Evidence that human skill level is more reactive to developmental history than to genes or environment

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Abstract

Behaviour can be influenced by genes, environment, and developmental history, but quantifying these effects in humans and over our evolutionary past is challenging. Here we use a mathematical model that mechanistically recovers major aspects of human development and evolution to compute the causal effects on adult energy extraction skill (e.g., at food acquisition or processing) of point interventions earlier in development. We find that, in this model, adult skill is little reactive to genetic change due to trade-offs in energy allocation to tissue growth, and that this reactivity has decreased over evolution. Adult skill is more reactive to environmental changes and least reactive to social change, and these reactivities have increased over evolution. Adult skill is much more reactive to changes in developmental history, defined as developmentally earlier phenotypes, than to genetic, environmental, or social change, although this reactivity has decreased over evolution. For instance, the model predicts that developmental changes that increase either an individual's body size as an infant or their skill level as a child boost adult skill in the same individual proportionally much more than enhancing this individual's environment as an infant or as a child. Our analyses suggest that developmental history can be a major influence on human skill acting separately from genetic, environmental, and social factors.

Significance statement

We traditionally think of our behavior as being most influenced by either genes or the environment. However, a third possibility is that our behaviour is influenced by our own personal history. For instance, while your two hands may have the same genes and have experienced mostly the same environment, they have different fingerprints because of random events during development. Yet, quantifying the relevance of this personal history is challenging in real organisms. Here we quantify the effect of these three components, namely genes, environment, and developmental history, in a computational replica of human brain evolution. We find that human skill level responds most strongly to changes in developmental history than to changes in genes or the environment.

Introduction

The human brain is strongly influenced by environmental factors[1]. Multiple lines of evidence, including molecular[2–5], anatomical[6–9], and behavioural[10;11], point to the existence of human specialisations for neuroplasticity[1;12–14]. The high level of plasticity of the human brain helps explain human enhanced learning abilities[15] and makes it possible to develop intervention strategies that can change behavioural and cognitive outputs over life[16].

Observed differences in neuroplasticity between chimpanzees and humans indicate that increased neuroplasticity evolved in the human lineage after the divergence of the two lineages, together with an overall increase in brain size and a general reorganization of the brain[17]. Patterns of brain reorganization can be inferred to a certain extent from qualitative assessments and quantitative shape analyses of endocranial morphology, and they include a frontal expansion[18], upper parietal expansion[19], occipital reorganization including a reduction of the primary visual cortex[20], and cerebellar expansion[21;22], among others.

However, unlike brain size and reorganization, changes in neuroplasticity are extremely difficult to document in the fossil record. While differences and similarities in brain development have been described among modern humans and fossil hominins, including Neanderthals[23], *Homo erectus*[24;25], and even australopiths[26], quantifying the extent to which brain development is influenced by environmental factors in fossils remains out of reach. This is partly due to the small sample sizes for fossil endocasts, particularly for immature individuals, the limited information provided by the endocranial evidence, the high frequency of taphonomic alterations and deformations, and the impossibility of carrying out longitudinal studies.

Given the limitations posed by the empirical study of the endocranial fossil record, mathematical modelling approaches can be particularly useful to study the evolution of brain development. These approaches can use empirical data from the fossil record, for instance, by determining whether the output of mathematical models that describe brain changes over ontogenetic and evolutionary time match fossil data[27]. Modelling approaches

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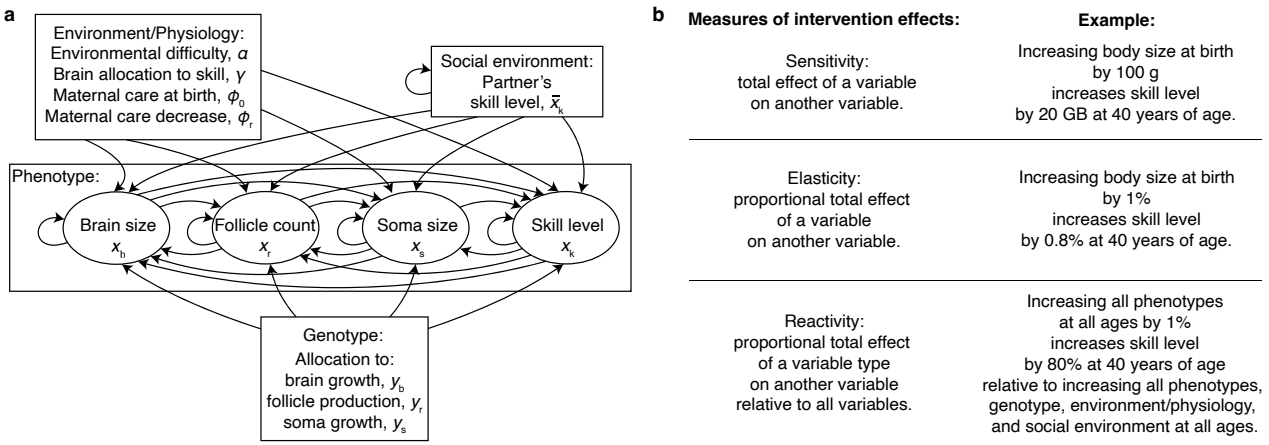

Figure 1: Causal diagram of the brain model and measures of the effects of intervention. **a,** Causal diagram of the model, where an arrow indicates a direct causal effect of a variable on another one or on itself at the next age. Variables are classified into genotypic, phenotypic, environmental/physiological, and social environmental. Genotypic variables are controlled by the individual's genotype. Phenotypic variables are constructed over development; the developmental history for a given age is defined here as the phenotypic variables at earlier ages. Environmental or physiological variables affect the development of phenotypic variables. Social environmental variables are the skill level (and so phenotypic variables) of social partners, which affect the development of the focal individual. **b,** We quantify the effect of intervention, that is, of altering a variable at a given age, on adult skill level, using three measures: sensitivity $\mathrm{d}x/\mathrm{d}y$, which measures total effects, that is, the effect of altering a variable over all causal pathways linking it to the response variable; elasticity $(y/x)\mathrm{d}x/\mathrm{d}y$, which measures the proportional total effects; and reactivity, which normalizes elasticity over all the possible interventions over development.

have been used to link developmental and evolutionary change in mammalian[28] and primate dentition[29], and they hold substantial promise to further our understanding of the relationship between brain development and evolution[30;31].

