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

Mauricio González-Forero*[1] and Aida Gómez-Robles†[2]

[1]Konrad Lorenz Institute for Evolution and Cognition Research, Klosterneuburg A-3400, Austria
[2]Department of Anthropology, University College London, 14 Taviton St, London, WC1H 0BW, UK

Abstract

The human brain is influenced by genes [1], environment [2], and developmental history [3]. Extensive efforts seek to identify the causes of observed variation in behaviour and disease [4–6]. Moreover, it is of primary interest to predict the brain's response to influences, both at present and over our evolutionary history [2;7–9]. Here we use a mathematical model that mechanistically recovers major aspects of human brain development and evolution [10–12] to predict the response of adult skill level to point influences earlier in development. We show that adult skill level is much more reactive to changes in developmental history than to genetic, environmental, or social changes over life during the recovered evolutionary trajectory from *H. habilis* to *H. sapiens*. This occurs despite our finding that adult skill level has evolved an increased reactivity to environmental and social change, and a decreased reactivity to genetic change, which is consistent with observation [13–15]. We further show that adverse genetic and environmental conditions that would developmentally yield low adult skill level can be more than compensated by time-specific changes in the environment or by slight changes in developmental history, yielding outstanding adult skill level. Our analyses identify developmental history as having a major ability to influence adult human skill level.

The adult human brain and its behavioural outputs are the product of a complex interplay of genetic, developmental, environmental, and social factors. While the brain expresses thousands of genes [1], multiple lines of evidence point to the existence of human specialisations for neuroplasticity [2;16]. Moreover, the brain is affected by developmental history as evidenced by studies of genetically identical individuals growing in the same environment who develop different behaviours, indicating that developmental noise may be an important source of behavioural variation [17;18]. Indeed, up to 50% of variation in human cognitive abilities has been estimated to stem from developmental noise [9]. The high level of plasticity of the human brain helps explain human learning abilities [19] and makes it possible to develop intervention strategies that can change behavioural and cognitive outputs over life [20]. Observed differences between chimpanzees and humans indicate that increased neuroplasticity evolved in the human lineage after the two lineages diverged, together with an overall increase in brain size and a general reorganization of the brain [2].

It is of primary importance to understand the causes of variation in human brain development and to predict the brain's response to changes in influencing factors [8]. The two goals of causal understanding and prediction are sometimes seen as trading-off [21], as this happens in predictive but causally opaque approaches based on regression and machine learning [22–24]. An additionally important aim is to predict the effects of interventions before data on their effects has become available [8]. Moreover, unlike brain size and reorganization, which can be inferred to a certain extent from endocranial size and shape, changes in neuroplasticity are extremely difficult to document in the fossil record. While differences and similarities in brain developmental trajectories have been described among modern humans and fossil hominins, including Neanderthals [25], *Homo erectus* [26], and even australopiths [27], quantifying the extent to which brain development is influenced by environmental factors in fossils remains out of reach. In addition, skill level is thought to be linked to brain size and organization, and it is usually inferred from the archaeological record, but the relationship between skill level and brain anatomy is not straightforward and they do not necessarily respond in the same way to modifications of underlying variables.

Here we formulate an approach for both causal understanding and prediction of human brain developmental trajectories. We use evolutionary developmental (evo-devo) dynamic mechanistic modelling to predict how human skill level responds to developmental interventions, understood broadly to include any endogenous or exogenous change over development, and how these responses 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 [10;28] (Fig. 1a; conceptually described in ref. [29]). 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 [10] and

*Mauricio.GonzalezForero@gmail.com
†a.gomez-robles@ucl.ac.uk

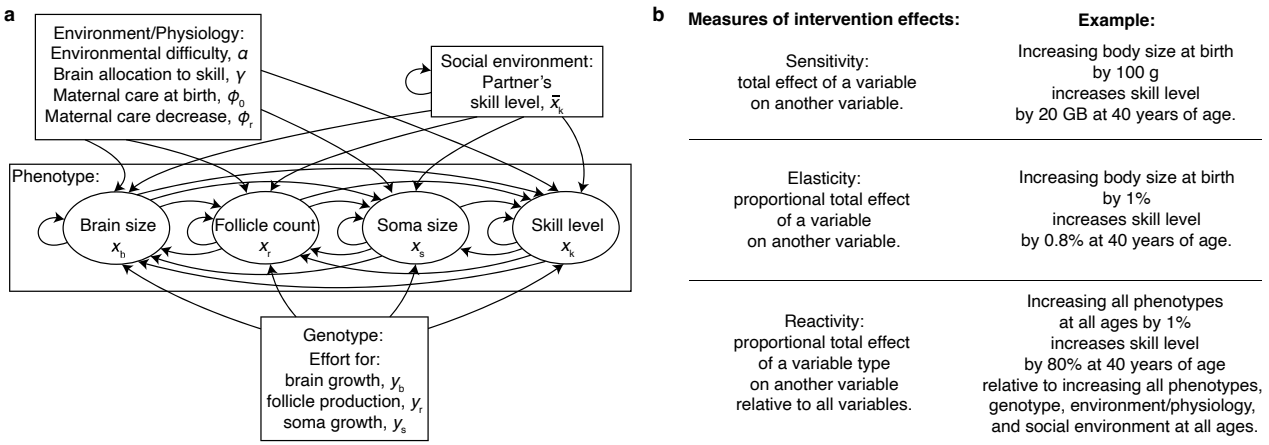

Figure 1: Causal diagram of the brain model and measures of the effects of intervention. **a,** Causal diagram of the model, where an arrow indicates a direct causal effect of a variable on another one or on itself at the next age. Variables are classified into genotypic, phenotypic, environmental/physiological, 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 are 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.

it is measured in information units[11]. It has been previously found that this model recovers major aspects of human development and evolution[10–12;29]. 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)[11;12]. Moreover, this model identifies a transition to seemingly cumulative culture in the Middle Pleistocene[11;29], which is consistent with the archaeological record[30–32]. We use this brain model to measure the effects on skill level of interventions performed earlier in life in the same individual. We quantify this effect through three different measures: sensitivity, or the total effect of a variable on another variable; elasticity, or the proportional total effect of a variable on another variable; and reactivity, or the proportional total effect of a variable on another variable relative to all variables affecting development (Fig. 1b).

