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Socio-economic status is a social construct with heritable components and genetic consequences

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In civilizations, individuals are born into or sorted into different levels of socio-economic status (SES). SES clusters in families and geographically, and is robustly associated with genetic effects. Here we first review the history of scientific research on the relationship between SES and heredity. We then discuss recent findings in genomics research in light of the hypothesis that SES is a dynamic social construct that involves genetically influenced traits that help in achieving or retaining a socio-economic position, and can affect the distribution of genes associated with such traits. Social stratification results in people with differing traits being sorted into strata with different environmental exposures, which can result in evolutionary selection pressures through differences in mortality, reproduction and non-random mating. Genomics research is revealing previously concealed genetic consequences of the way society is organized, yielding insights that should be approached with caution in pursuit of a fair and functional society.

Human societies throughout history have often been stratified by socio-economic status (SES), with different groups of people having access to different levels of power, prestige, wealth, health, freedom and overall quality of life[1] (Box 1). Some believe that, in a meritocratic system, inequality can serve as an incentive for individuals to be more productive, but inequality—or an excess thereof—is also considered disruptive, with detrimental effects on social cohesion[2–4]. Rising inequality is generally accompanied by growing disparities in mental and physical health[4,5]. Although in recent times, inequality between countries has broadly declined, inequality within many countries has been increasing, especially since the 1980s[6,7]. Social inequality is an inherently societal phenomenon driven by cultural, structural, economic, political and technological forces, although, as we show here, it is also associated with genetic variation. Although behavioural genetics research is actively examining the relationship between genetics and SES[8], most studies within the broader social sciences aiming to understand social

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BOX 1

Definitions of socio-economic concepts

- Social stratification: the hierarchical organization of societies based on access to resources, power and SES, ranging from high to low. Group membership may or may not persist across generations.
- Socio-economic status: a social construct reflecting how society assigns value to certain outcomes, skills, traits, behaviours, achievements and assets. SES captures and influences cultural norms within a specific society. In social science research, SES is typically described across four correlated dimensions:
  - Income: total earnings from all sources (for example, wages, bonuses, investments and rental income), typically averaged over multiple years and measured at the individual or household level.
  - Education: the formal acquisition of knowledge and skills through schooling, training or self-directed learning. Education is both a cause and a consequence of cognitive ability. It is often measured by years of attainment, earned credentials and standardized test scores.
  - Occupation: employment status, which can fluctuate over adulthood. Typically measured through occupational classes, continuous status scales or ranking systems that combine education and occupational status (for example, ISEI, SIOPS and CAMSIS)[58].
  - Wealth: net worth, defined as the value of assets (for example, property, financial holdings and possessions) minus liabilities. Wealth includes both self-generated and intergenerational components and is particularly difficult to measure at the top of the distribution.
- (Intergenerational) social mobility: the degree to which an individual's SES rank correlates with that of their parents, reflecting changes in SES across generations.
- Social inequality: the unequal distribution of resources, opportunities and quality of life across individuals or households within a society at a given time.

inequalities tend to focus on societal factors[6,9]. By not including genetic effects, these studies omit a substantially contributing force that may be increasing in importance due to recent societal changes. When acknowledging these genetic effects, however, it is important to tread with caution. Recent history has shown that attempts to control the genetic make-up of populations—in the form of eugenics—can result in serious violations of human rights, including limiting access to education and labour markets, involuntary sterilization, infanticide, and genocide[10–12].

In the first half of this Perspective, we review the history of social stratification and the scientific study of its relationship with DNA. We summarize recent developments in genomics research that have provided us with a wealth of data on the relationships between genetic effects and socio-economic outcomes, albeit overwhelmingly in populations of European ancestry and with a bias towards individuals with higher SES (Box 2). In the second half of this Perspective, we discuss how these new data could be interpreted in the context of SES as a dynamic social construct that could exert natural selection pressures on genes associated with socially advantageous traits.

History of social stratification and the science of heredity

Social stratification

Civilizations are generally defined as complex societies with urban development, some form of government, symbolic systems of communication (for example, writing) and social stratification[13,14]. Although there are no known civilizations without some form of social stratification, it is not a defining characteristic of human societies in general. Most of the hunter-gatherer societies known today are relatively egalitarian[1,15,16]. Social stratification became more pronounced with the rise of larger and more complex human societies that arose during developments of the Neolithic era starting about 12,000 years ago[17]. Broadly speaking, the gradual shift from hunter-gatherer to sedentary agricultural societies enabled surplus resource accumulation, which led to an increase in population size and division of labour[18]. This allowed for different levels of prestige to develop through job specialization and more unequal accumulation of possessions. In numerous societies, elite classes arose that gained control over food supplies, land, means of production and the labour of much of their population. In many instances, legal and structural systems developed over time that codified these social hierarchies, reinforcing the power and privilege of the elites and cementing the stratification within societies[19].

Human history has known relatively rigid social stratification systems with little movement between socio-economic levels, often maintained through religious beliefs that legitimized the divine mandate of rulers[20]. Social status was often ascribed, with children inheriting their parents' status. Many of the phenomena discussed in this Perspective apply to systems that show at least some degree of merit-based social mobility. The earliest recorded example of a formal merit-based system emerged in China in the sixth century BC, where Confucian scholars advocated education for all and introduced the notion that those who govern should do so on the basis of individual merit rather than inherited status[21]. Such meritocratic principles were later applied by Genghis Khan in the thirteenth century, as he selected leaders on the basis of ability rather than family[22]. European scholars translated Confucian texts in the seventeenth century, exposing them to alternative perspectives on governance and social organization. These Confucian ideals probably contributed to the intellectual milieu of the Enlightenment movement, where merit-based social systems gained prominence[23]. The medieval European estate system, where noble or common status was largely determined by birth, made way for socio-economic orders that aimed for more equal opportunities. As the Industrial Revolution unfolded, bringing increased production, economic growth and social change, a modern, more merit-based socio-economic system began to emerge, transitioning to a new social order that could accommodate an ever-expanding population, while also increasing a visible underclass.

