Human skill level is more reactive to developmental history than to genes or environment

Mauricio González-Forero[1,2*] and Aida Gómez-Robles[3†]

[1]School of Biology, University of St Andrews, Dyers Brae, St Andrews, KY16 9TH, Fife, UK.

[2]Present address: Konrad Lorenz Institute for Evolution and Cognition Research, Klosterneuburg A-3400, Austria.

[3]Department of Anthropology, University College London, 14 Taviton St, London, WC1H 0BW, UK.

[*]Corresponding author. Email: Mauricio.Gonzalez-Forero@kli.ac.at

[†]Corresponding author. Email: a.gomez-robles@ucl.ac.uk.

Human behaviour is influenced by genes, environment, and developmental history, but causally disentangling these influences remains challenging. Here we quantify the causal response of adult skill level to point influences earlier in development in a mechanistic *in-silico* replica of major aspects of human development and evolution. We find that adult skill level is more reactive to changes in developmental history than to genetic, environmental, or social changes in the genus *Homo*. We also find that adverse genetic and environmental conditions that would developmentally yield low adult skill level in humans can be compensated by time-specific environmental changes or by slight changes in developmental history, potentially yielding outstanding adult skill level. Our analyses identify a major capability of developmental history to influence adult human skill level.

The adult human brain is the product of a complex interplay of genetic, developmental, environmental, and social factors. While the brain expresses thousands of genes (*1*) and extensive research efforts seek to identify genes contributing to its behavioural outputs (*2–4*), multiple lines

of evidence point to the existence of human specialisations for neuroplasticity (*5, 6*). Moreover, the brain is affected by developmental history as evidenced by studies of essentially genetically identical individuals growing in the same environment who develop different behaviours, indicating that developmental noise may be an important source of behavioural variation (*7–9*). Indeed, up to 50% of variation in human cognitive abilities has been estimated to stem from developmental noise (*10*). The high level of plasticity of the human brain helps explain human learning abilities (*11*) and makes it possible to develop intervention strategies that can change behavioural and cognitive outputs over life (*12*). 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 (*5*).

However, disentangling the causes of variation in human brain development remains challenging. Analyses are commonly correlational due to the typically observational nature of human data, with persistent challenges to identify causality (*4, 7, 13–18*). Moreover, unlike brain size and reorganization, which can be inferred to some extent from endocranial size and shape (*19–21*), quantifying the influence on brain development of environmental factors in fossils remains out of reach.

These challenges can be addressed by quantifying causal influences in *in-silico* replicas of development. This is made possible by a recent data-integrated evolutionary developmental (evo-devo) dynamic mathematical model, hereafter referred to as the brain model, that mechanistically describes the developmental dynamics of human skill level and tissue sizes from energy conservation considering social interactions and environmental influences (*22, 23*) (Fig. 1A; conceptually described in (*24*)). In this model, skill level is defined as the capacity of an individual to overcome ecological or social challenges to extract energy from their environment (*22*) and it is measured in information units (*25*). It has been previously found that this model recovers major aspects of human development and evolution (*22, 24–26*). Specifically, it recovers the expansion of adult brain and body sizes from *Homo habilis* to *H. sapiens* scale, and the evolution of childhood, adolescence, and adulthood periods with timing matching that observed in *H. sapiens* (Fig. S1) (*25, 26*). Moreover, this model identifies a transition to seemingly cumulative culture in the Middle Pleistocene (*24, 25*), which is consistent with the archaeological record (*27–29*).

Here we use this brain model to mechanistically quantify, predict and understand the responses

of an individual's skill level to changes experienced earlier in life, and how these responses have evolved from *H. habilis* to *H. sapiens*. We quantify these effects through four 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; reactivity, or the proportional total effect of a variable on another variable relative to all variables affecting development; and first-order contributions, which quantify the mechanistic (not correlational) genetic and environmental contributions to differences in phenotypes (Fig. 1B). This reveals relatively small genetic and large developmental effects on adult skill level, such that slightly intervening developmental history can compensate for genetic and environmental adversity.

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. 2A; the computer code that generated all the figures is in (*30*)), 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 (Fig. S1C, blue). 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 (approximately 12 yr; Fig. S1C, red) has more pronounced effects than at other ages (Fig. 2E).

Skill is more elastic to developmental change

Adult skill level is more elastic to change in 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. 2B,F). This is unexpected as both genotypic and phenotypic change induce developmental feedback, and both are implemented at a single age only. The analytical expressions of the sensitivity of a recurrence indicate that the sensitivities to genotypic change are relatively small because the direct effects of phenotypes on phenotypes at the next age are mostly positive (section S3.2.1 of (*26*)), whereas the direct effects of genotype on phenotypes at the next age

are often negative due to trade-offs in energy allocation to growth of the various tissues (section S3.2.2 of (*26*)). 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. 2B) confined to the short period of brain growth (Fig. S1B). With *H. sapiens* brain and body sizes, adult skill level is most elastic to perturbation in: body size in infancy and early childhood, skill level in mid-childhood, and brain size during most of late childhood and adulthood (Fig. 2F). 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 (approximately 12-19 yrs; Fig. S1C,D). This inelasticity of adult skill to adolescent perturbation arises seemingly because, by adolescence, brain growth has ceased in the model, as perturbations occurring after brain growth arrest have vanishing effects on adult skill level except if they are on brain or skill.