The analysis of brain evolution may focus on anatomical traits (e.g., brain size, brain organization, and neuron numbers) or on functional traits (e.g., skill level at particular tasks such as food processing, tool making, language use, and social performance). While brain anatomy and function are linked, their relationship is not straightforward and they do not necessarily respond in the same way to modifications of underlying variables. Most studies of human brain evolution, particularly those exploring brain evolution in fossil hominins, focus on anatomical traits because those are more readily measurable. Yet, functional traits are of central interest. Changes in skill level are often inferred qualitatively from archaeological evidence[32], but recent studies have quantitatively modelled the ontogenetic development and long-term evolution in skill levels during hominin evolution[31]. These studies open the possibility of studying the change in plasticity in skill level over hominin evolution.

While plasticity refers to the developmental response of a trait to environmental change, an individual's skill level is the product of a complex interplay of genetic, developmental, environmental, and social factors. Common approaches to quantify the effect of a variable on another one, such as regression and causal inference, use least-squares[47], which yields population-level estimates. However, it is also of interest to estimate individual-level effects, for instance, the extent to which an individual's skill level responds to genetic, developmental, environmental, or social changes experienced earlier in life by the same individual. This can be attained by means of mechanistic modelling of the individual's skill development. Sensitivity analyses of such a model[48] using causal derivatives[49;50] (see Methods) can then be used to estimate causal effects.

Here we use a mechanistic modeling approach to analyse the causal influences on human skill over development and how such influences have evolved from *Homo habilis* to *H. sapiens*. To do this, we use a mathematical model, hereafter referred to as the brain model, that mechanistically describes the developmental dynamics of human skill level from energy conservation analysis[51;52] (Fig. 1a; conceptually described in ref.[27]). In the model, skill level is defined as the capacity of an individual to overcome ecological or social challenges to extract energy from their environment[51] and it is measured in information units[30]. It has been previously found that this model recovers major aspects of human development and evolution[51;30;31;27]. Specifically, it recovers the expansion of adult brain and body sizes from *H. habilis* to *H. sapiens* scale, and the evolution of various aspects of human development, including childhood, adolescence, and adulthood periods with timing matching that observed in *Homo sapiens* (Fig. 2)[30;31]. Moreover, this model identifies a transition to seemingly cumulative culture in the Middle Pleistocene[30;27], which is consistent with the archaeological record[53–55]. We use this brain model to measure the effects on skill level of interventions performed earlier in life in the same individual (Fig. 1b).

We show that, in this model, for individuals with brain and body sizes of *H. sapiens* scale, marginally changing an individual's genetic composition has little effects