Compared with many pre-industrial socio-economic orders, merit-based hierarchies increase opportunities across the population, allocate talent more efficiently and stimulate progress through competition between people and between firms. The term 'meritocracy', however, was originally coined in a negative light in the 1958 satire *The Rise of the Meritocracy* by Michael Young[24]. This book describes a dystopian future, in which meritocracy has led to a newly stratified society, replacing an aristocracy of birth by an aristocracy of talent, with a disenfranchised lower class of the less meritorious. If behaviours associated with merit (for example, intelligence, persistence and creative talent) are partly heritable, variation in genetics within families could still facilitate social mobility. The enduring accumulation of resources within families, however, could limit this mobility, gradually reverting meritocracy back towards an aristocracy of birth.

Heredity

Contemporary research shows SES to be central to social stratification, focusing on intergenerational transmission of education, occupation, class, earnings and wealth, and variations across countries, history,

gender and ethnicity[25–28]. Social science research on social stratification and intergenerational transmission of SES has largely ignored or actively resisted the study of its relationship with genetic factors, partly due to ethical concerns and historical misuse of genetics in social policy[29]. To better understand this oversight, it is important to consider the history of genetics research and its societal impact.

Scientific research exploring connections between genetics and socio-economic success has a turbulent and controversial history. During the sixteenth century, early ideas about biological heredity were influenced by legal concepts of cross-generational inheritance of property and wealth[30]. The concept of heritability began to be formalized in the nineteenth and early twentieth centuries in the light of the work of Mendel and Darwin, which revealed the laws of inheritance and mechanisms of evolution. Charles Darwin's half-cousin, Francis Galton, explored the heritability of traits linked to merit and socio-economic success in his book, *Hereditary Genius* (1869). In this period, a prelude to the emergence of the field of genetics, Galton and his followers put more emphasis on 'nature' than on nurture. In his book, Galton applied statistics to show that offspring of 'eminent' figures had a higher chance of succeeding in what were perceived to be high-profile professions[31]. Inspired by these findings, Galton became a proponent of improving what became known as the 'genetic quality' of a population through selective parenthood, thus initiating and spearheading the emerging eugenics movement[32]. This movement became widely supported in many countries across the world and across the political spectrum by established intellectuals and medical authorities[10]. Eugenics proponents intended to explore and enact policies that would increase the overall well-being of majority populations or dominant social groups, but inevitably at the expense of others who were deemed economically costly or socially undesirable and who suffered stigmatization and persecution as a result[12]. In many cases, eugenic ideas resulted in state-sponsored violence against marginalized groups, primarily via enforced or coerced sterilization[33–35]. The destructive power of the eugenics movement reached genocidal levels in the Second World War, after which its public support declined. The legacy of involuntary sterilization is still detectable, with population register data revealing that individuals categorized with severe mental and physical disabilities (up to 1970 in Finland and 1976 in Sweden) often remained childless[36]. Enforced or coercive sterilization continues in several countries to this day, including China and India, the two most populous countries on Earth, often targeting lower socio-economic groups as a means of population control[12]. In the second half of the twentieth century, the scientific field of heredity became largely decoupled from social applications in most countries and made progress through decades of twin and family studies[37].

When it comes to socio-economic success, merit in contemporary industrialized societies typically involves strong performance in the educational system and/or labour market, both of which have intelligence—defined and measured in various ways—as one of their strongest predictors, alongside non-cognitive predictors such as parents' SES and individual-level traits such as conscientiousness[38–42]. Intelligence was the first trait studied using the classical twin method[43,44], which estimates heritability by comparing the resemblance of identical and fraternal twins. The considerable heritability of intelligence—that is, the extent to which genetic differences explain individual differences within a population—and its increase from childhood (about 0.43) to adulthood (about 0.65) have become among the most replicated findings in twin research[37,45,46]. A change in heritability can occur because genetic influences change over time or, more probably, because the variance in or influence of environments change.

Twin and family studies suggest that the heritability of traits affecting socio-economic outcomes varies with societal equality. Theoretically, equalizing opportunities would reduce heritability estimates if the genetic correlates of SES operate through traits associated with inherited privilege or social biases, rather than through traits that enhance an individual's ability to perform in a meritocratic system. If genetics mainly correlate with structural factors, such as ancestry-linked access to resources, power or social networks, then reducing these inequalities should weaken the correlation between

BOX 2

Factors affecting the estimation of genetic effects on socio-economic outcomes (part I)

Sources of bias in estimating genetic effects

GWASs can help to illuminate genetic architectures of complex traits, but they can also present a somewhat distorted view of this genetic architecture, particularly for behavioural and socio-economic outcomes. There are three main sources of bias:

- Population stratification: the largest patterns of genetic variation within a population generally reflect ancestry differences, often from a more distant past. When cultural or environmental differences align with these genome-wide allele frequency differences, GWASs can produce false positive associations across the genome. Although controlling for ancestry differences reduces bias, it is difficult to eliminate entirely[61,133]. Uncontrolled population stratification could affect polygenic score differences between groups and effect sizes at individual loci, but it should have less of an effect on genome-wide parameters estimated by LD score regression[62], which explicitly deals with this problem. Controlling for ancestry could also obscure real genetic signals when the causal genetic variants themselves correlate with ancestry differences. A more effective approach to control for population stratification involves within-family analyses, where genetic differences between siblings are compared[61,89], though these currently tend to have much smaller sample sizes than population-based GWASs and capture only direct genetic effects, excluding potentially causal indirect genetic effects (Box 3).

- Ancestry-related ascertainment bias: GWAS datasets lack global diversity[134], with 72% of GWAS discoveries (2005–2018) originating from the USA, the UK and Iceland[135]. To minimize false positives from population stratification, GWASs often exclude ethnic minority groups and control for small ancestry differences. This narrows the scope of GWASs on SES-related outcomes. Besides missing how SES interacts with genetics in non-Western societies, they fail to capture influential social factors within Western societies such as systemic racism or discrimination experienced by minority groups.

- SES-related ascertainment bias: people who are willing and able to participate in genetics research tend to be healthier and have higher SES, introducing a sampling bias[136]. UK Biobank, for example, shows smaller regional SES differences than census data[87]. This bias can distort our estimates of genetic and environmental contributions to complex traits across the full SES spectrum. Additionally, collider bias may arise when participation is influenced by both genetic factors and SES, potentially distorting associations between them. Using population-based weights to adjust for the overrepresentation of healthier, wealthier participants can help to correct some of the biases[137].

