The high heritability of educational achievement reflects many genetically influenced traits, not just intelligence

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Because educational achievement at the end of compulsory schooling represents a major tipping point in life, understanding its causes and correlates is important for individual children, their families, and society. Here we identify the general ingredients of educational achievement using a multivariate design that goes beyond intelligence to consider a wide range of predictors, such as self-efficacy, personality, and behavior problems, to assess their independent and joint contributions to educational achievement. We use a genetically sensitive design to address the question of why educational achievement is so highly heritable. We focus on the results of a United Kingdom-wide examination, the General Certificate of Secondary Education (GCSE), which is administered at the end of compulsory education at age 16. GCSE scores were obtained for 13,306 twins at age 16, whom we also assessed contemporaneously on 83 scales that were condensed to nine broad psychological domains, including intelligence, self-efficacy, personality, well-being, and behavior problems. The mean of GCSE core subjects (English, mathematics, science) is more heritable (62%) than the nine predictor domains (35–58%). Each of the domains correlates significantly with GCSE results, and these correlations are largely mediated genetically. The main finding is that, although intelligence accounts for more of the heritability of GCSE than any other single domain, the other domains collectively account for about as much GCSE heritability as intelligence. Together with intelligence, these domains account for 75% of the heritability of GCSE. We conclude that the high heritability of educational achievement reflects many genetically influenced traits, not just intelligence.

academic achievement | twin studies | behavioral genetics | general cognitive ability | personalized learning

Education is one of society's biggest and most expensive environmental interventions in children's development, accounting for more than 6% of the gross domestic product in many countries (1). Differences among children in their educational achievement, especially culminating at the end of compulsory schooling, propel children on different lifelong pathways that affect higher education, occupation, and even health and mortality (1–4). Not only are differences in educational achievement important to society and to children as individuals, they are also a focal concern for parents (5, 6). For these reasons, it is important to understand the causes and correlates of differences among children in their educational achievement.

Educational achievement refers to mastery of specific content, including knowledge and skills for subjects such as literacy, numeracy, and science. The word achievement, in contrast to ability, connotes accomplishments by dint of effort. It is often assumed that effort is relatively more environmentally influenced than ability and thus that differences between children in their educational achievement are environmental in origin, reflecting differences among classrooms, schools, and parents (7, 8). This assumption is reasonable because, for example, most children will not learn to read or do arithmetic unless they are taught. However, genetic research has shown that individual differences in educational achievement are substantially heritable (9–11). Indeed, we have shown that educational achievement is significantly more heritable than intelligence in the early school years (12). We have recently found high heritability (58%) for the results of a nationwide examination, the General Certificate of Secondary Education (GCSE), which is administered in the United Kingdom at the end of compulsory education at age 16 (13).

The present study asks why individual differences in educational achievement at the end of compulsory education are so highly heritable, focusing on children's characteristics. Most phenotypic studies of the correlates of educational achievement have investigated intelligence or working memory (14–16). Correlations between IQ and educational achievement range between 0.4 and 0.7 (17). However, dozens of other traits have also been shown to relate to educational achievement, such as self-efficacy and motivation (18–21), emotional intelligence (22–25), personality (26–29),

Significance

Differences among children in educational achievement are highly heritable from the early school years until the end of compulsory education at age 16, when UK students are assessed nationwide with standard achievement tests [General Certificate of Secondary Education (GCSE)]. Genetic research has shown that intelligence makes a major contribution to the heritability of educational achievement. However, we show that other broad domains of behavior such as personality and psychopathology also account for genetic influence on GCSE scores beyond that predicted by intelligence. Together with intelligence, these domains account for 75% of the heritability of GCSE scores. These results underline the importance of genetics in educational achievement and its correlates. The results also support the trend in education toward personalized learning.

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prosocial behavior (5), well-being (30), goals (31), curiosity (32), beliefs about intelligence (33), self-efficacy (34), behavior problems (35, 36), health (37), and children's perceptions of their home environment (38) and their school environment (39). These traits are intercorrelated, which suggests the need for multivariate studies that can consider their joint and separate contributions to educational achievement. However, few broad multivariate phenotypic studies have been reported, although several studies have included intelligence in addition to another variable in predicting educational achievement (28, 40, 41). Recently, a theoretical model that attempted to integrate research on predictors of educational achievement focused on intelligence, specific interests, and personality, especially intellectual curiosity and conscientiousness (42).

Phenotypic correlations between such traits and educational achievement can be mediated genetically or environmentally, which is important because environmentally driven associations may be better targets for intervention. Relatively few studies have used genetically sensitive designs that can disentangle genetic and environmental sources of phenotypic correlations between children's traits and their educational achievement. Genetically sensitive studies have largely focused on intelligence, consistently showing that the phenotypic correlation between intelligence and educational achievement is mediated genetically to a substantial extent (43–50). Only a handful of studies have considered genetic contributions to educational achievement from other traits in addition to intelligence, such as self-efficacy (51), motivation (52, 53), personality (54), behavior problems (55–58), and perceptions of home environment (59) and school environment (60). Because these behavioral traits are correlated with each other and with educational achievement, adding up their separate genetic contributions to educational achievement could exceed the heritability of educational achievement. Multivariate genetic research is needed that considers the joint and independent contributions of a wide range of predictors to the heritability of educational achievement, taking into account the intercorrelations among the predictors. The only example to date is a twin study of longitudinal stability of teachers' grades at ages 11–17 for 800 pairs of twins that also reported multivariate genetic analyses, in which the heritability of teachers' grades at age 11 were largely explained collectively by genetic factors involved in intelligence, engagement, and externalizing behavior problems (61). This report led us to hypothesize that the substantial heritability of test scores at the end of compulsory education could almost entirely be explained by a larger set of predictors that includes self-efficacy, personality, and well-being.

The Current Study

We included diverse behavioral correlates of educational achievement in a multivariate genetic design, which allowed us to consider the joint and separate contributions of these traits to the heritability of educational achievement, taking into account the intercorrelations among the traits. Our study was sufficiently large to achieve adequate power to discriminate genetic and environmental estimates of variance and covariance between these behavioral correlates and educational achievement. The sample was from the UK Twins Early Development Study (62) and included 6,653 pairs of twins assessed on a set of examinations of educational achievement, called the GCSE, administered nationwide under standardized conditions at the end of compulsory education, typically at age 16. We created a composite GCSE score based on the three compulsory core subjects of English, mathematics, and science, which correlated 0.70 on average (see *Methods* for details about the sample and measures).

We focused on nine broad domains of candidate correlates of educational achievement: intelligence, self-efficacy, personality, well-being, parent-rated behavior problems, child-rated behavior problems, health, perceived school environment, and perceived home environment. Each domain is represented by a general composite rather than analyzing each of the scales within each domain. The reason for using composite indices is that they make the multivariate genetic analyses manageable and they provide an overview of the extent to which these diverse domains of behavior—considered separately and jointly—explain the heritability of educational achievement. In addition, our study was limited to measures included in the assessment of 16-y-old twins in the Twins Early Development Study (TEDS). Although the TEDS assessment was extensive, including 83 scales, it did not include all of the dozens of variables that have been reported to be associated with educational achievement. These two limitations—the use of general composite indices and the noninclusion of some measures—are conservative in the sense that including more fine-grained measures and additional variables might explain even more of the heritability of educational achievement. Conversely, if, as we hypothesized, most of the heritability of educational achievement is accounted for by these composite indices, this suggests that other predictors do not make a major independent contribution to the heritability of educational achievement after accounting for the predictors in the current study.