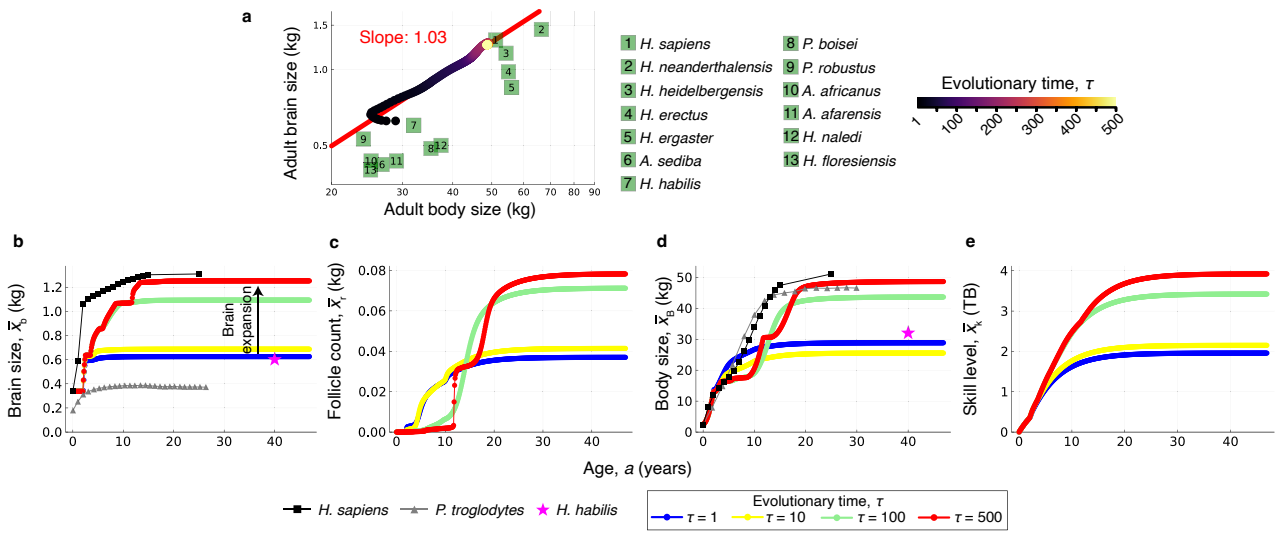

Figure 2: In-silico replica of the evolution and development of hominin brain size, body size, and skill level as obtained by the brain model. **a,** Green squares show the observed mean adult brain and body sizes in log-log scale for 13 hominin species (data from refs.[33–44] for females when possible). Coloured dots give the recovered evolution of adult brain and body sizes by the brain model, shifting from those of *H. habilis* to those of *H. sapiens*. **b-e,** Developmental dynamics underlying the trajectory in **a**: for (**b**) brain size, (**c**) pre-ovulatory ovarian follicle count (in mass units), (**d**) body size, and (**e**) skill level. Panels **b,d,** also show observed cross-sectional mean values of *H. sapiens* females (black squares; data from refs.[35;36]), *P. troglodytes* females (gray triangles; data from refs.[45;46]), and adult *H. habilis* of mixed sexes for brain size and of females for body size (pink stars: data from ref.[41]). One evolutionary time is the time from mutation to fixation, which would be 11.5 ky assuming fixation takes 500 generations and generation time is 23 years. All panels are taken with modification from ref.[31].

on her adult skill level, whereas marginally changing her body size or skill level during early childhood has much stronger effects on her adult skill level, in accordance with previously successful empirical interventions[56;57]. We introduce the notion of reactivity to quantify how sensitive a variable is to another variable relative to its sensitivity to all the other variables it depends on over development. This shows that over the recovered evolution from *H. habilis*, skill level has become less reactive to changes in the individual's genotype and developmental history (i.e., developmentally earlier phenotypes), and more reactive to environmental, physiological, and social changes. Yet, adult skill level in the model is substantially more reactive to phenotypic changes earlier in development than to any other type of intervention.

Results

Brain model overview

The brain model has the following structure (Fig. 1a; full details are in the Supplementary Information, SI). It considers females only and clonal reproduction for simplicity. Each female has three "genotypic traits" (y_{ia}), that is, traits that are under direct genetic control: $y_{\mathrm{b}a}$ gives the effort devoted to brain growth at age $a \in \{1,\ldots,N_{\mathrm{a}}\}$, $y_{\mathrm{r}a}$ is the effort devoted to producing mature ovarian follicles at age a which determine fertility, and $y_{\mathrm{s}a}$ is the effort devoted to growth of remaining somatic tissue at age a (the number of age bins is $N_{\mathrm{a}} = 470$ since we track development from birth to 47 years of age with 10 age bins per year). Such growth efforts can be understood as deviations from a reference value and so can take negative or positive values. The growth effort y_{ia} for each tissue i and each age a can be understood as being determined by a separate genetic locus, so there are $N_{\mathrm{g}}N_{\mathrm{a}} = 3 \times 470 = 1410$ loci. Because reproduction is clonal, it is unnecessary to specify how these loci are linked or whether they are in autosomes or sex chromosomes.

Each female also has four "phenotypic traits" (x_{ia}), which are constructed over development, and give the brain mass, mature ovarian follicle count in mass units, remaining somatic tissue mass, and skill level (for $i \in \{\mathrm{b},\mathrm{r},\mathrm{s},\mathrm{k}\}$) at each age a. Body size is the sum of the mass of all tissues ($x_{\mathrm{B}a} = x_{\mathrm{b}a} + x_{\mathrm{r}a} + x_{\mathrm{s}a}$). The key difference between genotypic traits (y_{ia}) and phenotypic traits (x_{ia}) is that the latter are not given by the genotype alone but are constructed over development. The development of phenotypic traits depends on the skill level of social partners ($\bar{x}_{\mathrm{k}a}$, where the overbar denotes resident or mean trait values) due to cooperation and competition for resources.

The development of phenotypic traits also depends on 19 parameters describing the environment or physiology of the organism including the environmental difficulty α, maternal care at birth φ_1, rate of maternal care decrease φ_{r}, and skill decisiveness γ at overcoming challenges (Eq. S10; the value of the i-th parameter at age a is ϵ_{ia}).

In vector notation (e.g., $\mathbf{x}_a = (x_{\mathrm{b}a}, x_{\mathrm{r}a}, x_{\mathrm{s}a}, x_{\mathrm{k}a})^{\mathsf{T}}$), the

development of phenotypic traits is given by

$$\mathbf{x}_{a+1} = \mathbf{g}_a(\mathbf{x}_a, \mathbf{y}_a, \boldsymbol{\epsilon}_a, \bar{x}_{\mathrm{k}a}) \tag{1}$$

where $\mathbf{g}_a$ is a function termed the developmental map. The brain model specifies $\mathbf{g}_a$ which is derived from energy conservation and phenomenological considerations of how skill level translates into energy extraction. Evolution occurs as the resident genotype $\bar{\mathbf{y}}$ evolves, which results in evolution of the resident phenotype $\bar{\mathbf{x}}$ (where the phenotype over life is $\mathbf{x} = (\mathbf{x}_1; \ldots; \mathbf{x}_{N_\mathrm{a}})$, the semicolon indicates a linebreak, and similarly for $\bar{\mathbf{y}}$).