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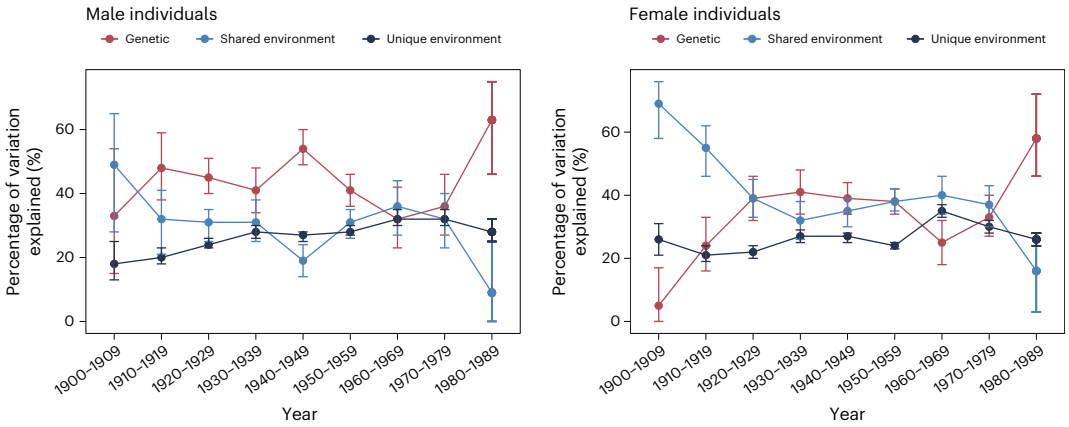

Fig. 1 | Changes in the heritability of educational attainment over time in Europe. The plots show the percentage of variation in educational attainment explained by genetic and environmental influences with 95% confidence intervals, as estimated in a meta-analysis of 28 European twin cohorts. Shared environmental influences are environmental factors that make siblings more similar to each other, and unique environmental influences are factors that do not, and also include measurement error. In twin studies, shared environmental influences can be overestimated at the expense of genetic influences when assortative mating occurs, as it increases the genetic similarity between siblings, mimicking shared environmental influences. This figure is based on data from table 2 in Silventoinen et al.[48].

genetics and SES. Alternatively, if genetic correlates of SES tend to reflect traits that improve an individual's competence in domains that influence social or economic mobility—such as cognitive or problem-solving abilities—then reducing environmental barriers should increase heritability by making genetic differences more predictive of life outcomes. In welfare states with more equal opportunities and high intergenerational mobility, the heritability of education has seemed to increase, primarily owing to reduced environmental influences and a relative constancy of genetic factors, increasing the relative contribution of genetics to the total variance[47]. In Europe, the heritability of educational attainment for women grew from 5% for those born 1900–1909 to 58% by 1980–1989 (ref. 48) (Fig. 1), possibly owing to improving educational systems and reduced societal barriers.

The genomics era

Advances in genotyping technologies in the twenty-first century enabled genome-wide association studies (GWASs), first applied in 2005 after being proposed in 1996 (refs. 49–52). In a GWAS, millions of genetic variants capturing most common genetic variation in a population are measured, and the effect of each variant on a trait of interest is estimated. The effects of individual genetic variants on complex traits turned out to be hard to distinguish from noise, owing to their extremely small effect sizes and the heavy multiple testing burden. Pooling data from many cohorts has enabled GWAS consortia to reach sample sizes of millions, identifying tens of thousands of variants linked to hundreds of physical, mental and behavioural traits[53].

The first large-scale GWAS on educational attainment was published in 2013 (ref. 54); it was conducted in about 125,000 individuals and identified only three associated genetic variants explaining around 0.02% of individual differences—a small harvest for such a large study, but enough to prompt further increases in sample size. Subsequent larger-scale GWASs reached up to three million individuals, identifying thousands of significantly associated variants[55–57]. GWASs on income and occupational status, each with sample sizes in the hundreds of thousands, found hundreds of associations, with very similar polygenic signals across these SES indicators[58–60]. Recent evidence indicates that a substantial portion of the signals detected in GWASs on SES-related traits may be confounded by ancestry differences (that is, population stratification), which could affect downstream analyses[61] (Box 2). Genetic correlations (r_g) estimated via methods such as linkage disequilibrium (LD) score regression (which leverages correlational patterns of genetic variants to adjust for population stratification)[62] are more likely to reflect the portion of the GWAS signal that is less affected by these confounds[63]. This less-confounded part of the income GWAS is consistent across Western countries, including the UK, the Netherlands, Norway, Estonia and the USA (average $r_g = 0.88$)[60]. Recently, the first educational attainment GWAS was conducted in a large East Asian dataset—South Korean and Taiwanese—which, after adjusting for population stratification, found a remarkably similar genetic signal to the European GWASs ($r_g = 0.87$)[64].

As with many behavioural traits, increasing sample sizes reveal progressively smaller effect sizes in line with the infinitesimal model, which builds on concepts proposed in the early days of quantitative genetics[65]. Modern GWASs confirm that complex traits are influenced by a large number of genetic variants, each with a small effect[66,67], but collectively explaining substantial variance. One way to harness the power of the aggregate genetic effects is through so-called polygenic scores, in which the alleles an individual carries are weighted by their estimated effects on a trait and then summed to produce genetic predictors. Many of the studies discussed below are based on polygenic scores constructed from the educational attainment GWASs, as these currently provide the most predictive scores due to their statistical power. Polygenic scores for educational attainment from a three-million-individual GWAS explain 12–16% of educational differences in European individuals[57], with about half attributable to the clustering of economic resources in families and assortative mating[61] (Box 3). Larger GWASs are expected to improve the precision of genetic effect estimates and thereby the predictive power of polygenic scores, although this depends on similarities between the discovery GWAS and the target datasets[68]. These polygenic scores capture part of the heritability of socio-economic outcomes, reflecting biological, social and demographic processes and correlated environmental exposures (Box 3). Genetic scores computed from birth with predictive power on future socio-economic outcomes hold potential value for research as well as policy development. Currently, however, their predictive power arises from a largely elusive combination of underlying traits and environmental influences[69]. Analyses of GWAS signals for SES outcomes using enrichment tools applied to diseases show the strongest enrichment in brain and neuronal processes[55,60], consistent with the role of cognitive and behavioural traits in SES. Rather than solely striving to expose underlying biology and perfect genetic effect size estimates, the field has greater potential when also striving to