Results

The twin method was used to conduct univariate, bivariate, and multivariate analyses of genetic and environmental influences on the variance and covariance of the GCSE core subjects composite (henceforth just GCSE) and its correlates (see *Methods* for a description of the twin method and analyses). Table S1 shows means and SDs for the unadjusted GCSE core measure by the five twin groups arising from sex and zygosity. The observed mean sex differences are very small [males 8.86 (1.23), females 8.96 (1.21)]; the difference is statistically significant because of the very large sample size. Sex, zygosity, and their interaction account for less than 1% of the variance, and for subsequent analyses, after outliers were removed, variables were age and sex regressed and normalized using van der Waerden transformation as explained in *Methods.* Full sex limitation genetic modeling has previously been reported for GCSE and found only very minor sex differences in genetic and environmental estimates (13). In addition, the only other multivariate genetic analysis of this type found little evidence of sex differences (61). For these reasons and to increase power, the present analyses are based on the total sample, combining sexes.

Univariate Genetic Analyses. GCSE is more highly heritable (62%) than any of the nine predictor variables (35–58%), as summarized in Fig. 1. Shared environmental influence, which could be due to shared family or school environments, accounted for about a quarter of the variance of GCSE (26%) and were 0% for

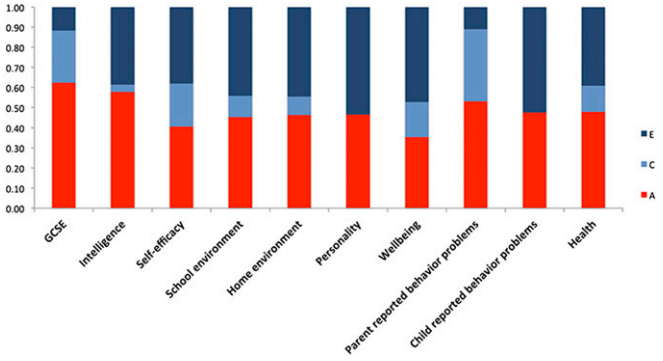

Fig. 1. Model fitting results for additive genetic (A), shared environment (C), and nonshared environment (E) components of variance for GCSE and nine predictors.

personality and child-rated behavior problems, 4% for intelligence, 21% for self-efficacy, and 36% for parent-rated behavior problems. Twin correlations are shown in Table S2 and model-fitting univariate estimates are presented in Table S3 for the standard ACE model that estimates additive genetic (A), shared environmental (C), and nonshared environmental (E) components of variance.

Bivariate Genetic Analyses. Fig. 2 illustrates the results of bivariate genetic analyses, which estimate the extent to which the phenotypic correlations between GCSE and each of the nine domains are mediated by genetic and environmental influences. The total length of the bar represents the phenotypic correlation between each of the domains and GCSE. The highest correlations with GCSE emerged for intelligence (0.58), self-efficacy (0.49), parent-rated behavior problems (0.33), and perceptions of school environment (0.34). The full correlation matrix is presented in Table S4.

Fig. 2 shows the proportion of the phenotypic correlation between GCSE and each domain that is explained by genetic, shared environmental, and nonshared environmental influences. For most of these domains, genetic influences in common with GCSE accounted for more than half of their correlation: intelligence (75%), self-efficacy (64%), perceptions of school environment (59%), personality (92%), well-being (53%), and behavior problems (81% for parent-rated, 89% for child-rated). Shared environment significantly mediated the phenotypic correlation with GCSE for intelligence (15%), self-efficacy (21%), school environment (31%), home environment (81%), well-being (34%), and health (28%). Cross-twin cross-trait correlations are shown in Table S2, and model-fitting estimates are included in Table S5.

Fig. 3 reorganizes the nine bivariate genetic analyses using Cholesky analysis (*Methods*) to show the extent to which the heritability of GCSE can be attributed to each predictor, in nine separate bivariate analyses. The length of the bar indicates the heritability of GCSE, which is estimated at 63% on average across the nine bivariate genetic analyses. The Cholesky analysis divides the heritability of GCSE into variance attributed to the predictor variable and residual variance, which indicates genetic influences on individual differences in GCSE independent of the predictor. The greatest contributions to GCSE heritability are from intelligence (51%) and self-efficacy (37%), with additional contributions from child-rated school environment (20%), personality (21%), well-being (8%), and behavior problems, both parent-rated (21%) and child-rated (16%). Child-rated health and home environment do not contribute to the heritability of GCSE. Model-fitting estimates for Fig. 3 are included in Table S6.

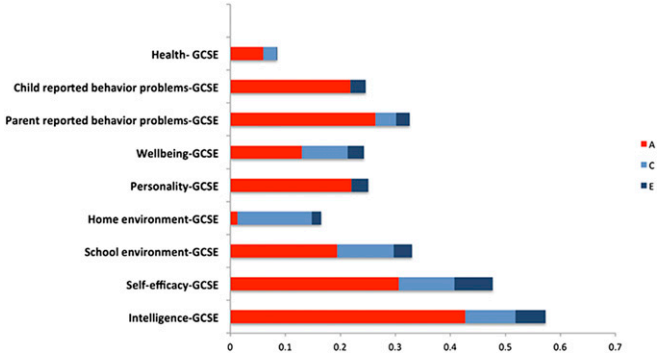

Fig. 2. Bivariate estimates for additive genetic (A), shared environmental (C), and nonshared environmental (E) contributions to the correlations between GCSE and nine predictors. The total length of the bar indicates the phenotypic correlations.

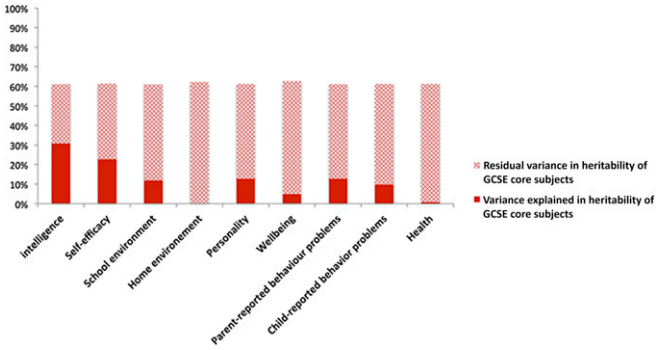

Fig. 3. Bivariate estimates of the extent to which the heritability of GCSE can be accounted for by each of the nine predictors, respectively (path a_{12} from the Cholesky decomposition; Fig. S1).

Multivariate Genetic Analyses. In summary, although intelligence accounts for most GCSE heritability, other domains also contribute significantly to GCSE heritability. Because the predictor variables correlate with each other (e.g., intelligence and self-efficacy correlate 0.35; see Table S4 for the full correlation matrix), their contributions to GCSE heritability exceed 100% when summed across the nine separate bivariate genetic analyses. For this reason, we conducted a multivariate genetic analysis including all nine predictors simultaneously to estimate how much of the GCSE variance they explain jointly. Phenotypically, in a multivariate Cholesky (conceptually similar to multiple regression) of GCSE on the nine predictors, the nine predictors account for 45% of the variance of GCSE. Multivariate genetic analysis (Cholesky) revealed that 75% of the heritability of GCSE is explained jointly by the nine predictors. Table S7 provides details of the results of the phenotypic and genetic multivariate analyses, and Tables S8–S10 provide details for genetic, shared environmental, and nonshared environmental correlation matrices.

We conducted an additional multivariate genetic analysis that asked whether, independent of intelligence, the other predictors collectively account for GCSE heritability. The eight predictors other than intelligence explain 50% of the GCSE heritability; adding intelligence raised this to 75%. Conversely, intelligence by itself explains 51% of GCSE heritability (Fig. 3 and Table S7).