Skill is intermediately elastic to environmental change

Altering the environment and physiology has moderate proportional effects on adult skill. With *H. habilis* brain and body sizes, increasing the environmental difficulty (i.e., individuals need higher skill level to extract energy) has mostly negative proportional effects on adult skill, with a marginally positive proportional effect of environmental difficulty at menarche (Fig. 2C, blue). Instead, with *H. sapiens* brain and body sizes, interventions making the environment more difficult during the year after menarche but less difficult in the subsequent year enhance adult skill level (Fig. 2G, blue). The decisiveness of skills at energy extraction has largely opposite proportional effects to those of environmental difficulty (Fig. 2C,G, pink). Either increasing maternal care or decreasing the rate of maternal care decrease early in childhood has positive effects on adult skill level (Fig. 2C,G, orange and green). Importantly, these developmental effects 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 (*22*)). This indicates that developmental drivers of cognition are not reliable indicators of its evolutionary drivers, contrasting previous expectations (*31*).

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 menarche (Fig. 2D). With *H. sapiens* brain and body sizes, increasing the skill level of social partners with whom the individual interacts at menarche has marked negative proportional effects on adult skill level (Fig. 2H, green). Instead, increasing the skill level of social partners with whom the individual interacts at other ages typically has positive proportional effects on adult skill level (Fig. 2H, green).

Evolution of developmental reactivity from *Homo habilis* to *H. sapiens*

To summarise the relative response to changes of a given type earlier in life, we construct an index using developmental sensitivities. We term this index "reactivity", which measures a phenotype's elasticity to change in all variables of a given type relative to the phenotype's total elasticity when changing all variables during development. Thus, reactivity ranges from 0 to 1, as heritability, but it is an individual-level rather than a population-level quantity, is causal rather than correlational, and measures how phenotypes would respond to potential interventions rather than 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 (but such contributions are not independent (*32*), p. 229).

The genotypic reactivity of skill level is small and has decreased over evolution. With brain and body sizes of *H. habilis* scale, the small reactivity of skill level to genotypic change peaks at around 5 yr (Fig. 3A, blue). Over evolution to *H. sapiens* brain and body sizes, genotypic reactivity decreases further, with a peak at around 18 yr (Fig. 3A, red).

The reactivity of skill level to developmental history is much higher despite having decreased over evolution. With *H. habilis* brain and body sizes, skill level throughout life reacts strongly to phenotypic change, and this reactivity increases with age (Fig. 3B, blue). Over evolution to

H. sapiens brain and body sizes, the reactivity of skill level to developmental history decreases but remains substantial still increasing with age (Fig. 3B, red).

The reactivity of skill level to environmental or physiological change is moderate and has increased over evolution. With *H. habilis* brain and body sizes, skill level reacts less strongly to environmental or physiological change than to developmental history particularly after childhood, and this reactivity decreases with age (Fig. 3C, blue). Over the evolution to *H. sapiens* brain and body sizes, the reactivity of skill level to environmental or physiological change increases, but still decreases with age (Fig. 3C, red).

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 yr (Fig. 3D, blue). Over evolution to *H. sapiens*, the social reactivity of skill level more than doubles, with a developmental peak at around 9 yr (Fig. 3D, red). 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 (Fig. 3A-D, red). Such small reactivity to social change may result partly because we do not consider the developmental feedback in social partners nor do we model social learning explicitly (Materials and Methods).

Some of these reactivities are summarised in Fig. 3I. The sensitivities and elasticities underlying the reactivities presented are shown in Figs. S2-S17. The reactivities of the mature ovarian follicle count and somatic tissue size over the recovered human brain expansion are shown in Fig. S18.

Identifying interventions that compensate for adversity

We now introduce genetic and environmental variation around the *H. sapiens* means, which leads some individuals to develop low or high adult skill level (Fig. 4A-D), yielding a predicted skill heritability that increases with age (Fig. 4H), consistent with observation (*33*). 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 intervention.

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 (*34*)—p. 261 of (*32*)—with formulas for the sensitivity of a recurrence (*35*)). Under the genotypic and environmental variation used, genes causally contribute over twice as much as the environment to the developed differences in adult skill level between average top- and bottom-skilled individuals (i.e., the genes of the average top-skilled individual increase their adult skill level by ≈ 0.2 TB, whereas the environment increases it by ≈ 0.09 TB; Fig. 4I,J, pink at 40 yr—the genes and environment of the average bottom-skilled individual decrease their adult skill level by similar magnitudes; Fig. 4I,J, blue). 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 between average top- and bottom-skilled individuals during childhood is nearly zero as skill differences only become appreciable after menarche (Fig. 4I). Instead, the first-order contribution of the environment to the differences in skill level becomes non-zero earlier in childhood.