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BOX 3

Factors affecting the estimation of genetic effects on socio-economic outcomes (part II)

Inflation of genetic effect estimates due to socio-economic structures

A substantial part of SES is inherited through the family, household and community one is born into, intertwining environmental and genetic influences. This can inflate genetic effect estimates in population-based GWASs. Polygenic scores for educational attainment based on population-based GWASs predict outcomes twice as strongly in children raised by biological families as in adoptees[138], and about 1.6 times more between families than within families[139]. Conducting GWASs within families[61,89] or geographical regions[91] significantly reduces the magnitude of genetic effects on SES-related outcomes. There are three main sources of inflation of genetic effects on SES as estimated in population-based GWASs:

- Indirect genetic effects: these occur when genetic influences arise not from an individual's own genes but from the shared loci of family members. These effects can arise from parents (for example, through parental investment or the prenatal environment), extended family members or even multi-generational/dynastic effects (for example, great-grandparents or beyond)[140]. Parents pass down half of their genes to their offspring. Polygenic scores for educational attainment constructed from untransmitted parental alleles predict socio-economic outcomes in children[90], probably reflecting indirect effects and/or assortative mating. As genetic effects cluster geographically[87], indirect effects from individuals outside the direct family that are geographically and socially nearby could influence GWAS signals as well[91].
- Gene–environment correlations: genes cluster within families, communities and neighbourhoods that provide favourable or unfavourable environmental circumstances. Living circumstances differ greatly between poorer and richer households and neighbourhoods. Genes that affect traits that influence socio-economic outcomes therefore correlate with effects that come from the environments that people are born in (passive gene–environment correlations) or are able to move to throughout their lives (active gene–environment correlations). This can make genetic effects seem stronger than they are and can induce a correlation between genetic variants and other traits (for example, body mass index and height) that are influenced by the environmental factors that come with higher or lower socio-economic outcomes.
- Assortative mating: socio-economic outcomes show strong positive assortative mating, where people select mates who resemble them. These outcomes are highly polygenic—that is, influenced by many alleles with small effects. When trait-increasing alleles, which can be found throughout the whole genome, are transmitted together to the offspring due to assortative mating, they become increasingly correlated with each other in the population, leading to overestimation of their individual genetic effects. This is analogous to, for example, the effects of a good teacher on a student's education being overestimated because good teachers cluster together in schools in better neighbourhoods, and thus children in the class of one good teacher tend to have classes from other good teachers as well.

understand what causes genetic effects to vary across different environments and populations—a direction we explore in the following sections on SES as a selection pressure.

Social stratification as a natural selection pressure

When Charles Darwin presented his theory of evolution by natural selection[70], he wrote that nature selects for adaptations that give organisms an advantage in the three struggles of life: with the physical environment, with other species and with members of one's own species. Over time, humans probably reduced the first two selection pressures while intensifying the third through social and economic competition, where winners are rewarded with the favourable social and environmental circumstances that come with higher SES.

GWASs on socio-economic outcomes produce polygenic signals that contain genetic effects on a mixture of traits and environmental effects[69]. Here we describe social forces that bind these traits and environmental effects together. In human societies, people become sorted into more favourable or less favourable environments, depending on a combination of their inherited privileges and their performance in the socio-economic system. This sorting could create selection pressures through differential mortality, reproductive success and mate choice. SES is a dynamic social construct that could target different associated traits and genes across time and space. In more merit-based systems, positive selection would probably act on genes associated with attributes and skills deemed beneficial by that particular society, assuming these translate to greater reproductive success; however, as we explain further in the section 'Reproductive success', in practice, the direction of the selection effect may have varied over time, and there can be nonlinear effects[71]. In particular, formal education and the introduction of money as a standardized medium of exchange could be expected to have sharpened this selection pressure. An educational system is a relatively efficient way to nurture talents and stratify the population on the basis of those talents at a relatively early age, and money offers a more efficient way to keep score of a person's 'earned' SES than earlier barter systems or reliance on resources such as land.

Historically, varying environmental and social conditions affected mortality and reproductive success, leading to complex patterns in the genetic architecture of complex traits. The highly polygenic and pleiotropic nature of complex traits, in addition to complex population structures, complicates the detection of past selection pressures at the DNA level[72]. Selection pressures on complex traits are dispersed across many minor effects. The most pervasive selection pressures detectable in genetic data across most complex traits, including educational attainment, are negative selection pressures, indicated by genetic variants with larger effects being kept at lower frequencies[73,74]. This pattern reflects a consistent constraint on extreme phenotypes (stabilizing selection), flattening the distribution of effect sizes and resulting in high polygenicity[75]. Thousands of genes show a strong signature of historical negative selection against damaging variants[76–78], and in contemporary populations, rare damaging variants in these genes have been associated with lower intelligence and educational attainment[79–81]. Modern selection pressures are expected to be detectable through associations between polygenic effects and patterns of mortality, reproduction and non-random mating, all of which appear

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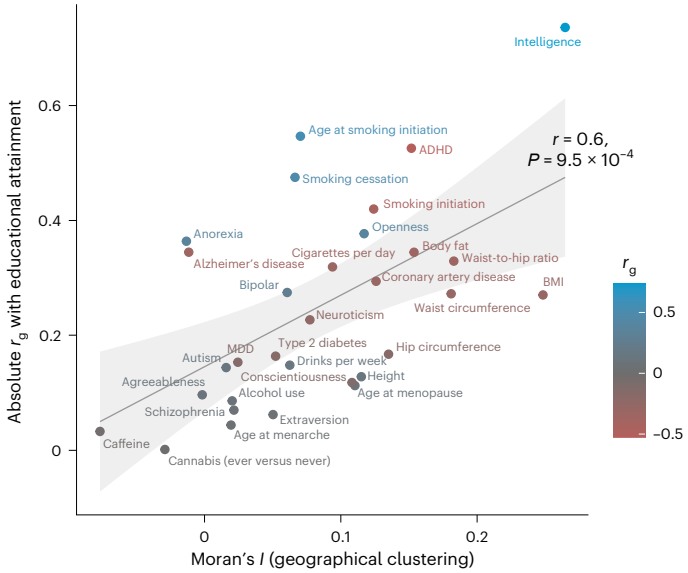