Skill is little elastic to genetic change

We find that adult skill level is little elastic to change in genotypic traits. With *H. habilis* brain and body sizes, perturbing genotypic traits at any age typically has small effects on adult skill level (Fig. 3a; the Computer Code that generated all the figures is in ref.[33]), with sensitive periods to genetic change occurring early in development and coinciding with the onset of reproductive ability, or menarche defined as the developmental emergence of mature ovarian follicles (blue dots in Fig. 2c). With *H. sapiens* brain and body sizes, perturbing genotypic traits also has small effects on adult skill level, but perturbing genotypic traits around the age of menarche has more pronounced effects than at other ages (Fig. 3e). Menarche here occurs at around 12 years of age (red dots in Fig. 2c).

Skill is more elastic to developmental change

Adult skill level is more elastic to change in the developmentally earlier phenotypes, exceeding the elasticity to genetic change by two orders of magnitude with either *H. habilis* or *H. sapiens* brain and body sizes (Fig. 3b,f). This is unexpected as both genotypic and phenotypic change induce developmental feedback and both are perturbed at a single age only. Inspection of the analytical expressions of the sensitivity of a recurrence 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.[12]), 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.[12]). 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. habilis* brain and body sizes, adult skill level is most elastic to perturbation in skill level or brain size at earlier ages, with a window of elasticity to change in body size (Fig. 3b) confined to the short period of brain growth (Fig. 2b). With *H. sapiens* brain and body sizes, adult skill

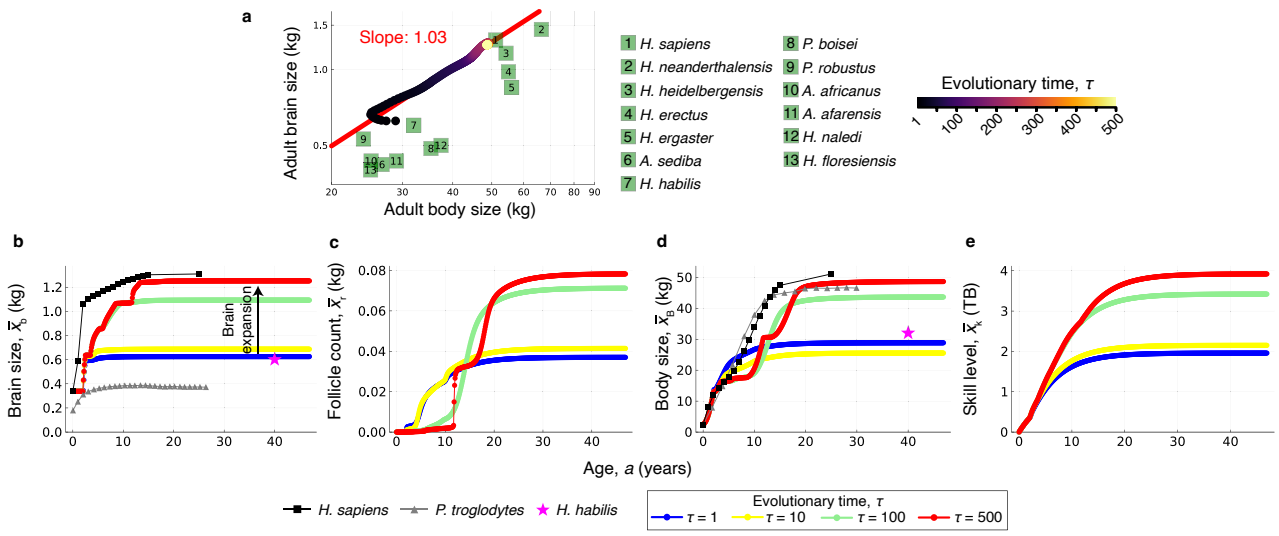

Figure 2: In-silico replica of the evolution and development of hominin brain size, body size, and skill level as obtained by the brain model. **a,** Green squares show the observed mean adult brain and body sizes in log-log scale for 13 hominin species (data for females when possible). Coloured dots 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), *P. troglodytes* females (gray triangles), and adult *H. habilis* of mixed sexes for brain size and of females for body size (pink stars). One evolutionary time step 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.[12], which lists references to the data sources.

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. 3f). 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.

Skill is intermediately elastic to environmental change

Altering the environment and physiology has moderate proportional effects on adult skill under both *H. habilis* and *H. sapiens* brain and body sizes. 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 dots in Fig. 3c). 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. 3g). Yet, increasing the environmental difficulty at other ages often has strong negative proportional effects on adult skill level (blue 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., increasing environmental difficulty often *decreases* skill level over development, whereas increasing environmental difficulty *increases* skill level over evolution; Figs. 6 and V of ref.[10]). This suggests that developmental drivers of cognition may not reliably indicate its evolutionary drivers[34].

Social interactions also have moderate developmental effects. 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. 3d). 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. 3h). 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. 3h).