Developmental sensitivity and elasticity

For this model, we analyse the sensitivity of skill level to changes earlier in development, and how such sensitivity has changed over evolution (Fig. 1b). We compute the sensitivity of skill level to developmentally earlier point perturbations in genotypic traits, phenotypic traits, social partners' skill level, or the parameters, each at a given age. For instance, we compute the effect on the skill level of a 40-year-old individual caused by a genotypic change that marginally changed her energy allocation to brain growth at 1 year of age but only at that 1 year of age (i.e., the total derivative $\mathrm{d}x_{\mathrm{k}40}/\mathrm{d}y_{\mathrm{b}1}$). Similarly, we also compute the effect on the skill level of a 40-year-old caused by a phenotypic change that marginally changed, for instance, her body size when she was 2 years old. We let these phenotypic changes occur independently of genetic or environmental changes; for instance, via developmental noise, although our approach does not need to specify how those changes occur [p. 6 of ref.[48]].

To compute these sensitivities, we use general formulas for the sensitivity of a recurrence derived previously[50]. These formulas give causal effects by implementing the perturbation at a given age and following its effects onwards over development (so, $\mathrm{d}x_{ia}/\mathrm{d}y_{kj} = 0$ if the perturbation age is later than the response age, $j > a$). These formulas also include causal feedbacks (e.g., $\mathrm{d}x_{ia}/\mathrm{d}y_{kj}$ depends on the developmental feedback matrix[58;50] $(2\mathbf{I} - \partial\mathbf{x}^\intercal/\partial\mathbf{x})^{-1}$ that in turn depends on direct causal effects $\partial x_{lr}/\partial x_{np}$ given by the recurrence (1) that are zero if the perturbation age p is later than the response age r). The units of the sensitivities depend on the perturbed and response variables. To compare sensitivities despite their different units, a standard approach is to compute the elasticity[48], which is the proportional sensitivity, so is dimensionless and is defined only for positive variables. As genotypic traits can take negative values (e.g., defined as deviations from a reference value), we compute a slight modification of elasticity in this situation (e.g., $(|y_{\mathrm{b}1}|/x_{\mathrm{k}40})\mathrm{d}x_{\mathrm{k}40}/\mathrm{d}y_{\mathrm{b}1}$). Thus, to compare sensitivities, we analyse the developmental elasticities of skill level over the evolutionary brain expansion from *H. habilis* to *H. sapiens* as recovered by the brain model.

We find that adult skill level is little elastic to change in genotypic traits, and much more elastic to change in phenotypic traits. With *H. sapiens* brain and body sizes, perturbing the genotypic traits at any age typically has small effects on the adult skill level, but perturbing the genotypic traits around the age of menarche has more pronounced effects (Fig. 3a; the Computer Code that generated the figures is in ref.[59]). Menarche is here defined as the age displaying developmental emergence of mature ovarian follicles (at around 12 years of age; Fig. 2c). With *H. habilis* brain and body sizes, perturbing genotypic traits also has small effects on adult skill, with sensitive periods to genetic change occurring earlier in development but also coinciding with the onset of reproductive ability (Fig. 3e).

In contrast, adult skill level is more elastic to change in the developmentally earlier phenotypes by two orders of magnitude with either *H. sapiens* or *H. habilis* brain and body sizes (Fig. 3b,f). This is unexpected as both genotypic ($\mathrm{d}\mathbf{x}^\intercal/\mathrm{d}\mathbf{y}$) and phenotypic ($\mathrm{d}\mathbf{x}^\intercal/\mathrm{d}\mathbf{x}$) change induce developmental feedback ($(2\mathbf{I} - \partial\mathbf{x}^\intercal/\partial\mathbf{x})^{-1}$) and both are perturbed at a single age only. Inspection of the analytical expressions of the sensitivity of a recurrence ($\mathrm{d}\mathbf{x}^\intercal/\mathrm{d}\mathbf{y}$ and $\mathrm{d}\mathbf{x}^\intercal/\mathrm{d}\mathbf{x}$) shows that the reason that sensitivities to genotypic change are relatively small is that the direct effects of phenotype on phenotype are mostly positive (section S3.2.1 of ref.[31]), whereas the direct effects of genotype on phenotype are often negative due to the trade-offs in energy allocation to growth of the various tissues (section S3.2.2 of ref.[31]). These positive and negative direct effects mean that the total effect of genotype on phenotype may be positive via a path on the causal diagram but negative via another one, and the two effects cancel each other yielding little total genotypic effect on phenotype.

With *H. sapiens* brain and body sizes, adult skill level is most elastic to perturbation in body size in infancy and early childhood, in skill level in mid-childhood, and in brain size during most of late childhood and adulthood (Fig. 3b). During adolescence, interventions on variables other than brain size have essentially no proportional effect on adult skill level in the model. Here adolescence is defined as the period where the individual is reproductively capable but keeps growing, which occurs between 12 to 19 years of age (Fig. 2c,d). It seems that this inelasticity of adult skill to adolescent perturbation is because by adolescence the brain has stopped growing in the model, as the perturbation effects on adult skill level vanish when the perturbations are implemented after the brain has stopped growing. With *H. habilis* brain and body sizes, the window of elasticity of adult skill to developmentally earlier change in skill level and body size is much shorter (Fig. 3f) and confined to the short period of brain growth (Fig. 2b).