Next, we identify interventions that compensate for the simulated adversity. We implement environmental interventions in the direction of steepest increase in adult skill level, given by the sensitivity of adult skill with respect to environment. These interventions make the environment less difficult during childhood, but more difficult at the ages of sharpest brain growth and of menarche. Consequently, bottom-skilled individuals, who have genes that are detrimental to their skill, develop higher skill level than otherwise top-skilled individuals (Fig. 5A-E).

Now, rather than intervening the environment, slightly intervening skill level earlier in life produces larger compensation (Fig. 5F-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 occur exogenously, for instance, via a supportive learning environment, or 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 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 how they all interact during development) have had on the individual until a given age.

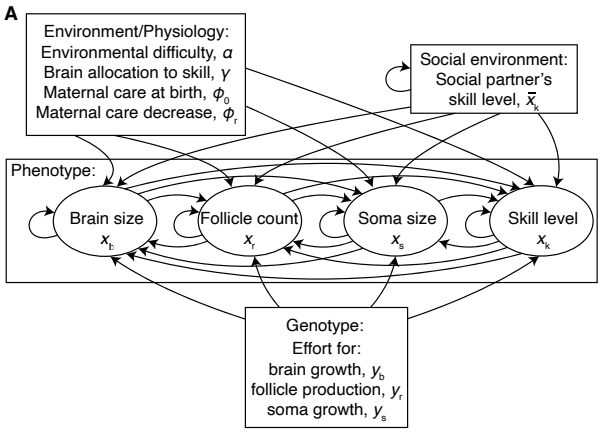
Our finding that the genotype has limited effects on adult skill level is surprising but consistent with recent findings of very small genetic effects on complex phenotypes in humans, where confounders have been increasingly removed from classic estimates of larger effects (*36,37*). Specifically, recent estimates for cognitive performance find a direct genetic effect heritability of 0.233 and a polygenic index with a coefficient of determination of 0.002 (Supplementary Tables 5 and 9 of (*38*)). Our finding that developmental history has the strongest effects on adult skill level is agnostic to what factors change developmental history. Those factors may be endogenous, such as developmental noise (*7–10*), or exogenous, such as targeted interventions (*12*). The strong sensitivity to developmental history entails that intervening it can be beneficial if done sufficiently slightly but detrimental if done too strongly. For instance, interventions in skill level in the same direction of Fig. 5F but of greater magnitude disrupt development (Fig. S20). This highlights the need of predictive approaches to identify desired interventions.

Our results indicate that developmental history and environmental change have an opposite age-specific pattern in influencing skill level. In early life in *H. sapiens* (up to approximately 10 yr), skill level is influenced by developmental history and environmental variation in roughly equal proportions (approximately 50%) (Fig. 3B,C). As individuals age, skill level becomes increasingly reactive to developmental history and insensitive to environmental change, with only 20% of skill-level reactivity to environmental change at 40 yr.

Our results suggest that the predominant role of developmental history in affecting skill level may have been shared with many *Homo* species. According to our results, the proportion of factors influencing skill level in *H. sapiens* would have been similar 1.1 Myrs after the origin of the genus *Homo*, roughly corresponding with the *H. erectus* time period (at evolutionary time $\tau = 100$; Fig. 3).

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 increased plasticity in skill level and brain anatomy (*5, 39–42*). Our results are also consistent with the view that a protracted brain development in *H. sapiens* allows for stronger environmental influences (*43*). At *H. habilis* brain size, skill level substantially reacts to environmental and social factors particularly up to 5 yr, roughly coinciding with the estimated weaning age (*44*). Skill level in early *Homo* species would then be comparatively little influenced by environmental factors after weaning, which is consistent with findings that many primate species reach adult skill levels around weaning age due to the lack of post-weaning provisioning (*45*). However, at *H. sapiens* brain size, environmental and, to a lesser extent, social factors can influence skill level until almost 15 yr, well into adolescence. This finding is consistent with previous studies indicating late brain maturation in humans (*46, 47*) associated with late acquisition of adult skill levels (*48, 49*). That is, *H. sapiens* brains and skill levels can be strongly influenced by environmental factors at an age at which *H. habilis* skill level would have been largely insensitive to influences other than developmental history (Fig. 3I).

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



B

Measures of perturbation effects:	Example:
Sensitivity: total effect of a variable on another variable.	Increasing body size at birth by 100 g increases skill level by 20 GB at 40 years of age.
Elasticity: proportional total effect of a variable on another variable.	Increasing body size at birth by 1% increases skill level by 0.8% at 40 years of age.
Reactivity: proportional total effect of a variable type on another variable relative to all variables.	Increasing all phenotypes at all ages by 1% increases skill level by 80% at 40 years of age relative to increasing all phenotypes, genotype, environment/physiology, and social environment at all ages.
First-order contribution: amount of difference between traits caused by a given variable type.	Genetic differences in an individual cause a 0.2 TB increase in their skill level at a given age relative to a reference (here, average) individual.