Discussion

We found that, although intelligence accounts for more of the heritability of educational achievement at age 16 than any of the other domains, the other domains collectively accounted for about as much GCSE heritability as intelligence. Collectively, all cognitive and noncognitive predictors accounted for 75% of the heritability of GCSE. These genetic results turn some fundamental assumptions about education upside down. For example, one of the reasons that the contribution of intelligence is sometimes considered controversial when discussing educational outcomes is that intelligence is viewed as genetic, whereas achievement is thought to be due to environmentally driven influences from home and school. In addition, other behavioral traits such as self-efficacy are presumed to contribute to educational achievement for environmental reasons. However, our results suggest the opposite: Genetic influence is greater for achievement than for intelligence, and other behavioral traits are related to educational achievement largely for genetic reasons.

Although correlates of educational achievement have been the target of much research, there have been few multivariate studies, especially using genetically sensitive designs. With nine broad cognitive and noncognitive domains of children's behavior distilled from 83 scales, our phenotypic results show that educational achievement is correlated with many characteristics of children,

not just intelligence. Our bivariate genetic results indicate that these phenotypic correlations are largely mediated by genetic factors. That is, to the extent that children's traits predict educational achievement, they do so largely for genetic reasons, for example, for personality (92%), behavior problems (81% for parent-rated, 89% for child-rated), intelligence (75%), self-efficacy (64%), and well-being (53%). Although intelligence accounts for more GCSE heritability than any other single domain, almost as much of the genetic contribution to GCSE heritability comes from the joint contribution of children's self-efficacy, behavior problems, personality, well-being, and their perceptions of school environment. In our multivariate genetic analyses across the nine domains, we were able to account for 75% of the high heritability (62%) of differences between children in their educational achievement at the end of compulsory schooling on the United Kingdom-wide GCSE examinations. The only previous relevant study was primarily a longitudinal genetic analysis of teachers' grades in a sample one-sixth the size of the present study (61). Although not the focus of that study, it included multivariate genetic results for teachers' grades at age 11 that were similar to those presented here for test scores at the end of compulsory education at age 16. What these findings mean is that children differ for genetic reasons in how easily they learn and perform at the examinations, and not just because of differences in intelligence, but because of a whole package of genetically related characteristics including self-efficacy, personality, and behavior problems, as well as intelligence.

In this study, our goal was to describe the general genetic landscape of educational achievement using broad behavioral domains. The next step in this program of research is to zoom in for more fine-grained analyses within each domain, both phenotypically and especially genetically, which is the unique contribution of our large twin study. For example, within the domain of intelligence, what are the relative contributions of verbal and nonverbal abilities to GCSE heritability? Within personality, what are the relative contributions of the general "Big Five" personality traits such as extraversion and neuroticism, as well as traits more specific to educational achievement such as grit, confidence, and optimism? For behavior problems, phenotypic research suggests, for example, that inattention symptoms are more predictive of educational outcomes than hyperactivity symptoms (36), and genetic research suggests that externalizing problems such as inattention are more predictive than internalizing problems such as depression (61).

Although we focused on the genetic findings from this study to address the question of why educational achievement is so highly heritable, the results are also instructive about environmental influences, which can only be disentangled from genetic influences in genetically sensitive designs such as the twin method. Most notably, shared environmental influence, which could be due to the effects of shared family environment or shared schools, accounts for 26% of the variance of educational achievement. This shared environmental estimate could also be partially due to assortative mating, as educational achievement and intelligence have been reported to be subject to assortative mating where mate selection depends on trait similarity between spouses (63). However, if the sources of the variance are indeed shared environmental factors, a question for future research is the source of this influence that accounts for a quarter of the variance in GCSE test scores and would appear to be an especially good target for intervention. At first glance, from our results, family and school environment are both important candidates to explain shared environmental influences on GCSE. More fine-grained studies will be needed to identify precise environmental predictors.

It is important to emphasize that finding genetic influence is not a counsel of despair in terms of helping children who find learning difficult—heritability does not imply immutability. Heritability describes the extent to which phenotypic variance can be ascribed to DNA differences, on average, in a particular population at a particular time. In other words, heritability describes what is; it does not predict what could be. For example, despite high heritability, with sufficient educational effort, nearly all children could reach minimal levels of literacy and numeracy, which is an explicit goal of education in Finland (64). Success in achieving that goal would reduce phenotypic variance, which could change heritability. Another example is greater equality of opportunity in education would decrease environmental sources of variance and thus increase heritability, which has been demonstrated empirically (65). Nonetheless, our results are important for education in pointing to the pervasive role of genetics and not just for educational achievement itself, nor just for intelligence, but also for most of the other correlates of educational achievement. The ubiquitous impact of genetics in education suggests the need for a new model for education that moves from a passive model of schooling as instruction (instruere, meaning "to build in") to an active model of education (educare, meaning "to bring out") (7). That is, education is more than what happens to a child passively; children are active participants in selecting, modifying, and creating their experiences that are correlated with their genetic propensities, known in genetics as genotype–environment correlation.

No policy implications necessarily follow from finding that genetics permeates educational achievement, because policy depends on values and knowledge. However, it is to be hoped that better policy decisions can be made with knowledge of genetic influence rather than assuming that all differences are environmental in origin (7). For example, it is worth knowing that the successful realization of values such as equality of educational opportunity will not get rid of genetic differences between children. To the contrary, heritability is likely to increase as environmental differences such as educational inequalities are removed; in this sense, heritability can be considered as an index of equality. Philosophically, it is important to recognize that children differ for genetic reasons in how easy and enjoyable they find learning. For example, genetic thinking counters the deplorable tendency to blame teachers and parents rather than recognizing that learning is inherently more difficult for some children and that differences in children's educational achievement are more a matter of genes than schools or home environments. At the practical level of curricula, the active genotype–environment correlation model of education adds support for the trend in education toward personalized learning. This trend toward personalized learning has become more practical with rapid advances in technology and educational software to supplement or supplant one-size-fits-all traditional systems of education. More specifically, our results showing strong connections between noncognitive domains and educational achievement suggest that these domains are also plausible candidates for intervention, although there is a need for longitudinal research such as cross-lagged analysis to explore causality more explicitly.

Methods

Participants. TEDS is a multivariate longitudinal study that recruited more than 11,000 twin pairs born in England and Wales in 1994, 1995, and 1996. The recruitment process and the sample are described in detail elsewhere (62). The TEDS sample is representative of the UK population compared with the data obtained by the Office of National Statistics (46). The project received approval from the King's College London Institute of Psychiatry ethics committee, and parental consent was obtained before data collection.

The sample for the present study included all individuals who had GCSE and other measures available at the age of 16. GCSE results at age 16 were available for 13,306 individuals. Children with major medical or psychiatric problems or severe perinatal medical problems were excluded from the analyses. Additionally, children whose first language was not English and whose zygosity was unknown or uncertain were excluded. Zygosity was assessed through a parent questionnaire of physical similarity, shown to be 95% accurate when validated against DNA testing (66). DNA testing was conducted where zygosity was unclear from this questionnaire. The present analyses were thus conducted on 13,306 individuals comprising 6,653 twin pairs: 2,362 monozygotic (MZ) pairs, 2,155 same-sex dizygotic (DZ) twin pairs, and 2,136 opposite-sex DZ twin pairs.

GCSE Measures. The GCSE is a UK nationwide examination taken at the end of the compulsory education. GCSE courses start typically at the age of 14, and the examinations are taken at the age of 16. The courses include a variety of subjects from traditional core academic subjects such as English and mathematics, to geography, history, music, modern foreign languages, physical education, and information and communication technology (ICT). Typically, students take 10 or more GCSE examinations at the end of compulsory education. English, mathematics, and science (composed of single-weighted or double-weighted science, or when taken separately, physics, chemistry, and biology) are compulsory courses. Many schools also require students to take English literature and one modern foreign language. The data for the present study were collected by questionnaires sent by mail and by telephone interview of parents and twins themselves. After completed forms were received from the families, the grades were coded from 11 (the highest grade, A*) to 4 (the lowest pass grade, G); no information about failed results was available. For 7,367 twins, self- and parent-reported GCSE results were verified using data obtained from the National Pupil database (NPD; www.gov.uk/government/uploads/system/uploads/attachment_data/file/251184/SFR40_2013_FINALv2.pdf), yielding correlations of 0.98 for English, 0.99 for mathematics, and >0.95 for all sciences.