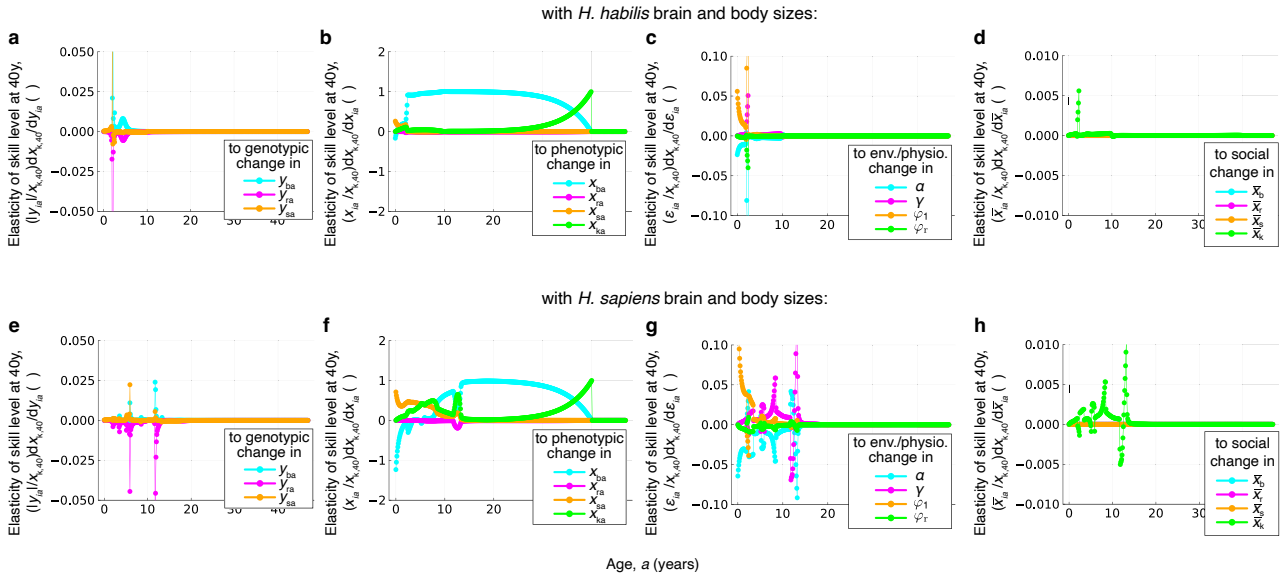

Figure 3: Recovered elasticity of skill level for the brain and body sizes of *H. habilis* and *H. sapiens*. Elasticity of adult skill level to (**a**,**e**) genotypic, (**b**,**f**) phenotypic, (**c**,**g**) environmental or physiological, and (**d**,**h**) social change: plots give the change in skill 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. habilis* (i.e., for blue dots in Fig. 2b-e). Panels **e-h** are for individuals with brain and body sizes of *H. sapiens* scale (i.e., for red 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.

Skill has been most reactive to developmental history throughout the evolution of the genus *Homo*

To quantify the extent to which a phenotype causally responds to genetic, developmental, or environmental changes experienced by the individual earlier in life, 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, similarly to heritability, but it is an individual-level quantity rather than a population-level one, it is causal rather than correlational, and it refers to how phenotypes would respond to potential interventions rather than to realised variation. The reactivities to the different variables add up to 1, such that each reactivity may be interpreted as the contribution of each variable type to the total elasticity of skill level at a given age.

With brain and body sizes of *H. habilis* scale, the reactivity of skill level to genotypic change is small, peaking at around 5 years of age (blue dots in Fig. 4a). As evolution proceeds and brain and body sizes reach *H. sapiens* scale, genotypic reactivity decreases further, 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, and this reactivity increases 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, but 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 level 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 still 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 (Methods). 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.

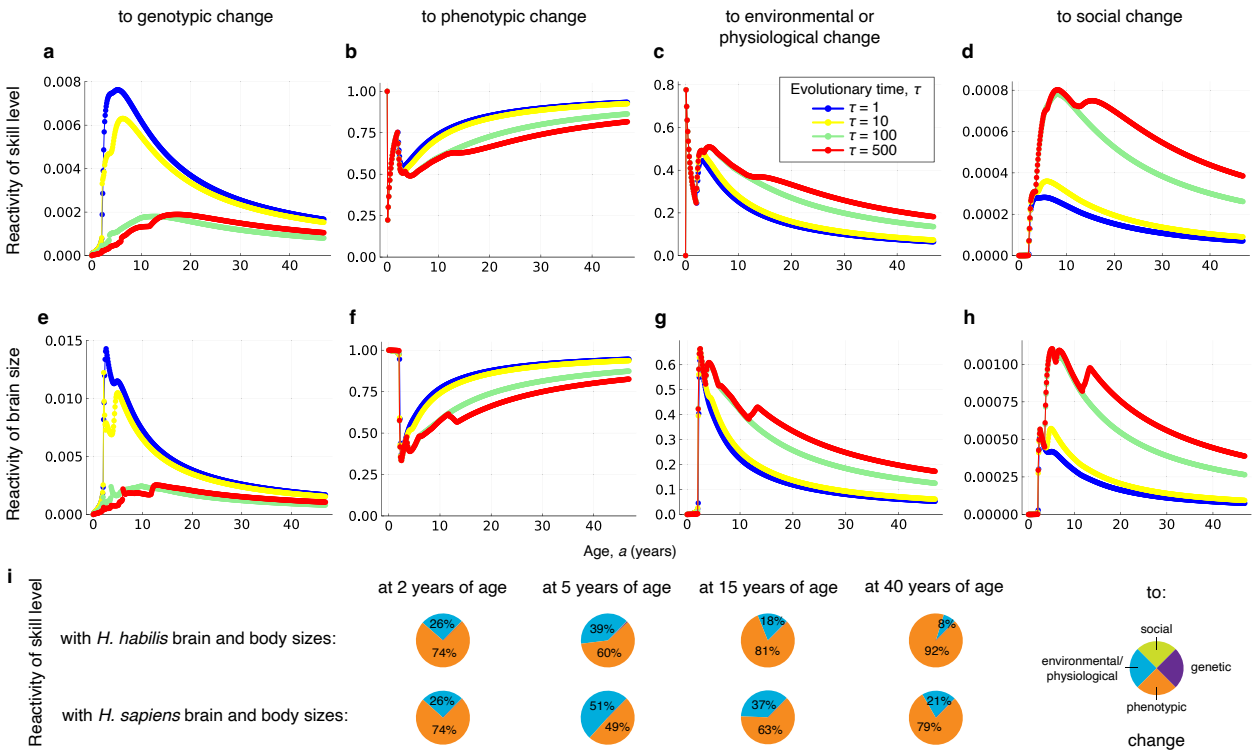

Figure 4: Developmental reactivity of skill level and brain size over the recovered hominin brain expansion. **a,** Reactivity of skill level to genotypic change; for instance, the red dot at the age $a = 10$ y gives how much the skill level of a 10 year old is affected by marginally increasing all genotypic 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.

Identifying interventions that compensate for adversity

The sensitivities described above identify interventions that can increase adult skill level. We use these sensitivities to implement simulated interventions to address the goal of compensating for adversity that leads to diminished adult skill level, where adversity refers to having either genes or an environment that are detrimental to adult skill level. For the average *H. sapiens* female in the brain model (red lines in Fig. 2b-e), introducing random variation in the genes and environmental difficulty results in a distribution of phenotypes over life (Fig. 5a-d). We use an amount of genotypic and environmental variation that yields a heritability of adult skill level approximately matching that observed for general cognitive ability in adults[35] (Fig. 5h), resulting in a predicted developmental trajectory of heritability that increases with age (Fig. 5h), consistent with observation[35]. The predicted heritabilities of adult brain and body sizes also fall within empirically estimated ranges (66-97% for total brain volume[36] and 47-90% for body mass index[37], although the heritability of body mass index appears to be larger in childhood than in adulthood[37]). The predicted increasing heritability of skill level with age contrasts with the decreasing reactivity of skill level to genetic change with age. This exemplifies that the two quantities measure different properties: heritability refers to realised variation, whereas reactivity refers to what would happen after an intervention.