Figure 1: **Causal diagram of the brain model and measures of the effects of perturbation.** (**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 perturbation, that is, of altering a variable at a given age, on adult skill level, using four 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; reactivity, which normalizes elasticity over all the possible interventions over development; and first-order contributions, which quantify the mechanistic contribution of phenotypic differences caused by differences in underlying factors.

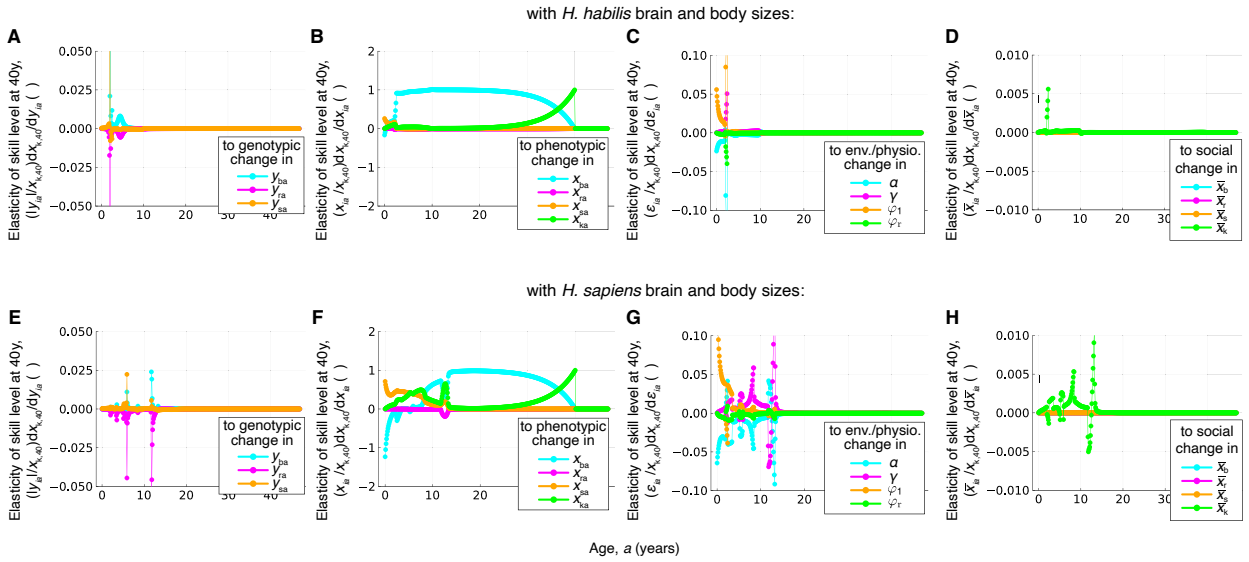


Figure 2: **Adult skill level is most elastic to change in developmental history.** Elasticity of adult skill level to (**A**,**E**) genotypic, (**B**,**F**) phenotypic, (**C**,**G**) environmental or physiological, and (**D**,**H**) social change: plots give the change in skill level at 40 yr when the 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* scale (i.e., for blue dots in Fig. S1B-E). Panels **E-H** are for individuals with brain and body sizes of *H. sapiens* scale (i.e., for red dots in Fig. S1B-E). In **B** and **F**, the elasticity after 40 yr is zero because the skill level at 40 yr is not affected by changes implemented in the future. The elasticity to phenotypic change is largest (**B**,**F**), followed by the elasticity to environmental (**C**,**G**), genetic (**A**), and social (**D**,**H**) change (see vertical axes).