For the present study, a composite measure of the compulsory core subjects was calculated and used in all analyses, because the scores on the core subjects were highly correlated (average of 0.70). This GCSE core measure was constructed as the mean of English, mathematics, and science scores: the mean of the English grade (the English language grade, or the mean of the English language grade and the English literature grade if both were taken), the science mean composite (the mean of all science GCSEs taken), and the mathematics grade. A GCSE core composite was created only if at least two of the three measures were available.

The GCSE measure was corrected for the small mean effects of age and sex (Table S1) by rescoring the variable as a standardized residual correcting for age and sex, as is standard practice in the analysis of twin data because members of a twin pair are identical in age and MZ twins are identical for sex, which would otherwise inflate twin estimates of shared environment (67). Finally, before conducting twin analyses, the GCSE measure was corrected for skew because the measure was negatively skewed, showing a ceiling effect similar to that observed in UK national statistics (NPD; www.gov.uk/government/uploads/system/uploads/attachment_data/file/251184/SFR40_2013_FINALv2.pdf). The GCSE measure was corrected for skew by mapping it on to a standard normal distribution using the rank-based van der Waerden's transformation (68, 69).

Measures Used to Predict GCSE. Data obtained from the twins and their families at age 16 for a range of cognitive and noncognitive measures were used to predict GCSE scores. These 83 measures were reduced to nine domains for the purpose of data reduction only; it should be noted that each domain was not assumed to reflect a single underlying latent factor. The data were collected by web-testing and questionnaires sent by mail.

Before domain composites were created, scales that correlated negatively with GCSE (such as behavior problems) were reversed so that scales within each domain could be summed and averaged. As with GCSE, all 83 scales were rescored as standardized residuals correcting for mean effects of age and sex. The scales were standardized with a mean of 0 and a SD of 1.0 so that they contributed equally when summed and averaged for each domain. Mean scores were calculated in this way for nine domains: general intelligence (Raven's Progressive Matrices and Mill Hill Vocabulary test), educational self-efficacy (5 scales such as academic self-concept, interest/enjoyment, attitudes toward key subjects), child-reported personality (10 scales such as Big Five Factors, optimism, and grit), child-reported well-being (17 scales, such as life satisfaction, happiness, hopefulness), parent-reported behavioral problems (12 scales such as hyperactivity, impulsivity, emotional lability), child-reported behavioral problems (8 scales such as peer problems, antisocial behavior, depression), child-reported health (9 scales such as body mass index, puberty status, sleep problems), child-reported school environment (10 scales such as engagement with school, attitudes to school, classroom environment), and child-reported home environment (10 scales such as chaos, monitoring, support). A more detailed description of the scales used to create composites is available at www.teds.ac.uk/downloads/Description83scales9domains.pdf. Composite scores were coded as missing when more than 40% of scales within that domain were missing.

Analyses. The twin method was used to conduct univariate, bivariate, and multivariate analyses of genetic and environmental influences on the variance and covariance of GCSE and the predictors of GCSE. The twin method assumes that twins reared together resemble each other due to the additive effects of shared genes or shared environmental factors. Identical, or MZ, twins share all segregating genes and are therefore 100% similar genetically. Nonidentical, or DZ, twins, on average, share half their segregating alleles, resulting in 50% genetic resemblance (like nontwin siblings). The correlation between twins for shared environmental effects is assumed to be 1.0 for both MZ and DZ twins growing up in the same family. Nonshared environmental influences are uncorrelated between twins and contribute to differences between them. On this basis, it is possible to decompose phenotypic variance and covariance into additive genetic (A), shared environmental (C), and nonshared environmental (E) etiologies (11).

We began by comparing intraclass correlations for MZ and DZ twins. To the extent that MZ twins correlate more highly than DZ twins, genetic influences (A) are implied. Shared environmental effects (C) are inferred from the residual familial resemblance not explained by heritability and can be estimated by subtracting the estimate of heritability from the MZ correlation. The difference between the MZ twin correlation and unity represents an estimate of nonshared environmental effects and measurement error (E). The ACE model parameters, together with confidence intervals, can be calculated more accurately using structural equation modeling with maximum-likelihood estimation, which also provides formal model fit statistics (70). Models were fit using the structural equation modeling program OpenMx (71). All fit statistics are available from the corresponding author on request.

Bivariate genetic analysis of covariance between variables is an extension of the univariate genetic analysis of variance. MZ and DZ cross-trait cross-twin correlations are examined to decompose the covariance between traits into additive genetic (A), shared environmental (C), and nonshared environmental (E) components. The bivariate genetic model estimates genetic and environmental mediation of the phenotypic correlation between variables (Fig. S1). Central to bivariate genetic analysis is the genetic correlation, which is the extent to which genetic effects on one variable are correlated with genetic effects on another variable, which is an index of pleiotropy. Genetic mediation of the phenotypic correlation between two variables is the genetic correlation weighted by the heritabilities of the two variables (Fig. S1*A*). An alternative representation of bivariate model-fitting is Cholesky decomposition (Fig. S1*B*), which focuses on how much of the variance of one variable can be accounted for by another variable, which is well suited to addressing our central question of the extent to which the heritability of GCSE can be explained by each of the nine predictor domains.

A series of nine bivariate analyses addressed the question of how much of the phenotypic variance and how much of the heritability of GCSE scores can be explained by each of the domains. Additionally, the proportion of phenotypic correlation between the GCSE core measure and nine domains was decomposed into additive genetic (A), shared environmental (C), and nonshared environmental (E) factors. This series of bivariate analyses did not control for variance explained by the other domains. Therefore, the phenotypic and genetic variance in GCSE explained by these individual bivariate analyses was expected to exceed 100% across the nine domains. A multivariate genetic extension of Cholesky analysis was used to estimate the extent to which the nine domains jointly explain the heritability of GCSE, taking into account the covariance among the nine domains.

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1. OECD (2013) *Education at a Glance 2013* (Organisation for Economic Co-operation and Development, Paris).
2. Bronfenbrenner U (1996) *The State of Americans: This Generation and the Next* (Simon and Schuster, New York).
3. Cutler DM, Lleras-Muney A (2008) *Making Americans Healthier: Social and Economic Policy as Health Policy*, eds Schoeni RF, House JS, Kaplan GA, Pollack H (Russell Sage Foundation, New York).
4. Cutler DM, Lleras-Muney A (2012) *Education and Health: Insights from International Comparisons* (National Bureau of Economic Research, Cambridge, MA).
5. Caprara GV, Barbaranelli C, Pastorelli C, Bandura A, Zimbardo PG (2000) Prosocial foundations of children's academic achievement. *Psychol Sci* 11(4):302–306.
6. Buchmann C, Dalton B (2002) Interpersonal influences and educational aspirations in 12 countries: The importance of institutional context. *Sociol Educ* 75(2): 99–122.