Fig. 2 | Traits that show higher genetic correlations with educational attainment tend to show stronger regional differences. Genetic correlations (r_g) can vary from −1 (100% shared variance due to the same genetic effects in the opposite direction) through 0 (no overlap in genetic effects) to 1 (100% of shared variance due to the same genetic effects in the same direction). The *y* axis indicates the absolute genetic correlation of the trait with educational attainment[55], excluding all British people. The genetic correlations were computed with LD score regression[63]. The *x* axis shows Moran's *I*, a measure of geographical clustering, of 31 polygenic scores in about 320,000 individuals in Great Britain. The Moran's *I* of the educational attainment polygenic score is 0.6 (not shown). ADHD, attention deficit hyperactivity disorder; BMI, body mass index; MDD, major depressive disorder. Figure adapted from ref. 87, Springer Nature Limited.

to be strongly driven by SES-related outcomes that vary across time and space, as discussed in more detail below.

Genes, geography and mortality

After the advent of agriculture, societies became larger and more complex[82]. Over the millennia of the Neolithic era, population growth led to urbanized settlements that covered geographical regions orders of magnitude larger than their predecessors. Different populations covered different geographical areas, and within populations and cities, socio-economic strata covered different regions[83]. The largest patterns of genetic variation align with differences between ancestral populations, which correlate strongly with geography, because individuals tend to reproduce with people who live closer to them geographically[84–86] (see 'Population stratification' in Box 2). If socio-economic outcomes are based on heritable merit-based outcomes, such as the performance in an educational system, it would be expected that the genes associated with these outcomes would show regional differences within populations as well.

Analyses of the geographical distribution of polygenic scores for a variety of behavioural, cognitive and health-related outcomes across Great Britain and across Estonia revealed that, once ancestry differences were minimized, the strongest regional differences were in educational attainment polygenic scores[87,88]. These geographical differences aligned with regional socio-economic differences; in Great Britain, lower polygenic scores clustered in economically disadvantaged coal mining areas[87], and in Estonia, higher polygenic scores were more concentrated in the two prospering university towns[88]. Migration contributed to these differences by increasing the geographical clustering of polygenic scores throughout the twentieth century while simultaneously reducing the correlation between ancestry and geography, as individuals with higher polygenic scores tended to move to more prosperous regions[87,88].

Polygenic scores for educational attainment and other SES outcomes are derived from GWASs estimating genetic effects on traits relevant to contemporary socio-economic systems. We can get a sense of the contributions of the underlying traits by estimating genetic correlations (r_g) between traits. The genetic correlations between educational attainment, income and occupational status are about 0.9 in many developed countries, suggesting that mostly the same genetic signal is being picked up for all three traits—namely, that of SES in those specific societies[58–60,64]. Of all traits investigated so far, intelligence shows the highest genetic correlation with SES (r_g with educational attainment, income and occupational status about 0.7); personality dimensions and mental and physical health outcomes also share a significant portion of their genetic effects with these SES outcomes[55,58–60]. Polygenic scores of traits that contribute more to socio-economic success tend to show stronger regional differences in Great Britain (Fig. 2) and in Estonia than those of traits that do not[87,88].

Genetic effects associated with educational attainment, income or occupational status are a patchwork of many underlying heritable outcomes, but these effects, as estimated in a GWAS, are also intertwined with environmental effects. Environmental factors are diverse and include cultural, social, economic and geographical contexts, ranging from societal structures such as housing quality, dietary options, healthcare and education systems to natural conditions such as air pollution. The increased efficiency with which populations in modern societies are stratified according to these heritable outcomes into different layers of environmental exposures leads to correlations between genes and environment that result in both 'double advantages' and 'double disadvantages'. As a result, conducting a standard population-based GWAS on an SES-related outcome is partially equivalent to doing a GWAS on being born into a better environment and/or the ability to move to a better environment. Molecular genetic evidence shows that SES-related genetic effects and environmental influences cluster on both a family level[61,89,90] and a regional level[87,91], resulting in systematic differences in these environmental exposures. These environmental factors that correlate with genetics can cause additional (regional) differences in mental and physical health outcomes, such as substance use and body mass index[91], but also more heritable traits, such as height (heritability about 80%; Fig. 3).

People with genetic variants that make it easier for them to get a better education are more likely to move to better neighbourhoods, whereas the people left behind are in worse living circumstances with higher mortality rates and greater risk for health problems such as obesity, diabetes[87] and infectious diseases. Regional differences in COVID-19 infection and mortality rates, for instance, also show significant genetic correlations with SES (Fig. 4), as it was easier for people in certain occupations, smaller households and better housing to avoid the disease by working from home and to be physically better prepared for infection through healthy diet and exercise[92]. COVID-19 was not likely to exert a strong selection pressure, because mortality was high only among those past the reproductive age. Previous pandemics, however, with higher mortality rates for younger people (such as the Black Death, smallpox and the Spanish flu) had higher mortality in areas of lower SES as well[93]. Besides the typical health consequences associated with lower-quality living conditions, deadly pandemics—whose frequency has increased since the advent of agriculture and cities[94,95]—could affect allele frequencies of genetic variants that affect traits that are more consistently associated with social stratification.