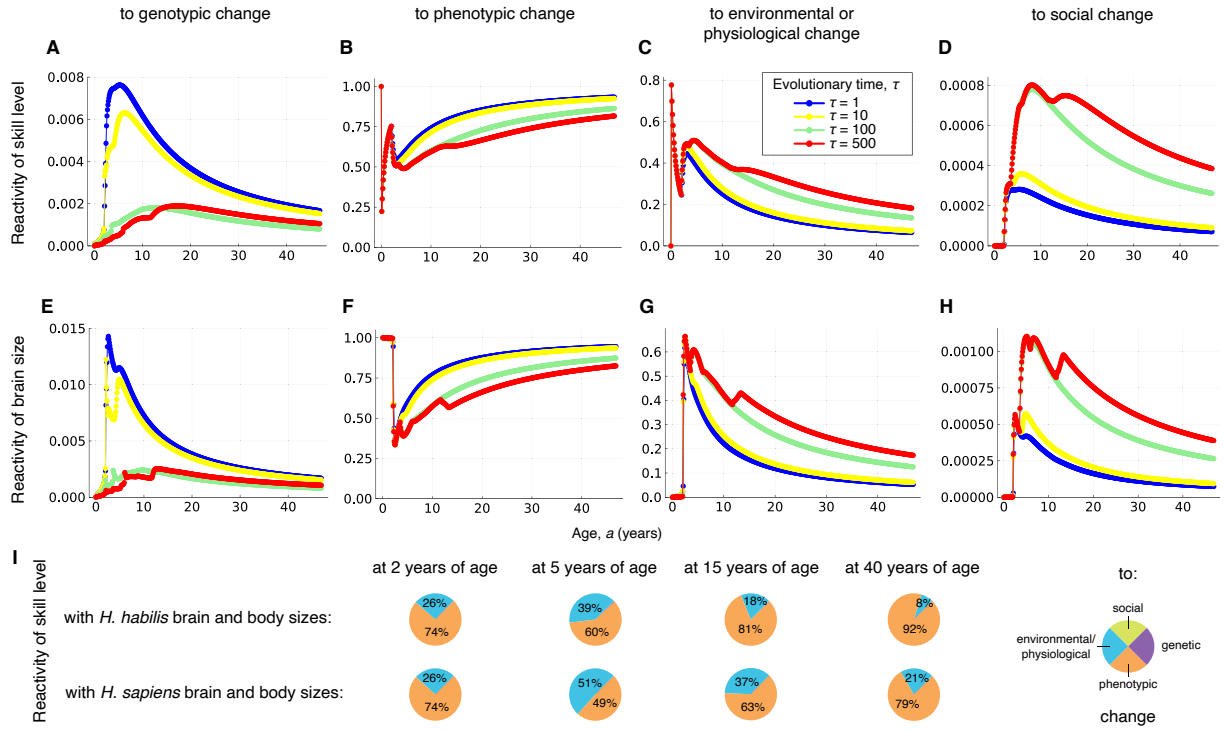


Figure 3: **Evolution of developmental reactivity of skill level and brain size over hominin brain expansion.** (**A**) Reactivity of skill level to genotypic change; for instance, the red dot at the age $a = 10$ yr gives the response of a 10-year-old's skill level to marginally increasing all genotypic 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. Skill level reactivity to genotypic and phenotypic change has decreased over evolution (**A**, **B**) and that to environmental, physiological, and social change has increased over evolution (**C**, **D**). Adult skill level remains most reactive to phenotypic change (**B**), environmental or physiological change (**C**), genotypic change (**A**), and social change (**D**) in decreasing order (see vertical axes). (**E-H**) Brain size reactivities have similar patterns. (**I**) Pie charts illustrating skill level reactivities in **A-D** at *H. sapiens* ($\tau = 500$) and *H. habilis* ($\tau = 1$) brain and body sizes for some ages.