Altering the environment and physiology has moderate proportional effects on adult skill under both *H. sapiens* and *H. habilis* brain and body sizes. With *H. sapiens* brain and body sizes, increasing the environmental difficulty at the age of menarche has a strong positive proportional effect on adult skill level (blue dots in Fig. 3c). Yet, increasing the environmental difficulty at other ages often has strong negative proportional effects on adult skill level (blue dots in Fig. 3c). With *H. habilis* brain and body sizes, increasing the environmental difficulty has mostly negative proportional effects on adult skill, with the proportional effect of changing the environmental difficulty at the age of menarche being only slightly positive (blue

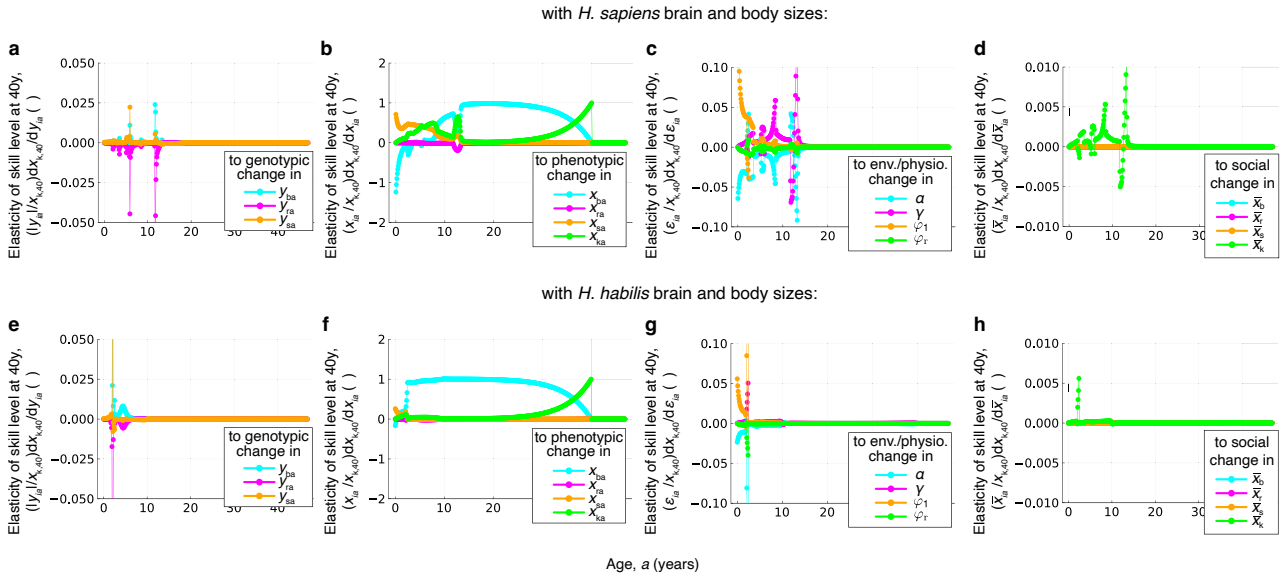

Figure 3: Recovered elasticity of skill level for the brain and body sizes of *H. sapiens* and *H. habilis*. Elasticity of adult skill level to (**a**,**e**) genotypic, (**b**,**f**) phenotypic, (**c**,**g**) environmental or physiological, and (**d**,**h**) social change: plots give the change in skill level at 40 y of age when the indicated variable is marginally increased at the age and only at the age in the horizontal axis. Panels **a-d** are for individuals with brain and body sizes of *H. sapiens* scale (i.e., for red dots in Fig. 2b-e). Panels **e-h** are for individuals with brain and body sizes of *H. habilis* (i.e., for blue dots in Fig. 2b-e). In **b** and **f**, the elasticity after 40 years of age is zero because the skill level at 40 years of age is not affected by changes implemented in the future.

dots in Fig. 3g). The decisiveness of skills at energy extraction has largely opposite proportional effects to those of environmental difficulty (pink dots in Fig. 3c,g). Either increasing maternal care or decreasing the rate of maternal care decrease early in childhood has positive effects on adult skill level (orange and green dots in Fig. 3c,g). Importantly, these developmental effects of changing parameters can be opposite to the evolutionary effects of changing such parameters (e.g., whereas increasing environmental difficulty often decreases skill level over development, increasing environmental difficulty *increases* skill level over evolution; Figs. 6 and V of ref.[51]). This suggests that developmental drivers of cognition may not reliably indicate its evolutionary drivers[60].

Social interactions also have moderate developmental effects. With *H. sapiens* brain and body sizes, increasing the skill level of social partners with whom the individual interacts at the age of menarche has marked negative proportional effects on adult skill level (green dots in Fig. 3d). Increasing the skill level of social partners with whom the individual interacts at other ages typically has positive proportional effects on adult skill level (green dots in Figs. 3d). With *H. habilis* brain and body sizes, increasing the social partners' skill level typically has positive proportional effects on the focal individual's skill, particularly around the age of menarche (Fig. 3h). Although a perturbation on a social partner's trait releases a developmental feedback in the social partner, we only consider the direct effect of the perturbation of social partners' traits on the focal individual, without considering the released developmental feedback in social partners that can then affect the focal individual. Hence, as skill level is the only trait in social partners assumed to directly affect the development of the focal individual, perturbing phenotypes other than skill level in social partners has no effect on the focal individual's adult skill (blue, pink, and orange dots are all zero in Fig. 3d,h). These perturbations in social interactions do not consider the effects of social learning, which has not yet been explicitly incorporated in the model.

Developmental reactivity

We are interested in quantifying the plasticity of skill level in the recovered hominin brain expansion. A common way to assess plasticity is via heritability: a trait with higher heritability is sometimes interpreted as being less plastic. Heritability, however, depends on population-level phenomena such as genetic, phenotypic, and environmental variation.

To quantify the extent to which a phenotype responds to genetic, developmental, or environmental change considering only individual-level properties, we construct an index using the developmental sensitivities computed above. We term this index "reactivity", which measures the elasticity of a phenotype to change in a variable relative to the phenotype's total elasticity when changing all variables during development. Thus, reactivity ranges from 0 to 1 and the reactivities to the different variables add up to 1. Robustness (or canalisation) to changing a variable is given by one minus reactivity. Reactivity and robustness defined in this manner are individual-level properties rather than population-influenced quantities such as heritability.