Using sensitivities, we quantify the causal, mechanistic contribution of genes and environment to the simulated differences in adult skill level (by combining the logic of Caswell's life table response experiments[38]—p. 261 of ref.[39]—with formulas for the sensitivity of a recurrence[40]). To do this, we compare the adult skill level of the average individual among either the top or bottom 1000th quantiles in adult skill level with that of the average individual in the population. Under the genotypic and environmental variation used, genes contribute over twice as much as the environment to the developed differences in adult skill level among these individuals (i.e., genotypic variation causally contributes an increase of ≈ 0.2 TB to the adult skill level of the average top skill indi-

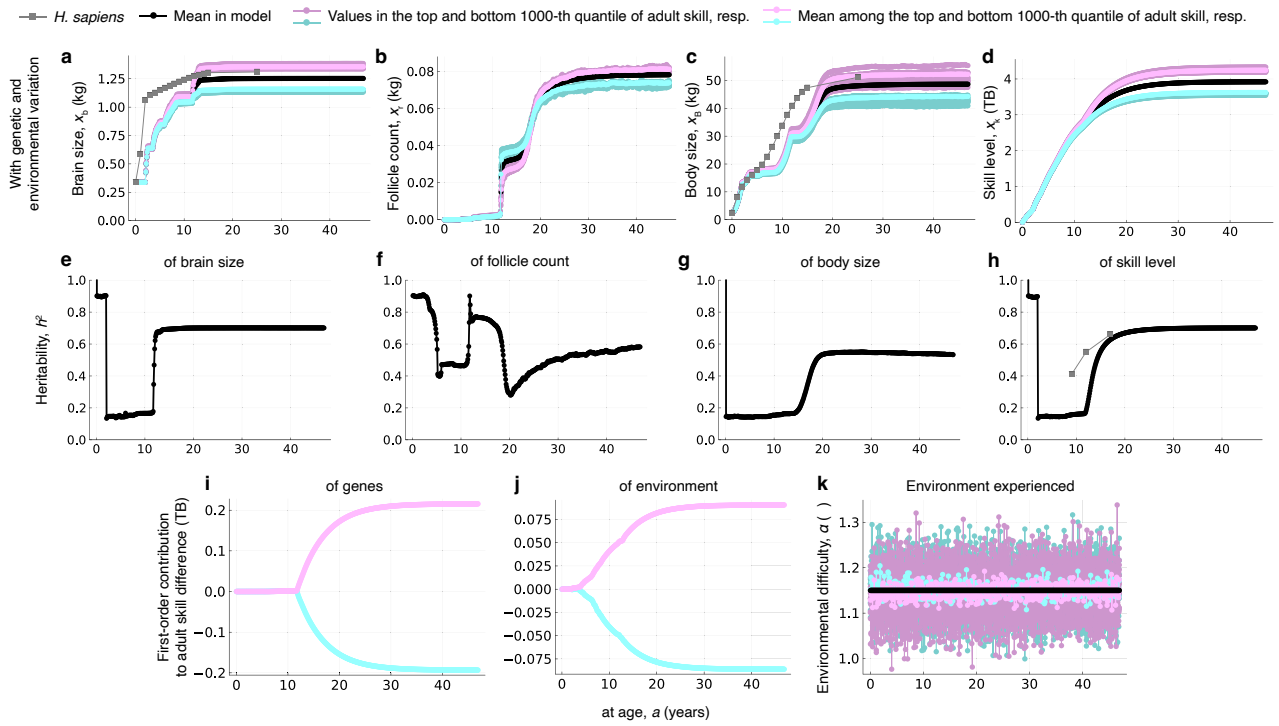

Figure 5: Simulating genetic and environmental adversity. We draw 10000 individuals experiencing normally distributed variation in their genotype (variance of $\sigma_g^2 = 0.1$) and environment (variance of $\sigma_e^2 = 0.002$) around the mean given by the *H. sapiens* scenario in Fig. 2, so they develop different phenotypes. **a-d,** Coloured lines show the phenotypes of individuals in the top (dark pink) and bottom (dark blue) 1000th quantile of adult skill level (with respective means in light pink and light blue). Bottom quantile individuals develop (**a**) smaller adult brain size, (**b**) higher fertility earlier and lower fertility later in life, (**c**) smaller adult body size, and (**d**) smaller adult skill level than the mean (black). **e-h,** Resulting heritabilities across the 10000 individuals; these heritabilities depend on σ_g^2 and σ_e^2 (Extended Data Fig. 1), whose values are chosen so that the resulting heritability for adult skill level (**h**, black) roughly matches the estimated value for general cognitive ability (**h**, gray); observed values taken from ref.[35]. First-order contribution of (**i**) genes and (**j**) environment to the differences in skill level between the average individuals among either the top (light pink) or bottom (light blue) 1000-th quantiles of adult skill level and average individuals, showing that genes contribute more to the simulated differences than the environment. **k,** Environment experienced.

vidual, and environmental variation causally contributes ≈ 0.09 TB; genes and environment contribute a decrease in adult skill level of similar magnitude for the average bottom skill individuals, shown in lines at 40 y in Fig. 5i). This further exemplifies that heritability, reactivity, and genetic contributions measure different properties: reactivity refers to what would happen after intervention, whereas both heritability and genetic contributions refer to realised variation; however, heritability refers to the fraction of phenotypic variation that is additive genetic, whereas genetic contributions refer to how much phenotypic differences are caused by differences in genes. The first-order contribution of genes to the differences in skill level in average individuals in the top and bottom quantiles during childhood is zero (Fig. 5i). During adolescence, this contribution becomes non-zero so that the genes of individuals at the top 1000th quantile in adult skill contribute positively to their skill, and negatively for those at the bottom 1000-th quantile. In contrast, the first-order contribution of the environment to the differences in skill level becomes non-zero earlier in childhood.

We now use the sensitivities to implement simulated interventions that compensate for adversity thereby alleviating the differences in skill. We implement simulated interventions in the environment in the direction of steepest increase in adult skill level with respect to the environment, which is given by the sensitivity of adult skill with respect to the environment. In particular, these interventions make the environment less difficult (i.e., requiring less of the individual's own skills to succeed at energy extraction) during childhood, but making it more difficult at the ages of sharpest brain growth and of menarche. As a result, individuals in the bottom 1000th quantile of adult skill level, which have genes that are detrimental to their skill, develop higher skill level than individuals in the top quantile (Fig. 6a-e).