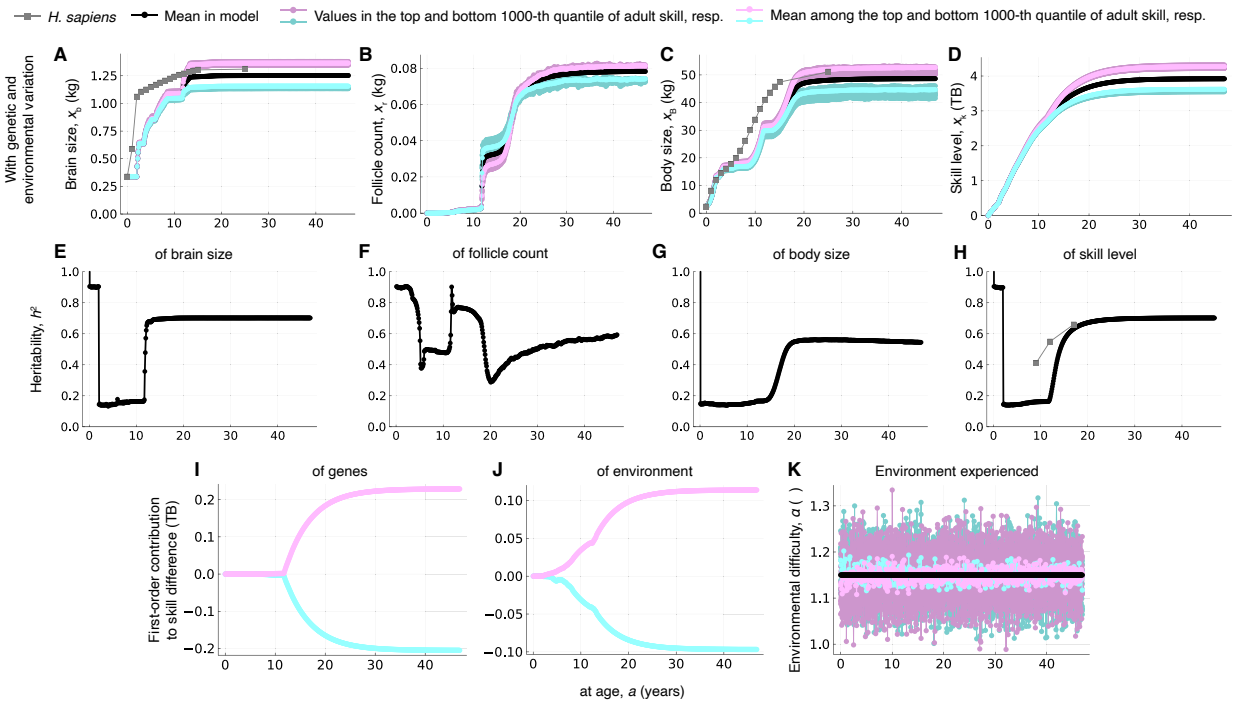


Figure 4: **Simulating genetic and environmental adversity.** For each age, we draw 10000 genotypic traits and environmental difficulties, normally distributed (variance $\sigma_g^2 = 0.1$ and $\sigma_e^2 = 0.002$, respectively) around their means given by the *H. sapiens* scenario in Fig. S1, under which we develop 10000 individuals. (**A-D**) 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 (Fig. S19), whose values are chosen so the heritability of adult skill level (**H**, black) roughly matches the empirically estimated heritability of general cognitive ability (**H**, gray) by (*33*). First-order contribution of (**I**) genes and (**J**) environment to the skill-level difference between the average individual among the top (light pink) or bottom (light blue) 1000-th quantiles in adult skill level and the population-average individual, showing that genes contribute more to the simulated differences than the environment. (**K**) Environment experienced.

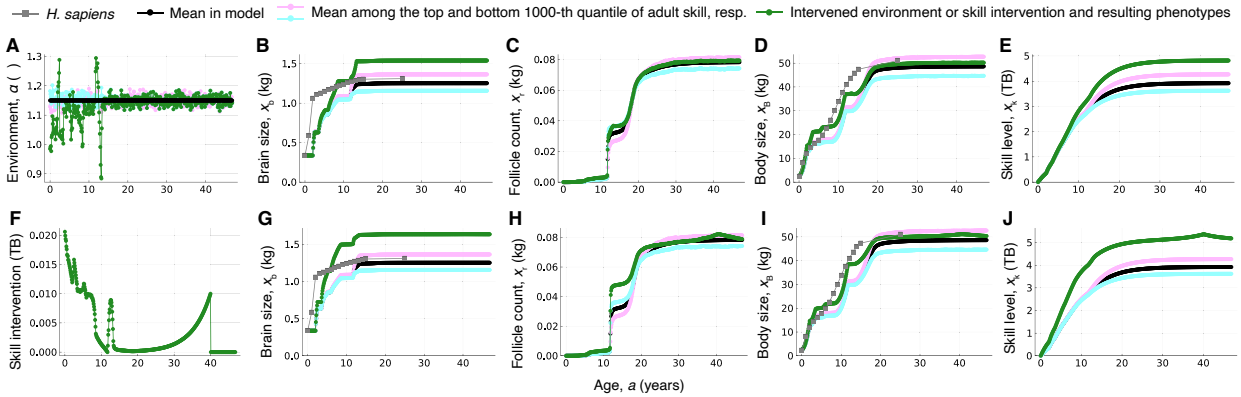


Figure 5: **Predicting interventions that compensate for adversity.** (**A**) Environmental difficulty on average: across the population (black), among the bottom (light blue) and top (light pink) 1000th quantile of adult skill level, and among the bottom 1000th quantile of adult skill level facing environmental intervention (green). The environment is intervened in the direction of steepest increase of adult skill level (i.e., the environment in green is that in blue plus 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 top-quantile individuals (pink). (**F**) Slight intervention in skill level in the direction of steepest increase of adult skill level (skill level at each age is increased by 0.01 times the sensitivity of adult skill level with respect to skill level at that age). (**G-J**) Corresponding phenotypes developed. 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 top-quantile individuals (pink).

References and Notes

1. H. Zeng, *et al.*, Large-Scale Cellular-Resolution Gene Profiling in Human Neocortex Reveals Species-Specific Molecular Signatures. *Cell* **149**, 483–496 (2012).

2. O. Canela-Xandri, K. Rawlik, A. Tenesa, An atlas of genetic associations in UK Biobank. *Nat. Genet.* **50**, 1593–1599 (2018).

3. S. Sakaue, *et al.*, A cross-population atlas of genetic associations for 220 human phenotypes. *Nat. Genet.* **53**, 1415–1424 (2021).

4. E. Uffelmann, *et al.*, Genome-wide association studies. *Nat. Rev. Methods Primers* **1**, 59 (2021).

5. C. C. Sherwood, A. Gómez-Robles, Brain plasticity and human evolution. *Annu. Rev. Anthropol.* **46**, 399–419 (2017).

6. K. R. Rosenberg, The evolution of human infancy: why it helps to be helpless. *Ann. Rev. Anthropol.* **50**, 423–440 (2021).

7. E. Turkheimer, M. Waldron, Nonshared Environment: A Theoretical, Methodological, and Quantitative Review. *Psychol. Bull.* **126**, 78–108 (2000).

8. J. Freund, *et al.*, Emergence of Individuality in Genetically Identical Mice. *Science* **340**, 756–759 (2013).

9. K. J. Mitchell, *Innate: How the Wiring of Our Brains Shapes Who We Are* (Princeton Univ. Press, Princeton, NJ, USA) (2018).