7. Asbury K, Plomin R (2013) *G is for Genes: The Impact of Genetics on Education and Achievement* (Wiley-Blackwell, Chichester, UK).
8. Nye B, Konstantopoulos S, Hedges LV (2004) How large are teacher effects? *Educ Eval Policy Anal* 26(3):237–257.
9. Gill CE, Jardine R, Martin NG (1985) Further evidence for genetic influences on educational achievement. *Br J Educ Psychol* 55(Pt 3):240–250.
10. Martin NG, Martin PG (1975) The inheritance of scholastric abilities in a sample of twins. I. Ascertainments of the sample and diagnosis of zygosity. *Ann Hum Genet* 39(2):213–218.
11. Plomin R, DeFries JC, Knopik VS, Neiderhiser JM (2013) *Behavioral Genetics* (Worth Publishers, New York), 6th Ed.
12. Kovas Y, et al. (2013) Literacy and numeracy are more heritable than intelligence in primary school. *Psychol Sci* 24(10):2048–2056.
13. Shakeshaft NG, et al. (2013) Strong genetic influence on a UK nationwide test of educational achievement at the end of compulsory education at age 16. *PLoS ONE* 8(12):e80341.
14. Alloway TP, Alloway RG (2010) Investigating the predictive roles of working memory and IQ in academic attainment. *J Exp Child Psychol* 106(1):20–29.
15. Deary IJ, Strand S, Smith P, Fernandes C (2007) Intelligence and educational achievement. *Intelligence* 35(1):13–21.
16. Gathercole SE, Pickering SJ, Knight C, Stegmann Z (2004) Working memory skills and educational attainment: Evidence from national curriculum assessments at 7 and 14 years of age. *Appl Cogn Psychol* 18(1):1–16.
17. Mackintosh N (2011) *IQ and Human Intelligence* (Oxford Univ Press, Oxford, UK).
18. Diseth Å (2011) Self-efficacy, goal orientations and learning strategies as mediators between preceding and subsequent academic achievement. *Learn Individ Differ* 21(2):191–195.
19. Eccles JS, Roeser R, Wigfield A, Freedman-Doan C (1999) *Child Psychology: A Handbook of Contemporary Issues*, eds Balter L, Tamis-LeMonda CS (Psychology Press, Philadelphia), pp 287–317.
20. Spinath B, Spinath FM, Harlaar N, Plomin R (2006) Predicting school achievement from general cognitive ability, self-perceived ability, and intrinsic value. *Intelligence* 34(4): 363–374.
21. Zuffianò A, et al. (2013) Academic achievement: The unique contribution of self-efficacy beliefs in self-regulated learning beyond intelligence, personality traits, and self-esteem. *Learn Individ Differ* 23:158–162.
22. Downey LA, Mountstephen J, Lloyd J, Hansen K, Stough C (2008) Emotional intelligence and scholastic achievement in Australian adolescents. *Aust J Psychol* 60(1): 10–17.
23. Mavroveli S, Sánchez-Ruiz MJ (2011) Trait emotional intelligence influences on academic achievement and school behaviour. *Br J Educ Psychol* 81(Pt 1):112–134.
24. Parker JDA, et al. (2004) Academic achievement in high school: Does emotional intelligence matter? *Pers Individ Dif* 37(7):1321–1330.
25. Pope D, Roper C, Qualter P (2012) The influence of emotional intelligence on academic progress and achievement in UK university students. *Assess Eval High Educ* 37(8):907–918.
26. Briley DA, Domiteaux M, Tucker-Drob EM (2014) Achievement-relevant personality: Relations with the Big Five and validation of an efficient instrument. *Learn Individ Differ* 32:26–39.
27. Laidra K, Pullmann H, Allik J (2007) Personality and intelligence as predictors of academic achievement: A cross-sectional study from elementary to secondary school. *Pers Individ Dif* 42(3):441–451.
28. Stankov L (2013) Noncognitive predictors of intelligence and academic achievement: An important role of confidence. *Pers Individ Dif* 55(7):727–732.
29. Tough P (2013) *How Children Succeed* (Random House, London, UK).
30. Marques SC, Pais-Ribeiro JL, Lopez SJ (2011) The role of positive psychology constructs in predicting mental health and academic achievement in children and adolescents: A two-year longitudinal study. *J Happiness Stud* 12(6):1049–1062.
31. Senko C, Hulleman CS, Harackiewicz JM (2011) Achievement goal theory at the crossroads: Old controversies, current challenges, and new directions. *Educ Psychol* 46(1):26–47.
32. von Stumm S, Hell B, Chamorro-Premuzic T (2011) The hungry mind: Intellectual curiosity is the third pillar of academic performance. *Perspect Psychol Sci* 6(6):574–588.
33. Blackwell LS, Trzesniewski KH, Dweck CS (2007) Implicit theories of intelligence predict achievement across an adolescent transition: A longitudinal study and an intervention. *Child Dev* 78(1):246–263.
34. Zimmerman BJ, Bandura A, Martinez-Pons M (1992) Self-motivation for academic attainment: The role of self-efficacy beliefs and personal goal setting. *Am Educ Res J* 29(3):663–676.
35. Hinshaw SP (1992) Externalizing behavior problems and academic underachievement in childhood and adolescence: Causal relationships and underlying mechanisms. *Psychol Bull* 111(1):127–155.
36. Pingault J-B, et al. (2011) Childhood trajectories of inattention and hyperactivity and prediction of educational attainment in early adulthood: A 16-year longitudinal population-based study. *Am J Psychiatry* 168(11):1164–1170.
37. De Ridder KAA, et al. (2013) Adolescent health and high school dropout: A prospective cohort study of 9000 Norwegian adolescents (the Young-HUNT). *PLoS ONE* 8(9):e74954.
38. Hill NE, Tyson DF (2009) Parental involvement in middle school: A meta-analytic assessment of the strategies that promote achievement. *Dev Psychol* 45(3):740–763.
39. Marchant GJ, Paulson SE, Rothlisberg BA (2001) Relations of middle school students' perceptions of family and school contexts with academic achievement. *Psychol Sch* 38(6):505–519.
40. Leeson P, Ciarrochi J, Heaven PCL (2008) Cognitive ability, personality, and academic performance in adolescence. *Pers Individ Dif* 45(7):630–635.
41. Roskam I, Nils F (2007) Predicting intra-individual academic achievement trajectories of adolescents nested in class environment: Influence of motivation, implicit theory of intelligence, self-esteem and parenting. *Psychol Belg* 47(1):119–143.
42. Schmidt FL (2014) A general theoretical integrative model of individual differences in interests, abilities, personality traits, and academic and occupational achievement: A commentary on four recent articles. *Perspect Psychol Sci* 9(2):211–218.
43. Bartels M, Rietveld MJH, Van Baal GCM, Boomsma DI (2002) Heritability of educational achievement in 12-year-olds and the overlap with cognitive ability. *Twin Res* 5(6):544–553.
44. Calvin CM, et al. (2012) Multivariate genetic analyses of cognition and academic achievement from two population samples of 174,000 and 166,000 school children. *Behav Genet* 42(5):699–710.
45. Johnson W, Deary IJ, Iacono WG (2009) Genetic and environmental transactions underlying educational attainment. *Intelligence* 37(5):466–478.
46. Kovas Y, Haworth CMA, Dale PS, Plomin R (2007) The genetic and environmental origins of learning abilities and disabilities in the early school years. *Monogr Soc Res Child Dev* 72(3):vii, 1–144.
47. Petrill SA, Wilkerson B (2000) Intelligence and achievement: A behavioral genetic perspective. *Educ Psychol Rev* 12(2):185–199.
48. Thompson LA, Detterman DK, Plomin R (1991) Associations between cognitive abilities and scholastic achievement: Genetic overlap but environmental differences. *Psychol Sci* 2(3):158–165.
49. Wadsworth SJ, DeFries JC, Fulker DW, Plomin R (1995) Cognitive ability and academic achievement in the Colorado Adoption Project: A multivariate genetic analysis of parent-offspring and sibling data. *Behav Genet* 25(1):1–15.
50. Wainwright MA, Wright MJ, Geffen GM, Luciano M, Martin NG (2005) The genetic basis of academic achievement on the Queensland Core Skills Test and its shared genetic variance with IQ. *Behav Genet* 35(2):133–145.
51. Greven CU, Harlaar N, Kovas Y, Chamorro-Premuzic T, Plomin R (2009) More than just IQ: school achievement is predicted by self-perceived abilities—but for genetic rather than environmental reasons. *Psychol Sci* 20(6):753–762.
52. Gottschling J, Spengler M, Spinath B, Spinath FM (2012) The prediction of school achievement from a behavior genetic perspective: Results from the German twin study on Cognitive Ability, Self-Reported Motivation, and School Achievement (CoSMoS). *Pers Individ Dif* 53(4):381–386.
53. Spinath FM, Spinath B, Plomin R (2008) The nature and nurture of intelligence and motivation in the origins of sex differences in elementary school achievement. *Eur J Pers* 22(3):211–229.
54. Hicks BM, Johnson W, Iacono WG, McGue M (2008) Moderating effects of personality on the genetic and environmental influences of school grades helps to explain sex differences in scholastic achievement. *Eur J Pers* 22(3):247–268.
55. Johnson W, McGue M, Iacono WG (2005) Disruptive behavior and school grades: Genetic and environmental relations in 11-year-olds. *J Educ Psychol* 97(3):391–405.
56. Newsome J, Boisvert D, Wright JP (2014) Genetic and environmental influences on the co-occurrence of early academic achievement and externalizing behavior. *J Crim Justice* 42(1):45–53.
57. Polderman TJC, Boomsma DI, Bartels M, Verhulst FC, Huizink AC (2010) A systematic review of prospective studies on attention problems and academic achievement. *Acta Psychiatr Scand* 122(4):271–284.
58. Saudino KJ, Plomin R (2007) Why are hyperactivity and academic achievement related? *Child Dev* 78(3):972–986.
59. Hanscombe KB, Haworth CMA, Davis OSP, Jaffee SR, Plomin R (2011) Chaotic homes and school achievement: A twin study. *J Child Psychol Psychiatry* 52(11):1212–1220.
60. Haworth CMA, et al. (2013) Understanding the science-learning environment: A genetically sensitive approach. *Learn Individ Differ* 23:145–150.
61. Johnson W, McGue M, Iacono WG (2006) Genetic and environmental influences on academic achievement trajectories during adolescence. *Dev Psychol* 42(3):514–532.
62. Haworth CMA, Davis OSP, Plomin R (2013) Twins Early Development Study (TEDS): A genetically sensitive investigation of cognitive and behavioral development from childhood to young adulthood. *Twin Res Hum Genet* 16(1):117–125.
63. Vinkhuyzen AA, van der Sluis S, Maes HH, Posthuma D (2012) Reconsidering the heritability of intelligence in adulthood: Taking assortative mating and cultural transmission into account. *Behav Genet* 42(2):187–198.
64. Sahlberg P (2011) *Finnish Lessons: What Can the World Learn from Educational Change in Finland?* (Teachers College Press, New York).
65. Heath AC, et al. (1985) Education policy and the heritability of educational attainment. *Nature* 314(6013):734–736.
66. Price TS, et al. (2000) Infant zygosity can be assigned by parental report questionnaire data. *Twin Res* 3(3):129–133.
67. McGue M, Bouchard TJ, Jr (1984) Adjustment of twin data for the effects of age and sex. *Behav Genet* 14(4):325–343.
68. Lehmann E (1975) *Nonparametric Statistical Methods Based on Ranks* (Holden-Day, San Francisco).
69. Van Der Waerden BL (1975) On the sources of my book Moderne Algebra. *Hist Math* 2(1):31–40.
70. Rijsdijk FV, Sham PC (2002) Analytic approaches to twin data using structural equation models. *Brief Bioinform* 3(2):119–133.
71. Boker S, et al. (2011) OpenMx: An open source extended structural equation modeling framework. *Psychometrika* 76(2):306–317.