Moreover, rather than intervening the environment, slightly intervening skill level earlier in life can produce an even larger compensation (Fig. 6f-j). This requires slighter interventions because of the higher elasticity of skill level to developmental history than to the environment. These interventions increase skill level during childhood, at the age of menarche, and as the assessed adult age approaches. In practice, these interventions may be implemented exogenously, for instance, via a supportive learning environment; however, the interventions might also

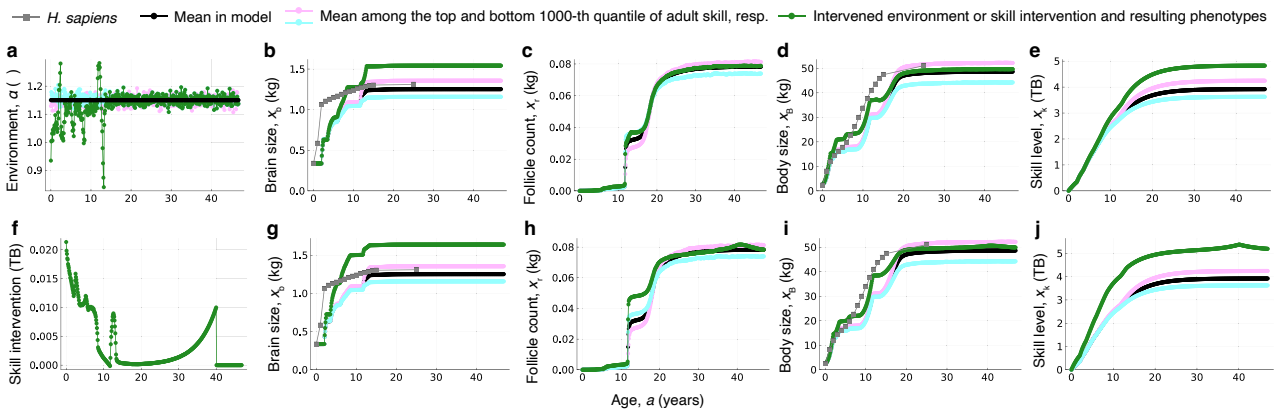

Figure 6: Predicting interventions that can more than compensate for adversity. **a,** Environment experienced by individuals on average: across the population (black), among the bottom (light blue) and top (light pink) 1000th quantile of adult skill level, and among the bottom 1000th quantile facing intervention (green). The environment is intervened in the direction of steepest increase of adult skill level (i.e., changing the environment by the amount given the sensitivity of adult skill level with respect to environmental difficulty). **b-e,** Corresponding phenotypes developed. Under the intervened environment, bottom quantile individuals (green) develop (**b**) larger adult brain size, (**c**) average fertility, (**d**) average adult body size, and (**e**) higher adult skill level than the individuals in the top quantile of adult skill level (pink). **f,** Rather than intervening the environment, here we intervene skill level, with the intervention shown in **f**. Skill level is intervened in the direction of steepest increase of adult skill level (i.e., changing skill level by 0.01 times the amount given by the sensitivity of adult skill level with respect to skill level at earlier ages). That is, a relatively slight intervention in skill level is implemented. **g-j,** Corresponding phenotypes developed. Despite the slight intervention in skill, the bottom quantile individuals (green), who have genes detrimental to adult skill, develop (**g**) larger adult brain size, (**h**) high early fertility, (**i**) slightly larger adult body size, and (**j**) substantially higher adult skill level than the individuals in the top quantile (pink).

happen endogenously, via developmental noise in brain connectivity.

Discussion

Our results indicate 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.

Our finding that the genotype has limited effects on adult skill level is surprising but consistent with recent findings of very small small genetic effects on complex phenotypes in humans, where confounders have been increasingly removed from classic estimates of larger effects[41;42], including the recent finding of a direct genetic effect heritability of 0.233 and a polygenic index with a coefficient of determination of 0.002 both for cognitive performance (Supplementary Tables 5 and 9 of ref.[43]). Our finding that developmental history has the strongest effects on adult skill level is agnostic regarding what factors can change developmental history. Those factors may be endogenous, such as developmental noise[3;17;18;9], or exogenous, such as targeted interventions[20]. The strong sensitivity to developmental history entails that intervening it can be beneficial but also detrimental. For instance, interventions in skill level in the same direction we used in Fig. 6f but of greater magnitude disrupt development. This highlights the need for predictive approaches to identify desired interventions.

Our results indicate that developmental history and environmental change have an opposite pattern in influencing skill level. During the first period of life (up to approximately 10 years of age), 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.

Our results suggest that the predominant role of developmental history in affecting skill level may be shared by many species within the genus *Homo*. 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*, which would roughly correspond with the *H. erectus* time period (at evolutionary time $\tau = 100$; Fig. 4).

We 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[2;44;13]. Later hominin species also show an increased window of opportunity for environmental and social factors to influence skill level in the

model. 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[45]. 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[46]. 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 finding is consistent with previous studies indicating late brain maturation in humans[47;48] associated with late acquisition of adult skill levels[49;50]. In other words, *H. sapiens* brains and skill levels can still be 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).

Overall, our results identify a predominant role of developmental history in shaping adult skill level in humans and identify opportunities for intervention with specific timing to mitigate and overcome adverse conditions that would otherwise yield detrimental developmental outcomes.

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Methods

Brain model overview. The brain model has the following structure (Fig. 1a; full details are in the 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., $\mathbf{x}_a = (x_{\mathrm{b}a}, x_{\mathrm{r}a}, x_{\mathrm{s}a}, x_{\mathrm{k}a})^{\intercal}$), 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 was derived from energy conservation and phenomenological considerations of how skill level translates into energy extraction[10;11]. 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 $\mathbf{y}$ and $\boldsymbol{\epsilon}$).

Sensitivity. 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, is correlational and depends on population-level phenomena such as genetic, phenotypic, and environmental variation.

Sensitivity analyses of a model[51] can be used to estimate causal effects. Such causal effects can be computed with derivatives, provided that such derivatives are causal[52]. 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 $x \not\to y$, then causal derivatives of x with respect to y must be zero even if f is invertible[53]. Recently derived formulas for the sensitivity of a recurrence[40] 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 perturbation onwards, specifically, by applying a perturbation at such initial time and following the effects of the perturbation from that time[40]. In particular, this approach implies the causal arrow of time, such that perturbations can only causally affect the future, but not the past.

