Intelligence 56 (2016) 10–15

Contents lists available at ScienceDirect

Intelligence

When does socioeconomic status (SES) moderate the heritability of IQ? No evidence for g × SES interaction for IQ in a representative sample of 1176 Australian adolescent twin pairs

Timothy C. Bates [a,*], Narelle K. Hansell [b], Nicholas G. Martin [b,*], Margaret J. Wright [b]

[a] The University of Edinburgh, Edinburgh, UK
[b] Queensland Institute of Medical Research, Brisbane, Australia

ARTICLE INFO

Article history:
Received 27 November 2015
Received in revised form 11 February 2016
Accepted 12 February 2016
Available online xxxx

Keywords:
IQ
Child development
Cognitive development
G × SES
Gene–environment interaction

ABSTRACT

Bioecological theory predicts that cognitive ability is more heritable among those raised in higher socioeconomic status (SES) families. However, the mechanism of this effect is unclear, and the effect may not be universal. We tested for gene × SES interaction effects on Full-scale IQ in 2307 adolescent Australian twins (mean age 16.2 years). Mean IQ scores were modestly higher among those from higher SES backgrounds, but the magnitude of genetic influences on IQ was uniformly high across the range of SES. Research identifying the conditions under which expressed genetic potential can become decoupled from parental SES, as seen here, is needed. We speculate that school provision may be key.

1. Introduction

Mainstream models of cognitive development include not only main effects of genes and of environments, but also clearly specified relations between genes and environments, i.e. a "*bioecological*" approach (Bronfenbrenner & Ceci, 1994). Testing these mechanisms is central to understanding cognitive development and requires studies of both genes and environments. In a now classic study, Rowe, Jacobson, and Van den Oord (1999) reported that IQ differences among US children raised in high socioeconomic status (SES) families largely reflected genetic factors, but that differences in family environment were the predominant causes of variance among children raised in low SES families: the so-called Scarr–Rowe effect. Gene × SES interactions have now been reported in US infants (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011) and children (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), adolescents (Harden, Turkheimer, & Loehlin, 2007) and mature adults (Bates, Lewis, & Weiss, 2013). It is important to note that a number of non-significant findings have been reported in the US (Grant et al., 2010; Soden-Hensler, 2012) and null findings from outside the US, e.g., in the UK (Hanscombe et al., 2012) and Netherlands (van der Sluis, Willemsen, de Geus, Boomsma, & Posthuma, 2008). The moderation of genetic expression in intelligence may, then, be smaller in size than early estimates suggested, or even be absent in some populations Tucker-Drob and Bates (2015).

These methods can detect not only gene × SES interactions, but also interactions of SES with unique and with shared-environment variance. No study has supported interactions of SES with unique environment. Significant C × SES interactions have been reported only in the youngest samples (Tucker-Drob et al., 2011; Turkheimer et al., 2003). Studies of adolescents (Harden et al., 2007) and mature adults (Bates et al., 2013), however, indicate no evidence of C × SES interactions. Developmentally, the heritability of intelligence increases from infancy onwards (Haworth et al., 2010). It may be that C × SES are present in at least some groups, but that these wash out along with C itself over time. Importantly, heritability asymptotes at higher levels in high (compared to low) socioeconomic status groups (Tucker-Drob, Briley, & Harden, 2013). We suggested that this combination of increased genetic variance, increased means, and increased heritability which accompany higher SES reflects a "Matthew" effect — an effect where initial behavioral advantages grow over time (Stanovich, 1986). For IQ, a genetic variant of this effect which we term a "*Genomic Matthew effect*" occurs when environments influence IQ development such that the genetically rich profit *more* from enriched environments than do those with reduced genetic potential(s). This implies that additional resources, often coupled with parental SES, act not to minimize gaps between children (Ceci & Papierno, 2005), but to amplify them (Bates et al., 2013).

