gerhart, The Plausibility of Life: Resolving Darwin's Dilemma, Yale University Press, New Haven, CT, 2005.
[85] J. Kozlowski, R.G. Weigert, Optimal allocation of energy to growth and reproduction, Theor. Popul. Biol. 29 (1987) 16–37.
[86] J. Kozlowski, J. Weiner, Interspecific allometries are byproducts of body size optimization, Am. Nat. 149 (1997) 352–380.
[87] L. Krubitzer, J.C. Dooley, Cortical plasticity within and across lifetimes: how can development inform us about phenotypic transformations?, Front. Hum. Neurosci. 7 (2013) https://doi.org/10.3389/fnhum.2013.00620.
[88] C.W. Kuzawa, Developmental origins of life history: growth, productivity, and reproduction, Am. J. Hum. Biol. 19 (5) (2007) 654–661.
[89] C.W. Kuzawa, H.T. Chugani, L.I. Grossman, L. Lipovich, O. Muzik, P.R. Hof, D.E. Wildman, C.C. Sherwood, W.R. Leonard, N. Lange, Metabolic costs and evolutionary implications of human brain development, Proc. Natl. Acad. Sci. USA 111 (36) (2014) 13010–13015.
[90] C.W. Kuzawa, Z.M. Thayer, Timescales of human adaptation: the role of epigenetic processes, Epigenomics 3 (2) (2011) 221–234.
[91] M. Lahdenpera, V. V Lummaa, S. Helle, M. Tremblay, A.R. Russell, Fitness benefits of prolonged post-reproductive lifespan in women, Nature 428 (2004) 178–181.
[92] L. Lefebvre, Brains, innovations, tools and cultural transmission in birds, non-human primates, and fossil hominins, Front. Hum. Neurosci. 7 (2013) https://doi.org/10.3389/fnhum.2013.00245.
[93] W.R. Leonard, M.L. Robertson, Comparative primate energetics and hominid evolution, Am. J. Phys. Anthropol. 102 (1997) 265–281.
[94] W.R. Leonard, M.L. Robertson, J.J. Snodgrass, C.W. Kuzawa, Metabolic correlates of hominid brain evolution, Comp. Biochem. Physiol. A 136 (2003) 5–15.
[95] S.L. Lindstedt, W.A. Calder, Body size, physiological time and longevity in homothermic animals, Q. Rev. Biol. 56 (1981) 1–16.
[96] J.L. Locke, B. Bogin, Language and life history: a new perspective on the development and evolution of human language, Behav. Brain Sci. 29 (2006) 259–325.
[97] B.S. Low, A. Hazel, N. Parker, K.B. Welch, Influences on women's reproductive lives: unexpected ecological underpinnings, Cross-Cult. Res. 42 (2008) 201–219.
[98] E.L. MacLean, B. Hare, C.L. Nunn, E. Addessi, F. Amici, R.C. Anderson, … Y. Zhao, The evolution of self-control, Proc. Natl. Acad. Sci. USA 111 (20) (2014) E2140–E2148, https://doi.org/10.1073/pnas.1323533111.
[99] L. Magrassi, K. Leto, F. Rossi, The lifespan of neurons is uncoupled from organismal lifespan, Proc. Natl. Acad. Sci. USA 110 (11) (2013) 4374–4379, https://doi.org/10.1073/pnas.1217505110.
[100] A. Malamitsi-Puchner, K.E. Nikolaou, K.P. Puchner, Intrauterine growth restriction, brain-sparing effect, and neurotrophins, Ann. N. Y. Acad. Sci. 1092 (1) (2006) 293–296, https://doi.org/10.1196/annals.1365.026.
[101] J.S. Millar, R.M. Zammuto, Life histories of mammals: and analysis of life tables, Ecology 64 (1983) 631–635.
[102] D. Nettle, Dying young and living fast: variation in life history across English neighborhoods, Behav. Ecol. 21 (2) (2010) 387–395.

[103] D. Nettle, W.E. Frankenhuis, I.J. Rickard, The evolution of predictive adaptive responses in human life history, Proc. R. Soc. B 280 (1766) (2013) https://doi.org/10.1098/rspb.2013.1343.
[104] J.F. O'Connell, K. Hawkes, N.G. Blurton Jones, Grandmothering and the evolution of Homo erectus, J. Hum. Evol. 36 (1999) 461–485.