Supporting Information

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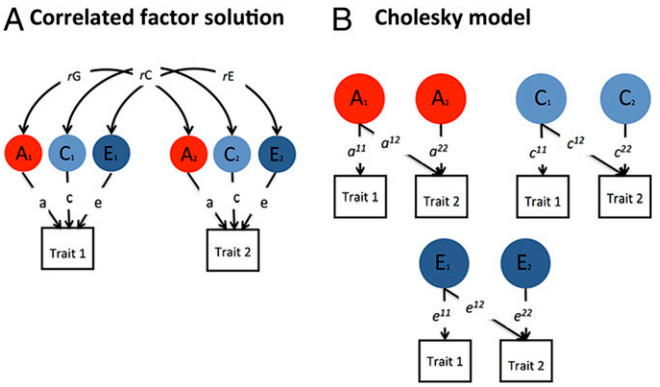

Fig. S1. Bivariate model of additive genetic (A), shared environmental (C), and nonshared environmental (E) contributions to the correlations between traits. Two algebraically equivalent representations of the bivariate model are shown: (*A*) correlated factor solution of genetic correlation (*r*G), shared environmental correlation (*r*C), and nonshared environmental correlation (*r*E) and (*B*) Cholesky decomposition.

Table S1. Descriptive statistics

	N	Whole sample	Male	Female	MZm	DZm	MZf	DZf	DZos	Sex	Zygosity	Sex × zygosity	R^2
GCSE core subjects mean grade	12,103	8.91 (1.23)	8.86 (1.23)	8.96 (1.21)	8.83 (1.23)	8.90 (1.21)	8.95 (1.16)	8.95 (1.24)	8.93 (1.24)	20.26*	1.91	0.13	<0.01

GCSE core subjects mean grade have a maximum of 11 and a minimum of 4, representing grades A* to G. *n* = sample size after exclusions (individuals). ANOVA performed (one randomly selected twin per pair) to test main and interaction effects of sex and zygosity: results = *F* statistic. R^2 = proportion of variance explained by sex, zygosity, and their interaction. DZ, dizygotic; f, female; m, male; MZ, monozygotic; os, opposite sex. *$P < 0.01$.

Table S2. Twin correlations for all nine predictors and GCSE and cross-correlations for all nine predictors with GCSE

	Twin correlations within trait		Cross-correlations with GCSE	
	MZ	DZ	MZ	DZ
GCSE	0.85 (0.83–0.87) *n* = 2115	0.54 (0.51–0.56) *n* = 3794		
Intelligence	0.60 (0.55–0.66) *n* = 760	0.32 (0.27–0.38) *n* = 1182	0.53 (0.47–0.59) *n* = 752	0.29 (0.23–0.33) *n* = 1209
Self-efficacy	0.62 (0.54–0.64) *n* = 830	0.40 (0.37–0.47) *n* = 1326	0.40 (0.33–0.45) *n* = 807	0.21 (0.16–0.26) *n* = 1316
School environment	0.45 (0.39–0.51) *n* = 826	0.29 (0.24–0.34) *n* = 1322	0.32 (0.25–0.38) *n* = 804	0.16 (0.10–0.21) *n* = 1314
Home environment	0.54 (0.50–0.62) *n* = 786	0.33 (0.28–0.39) *n* = 1233	0.19 (0.11–0.25) *n* = 766	0.12 (0.07–0.17) *n* = 1244
Personality	0.64 (0.42–0.55) *n* = 764	0.21 (0.15–0.26) *n* = 1188	0.25 (0.18–0.32) *n* = 752	0.07 (0.01–0.16) *n* = 1203
Well-being	0.54 (0.48–0.62) *n* = 704	0.35 (0.29–0.40) *n* = 1106	0.25 (0.18–0.34) *n* = 679	0.14 (0.08–0.19) *n* = 1091
Parent-reported behavior problems	0.87 (0.87–0.91) *n* = 1661	0.63 (0.60–0.65) *n* = 1963	0.28 (0.23–0.33) *n* = 1460	0.16 (0.12–0.19) *n* = 2568
Child-reported behavior problems	0.48 (0.44–0.53) *n* = 1639	0.22 (0.18–0.25) *n* = 1923	0.19 (0.15–0.25) *n* = 1448	0.10 (0.06–0.14) *n* = 2547
Health	0.61 (0.57–0.65) *n* = 1237	0.36 (0.33–0.40) *n* = 2286	0.10 (0.04–0.16) *n* = 1103	0.06 (0.01–0.10) *n* = 1992

DZ, dizygotic; MZ, monozygotic.