A lack of research on gene × environment interactions, however, means that it is far from clear how universal are gene × SES effects on

* Corresponding authors.
E-mail address: tim.bates@ed.ac.uk (T.C. Bates).

http://dx.doi.org/10.1016/j.intell.2016.02.003
0160-2896/

cognition. It has been suggested that failures to find gene × SES effects are due to either sample selection — for instance twin pairs pre-selected for passing IQ criteria for admission to the US Armed Forces (Grant et al., 2010) or low sample size. This cannot explain, however, unpublished data in a representative sample of Florida children which failed to find gene × SES interaction (Soden-Hensler, 2012), although a significant gene interaction with a measure of school quality based on number of deprived students in the school was reported (Hart, Soden, Johnson, Schatschneider, & Taylor, 2013). Relatively few studies have been undertaken outside the US, but evidence for gene × SES interactions on the heritability of IQ have largely been negative. In particular, in the largest study to date – 8716 twin pairs of the UK TEDS study – gene × SES interactions were present in only one of multiple measures of ability from ages 2 through 14, and then in the opposite direction to that predicted by the bioecological model (Hanscombe et al., 2012). Research in the Netherlands also failed to find evidence for an interaction of a binary measure of parental education with intelligence in 370 adult twins and siblings aged 36–65 (van der Sluis et al., 2008). However the small n and the conservative nature of the gene × SES design (van der Sluis, Posthuma, & Dolan, 2012) gave this study low power to detect G × E effects.

Testing to date, then, suggests no gene × SES interactions outside US samples. Such national differences in the moderation of the heritability of IQ may provide insights into the mechanisms via which environments restrict or promote phenotypic ability. We previously predicted that, "*in societies where provision of intellectual resources is universal* (*independent of income*)*…no G × SES effects* [*will be observed*]" (Bates et al., 2013). For such ideas to be tested, analyses of heritability of IQ must be conducted in different nations and educational systems to assess the circumstances fostering the expression of talent. Here, we extend the range of nations in which gene × SES interactions have been tested to a large representative sample of Australian adolescent twins. Reflecting large-scale studies (Sackett et al., 2012), we predicted that higher SES would be weakly associated with higher intelligence. In our second model, we examined whether main effects of genetic (A), between-family environment (C), and unique environment variance (E) are moderated by SES.

It is important in running and interpreting a G × SES study to understand the power of the analyses in order to interpret a null result (Hanscombe et al., 2012; van der Sluis et al., 2012). Power calculations for the present study were undertaken in R (code available as a public gist on Github https://goo.gl/QypvQS).

Our goal was to estimate the degree to which genetic influences on IQ present in the sample were moderated with respect to SES level. In the simulations, magnitude of change in heritability of IQ moving from 2-SDs below the mean in SES to 2-SDs above the mean SES was varied. SES was simulated as a normally distribute variable so as not to over estimate the numbers of families at levels of either side of the mean. Because the sample consisted of adolescents, in line with a range of studies, we simulated data with no C, substantial additive genetic effects at the mean level of SES, and with a modest residual E variance equal across the sample (Deary, Spinath, & Bates, 2006). To estimate power, we conducted two types of test: Power to detect a loss of fit from the free model to on in which all moderation was dropped (a′, c′, and e′); and 1-degree of freedom tests comparing a base model with C, c′, and e′ fixed at zero to a model in which a′ was also set to zero. All simulations consisted of 1000 runs, with nominal p-value set to .05.

When simulating a large effect (mean 'a' of 0.5 (falling to 0.3 at −2 SDs below the mean SES, and rising to 0.7 at +2 SDs, i.e., comparable but somewhat smaller than that reported by Turkheimer et al., 2003) power to detect loss of fit on dropping a′ was >99%, power remained at 98% for much smaller swings of a across the range of SES (mean 'a' of 0.5 and a min and max 0.4 and 0.6 respectively), and was at 84.9% given a mean path-coefficient 'a' of 0.5, swinging from a minimum of 0.43 at −2 SDs of SES, and rising to 0.57 at +SD above mean SES, which we classified as a small difference in heritability across the range of SES. This suggests we had excellent power to detect moderation at levels reported previously. Power to detect the presence of moderation (i.e. dropping all of a′, c′, and e′) was 97% given mean 'a' of 0.5 (min and max 0.35 and 0.65 respectively). The sample thus had good power to detect effects at levels compatible with proposed dependencies of IQ on SES.