[105] B.A. Olshausen, D.J. Field, Emergence of simple-cell receptive field properties by learning a sparse code for natural images, Nature 381 (6583) (1996) 607–609.
[106] M. Palecek, Modularity of mind: is it time to abandon this ship?, Philos. Soc. Sci. 47 (2) (2016) 132–144, https://doi.org/10.1177/0048393116672833.
[107] M.D. Pagel, P.H. Harvey, Evolution of the juvenile period in mammals, in: M.E. Pereira, L.A. Fairbanks (Eds.), Juvenile Primates: Life History, Development, and Behavior, Oxford University Press, New York, 1993, pp. 28–37.
[108] R.E. Passingham, Rates of brain development in mammals including man, Brain Behav. Evol. 26 (1985) 167–175.
[109] R.E. Passingham, J.B. Smaers, Is the prefrontal cortex especially enlarged in the human brain allometric relations and remapping factors, Brain Behav. Evol. 84 (2014) 156–166.
[110] J.C. Pearson, D. Lemons, W. McGinnis, Modulating Hox gene functions during animal body patterning, Nat. Rev. Genet. 6 (2005) 893, https://doi.org/10.1038/nrg1726.
[111] R.H. Peters, The Ecological Implications of Body Size, Cambridge University Press, Cambridge, 1983.
[112] J.E. Pettay, S. Helle, J. Jokela, V. Lummaa, Natural selection on female life-history traits in relation to socio-economic class in preindustrial human populations, PLoS One 2 (2007), e606.
[113] C.D. Placek, R.J. Quinlan, Adolescent fertility and risky environments: a population-level perspective across the lifespan, Proc. R. Soc. B 279 (2012) 4003–4008.
[114] R.A. Poldrack, M.J. Farah, Progress and challenges in probing the human brain, Nature 526 (7573) (2015) 371–379, https://doi.org/10.1038/nature15692.
[115] H. Pontzer, Energy expenditure in humans and other primates: a new synthesis, Annu. Rev. Anthropol. 44 (2015) 169–187, https://doi.org/10.1146/annurev-anthro-102214-013925.
[116] H. Pontzer, D.A. Raichlen, A.D. Gordon, K.K. Schroepfer-Walker, B. Hare, S.R. Ross, Primate energetics and life history, Proc. Natl. Acad. Sci. USA 111 (2014) 1433–1437.
[117] D.E.L. Promislow, P.H. Harvey, Living fast and dying young; A comparative analysis of life history variation among mammals, J. Zool. (Lond.) 220 (1990) 417–437.
[118] J.W. Prothero, K.D. Jurgens, Scaling of maximum lifespan in mammals: a review, in: A.D. Woodhead, K.H. Thompson (Eds.), Evolution of Longevity in Animals: A comparative approach, Plenum Press, New York, 1987, pp. 49–74.
[119] A. Purvis, P.H. Harvey, Mammal life-history evolution: a comparative test of Charnov's model, J. Zool. (Lond.) 237 (1995) 259–283.
[120] A.F. Read, P.H. Harvey, Life history differences among the eutherian radiations, J. Zool. (Lond.) 219 (1989) 329–353.
[121] R. Reep, R.B. Darlington, B.L. Finlay, The limbic system in mammalian brain evolution, Brain Behav. Evol. 70 (2007) 57–70.
[122] J.K. Rilling, M.F. Glasser, S. Jbabdi, J. Andersson, T.M. Preuss, Continuity, divergence, and the evolution of brain language pathways, Front. Evol. Neurosci. 3 (2012) https://doi.org/10.3389/fnevo.2011.00011.
[123] S.L. Robson, C.P. van Schaik, K. Hawkes, The derived features of human life history, in: K. Hawkes, R. Paine (Eds.), The Evolution of Human Life History, School of American Research Press, Santa Fe, 2006, pp. 17–44.
[124] C. Ross, K.E. Jones, Socioecology and the evolution of primate reproductive rates, in: P.C. Lee (Ed.), Comparative Primate Socioecology, Cambridge University Press, Cambridge, 1999, pp. 73–110.
[125] G.A. Sacher, Relation of lifespan to brain weight and body weight in mammals, in: G.E.W. Wolstenholme, M. O'Connor (Eds.), The Lifespan of Animals, Ciba Foundation Colloquia on Ageing, vol. 5, Churchill, London, 1959, pp. 115–141.