With brain and body sizes of *H. habilis* scale, the reactivity of skill level to genotypic change is small, with skill level at around 5 years of age being most reactive to geno-

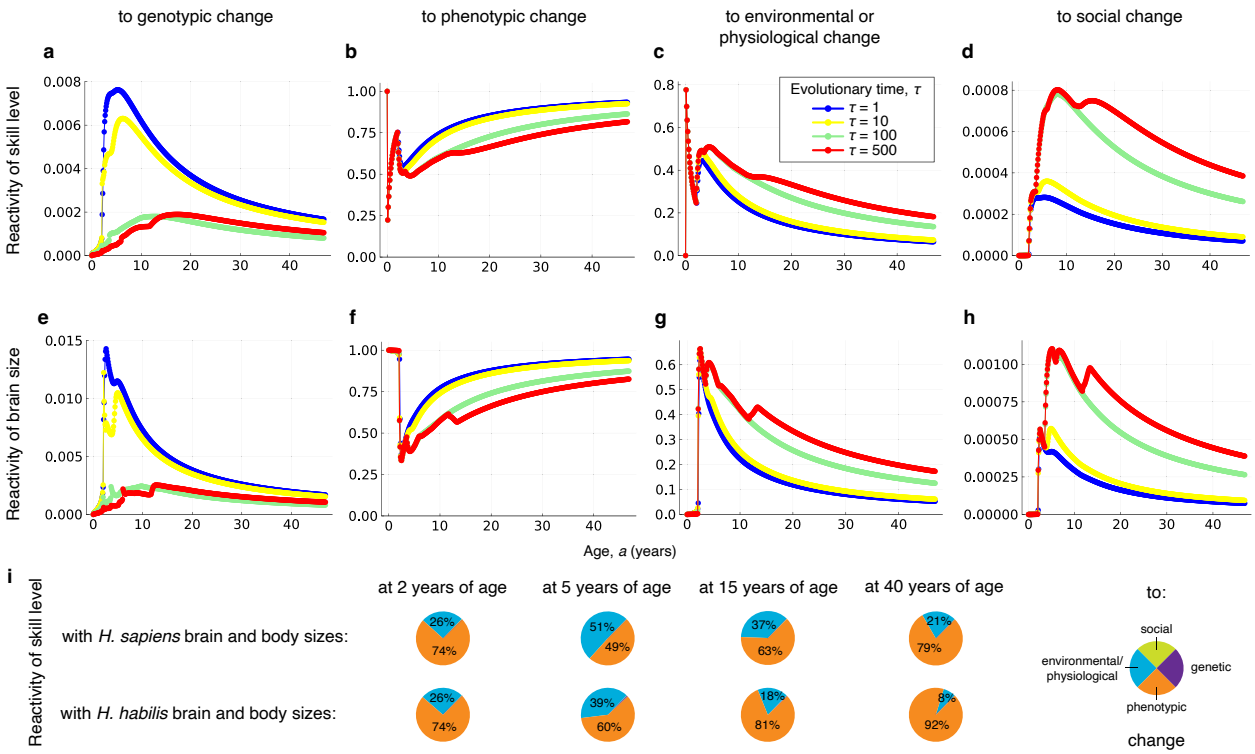

Figure 4: Developmental reactivity of skill level and brain size over the recovered hominin brain expansion. **a,** Reactivity of skill level to genotypic change; for instance, the red dot at the age $a = 10$ y gives how much the skill level of a 10 year old is affected by marginally increasing all genotypic traits across all ages relative to changing all variables developmentally affecting skill level at the evolutionary time where adult brain and body sizes are of *H. sapiens* scale. The blue dot at the same age is for the evolutionary time when adult brain and body sizes are of *H. habilis* scale. Reactivity of skill level to **b,** phenotypic, **c,** environmental or physiological, and **d,** social change. Reactivities add up to 1 (e.g., the red lines at a given age in **a**, **b**, **c**, and **d** add up to 1). The largest reactivity is to phenotypic change (**b**), followed by the reactivity to environmental or physiological change (**c**), genotypic change (**a**), and social change (**d**) (see scale in vertical axes). The reactivity of skill level to genotypic and phenotypic change has decreased over evolution (**a**, **b**). In contrast, the reactivity of skill level to environmental, physiological, and social change has increased over evolution (**c**, **d**). **e-h,** The reactivities of brain size have similar patterns. **i,** Pie charts illustrating the reactivities of skill level in **a-d** at *H. sapiens* ($\tau = 500$) and *H. habilis* ($\tau = 1$) brain and body sizes for some ages.

typic change (blue dots in Fig. 4a). As evolution proceeds and brain and body sizes reach *H. sapiens* scale, genotypic reactivity decreases substantially with a peak at around 18 years of age (red dots in Fig. 4a).

Skill level is much more reactive to changes in developmental history than to genotypic change. With *H. habilis* brain and body sizes, skill level throughout life reacts strongly to phenotypic change, increasing with age (blue dots in Fig. 4b). Over the evolution to *H. sapiens* brain and body sizes, the reactivity of skill level to developmental history decreases but remains substantial (red dots in Fig. 4b).

Skill level is moderately reactive to environmental or physiological change. With *H. habilis* brain and body sizes, skill level reacts less strongly to environmental or physiological change than to developmental history, and this reactivity decreases with age (blue dots in Fig. 4c). Over the evolution to *H. sapiens* brain and body sizes, the reactivity of skill level to environmental or physiological change increases (red dots in Fig. 4c).