For the brain 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 devel-

opmental noise, although our approach does not need to specify how those changes occur (p. 6 of ref.[51]).

To compute these sensitivities, we use general formulas for the sensitivity of a recurrence derived previously[40]. To do that, we first build the matrix of direct effects of the phenotype on the phenotype at age a (all derivatives are evaluated at $\mathbf{y} = \bar{\mathbf{y}}$)

$$\frac{\partial \mathbf{x}_{a+1}^{\mathsf{T}}}{\partial \mathbf{x}_a} = \begin{pmatrix} \frac{\partial x_{\mathrm{b},a+1}}{\partial x_{\mathrm{b}a}} & \frac{\partial x_{\mathrm{r},a+1}}{\partial x_{\mathrm{b}a}} & \frac{\partial x_{\mathrm{s},a+1}}{\partial x_{\mathrm{b}a}} & \frac{\partial x_{\mathrm{k},a+1}}{\partial x_{\mathrm{b}a}} \\ \frac{\partial x_{\mathrm{b},a+1}}{\partial x_{\mathrm{r}a}} & \frac{\partial x_{\mathrm{r},a+1}}{\partial x_{\mathrm{r}a}} & \frac{\partial x_{\mathrm{s},a+1}}{\partial x_{\mathrm{r}a}} & \frac{\partial x_{\mathrm{k},a+1}}{\partial x_{\mathrm{r}a}} \\ \frac{\partial x_{\mathrm{b},a+1}}{\partial x_{\mathrm{s}a}} & \frac{\partial x_{\mathrm{r},a+1}}{\partial x_{\mathrm{s}a}} & \frac{\partial x_{\mathrm{s},a+1}}{\partial x_{\mathrm{s}a}} & \frac{\partial x_{\mathrm{k},a+1}}{\partial x_{\mathrm{s}a}} \\ \frac{\partial x_{\mathrm{b},a+1}}{\partial x_{\mathrm{k}a}} & \frac{\partial x_{\mathrm{r},a+1}}{\partial x_{\mathrm{k}a}} & \frac{\partial x_{\mathrm{s},a+1}}{\partial x_{\mathrm{k}a}} & \frac{\partial x_{\mathrm{k},a+1}}{\partial x_{\mathrm{k}a}} \end{pmatrix}, \quad (2)$$

which is obtained by differentiating Eq. (1). We then build the matrix of direct effects of the phenotype on the phenotype at all ages

$$\frac{\partial \mathbf{x}^{\mathsf{T}}}{\partial \mathbf{x}} = \begin{pmatrix} \mathbf{I} & \frac{\partial \mathbf{x}_2^{\mathsf{T}}}{\partial \mathbf{x}_1} & \cdots & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{I} & \cdots & \mathbf{0} & \mathbf{0} \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ \mathbf{0} & \mathbf{0} & \cdots & \mathbf{I} & \frac{\partial \mathbf{x}_{N_\mathrm{a}}^{\mathsf{T}}}{\partial \mathbf{x}_{N_\mathrm{a}-1}} \\ \mathbf{0} & \mathbf{0} & \cdots & \mathbf{0} & \mathbf{I} \end{pmatrix}. \quad (3)$$

The sensitivity of the phenotype to phenotypic change is then given by the developmental feedback matrix

$$\frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\mathbf{x}} = \left(2\mathbf{I} - \frac{\partial \mathbf{x}^{\mathsf{T}}}{\partial \mathbf{x}}\right)^{-1}. \quad (4)$$

Partitioning this matrix into blocks of size 4×4, the aj-th block entry gives the total effects of the phenotype at age a on the phenotype at age j (that is, $\mathrm{d}\mathbf{x}_j^{\mathsf{T}}/\mathrm{d}\mathbf{x}_a$, whose ik-th entry gives the total effect of the i-th phenotype at age a on the k-th phenotype at age j, $\mathrm{d}x_{kj}/\mathrm{d}x_{ia}$).

Proceeding similarly to compute the matrix of direct effects of the genotype on the phenotype ($\partial \mathbf{x}^{\mathsf{T}}/\partial \mathbf{y}$), the sensitivity of the phenotype to genotypic change is in turn given by

$$\frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\mathbf{y}} = \frac{\partial \mathbf{x}^{\mathsf{T}}}{\partial \mathbf{y}} \frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\mathbf{x}} \quad (5)$$

Partitioning this matrix into blocks of size 3×4, the aj-th block entry gives the total effects of the genotype at age a on the phenotype at age j (that is, $\mathrm{d}\mathbf{x}_j^{\mathsf{T}}/\mathrm{d}\mathbf{y}_a$, whose ik-th entry gives the total effect of the i-th genotypic trait at age a on the k-th phenotype at age j, $\mathrm{d}x_{kj}/\mathrm{d}y_{ia}$).

Also proceeding similarly to compute the matrix of direct effects of the social partners' phenotype on the focal individual's phenotype ($\partial \mathbf{x}^{\mathsf{T}}/\partial \bar{\mathbf{x}}$), the sensitivity of the phenotype to genotypic change is in turn given by

$$\frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\bar{\mathbf{x}}} = \frac{\partial \mathbf{x}^{\mathsf{T}}}{\partial \bar{\mathbf{x}}} \frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\mathbf{x}} \quad (6)$$

Partitioning this matrix into blocks of size 4×4, the aj-th block entry gives the total effects of the social partners' phenotype at age a on the focal individual's phenotype at age j (that is, $\mathrm{d}\mathbf{x}_j^{\mathsf{T}}/\mathrm{d}\bar{\mathbf{x}}_a$, whose ik-th entry gives the total effect of the i-th phenotype of the social partners at age a on the k-th phenotype at age j, $\mathrm{d}x_{kj}/\mathrm{d}\bar{x}_{ia}$).

Finally, proceeding similarly to compute the matrix of direct effects of the parameters on the phenotype ($\partial \mathbf{x}^{\mathsf{T}}/\partial \boldsymbol{\epsilon}$), the sensitivity of the phenotype to environmental or physiological change is in turn given by

$$\frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\boldsymbol{\epsilon}} = \frac{\partial \mathbf{x}^{\mathsf{T}}}{\partial \boldsymbol{\epsilon}} \frac{\mathrm{d}\mathbf{x}^{\mathsf{T}}}{\mathrm{d}\mathbf{x}} \quad (7)$$

Partitioning this matrix into blocks of size 19×4, the aj-th block entry gives the total effects of the parameters at age a on the phenotype at age j (that is, $\mathrm{d}\mathbf{x}_j^{\mathsf{T}}/\mathrm{d}\boldsymbol{\epsilon}_a$, whose ik-th entry gives the total effect of the i-th parameter at age a on the k-th phenotype at age j, $\mathrm{d}x_{kj}/\mathrm{d}\epsilon_{ia}$).