10. R. Plomin, K. Kawakami, Nurture and Nonshared Environment in Cognitive Development. *Psychol. Rev.* (2025), doi:https://doi.org/10.1037/rev0000551.

11. C. P. van Schaik, J. M. Burkart, Social learning and evolution: the cultural intelligence hypothesis. *Phil. Trans. R. Soc. B* **366**, 1008–1016 (2011).

12. J. Sparling, C. Dragomir, S. Ramey, L. Florescu, An educational intervention improves developmental progress of young children in a Romanian orphanage. *Infant Ment. Health J.* **26**, 127–142 (2005).

13. R. Whelan, *et al.*, Neuropsychosocial profiles of current and future adolescent alcohol misusers. *Nature* **512**, 185–189 (2014).

14. L. Jollans, R. Whelan, The Clinical Added Value of Imaging: A Perspective From Outcome Prediction. *BP:CNNI* **1**, 423–432 (2016).

15. R. A. Kievit, *et al.*, Developmental cognitive neuroscience using latent change score models: A tutorial and applications. *Dev. Cogn. Neurosci.* **33**, 99–117 (2018).

16. M. D. Rosenberg, B. J. Casey, A. J. Holmes, Prediction complements explanation in understanding the developing brain. *Nat. Commun.* **9**, 589 (2018).

17. O. N. Tikhodeyev, O. V. Shcherbakova, The Problem of Non-Shared Environment in Behavioral Genetics. *Behav. Genet.* **49**, 259–269 (2019).

18. M. Ota, *et al.*, Causal modeling of gene effects from regulators to programs to traits: integration of genetic associations and Perturb-seq. *bioRxiv* (2025), doi:https://doi.org/10.1101/2025.01.22.634424.

19. P. Gunz, S. Neubauer, B. Maureille, J.-J. Hublin, Brain development after birth differs between Neanderthals and modern humans. *Curr. Biol.* **20**, R921–R922 (2010).

20. H. Coqueugniot, J.-J. Hublin, F. Veillon, F. Houët, T. Jacob, Early brain growth in *Homo erectus* and implications for cognitive ability. *Nature* **431**, 299–302 (2004).

21. P. Gunz, *et al.*, *Australopithecus afarensis* endocasts suggest ape-like brain organization and prolonged brain growth. *Sci. Adv.* **6**, eaaz4729 (2020).

22. M. González-Forero, T. Faulwasser, L. Lehmann, A model for brain life history evolution. *PLOS Comp. Biol.* **13**, e1005380 (2017).

23. G. B. West, J. H. Brown, B. J. Enquist, A general model for ontogenetic growth. *Nature* **413**, 628–631 (2001).

24. M. González-Forero, A. Gómez-Robles, Why did the human brain size evolve? A way forward. *Phil. Trans. R. Soc. B* **380**, 20240114 (2025).

25. M. González-Forero, A. Gardner, Inference of ecological and social drivers of human brain-size evolution. *Nature* **557**, 554–557 (2018).

26. M. González-Forero, Evolutionary-developmental (evo-devo) dynamics of hominin brain size. *Nat. Hum. Behav.* **8**, 1321–1333 (2024).

27. W. Roebroeks, P. Villa, On the earliest evidence for habitual use of fire in Europe. *Proc. Natl. Acad. Sci.* **108**, 5209–5214 (2011).

28. L. Barham, *et al.*, Evidence for the earliest structural use of wood at least 476,000 years ago. *Nature* **622**, 107–111 (2023).

29. J. Paige, C. Perreault, 3.3 million years of stone tool complexity suggests that cumulative culture began during the Middle Pleistocene. *Proc. Natl. Acad. Sci.* **121**, e2319175121 (2024).

30. M. González-Forero, Computer code for: Human skill level is more reactive to developmental history than to genes or environment. *Zenodo* (2025), doi:https://doi.org/10.5281/zenodo.16612939.

31. B. J. Ashton, A. R. Ridley, E. K. Edwards, A. Thornton, Cognitive performance is linked to group size and affects fitness in Australian magpies. *Nature* **554**, 364–367 (2018).

32. H. Caswell, *Matrix Population Models* (Sinauer, Sunderland, MA, USA), 2nd ed. (2001).

33. C. M. A. Haworth, *et al.*, The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Mol. Psychiatry* **15**, 1112–1120 (2010).

34. H. Caswell, Analysis of life table response experiments I. Decomposition of effects on population growth rate. *Ecol. Model.* **46**, 221–237 (1989).

35. M. González-Forero, A mathematical framework for evo-devo dynamics. *Theor. Popul. Biol.* **155**, 24–50 (2024).

36. J. J. Berg, *et al.*, Reduced signal for polygenic adaptation of height in UK Biobank. *eLife* **8**, e39725 (2019).

37. M. Sohail, *et al.*, Polygenic adaptation on height is overestimated due to uncorrected stratification in genome-wide association studies. *eLife* **8**, e39702 (2019).

38. T. Tan, *et al.*, Family-GWAS reveals effects of environment and mating on genetic associations. *medRxiv* (2024), doi:https://doi.org/10.1101/2024.10.01.24314703.