Table S3. Model fitting estimates (and 95% CIs) for additive genetic (A), shared environment (C), and nonshared environment (E) components of variance for GCSE and nine predictors

	Variance components (95% CIs)		
	A	C	E
GCSE	0.62 (0.58–0.67)	0.26 (0.21–0.30)	0.12 (0.11–0.13)
Intelligence	0.58 (0.46–0.63)	0.04 (0.01–0.13)	0.39 (0.35–0.43)
Self-efficacy	0.40 (0.30–0.52)	0.21 (0.12–0.30)	0.38 (0.34–0.42)
School environment	0.45 (0.33–0.53)	0.11 (0.05–0.20)	0.44 (0.40–0.49)
Home environment	0.46 (0.33–0.55)	0.09 (0.03–0.20)	0.44 (0.40–0.49)
Personality	0.46 (0.36–0.51)	0.00 (0.00–0.08)	0.53 (0.49–0.58)
Well-being	0.35 (0.22–0.49)	0.17 (0.06–0.28)	0.47 (0.43–0.52)
Parent-reported behavior problems	0.53 (0.49–0.57)	0.36 (0.32–0.40)	0.11 (0.10–0.12)
Child-reported behavior problems	0.48 (0.42–0.51)	0.00 (0.00–0.04)	0.52 (0.49–0.56)
Health	0.48 (0.39–0.57)	0.13 (0.11–0.20)	0.39 (0.36–0.42)

Table S4. Phenotypic correlation matrix between GCSE and nine predictors (with 95% CIs)

	GCSE	Intelligence	Self-efficacy	School environment	Home environment	Personality	Well-being	Parent-reported behavior problems	Child-reported behavior problems	Health
GCSE	1.00									
Intelligence	0.58 (0.56–0.60)	1.00								
Self-efficacy	0.49 (0.46–0.51)	0.35 (0.33–0.38)	1.00							
School environment	0.34 (0.32–0.37)	0.24 (0.21–0.27)	0.46 (0.43–0.48)	1.00						
Home environment	0.17 (0.14–0.20)	0.13 (0.10–0.16)	0.30 (0.28–0.33)	0.52 (0.50–0.55)	1.00					
Personality	0.28 (0.25–0.31)	0.18 (0.15–0.21)	0.42 (0.39–0.45)	0.39 (0.37–0.42)	0.38 (0.36–0.41)	1.00				
Well-being	0.26 (0.23–0.28)	0.17 (0.14–0.20)	0.41 (0.38–0.44)	0.54 (0.52–0.56)	0.61 (0.59–0.63)	0.51 (0.49–0.54)	1.00			
Parent-reported behavior problems	0.33 (0.31–0.35)	0.26 (0.22–0.29)	0.26 (0.22–0.29)	0.29 (0.26–0.33)	0.31 (0.27–0.35)	0.22 (0.18–0.26)	0.38 (0.35–0.41)	1.00		
Child-reported behavior problems	0.25 (0.23–0.27)	0.18 (0.15–0.21)	0.36 (0.33–0.38)	0.39 (0.37–0.42)	0.42 (0.39–0.45)	0.30 (0.27–0.33)	0.54 (0.52–0.56)	0.38 (0.36–0.40)	1.00	
Health	0.08 (0.05–0.12)	0.07 (0.03–0.11)	0.14 (0.10–0.18)	0.23 (0.20–0.27)	0.26 (0.23–0.30)	0.08 (0.04–0.12)	0.32 (0.28–0.35)	0.17 (0.15–0.20)	0.42 (0.40–0.44)	1.00

Table S5. Bivariate model-fitting estimates (and CIs)

	Proportion of phenotypic correlation explained by A, C, and E (95% CIs)		
	A	C	E
Intelligence-GCSE	0.75 (0.63–0.86)	0.15 (0.06–0.26)	0.10 (0.07–0.13)
Self-efficacy-GCSE	0.64 (0.51–0.77)	0.21 (0.09–0.33)	0.15 (0.11–0.18)
School environment-GCSE	0.59 (0.37–0.80)	0.31 (0.12–0.50)	0.10 (0.04–0.16)
Home environment-GCSE	0.08 (-0.35–0.50)	0.81 (0.44–1.18)	0.10 (-0.01–0.26)
Personality-GCSE	0.92 (0.66–1.17)	(-0.05) (-0.27–0.17)	0.14 (0.06–0.21)
Well-being-GCSE	0.53 (0.22–0.85)	0.34 (0.06–0.61)	0.13 (0.04–0.21)
Parent-reported behavior problems-GCSE	0.81 (0.70–0.93)	0.11(-4.25E-03–0.22)	0.07 (0.06–0.10)
Child-reported behavior problems-GCSE	0.89 (0.70–1.08)	(-0.01) (-0.18–0.15)	0.12 (0.12–0.12)
Health-GCSE	0.71 (0.55–1.43)	0.28 (-0.37–0.85)	0.01 (-0.17–0.19)

Bivariate estimates (and 95% CIs) for additive genetic (A), shared environmental (C), and nonshared environmental (E) contributions to the correlations between GCSE and nine predictors.

Table S6. Bivariate model-fitting results of the extent to which the heritability of GCSE can be explained by the nine predictors (95% CIs)

	Heritability of GCSE	
	Shared	Independent
Intelligence	0.31 (0.22–0.37)	0.31 (0.25–0.41)
Self-efficacy	0.23 (0.15–0.33)	0.39 (0.15–0.32)
School environment	0.12 (0.05–0.25)	0.50 (0.37–0.59)
Home environment	0.00 (6E-18–0.02)	0.63 (6E-01–0.02)
Personality	0.13 (7E-02–0.22)	0.50 (4E-01–0.58)
Well-being	0.05 (0.01–0.12)	0.58 (0.50–0.65)
Parent-reported behavior problems	0.13 (0.13–0.16)	0.50 (0.44–0.54)
Child-reported behavior problems	1E-01 (6E-02–0.15)	5E-01 (6E-02–0.15)
Health	0.01 (5E-05–0.03)	0.62 (6E-01–0.67)

The graph displays the decomposition of heritability of GCSE into shared variance accounted for by genetic influences on the respective domain and independent variance, which is residual (i.e., unaccounted by the respective domain). As an example, for intelligence, the genetic loading of 0.31 on GCSE, estimated for the squared path a^{21} (Fig. 4), indicates that genetic influences on intelligence accounted for ~50% of the heritability of GCSE.