Reproductive success

Natural selection affects allele frequencies through differences in reproductive success, influenced by differential mortality rates, fertility rates and mate choice. Similar to other large mammals, human populations have historically faced density-dependent checks, where resource

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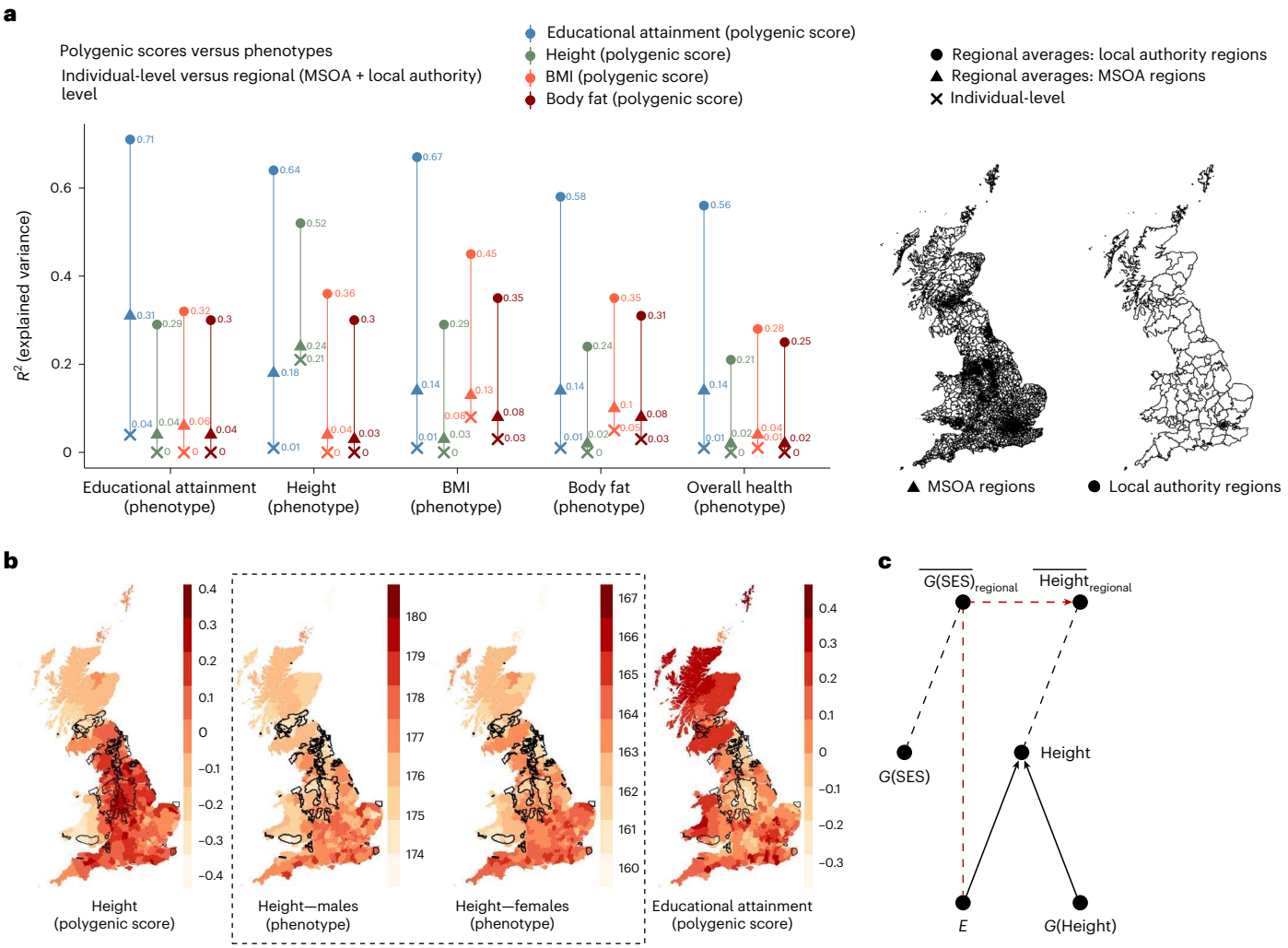

Fig. 3 | Polygenic prediction of average phenotypes per region probably captures environmental influences. a–c, Polygenic scores for educational attainment capture environmental effects on a regional level that are not visible when examining individual-level data. Panel **a** shows this on the basis of about 320,000 unrelated UK Biobank participants of European descent. The polygenic score for height explains 21% of individual differences in height, whereas the polygenic score for educational attainment explains only 1% of individual differences in height. When we consider the average scores per region, however, the polygenic score for educational attainment explains more regional differences in height (64%) than the polygenic score for height does (52%), presumably because the regional average of the educational attainment polygenic score better captures regional differences in poor versus rich environments, and these affect height. Panel **b** displays the geographical distributions of regional averages of polygenic scores for height and educational attainment, alongside the regional average for phenotypic height (in cm). The black lines denote coal mining regions, where environmental circumstances associated with socio-economic deprivation tend to cluster with polygenic scores for educational attainment. Without such clustering, coal mining regions would be among the taller regions of the country, which phenotypically they are not. For statistical analyses regarding these regional differences and the migration herein, see Abdellaoui et al.[87]. The hypothesized causal diagram in **c** illustrates environmental influences on height (*E*) that cluster regionally with genetic influences associated with SES (*G*(SES)), making the regional average of those genes ($\overline{G(SES)}_{regional}$) predictive of the regional average of phenotypic height ($\overline{Height}_{regional}$) – this pathway is indicated in red. *G*(Height) denotes the genetic influences on height on an individual level (Height). MSOA, middle layer super output area. For details on the data and quality control for panels **a** and **b**, see Abdellaoui et al.[91]; for details on polygenic score computation and geographical regions, see Abdellaoui et al.[87]. Panels **a** and **b** adapted from ref. 91, CC BY 4.0.

availability affected population density through mortality and fertility rates[96]. In humans, these dynamics are probably intertwined with SES.

A collection of about 15,000 English men's wills from the sixteenth to the twentieth century showed a positive relationship between men's income and net fertility in England, with the wealthiest individuals leaving nearly twice as many offspring as the poorest individuals[97,98]. This was probably influenced by higher child mortality rates in lower-SES groups[98,99] and greater mating opportunities for higher-SES male individuals, as women tend to prefer men with more resources across cultures with different mating systems, different levels of gender equality and different religions[100]. Pre-industrial data from the thirteenth to the twenty-first century across multiple countries confirm this positive income–fertility relationship[101]. As societies underwent major transformations due to the Industrial Revolution, including changes in population density, urbanization and industrialization, a general shift was observed across the world from a positive relationship to a negative relationship between income and reproductive success[101]. Several explanations posed for this reversal include changes in child mortality, birth control, and women's education and workforce participation[101–103]. Studies on industrialized societies have shown sex differences in the association between wealth and fertility[104].