39. A. Gómez-Robles, W. D. Hopkins, C. C. Sherwood, Increased morphological asymmetry, evolvability and plasticity in human brain evolution. *Proc. R. Soc. B.* **280**, 20130575 (2013).

40. B. Hrvoj-Mihic, T. Bienvenu, L. Stefanacci, A. R. Muotri, K. Semendeferi, Evolution, development, and plasticity of the human brain: from molecules to bones. *Front. Hum. Neurosci.* **7**, 707 (2013).

41. E. C. Snell-Rood, An overview of the evolutionary causes and consequences of behavioural plasticity. *Anim. Behav.* **85**, 1004–1011 (2013).

42. A. Gómez-Robles, W. D. Hopkins, S. J. Schapiro, C. C. Sherwood, Relaxed genetic control of cortical organization in human brains compared with chimpanzees. *Proc. Natl. Acad. Sci. USA* **112**, 14799–14804 (2015).

43. A. Gómez-Robles, C. Nicolaou, J. B. Smaers, C. C. Sherwood, The evolution of human altriciality and brain development in comparative context. *Nat. Ecol. Evol.* **8**, 133–146 (2024).

44. S. L. Robson, B. Wood, Hominin life history: reconstruction and evolution. *J. Anat.* **212**, 394–425 (2008).

45. S. A. Heldstab, K. Isler, C. Schuppli, C. P. van Schaik, When ontogeny recapitulates phylogeny: Fixed neurodevelopmental sequence of manipulative skills among primates. *Sci. Adv.* **6**, eabb4685 (2020).

46. D. J. Miller, *et al.*, Prolonged myelination in human neocortical evolution. *Proc. Natl. Acad. Sci. USA* **109**, 16480–16485 (2012).

47. A. Sotiras, *et al.*, Patterns of coordinated cortical remodeling during adolescence and their associations with functional specialization and evolutionary expansion. *Proc. Natl. Acad. Sci. USA* **114**, 3527–3532 (2017).

48. H. Kaplan, K. Hill, J. Lancaster, A. M. Hurtado, A theory of human life history evolution: diet, intelligence, and longevity. *Evol. Anthropol.* **9**, 156–185 (2000).

49. C. Schuppli, K. Isler, C. P. van Schaik, How to explain the unusually late age at skill competence among humans. *J. Hum. Evol.* **63**, 843–850 (2012).

50. H. Caswell, *Sensitivity Analysis: Matrix Methods in Demography and Ecology* (Springer Open, Cham, Switzerland) (2019).

51. J. M. Henshaw, M. B. Morrissey, A. G. Jones, Quantifying the causal pathways contributing to natural selection. *Evolution* **74**, 2560–2574 (2020).

52. J. Lehtonen, J. Otsuka, Evolutionary game theory of continuous traits from a causal perspective. *Phil. Trans. R. Soc. B* **378**, 20210507 (2023).

53. V. L. Greene, An algorithm for total and indirect causal effects. *Political Methodology* **4**, 369–381 (1977).

54. R. O. Deaner, K. Isler, J. Burkart, C. van Schaik, Overall Brain Size, and Not Encephalization Quotient, Best Predicts Cognitive Ability across Non-Human Primates. *Brain Behav. Evol.* **70**, 115–124 (2007).

55. S. A. Heldstab, *et al.*, Manipulation complexity in primates coevolved with brain size and terrestriality. *Sci. Rep.* **6**, 24528 (2016).

56. G. Nave, W. H. Jung, R. K. Linnér, J. W. Kable, P. D. Koellinger, Are Bigger Brains Smarter? Evidence From a Large-Scale Preregistered Study. *Psychol. Sci.* **30**, 43–54 (2019).

57. S. Cox, S. Ritchie, C. Fawns-Ritchie, E. Tucker-Drob, I. Deary, Structural brain imaging correlates of general intelligence in UK Biobank. *Intelligence* **76**, 101376 (2019).

58. R. M. B. Jiska S. Peper, D. I. Boomsma, R. S. Kahn, H. E. H. Pol, Genetic influences on human brain structure: A review of brain imaging studies in twins. *Hum. Brain Mapp.* **28**, 464–473 (2007).

59. C. E. Elks, *et al.*, Variability in the heritability of body mass index: a systematic review and meta-regression. *Front. Endocrinol.* **3** (2012).

60. M. González-Forero, How development affects evolution. *Evolution* **77**, 562–579 (2023).

Acknowledgments

We thank Nicole Walasek and an anonymous reviewer for comments on the manuscript.

Funding: 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).

Author contributions: MGF conceived the work and did the mathematical and computational analyses. MGF and AGR analysed the results and wrote the paper.

Competing interests: There are no competing interests to declare.

Data and materials availability: No data were collected in this study. All data used were previously published in references provided in the main text or in the Supplementary Materials. All codes have been deposited at https://doi.org/10.5281/zenodo.16612939 (ref. (*30*)).

Supplementary materials

Materials and Methods

Figs. S1 to S20

References *(50-60)*