The reactivity of skill level to social change is very small although it increases over evolution. With *H. habilis* brain and body sizes, skill level reacts slightly to changes in the skill level of social partners, with a developmental peak at around 5 years of age (blue dots in Fig. 4d). Over the evolution to *H. sapiens*, the social reactivity of skill more than doubles, with a developmental peak at around 9 years of age (red dots in Fig. 4d). However, for brain and body sizes of *H. sapiens* scale, the reactivity of skill level to social change is three orders of magnitude smaller than to phenotypic, environmental or physiological change, and one order of magnitude smaller than to genotypic change (red dots of Fig. 4a-d). Such small reactivity to social change may result partly because we do not consider the developmental feedback in social partners nor model social learning explicitly (see above). Some of these reactivities are visually summarised in Fig. 4i. The sensitivities and elasticities underlying the reactivities presented are shown in Figs. S1-S16. The reactivities of the mature ovarian follicle count and somatic tissue size over the recovered human brain expansion are shown in Fig. S17.

Discussion

Our mechanistic modelling approach enables calculation of how the developmental response of skill level to various factors has changed over hominin evolution and offers an approach to predict the effects of intervention on skill. Our results find a reduction in the reactivity to genetic change over evolution and an increase in the reactivity to non-genetic factors, which is consistent with the view that humans have evolved an increased level of plasticity in skill level and brain anatomy[1;61;8].

Our results suggest that adult human skill level is much more sensitive to the individual's developmental history than to any other factors, including genetic change, environmental change, and changes in the skill level of social partners. Developmental history is here captured by the phenotypes, which store the effects that all developmentally relevant factors (genotype, environment, social context, developmentally earlier phenotypes, and the way they all interact during development) have had on the individual until a given age. Although the sources of variation of behaviour are traditionally partitioned into genetic and environmental, a developmental source of variation that is separate from genetic and environmental is developmental noise. Our results, which quantify separately the genetic, environmental, and developmental effects on adult skill level, indicate that developmental noise, by its ability to affect developmental history separately from genetic and environmental variation, can have major effects on generating behavioural variation. Thus, our results are consistent with the increasing volume of work evidencing the relevance of developmental noise in phenotypic variation[62–71]. However, our calculations did not consider all biologically possible effects, for instance, by not including the developmental feedback in social partners or additional complexities such as social learning, which could in principle render the social environment more influential on an individual's adult skill level.

In our model, the two major factors that influence adult skill level are developmental history and environmental change (which includes factors such as environmental difficulty and maternal care, Fig. 1), with genetic change and social change having much smaller effects. Developmental history and environmental change have an opposite pattern in influencing skill level. During the first period of life (up to approximately 10 years old), skill level is influenced by developmental history and environmental variation in roughly equal proportions (approximately 50%) in *H. sapiens* (Fig. 4b,c). However, as individuals age, skill level becomes increasingly reactive to developmental history and increasingly insensitive to environmental change, with only 20% of the skill level reactivity being due to environmental change at 40 years of age.

Even if skill level differs substantially between earlier and later species of the genus *Homo*, with increased technological complexity identified in the archaeological record[72–75], our results suggest that the predominant role of developmental history in affecting skill level may be shared by many species within the genus, spanning from those of *H. habilis* brain and body sizes to those of *H. sapiens* brain and body sizes. The reactivity of skill level is rather similar across this brain and body size variation in the model, suggesting a lack of abrupt changes in the way that skill level is shaped over life despite changes in timing and extent of the effects (Fig. 3). However, later hominin species are predicted by the model to have evolved a relatively increased influence of point perturbations in environmental and social factors, an influence that has doubled with respect to earlier hominins, even if those factors still have an overall minor influence with respect to developmental history. According to our results, the combination of factors influencing skill level observed in *H. sapiens* would be already present in similar proportions 1.1 Myrs after the origin of the genus *Homo* (at evolutionary time $\tau = 100$; Fig. 4). Assuming a 2.5 Ma origin for the genus *Homo*, even if an earlier origin is possible[76], this would mean that a similar interplay of genetic, developmental, environmental and social factors as that observed in *H. sapiens* was already shaping skill level in the genus *Homo* at 1.4 Ma, a time period corresponding with *H. erectus* fossils[77], thus indicating that this combination of factors had already evolved in this species.

Later hominin species in the model also show an increased window of opportunity for environmental and social factors to influence skill level. At *H. habilis* brain size, environmental and social factors substantially modify skill level only until less than five years of age, roughly coinciding with the expected weaning age[78;79]. This indicates that skill level in early species of the genus *Homo* would be comparatively little influenced by environmental factors after the age of weaning, consistent with findings that many primate species reach adult skill levels around the age of weaning due to the lack of post-weaning provisioning[80]. However, at *H. sapiens* brain size, environmental and, to a lesser extent, social factors can modify skill level until almost 15 years of age, well into adolescence. This observation is consistent with previous studies indicating late brain maturation in humans[81;82] associated with late acquisition of adult skill levels[83;84]. Indeed, the early age at which *H. habilis* brains would stop to be influenced by environmental factors corresponds with a period of peak metabolic demand in *H. sapiens*[35] associated with neural plasticity and learning, and involving overproliferation of dendritic arbors and synapses before activity-dependent pruning[85;86]. In other words, *H. sapiens* brains and skill levels are still strongly influenced by environmental factors at an age at which *H. habilis* skill level would be largely insensitive to any influences beyond developmental history (Fig. 4i).