In contemporary Western populations, including in Great Britain, common genetic variants associated with higher SES show a negative correlation with offspring count[71,105–108]. This implies a recent decline

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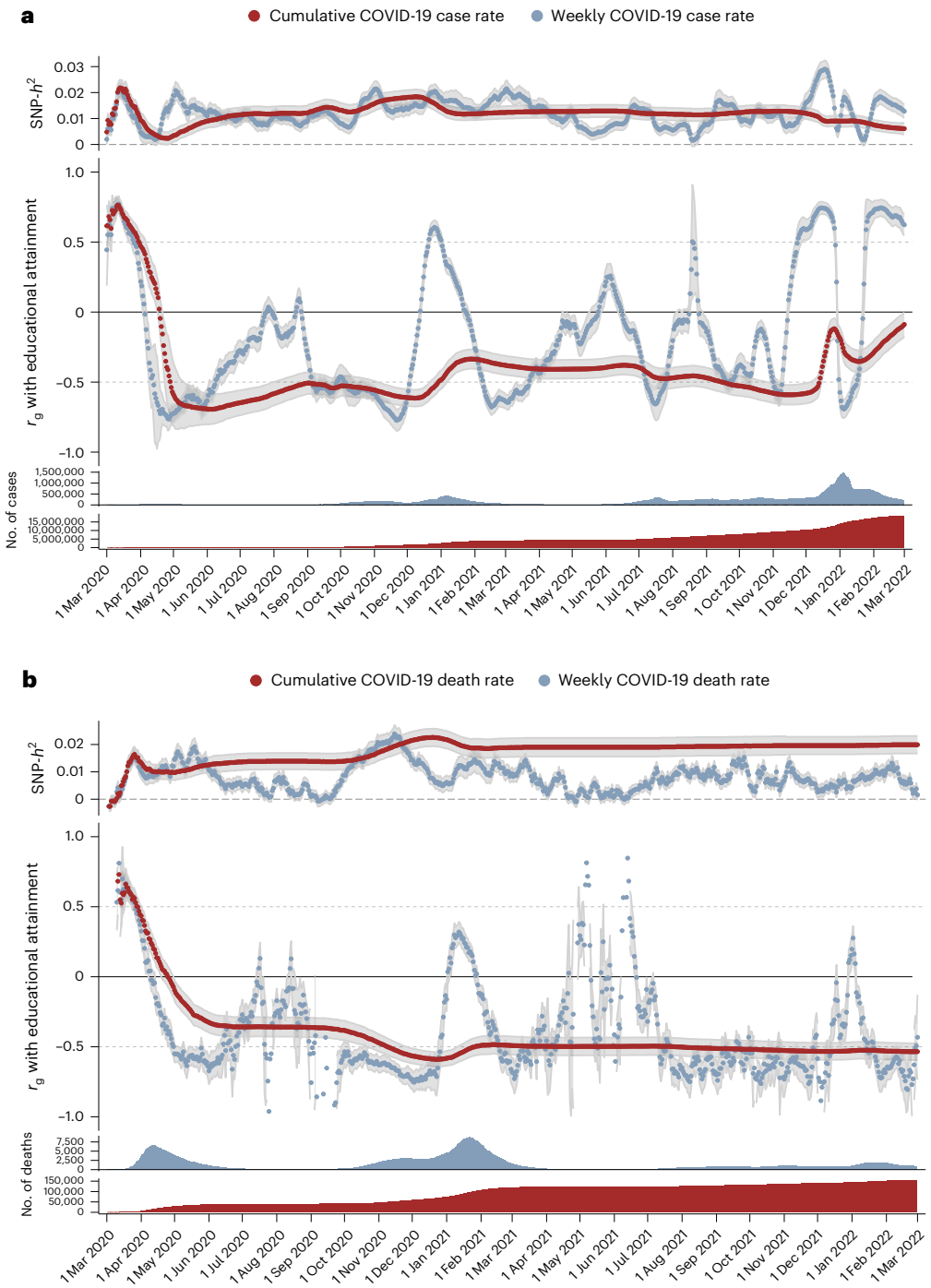

Fig. 4 | Genetic correlations show that COVID-19 infections and deaths in England originated in higher-SES regions and spread more widely in lower-SES regions. a,**b**, Data for COVID-19 cases (**a**) and deaths (**b**). The panels show the results from a total of 2,924 regional GWASs (four per day—cases and deaths, cumulative and weekly—for four years, or 731 days) performed on 1.2 million common single nucleotide polymorphisms (SNPs) from 396,042 individuals of European descent living in England. As opposed to a traditional GWAS, in a regional GWAS the participants are given the phenotype of the region they live in (315 regions), which often results in genetic signals associated with socio-economic outcomes due to their geographical clustering[87]. COVID-19 data on the 315 regions were obtained from Public Health England. Each dot is one regional GWAS (one day), for which either the weekly or the cumulative cases (**a**) or deaths (**b**) were analysed as the phenotype. The upper panel shows the variation explained by all 1.2 million SNPs (the SNP-based heritability or SNP-h^2). The large middle panel shows the genetic correlation (r_g) of the genetic signal with the educational attainment GWAS from Lee et al.[55]. The grey shaded areas around the points indicate 95% confidence intervals for both the SNP-h^2 and genetic correlations. These genetic correlations show several positive peaks, including at the start of the pandemic in March 2020 and around the start of the spread of the new and more contagious B1.1.7 variant in December 2020, both reflecting more infections in richer regions of the country (in or near London), after which the genetic correlation with education becomes negative again. The two bottom panels show the total number of cumulative and weekly COVID-19 cases (**a**) or deaths (**b**). For more information on quality control and statistical approaches used, see Abdellaoui[132], where the same results were reported for cases only for the first six weeks or so of the COVID-19 pandemic.

of these variants, despite their relationship with decreased mortality described in the previous paragraph. By contrast, rare genetic variants with stronger deleterious effects on intelligence, educational attainment and income negatively affect reproduction rates in Great Britain, especially in men[81]. This may reflect a nonlinear relationship between intelligence and reproductive success, with rare damaging variants being more predictive at the lower end of the intelligence spectrum.