2. Method

2.1. Participants

The sample consisted of all 2307 twins of the Brisbane Adolescent Twin Study (Wright & Martin, 2004) for whom the ability tests and the socioeconomic status definition variable were available. Females (n = 1233) had a mean age of 16.28 years (SD = 0.46) and males (n = 1119) a mean age of 16.24 years (SD = 0.43). Broken down by zygosity there were 244 pairs of MZ female twins, 211 MZ male pairs, 201 and 177 DZ female and male pairs respectively and 343 pairs of DZ opposite-sex twins. Exclusion criteria for entry to this cohort were a significant head injury, neurological or psychiatric illness, substance dependence, or chronic use of medications with central nervous system effects. Twins with these characteristics were, therefore unavailable for study but constitute a minority of the population. Written, informed consent was obtained from all participants and a parent or guardian for those aged less than 18 years and the study was approved by the Human Research Ethics Committee at QIMR. Zygosity was determined from DNA using a commercial kit (AmpFlSTR Profiler Plus Amplification Kit, ABI). This was later confirmed for >60% of the sample genotyped on the 610 K Illumina genome-wide SNP platform (Medland et al., 2009).

2.2. Measures

Full-scale Intelligence was assessed based on five IQ subtests of the Multidimensional Aptitude Battery (MAB: Jackson, 1984, 1998). These tests were modeled on the Wechsler scales and consisted of three verbal subtests (information, vocabulary, arithmetic) and two performance subtests (spatial, object assembly). All tests were computer administered with a 7-minute time limit. The subtests showed a significant positive manifold of correlations in the total sample (average 0.477, ranging from a maximum of 0.670 (between information and vocabulary) down to 0.278 between spatial and vocabulary) in line with the existence of a higher-order general factor underlying cognitive abilities (Deary, 2012). Childhood SES was assessed using the Australian Socioeconomic Index 2006 (AUSEI06) occupational status scale (McMillan, Beavis, & Jones, 2009). For each individual, AUSEI06 was ascertained for each parent, and childhood status for each twin pair was set to the maximum of their maternal and paternal values.

2.3. Analyses

To avoid shared-age inflating twin similarities and family-differences (McGue & Bouchard, 1984), intelligence scores were residualized for age, age^2, and sex. Interactions were tested in a G × E analysis (Purcell, 2002) implemented using the OpenMx 2 (Boker et al., 2011; Neale et al., 2015) and umx (Bates, Neale, & Maes, under review) packages under R (R Core Team, 2014). As in the standard ACE twin model (Neale & Maes, 1996) with latent variables representing additive genetic (A) and between-family (C), and unique (E) environmental variance components, and allows for the addition of interaction effects on these paths (see Fig. 1). The covariance of additive genetic effect within twin pairs was set to 1.0 in MZ twins, reflecting their near-complete genomic sharing and to 0.5 in DZ twins, reflecting the fact that these twins share (on average) half of their additive genetic inheritance. Between-family environment covariance was set to 1.0 to model variance shared at the family level. Covariance for unique environment effects was, of course set to zero, modeling influences unique

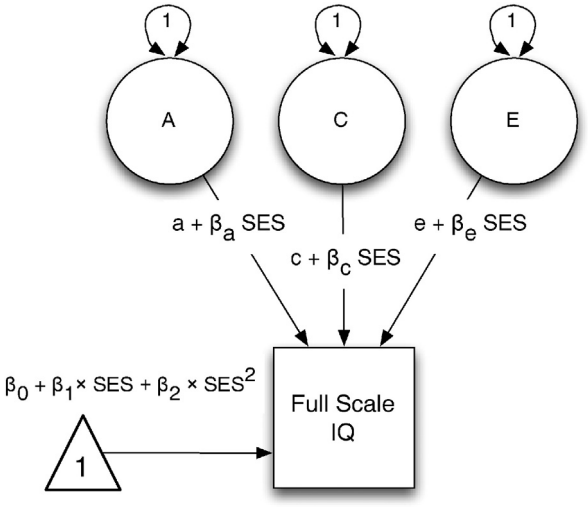

Fig. 1. Full SES interaction model for intelligence. Note: SES is Parental Socioeconomic Status. A = additive genetic effects, C = shared-environmental effects; E = unshared environmental effects. a, c, and e are genetic, shared-environmental, and non-shared environmental effects respectively. β_a, β_c, and β_e are corresponding SES-moderation effects. Mean effect (triangle symbol and path function) controls SES–IQ covariance, including shared genetic effects on SES & IQ. Double-headed arrows represent variances). One twin-member shown: For details on modeling, see Method.