These formulas automatically 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[54;40] $(2\mathbf{I} - \partial \mathbf{x}^{\mathsf{T}}/\partial \mathbf{x})^{-1}$ 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).

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.

Elasticity. 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[51], which is the proportional sensitivity, so is dimensionless and is defined only for positive variables. 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. 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., $(|y_{\mathrm{b}1}|/x_{\mathrm{k}40})\mathrm{d}x_{\mathrm{k}40}/\mathrm{d}y_{\mathrm{b}1}$). Therefore, we compute the elasticity of phenotype k at age j to change in $\zeta \in \mathbb{R}$ as

$$E_{\zeta kj} = \frac{1}{x_{kj}} \frac{\mathrm{d}x_{kj}}{\mathrm{d}\zeta} |\zeta|. \quad (8)$$

When ζ is positive, $E_{\zeta kj}$ is the standard elasticity of phenotype k at age j to change in ζ.

Reactivity. We define reactivities as follows. We extend the notion of elasticity 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

$$E_{\boldsymbol{\zeta}kj} = \frac{1}{x_{kj}} \frac{\mathrm{d}x_{kj}}{\mathrm{d}\boldsymbol{\zeta}^{\intercal}} |\boldsymbol{\zeta}| = \sum_i \frac{1}{x_{kj}} \frac{\mathrm{d}x_{kj}}{\mathrm{d}\zeta_i} |\zeta_i|, \tag{9}$$

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[40], 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

$$E_{\mathbf{t}kj} = E_{\mathbf{x}kj} + E_{\mathbf{y}kj} + E_{\boldsymbol{\epsilon}kj} + E_{\bar{\mathbf{x}}kj} + E_{\bar{\mathbf{y}}kj}. \tag{10}$$

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

$$R_{\boldsymbol{\zeta}\mathbf{t}kj} = \frac{E_{\boldsymbol{\zeta}kj}}{E_{\mathbf{t}kj}}, \tag{11}$$

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 ($R_{\mathbf{t}\mathbf{t}kj} = 1$).

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. Reactivity to genetic change is not "the genetic component" of a trait, similarly to heritability which is not the genetic component of a trait although this is a common misinterpretation.

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[10;12], which is consistent with brain size and skill level being positively correlated in primates[55;56], together with the consistent yet modest positive association between brain size and cognitive ability in humans[57;58]. This proportionality in the brain model predicts that additional variation in the observed relationship between brain size and cognitive ability could be due to variation in the parameters involved in the proportionality (i.e., the metabolic cost of brain tissue and of memory, as well as the fraction of brain metabolic rate allocated to the skill considered).

Heritability. We compute heritability using its standard definition as follows. We introduce genotypic variation by letting genotypic traits be given by $y_{ij}^{(r)} = \bar{y}_{ij} + \delta_{yij}^{(r)}$, where $\bar{y}_{ij}$ are the genotypic traits obtained by the brain model under the *H. sapiens* scenario and $\delta_{yij}^{(r)}$ is a value drawn from the normal distribution with mean zero and variance σ_{g}^2. We introduce environmental variation by letting the environmental difficulty faced at age j be given by $\alpha_j^{(r)} = \bar{\alpha} + \delta_{\alpha j}^{(r)}$, where $\bar{\alpha} = \alpha$ is the environmental difficulty used by the brain model under the *H. sapiens* scenario and $\delta_{\alpha j}^{(r)}$ is a value drawn from the normal distribution with mean zero and variance σ_{e}^2. Using the vector notation defined above, we then obtain a vector $\mathbf{y}^{(r)}$ of genotypic traits and a vector of environmental difficulty $\boldsymbol{\alpha}^{(r)}$ across life for replica r. We then compute the developed phenotypes $\mathbf{x}^{(r)}$ by solving the developmental dynamics in Eq. (1) evaluated at the genotypic traits $\mathbf{y}^{(r)}$ and environment $\boldsymbol{\alpha}^{(r)}$.

We form the matrix of predictor variables $\mathbf{X}$, whose first column contains ones and the r-th row in the remainder columns is the vector of genotypic differences $\mathrm{vec}(\boldsymbol{\delta}^{(r)})$ (the first column of ones is to estimate the intercepts, which yields a higher coefficient of determination). We also form the vector of the response variable $\mathbf{Y}_{ij}$, whose r-th entry is $x_{ij}^{(r)}$ for a given phenotype i at a given age j. Then, we consider the linear regression of phenotype on genotype

$$\mathbf{Y}_{ij} = \mathbf{X}\boldsymbol{\beta}_{ij} + \text{residual}, \tag{12}$$

where the k-th entry of the vector $\boldsymbol{\beta}_{ij}$ is the regression coefficient corresponding to the k-th entry of $\mathrm{vec}(\boldsymbol{\delta}^{(r)})$. The least-squares estimate of these coefficients is given by

$$\hat{\boldsymbol{\beta}}_{ij} = (\mathbf{X}^{\intercal}\mathbf{X})^{-1}(\mathbf{X}^{\intercal}\mathbf{Y}_{ij}). \tag{13}$$

The replicas of the breeding values of phenotype i at age j are listed by the vector $\mathbf{X}\hat{\boldsymbol{\beta}}_{ij}$. Denoting by $\mathrm{var}[\mathbf{Y}_{ij}]$ the variance among the entries of $\mathbf{Y}_{ij}$, the heritability of the phenotype i at age j is thus given by

$$h_{ij}^2 = \frac{\mathrm{var}[\mathbf{X}\hat{\boldsymbol{\beta}}_{ij}]}{\mathrm{var}[\mathbf{Y}_{ij}]}. \tag{14}$$

First-order contributions to phenotypic differences. We quantify the contributions of genes and environment to the differences in skill level among bottom and top quantile individuals in adult skill level as follows. Our approach combines the sensitivities we computed above (Eqs. 5 and 7), which use formulas for the sensitivity of a recurrence[40], with the logic of Caswell's life table response experiment method[38] (and p. 261 of ref.[39]).