Our results show a similar overall pattern of evolutionary change in the reactivity of skill level and brain size (Fig. 4). This result arises because of the emergence of a proportionality between adult brain size and adult skill level in the model[51;31] and is consistent with brain size and skill level being positively correlated in primates[87;88]. Indeed, complex manipulative skills in primates are built upon simpler skills and require a slow brain development, which is associated with large brain size[80]. More complex and later emerging skills take longer to develop than simpler and earlier emerging skills[80] in a similar way as later occurring neurodevelopmental events take longer than earlier occurring events in larger brained mammals[89;90],

primates[91], and hominins[14]. Therefore, the extended window of opportunity for environmental factors to influence skill level in later hominins, which is identified by our model, may have been particularly advantageous in the context of large and slowly maturing brains associated with late acquired adult skill levels. Accordingly, having a large brain size with larger numbers of neurons is considered to be a pre-requisite for species to have high levels of behavioural plasticity, as larger neural networks allow for a broader variety of behaviours[61].

The results of our mechanistic model indicate that skill level may be most strongly dependent on developmental history throughout the evolution of the genus *Homo*, with the effect of genetic change decreasing and the effect of environmental and social change increasing from earlier to later *Homo*. Our results also point to an association between brain expansion, late acquisition of adult skills, and heightened windows of opportunity for non-genetic factors to influence skill level in later *Homo*.

Methods

Sensitivity. Sensitivity analyses of a model[48] can be used to estimate causal effects. Such causal effects can be computed with derivatives, provided that such derivatives are causal[49]. Specifically, if a variable x causally affects $y = f(x)$ for some function f but y does not causally affect x, where these causal relationships are respectively denoted by $x \to y$ and $x \nrightarrow y$, then causal derivatives of x with respect to y must be zero even if f is invertible[92]. Recently derived formulas for the sensitivity of a recurrence[50] give the causal derivatives of variables mechanistically described by a system of recurrence equations and allow for arbitrarily complex and non-linear causal feedbacks. This causality from derivatives is achieved by considering the recurrence from the time of a pertubation onwards, applying a perturbation at such initial time, and following the effects of the perturbation from that time onwards[50]. In particular, this approach implies the causal arrow of time, such that perturbations can only causally affect the future, but not the past.

Reactivity. We define reactivities as follows. We compute the elasticity of phenotype k at age j to change in $\zeta \in \mathbb{R}$ as

$$E_{\zeta kj} = \frac{1}{x_{kj}} \frac{\mathrm{d}x_{kj}}{\mathrm{d}\zeta} |\zeta|. \tag{2}$$

When ζ is positive, $E_{\zeta kj}$ is the standard elasticity of phenotype k at age j to change in ζ. We extend this notion to let the perturbed variable be a vector, so the elasticity of phenotype k at age j to change in $\boldsymbol{\zeta} \in \mathbb{R}^{n\times 1}$ is

$$E_{\boldsymbol{\zeta} kj} = \frac{1}{x_{kj}} \frac{\mathrm{d}x_{kj}}{\mathrm{d}\boldsymbol{\zeta}^{\intercal}} |\boldsymbol{\zeta}| = \sum_i \frac{1}{x_{kj}} \frac{\mathrm{d}x_{kj}}{\mathrm{d}\zeta_i} |\zeta_i|, \tag{3}$$

where $|\boldsymbol{\zeta}|$ is the vector with the entries of $\boldsymbol{\zeta}$ in absolute value and ζ_i is the i-th entry of $\boldsymbol{\zeta}$. In the evo-devo dynamics framework we use[50], the developmental map is in general of the form $\mathbf{x}_{a+1} = \mathbf{g}_a(\mathbf{x}_a, \mathbf{y}_a, \boldsymbol{\epsilon}_a, \bar{\mathbf{x}}, \bar{\mathbf{y}})$. Then, letting $\mathbf{t} = (\mathbf{x}; \mathbf{y}; \boldsymbol{\epsilon}; \bar{\mathbf{x}}; \bar{\mathbf{y}})$ be the vector of all the variables affecting development, the total elasticity of phenotype k at age j to change in $\mathbf{t}$ is

$$E_{\mathbf{t}kj} = E_{\mathbf{x}kj} + E_{\mathbf{y}kj} + E_{\boldsymbol{\epsilon}kj} + E_{\bar{\mathbf{x}}kj} + E_{\bar{\mathbf{y}}kj}. \tag{4}$$

Hence, we define the reactivity of phenotype k at age j to change in $\boldsymbol{\zeta}$ relative to $\mathbf{t}$ as

$$R_{\boldsymbol{\zeta}\mathbf{t}kj} = \frac{E_{\boldsymbol{\zeta}kj}}{E_{\mathbf{t}kj}}, \tag{5}$$

which gives the contribution of change in $\boldsymbol{\zeta}$ to the total elasticity of phenotype k at age j. In particular, $R_{\mathbf{y}\mathbf{t}kj}$ is the contribution of genotype change to the total elasticity, $R_{\mathbf{x}\mathbf{t}kj}$ is the contribution of phenotype change to the total elasticity, $R_{\bar{\mathbf{x}}\mathbf{t}kj}$ is the contribution of change in social partners' phenotype to the total elasticity, and $R_{\boldsymbol{\epsilon}\mathbf{t}kj}$ is the contribution of change in environmental or physiological variables (depending on what the variables in $\boldsymbol{\epsilon}$ measure) to the total elasticity. Reactivities add up to 1 ($R_{\mathbf{t}\mathbf{t}kj} = 1$).

Acknowledgements

MGF was partly funded by an European Research Council Consolidator Grant to A. Gardner (grant no. 771387). AGR is supported by a Royal Society-Leverhulme Trust Senior Research Fellowship (SRF\R1\241038).

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