More recently, the relationship between SES and reproductive success, which seems to be nonlinear and sex-specific, seems to be reverting back to an overall positive one, both within and between a number of high-income countries; this is probably driven by multiple factors, such as an increased compatibility between women's careers and families due to more favourable family policies and social norms, cooperative fathers, and more flexible labour markets[109–111].

Assortative mating

Besides determining who propagates their genes, mate choice can also affect genetic variation when people choose partners similar to themselves. When people choose mates who resemble them for a trait—assortative mating—this affects genetic variation by widening the genetic distribution in the offspring population and increasing resemblance between family members[65,112]. Humans tend to meet and choose partners similar to themselves in terms of ethnicity, religion and SES[113,114]. The strongest DNA similarity in spouses is for ancestry-related variation[115], followed by polygenic effects associated with educational attainment, which show the highest assortative mating levels among studied traits so far[116–120]. Interestingly, the educational-attainment-associated loci show a higher correlation between spouses than expected on the basis of the phenotypic spousal correlations. Possible explanations include additional assortative mating on the basis of more heritable underlying traits (for example, intelligence), matching based on characteristics of both the mate and their family members, or inaccurate genetic effect estimates[118,121,122] (Boxes 2 and 3).

Although assortative mating on education may have been strengthened more recently by the increased heritability of educational attainment in women (Fig. 1) and more women joining the workforce[123], studies on partnership markets around the Industrial Revolution suggest that assortative mating on socio-economic outcomes is not a recent phenomenon[124,125]. Data on 422,374 British inhabitants from 1600 to 2022 show substantial assortative mating and persistence of social status across generations despite major social changes over time[126]. In Norway, assortative mating continues to increase genetic similarities within families for genetic variants associated with educational attainment, and thus has not yet reached equilibrium[119]. Equilibrium, where genetic similarities within families stabilize despite ongoing assortative mating, is expected to be achieved after many generations of stable assortative mating on this outcome. The ongoing increase in familial genetic similarity suggests a recent increase in assortative mating, potentially contributing to, or even due to, growing inequalities in contemporary Norwegian society.

Assortative mating probably increases with the geographical clustering of SES, as physical proximity increases the chance of finding a similar partner. SES has been clustering geographically since ancient times[83]. Distances travelled with SES-related migration increased in recent times, which may have increased geographical clustering of SES-associated alleles[87], increasing assortative mating. If current rates of assortative mating on traits that influence SES persist or increase, this could further increase social inequalities on both an economic and a genetic level over generations, making them harder to overcome. Assortative mating can also make economic factors become more intertwined with genetics over generations, as environmental advantages tied to SES influence mate selection. In both Great Britain and Norway, for example, it has been shown that earlier-born siblings, who have a higher SES due to environmental factors, marry spouses with higher polygenic scores for educational attainment[127]. The increasing correlation within individuals between SES-associated alleles across the genome, and between these alleles and environmental factors that influence SES, could have societal and evolutionary consequences, but also complicate the task of accurately quantifying genetic effects associated with SES (Box 3).

Conclusions

Social inequality has long been inherent in the way human societies are organized, arising because certain outcomes are more valued and rewarded than others, and reinforced by the familial clustering and transmission of status, resources and genetic predispositions. Populations become stratified into social environments with differing levels of health risks, safety and opportunity, leading to disparities in mental and physical health. When combining substantial life quality differences between social layers with a certain amount of social mobility, it becomes desirable to climb the social ladder, stimulating many to try but allowing only those with the most advantageous talents to succeed. Over time, this could influence the genetic make-up of populations through differential mortality, fertility and non-random mating.

The strength and nature of these selection pressures vary across time and cultural contexts. Certain cognitive abilities may have conferred a more consistent advantage throughout our recent evolutionary history, particularly in more merit-based societies, which could have contributed to the increasingly complex societies that make humans such a unique primate. Technological advances may have impacted the effects of SES on genetic variation in multiple ways, including through improved and more accessible educational systems. These developments may, as a side effect, have made humans more efficient at stratifying the population according to genetic talent, further inducing geographical clustering and assortative mating on SES-related genetic variants, potentially increasing genetic differences associated with social inequality. The more direct genetic effects, however, are significantly smaller than initially estimated in population-based genetic association studies, due to assortative mating and environmental influences that get entangled with genetic effects (Box 3).

At the heart of these discussions is not a call for genetic intervention, but rather a call for a deeper understanding and awareness that our social structures are part of an evolving environment that, over time, shapes both social and genetic outcomes[128,129]. The relationship between social stratification and genetic effects is complicated, consisting of a network of complex traits and environmental circumstances woven together by social and economic forces created by increasingly complex societies. Although precise estimates of genetic effects are important, this field of research holds greater potential in uncovering how correlations between genes and social outcomes shift across societies and time[47,48,130]. Processes such as migration, mate choice, reproductive success and mortality shape how populations develop and could create feedback loops that reinforce or reshape inequalities. Understanding these dynamics could help us to explore societal structures of the past through traces left in the genome by, for example, assortative mating[131]; reveal causes of present inequalities; and anticipate how inequalities might evolve with future social changes. This line of investigation can provide researchers across disciplines with a framework for studying the dynamic interplay between genetics, complex traits and social organization. Although mistakes from the past should keep us vigilant about the potential for harmful effects of genetically informed social policies, advances in interdisciplinary genomics research can help us to better understand the processes underlying the way societies are organized and their consequences.

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Author contributions

A.A. conceived the article, analysed the data, produced the figures and wrote the manuscript. H.C.M., M.K., A.R., M.M., F.C.T., M.C.M., B.P.Z., K.J.H.V. and P.M.V. provided comprehensive feedback on the manuscript.

Competing interests

M.C.M. is a trustee of the UK Biobank, is on the Scientific Advisory Board of Our Future Health and Lifelines Biobank, and is on the Data Management Advisory Board of the Health and Retirement Survey. F.C.T. is a research fellow at AnalytiXIN, which is a consortium of health-data organizations, industry partners and university partners in Indiana primarily funded through the Lilly Endowment, IU Health and Eli Lilly and Company. The remaining authors declare no competing interests.

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