Table S7. Phenotypic multivariate Cholesky and genetic multivariate Cholesky model-fitting estimates (and 95% CIs) for all nine predictors

	Phenotypic variance of GCSE		Heritability of GCSE	
Predictors of GCSE	Shared	Independent	Shared	Independent
Intelligence	0.34 (0.30–0.38)	0.66 (0.63–0.70)	0.31 (0.25–0.41)	0.31 (0.22–0.37)
Eight noncognitive predictors	0.28	0.72 (0.69–0.76)	0.30	0.31 (3E-16–0.38)
Eight noncognitive predictors and intelligence	0.45	0.55 (0.52–0.58)	0.45	0.15 (6E-16–0.24)

Decomposition of the phenotypic variance and of heritability of GCSE into shared variance accounted for by phenotypic or genetic influences on the respective predictors and independent variance, which is residual (i.e., unaccounted by the respective predictors). As an example, the eight noncognitive predictors alone account for 28% (0.28/1.0) of the phenotypic variance in GCSE and 49% (0.30/0.61) of the heritability of GCSE, leaving 72% (0.72/1.0) phenotypic and 51% (0.31/0.61) residual GCSE heritability. For the models with multiple predictors (i.e., eight noncognitive predictors and intelligence), the shared variance represents the sum of the GCSE variance/heritability explained by all predictors together. Hence, CIs cannot be computed for these summed estimates, but only for the independent GCSE variance/heritability or single predictors (i.e., intelligence).

Table S8. Genetic (*r*G) correlation matrices between the GCSE composite and the nine predictor composites (with 95% CIs)

	Genetic correlations (*r*G)									
	GCSE	Intelligence	Self-efficacy	School environment	Home environment	Personality	Well-being	Parent-reported behavior problems	Child-reported behavior problems	Health
GCSE	1.00									
Intelligence	0.76 (0.68–0.84)	1.00								
Self-efficacy	0.62 (0.52–0.73)	0.64 (0.49–0.81)	1.00							
School environment	0.43 (0.30–0.60)	0.40 (0.22–0.61)	0.62 (0.43–0.81)	1.00						
Home environment	0.10 (−0.02–0.21)	0.08 (−0.09–0.23)	0.25 (0.06–0.41)	0.53 (0.35–0.69)	1.00					
Personality	0.47 (0.35–0.61)	0.29 (0.15–0.43)	0.58 (0.42–0.72)	0.63 (0.46–0.82)	0.55 (0.41–0.69)	1.00				
Well-being	0.29 (0.17–0.42)	0.24 (0.07–0.41)	0.47 (0.29–0.65)	0.65 (0.49–0.83)	0.78 (0.66–0.89)	0.68 (0.55–0.83)	1.00			
Parent-reported behavior problems	0.46 (0.40–0.51)	0.36 (0.24–0.48)	0.43 (0.30–0.56)	0.44 (0.27–0.63)	0.35 (0.22–0.49)	0.30 (0.16–0.45)	0.35 (0.23–0.48)	1.00		
Child-reported behavior problems	0.39 (0.30–0.48)	0.25 (0.11–0.39)	0.57 (0.43–0.75)	0.70 (0.55–0.93)	0.55 (0.43–0.67)	0.45 (0.32–0.58)	0.74 (0.63–0.84)	0.45 (0.38–0.53)	1.00	
Health	0.09 (−0.01–0.19)	0.09 (−0.08–0.29)	0.13 (−0.09–0.36)	0.28 (0.01–0.56)	0.25 (0.06–0.46)	0.12 (−0.07–0.35)	0.38 (0.19–0.59)	0.15 (0.06–0.23)	0.54 (0.44–0.66)	1.00

Derived from the standardized multivariate Cholesky (correlated factors solution).

Table S9. Shared environmental (*r*C) correlation matrices between the GCSE composite and the nine predictor composites (with 95% CIs)

	Shared environment correlations (*r*C)									
	GCSE	Intelligence	Self-efficacy	School environment	Home environment	Personality	Well-being	Parent-reported behavior problems	Child-reported behavior problems	Health
GCSE	1.00									
Intelligence	0.65 (0.40–0.87)	1.00								
Self-efficacy	0.47 (0.28–0.65)	(−0.09) (−0.48–0.27)	1.00							
School environment	0.62 (0.32–0.98)	0.28 (−0.27–0.80)	0.55 (0.19–0.89)	1.00						
Home environment	0.66 (0.34–0.92)	0.49 (−0.04–0.86)	0.66 (0.31–0.90)	0.85 (0.39–1.00)	1.00					
Personality	(−0.03) (0.94–0.86)	0.09 (−0.97–0.91)	0.48 (−0.76–0.99)	0.48 (−0.81–0.99)	0.67 (−0.58–1.00)	1.00				
Well-being	0.46 (0.18–0.72)	0.13 (−0.31–0.63)	0.40 (0.05–0.67)	0.81 (0.39–0.98)	0.75 (0.40–0.95)	0.49 (−0.56–1.00)	1.00			
Parent-reported behavior problems	0.16 (0.04–0.27)	0.25 (−0.05–0.54)	0.09 (−0.13–0.28)	0.41 (0.12–0.76)	0.57 (0.28–0.82)	0.61 (−0.19–1.00)	0.79 (0.56–0.96)	1.00		
Child-reported behavior problems	0.19 (−0.18–0.54)	0.12 (−0.45–0.68)	0.52 (0.01–0.81)	0.63 (−0.04–0.95)	0.83 (0.42–0.98)	0.81 (−0.26–1.00)	0.80 (0.41–0.98)	0.80 (0.52–0.99)	1.00	
Health	0.17 (−0.09–0.45)	0.32 (−0.26–0.81)	0.45 (0.01–0.86)	0.43 (−0.28–0.91)	0.74 (0.13–0.98)	0.65 (−0.57–1.00)	0.33 (−0.16–0.76)	0.38 (0.20–0.62)	0.77 (0.25–0.98)	1.00

Derived from the standardized multivariate Cholesky (correlated factors solution).

Table S10. Nonshared environmental (*r*E) correlation matrices between the GCSE composite and the nine predictor composites (with 95% CIs)

	Nonshared environmental correlations (*r*E)									
	GCSE	Intelligence	Self-efficacy	School environment	Home environment	Personality	Well-being	Parent-reported behavior problems	Child-reported behavior problems	Health
GCSE	1.00									
Intelligence	0.24 (0.17–0.31)	1.00								
Self-efficacy	0.31 (0.24–0.37)	0.21 (0.15–0.28)	1.00							
School environment	0.13 (0.07–0.20)	0.09 (0.03–0.16)	0.31 (0.25–0.36)	1.00						
Home environment	0.05 (−0.02–0.13)	0.10 (0.03–0.17)	0.23 (0.17–0.30)	0.45 (0.40–0.50)	1.00					
Personality	0.14 (0.06–0.21)	0.08 (0.20–0.15)	0.33 (0.27–0.39)	0.24 (0.18–0.30)	0.23 (0.16–0.29)	1.00				
Well-being	0.10 (0.03–0.17)	0.12 (0.05–0.18)	0.36 (0.30–0.42)	0.39 (0.33–0.44)	0.43 (0.37–0.48)	0.41 (0.35–0.46)	1.00			
Parent-reported behavior problems	0.20 (0.15–0.25)	0.12 (0.04–0.20)	0.20 (0.12–0.28)	0.09 (0.02–0.17)	0.13 (0.05–0.22)	0.09 (0.01–0.17)	0.19 (0.11–0.26)	1.00		
Child-reported behavior problems	0.12 (0.06–0.17)	0.13 (0.06–0.20)	0.16 (0.09–0.22)	0.15 (0.09–0.22)	0.26 (0.19–0.32)	0.15 (0.06–0.24)	0.35 (0.30–0.41)	0.27 (0.22–0.31)	1.00	
Health	0.00 (−0.06–0.06)	(−0.04) (−0.13–0.05)	0.02 (−0.08–0.11)	0.14 (0.05–0.23)	0.15 (0.06–0.24)	(−0.02) (−0.11–0.07)	0.25 (0.17–0.33)	0.07 (0.02–0.12)	0.27 (0.22–0.31)	1.00

Derived from the standardized multivariate Cholesky (correlated factors solution).