[126] T. Sakai, S. Hirata, K. Fuwa, K. Sugama, K. Kusunoki, H. Makishima, H. Takeshita, Fetal brain development in chimpanzees versus humans, Curr. Biol. 22 (18) (2012) R791–R792, https://doi.org/10.1016/j.cub.2012.06.062.
[127] T. Sakai, M. Matsui, M. Mikami, I. Malkova, Y. Hamada, M. Tomonaga, T. Matsuzawa, Developmental patterns of chimpanzee cerebral tissues provide important clues for understanding the remarkable enlargement of the human brain, Proc. R. Soc. B 280 (1753) (2013) https://doi.org/10.1098/rspb.2012.2398.
[128] P.T. Schoenemann, Evolution of the size and functional areas of the human brain, Annu. Rev. Anthropol. 35 (2006) 379–406.
[129] R. Sear, R. Mace, Who keeps children alive? A review of the effects of kin on child survival, Evol. Hum. Behav. 29 (2008) 1–18.
[130] R. Sear, R. Mace, I.A. McGregor, Maternal grandmothers improve nutritional status and survival of children in rural Gambia, Proc. R. Soc. B 267 (2000) 1641–1647.
[131] K. Semendeferi, A. Lu, N. Schenker, H. Damasio, Humans and great apes share a large frontal cortex, Nat. Neurosci. 5 (2002) 272–276.
[132] C.C. Sherwood, J.B. Smaers, What's the fuss over human frontal lobe evolution?, Trends Cogn. Sci. 17 (9) (2013) 432–433.
[133] J.B. Smaers, D.K.N. Dechmann, A. Goswami, C. Soligo, K. Safi, Comparative analyses of evolutionary rates reveal different pathways to encephalization in bats, carnivorans, and primates, Proc. Natl. Acad. Sci. USA 109 (44) (2012) 18006–18011.
[134] B.H. Smith, R.L. Tompkins, Toward a life history of the Hominidae, Annu. Rev. Anthropol. 24 (1) (1995) 257–279.
[135] O. Sporns, Contributions and challenges for network models in cognitive neuroscience, Nat. Neurosci. 17 (5) (2014) 652–660, https://doi.org/10.1038/nn.3690.
[136] S.C. Stearns, The Evolution of Life Histories, Oxford University Press, Oxford, 1992.
[137] G. Striedter, Principles of Brain Evolution, Sinauer, New York, 2005.
[138] W.J. Sutherland, A. Grafen, P.H. Harvey, Life history correlations and demography, Nature 320 (1986) 88.
[139] S. Syal, B.L. Finlay, Thinking outside the cortex: social motivation in the evolution and development of language, Dev. Sci. 14 (2) (2011) 417–430.
[140] N. Tinbergen, On aims and methods of ethology, Zeitschrift für Tierpsychology 20 (1963) 410–433.
[141] A. Vernetti, T.J. Smith, A. Senju, Gaze-contingent reinforcement learning reveals incentive value of social signals in young children and adults, Proc. R. Soc. B 284 (1850) (2017) https://doi.org/10.1098/rspb.2016.2747.
[142] E. Voland, A. Chasiotis, W. Schiefenhovel (Eds.), Grandmotherhood: The Evolutionary Significance of the Second Half of Female Life, Rutgers University Press, Piscataway, NJ, 2005.
[143] J. Wallman, J. Winawer, Homeostasis of eye growth and the question of myopia, Neuron 43 (4) (2004) 447–468.
[144] M.J. West-Eberhard, Developmental Plasticity and Evolution, Oxford University Press, Oxford, 2003.
[145] D. Western, Size, life history, and ecology in mammals, Afr. J. Ecol. 17 (1979) 185–202.
[146] D. Western, J. Ssemakula, Life history parameters in birds and mammals and their evolutionary interpretations, Oecologia 54 (1982) 281–290.
[147] G.C. Williams, Pleiotropy, natural selection, and the evolution of senescence, Evolution 11 (1957) 398–411.
[148] G.C. Williams, Natural selection, the cost of reproduction, and a refinements of Lack's principle, Am. Nat. 199 (1966) 687–690.
[149] A.D. Workman, C.J. Charvet, B. Clancy, R.B. Darlington, B.L. Finlay, Modeling transformations of neurodevelopmental sequences across mammalian species, J. Neurosci. 33 (2013) 7368–7383.