Let $x_{ia}^{(r)}$ be the value of the i-th phenotype at age a for an individual that developed under conditions labelled r, where the individual has genotype $\mathbf{y}^{(r)}$ and experiences an environment $\boldsymbol{\alpha}^{(r)}$ over life. The quantity $x_{ia}^{(r)} - \bar{x}_{ia}$ gives the differences in the i-th phenotype at age a when the individual develops under conditions r relative to an average (resident) individual, who has genotype $\bar{\mathbf{y}}$ and experiences an environment $\bar{\boldsymbol{\alpha}} = \boldsymbol{\alpha}$ over life. From Taylor's

theorem, to first order of approximation, we have

$$x_{ia}^{(r)} \approx \bar{x}_{ia} + \left.\frac{\mathrm{d}x_{ia}}{\mathrm{d}\mathbf{y}^{\top}}\right|_{\mathbf{m}=\bar{\mathbf{m}}} (\mathbf{y}^{(r)} - \bar{\mathbf{y}}) + \left.\frac{\mathrm{d}x_{ia}}{\mathrm{d}\boldsymbol{\alpha}^{\top}}\right|_{\mathbf{m}=\bar{\mathbf{m}}} (\boldsymbol{\alpha}^{(r)} - \boldsymbol{\alpha}), \tag{15}$$

where $\mathbf{m} = (\mathbf{y}; \boldsymbol{\alpha})$ and $\bar{\mathbf{m}} = (\bar{\mathbf{y}}; \bar{\boldsymbol{\alpha}})$. We then have that

$$\left.\frac{\mathrm{d}x_{ia}}{\mathrm{d}\mathbf{y}^{\top}}\right|_{\mathbf{m}=\bar{\mathbf{m}}} (\mathbf{y}^{(r)} - \bar{\mathbf{y}}) \tag{16}$$

is the first-order genetic contribution to the phenotypic difference $x_{ia}^{(r)} - \bar{x}_{ia}$ and

$$\left.\frac{\mathrm{d}x_{ia}}{\mathrm{d}\boldsymbol{\alpha}^{\top}}\right|_{\mathbf{m}=\bar{\mathbf{m}}} (\boldsymbol{\alpha}^{(r)} - \boldsymbol{\alpha}) \tag{17}$$

is the first-order environmental contribution to the phenotypic difference $x_{ia}^{(r)} - \bar{x}_{ia}$.

In Fig. 5i,j, we plot these first-order genetic and environmental contributions to the differences in skill level $x_{\mathrm{k}a}^{(r)} - \bar{x}_{\mathrm{k}a}$, where the genotype $y_{ij}^{(r)}$ and environment $\alpha_j^{(r)}$ for a given age j are the mean among the values for individuals in the top or bottom 1000-th quantile of adult skill level. With these definitions, heritabilities, contributions to phenotypic differences, and the regression effects ($\hat{\boldsymbol{\beta}}$) depend on the population sample (the former two via $\mathbf{X}$ and $\mathbf{Y}_{ij}$, the latter via $\mathbf{y}^{(r)}$ and $\boldsymbol{\alpha}^{(r)}$) but the mechanistic effects (e.g., $\mathrm{d}x_{ia}/\mathrm{d}\mathbf{y}|_{\mathbf{m}=\bar{\mathbf{m}}}$) do not so in that sense they are individual-level properties. Mechanistic heritability may be defined with the mechanistic breeding value (i.e., genetic contribution (16) plus mean value $\bar{x}_{ia}$), but mechanistic heritability can be greater than one because mechanistic breeding values may be correlated with the mechanistic residual, whereas breeding values are not correlated with the residual because of least squares[40;59].

Data availability. No data were collected in this study. All data used were previously published in references provided in the main text or Methods.

Code availability. All codes have been deposited at https://doi.org/10.5281/zenodo.15038874 (ref.[33]).

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Author contributions

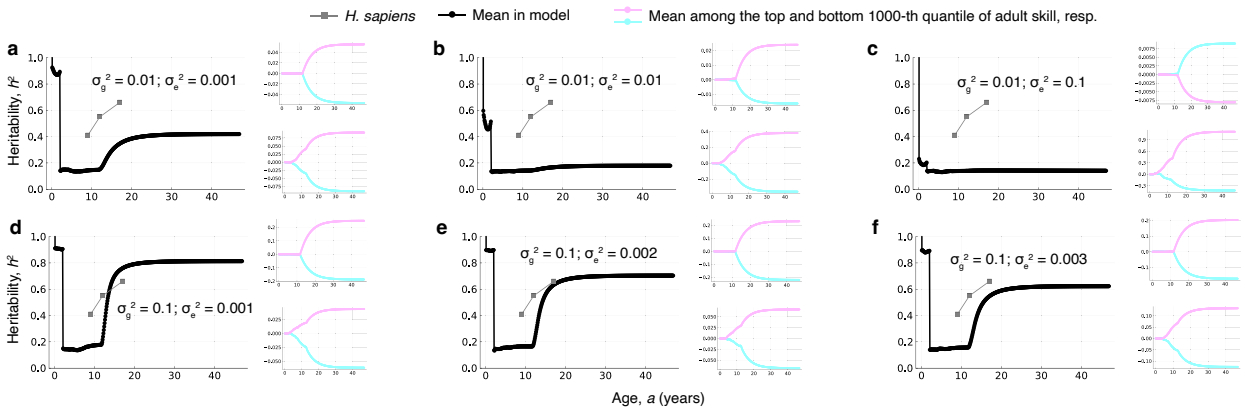
MGF conceived the work and did the mathematical and computational analyses. MGF and AGR wrote the paper.

Competing interests

The authors declare no competing interests.

Materials & Correspondence.

Correspondence and requests for materials should be addressed to Mauricio González-Forero and Aida Gómez-Robles.



Extended Data Figure 1: Predicted heritability of skill level and first-order contributions to adult skill differences under various values of genotypic and environmental variation. Each panel uses the indicated parameter values, where the large plot is that of heritability, the small top plot is that of the first-order contribution of genes, and the small bottom plot is that of the first-order contribution of the environment. Panel **e** corresponds to Fig. 5h,i,j, with slight differences due to random variation. In contrast to heritability, the sensitivities, elasticities, and reactivities in Figs. 3 and 4 do not depend on genotypic and environmental variation σ_{g}^2 and σ_{e}^2 as they are individual-level properties rather than population-level ones.