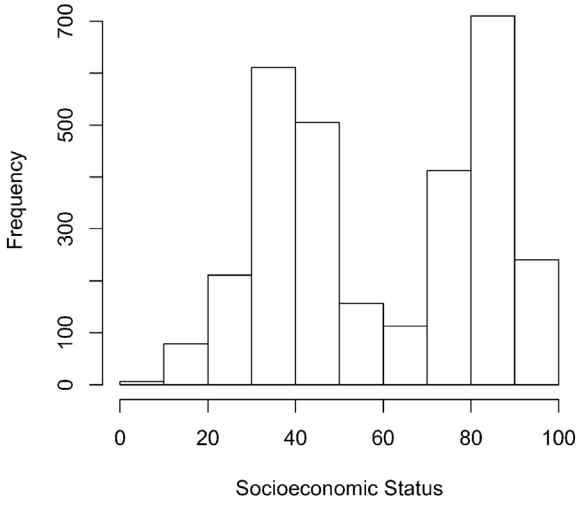

Fig. 2. Histogram of Socioeconomic Status.

to each twin – including measurement error – which make family members different from each other.

In the G × SES model, these A, C, and E main effects are supplemented by linear (β_1) and quadratic (β_2) effects of measured individual-level SES on intelligence means and also on the A, C, and E variance components, allowing both main and SES-moderated effects on genetic, between-family, and unique-environmental influence (see Fig. 1).

The net effect of each latent variance source is given by the sum of the main effects of a, c, & e, and the $\beta_a \times$ SES, $\beta_c \times$ SES, and $\beta_e \times$ SES moderated effects. A standardized solution of this model provides information regarding the relative values of the variance components for a given value of the moderator. However, this heritability-style standardization loses considerable information regarding the source of variance. As advocated by Purcell (2002), therefore we also report and focus on the unstandardized results.

3. Results

Mean IQ in the sample was 111.6. MZ and DZ correlations for IQ suggested significant, mostly additive, heritability ($r = 0.84$ and 0.52, respectively). As an initial test for the association of parental SES with adolescent intelligence, a regression model was constructed with IQ as the dependent variable and parental SES as the independent variable. Parental SES scores ranged from 6.2 to 97.9 ($M = 59.65$, SD = 24.0, scale range 0–100). SES was distributed bi-modally (see Fig. 2). This reflected the significant gap in educational attainment, prestige, and salary between a high-frequency cluster of professional occupations (primarily teaching and nursing) and the next significant cluster below this group in terms of SES. To take into account the clustering of the data (individuals nested within twin pairs violate the assumption of independence), multilevel models using lme4 (D. Bates, Maechler, & Bolker, 2011) were used to provide parameter estimates. Controlling family membership as a random effect, parental SES and offspring IQ-scores showed a moderate size relationship ($\beta = 0.27$, SE = 0.025: see Fig. 3), and a χ^2 likelihood ratio test demonstrated that including the fixed effect SES explained significantly greater variation in IQ than did a model excluding this variable ($\chi^2(1) = 103.2$, $p < 2.2 \times 10^{-16}$).

Because interactions have been reported as being stronger for performance than for verbal IQ (Turkheimer et al., 2003), we also tested these components of IQ separately. The results very closely resembled those for general ability: As with the general ability composite, all interaction effects could be dropped without significant loss of fit for both performance IQ ($\chi^2(1) < .001$, p = 1.000), and for Verbal IQ ($\chi^2(1) < .001$, p = 1.000). In all cases, the preferred model by AIC was that which dropped shared environment along with all moderation terms. In no case could additive genetic effects be dropped without dramatic loss of fit.

We next tested the gene × SES model of additive genetic, between and within-family environment ACE effects, including socioeconomic status interactions. The moderating effects of SES on each of these components were tested using χ^2 tests of the effects of dropping these parameters from the model. The standardized parameter estimates (and 95% confidence intervals) for the full g × SES Model of Adolescent IQ are shown in Table 1. Fig. 4 shows graphically the estimated values of additive genetic, shared environmental and unique environmental effects at each level of parental SES, presented in both unstandardized and standardized forms.

Dropping the effect of SES as a moderator of heritability did not significantly reduce model fit, $\chi^2(1) = .393$, p = 0.53, indicating no significant gene × SES interaction effect in the data (see Table 2, model 2). We

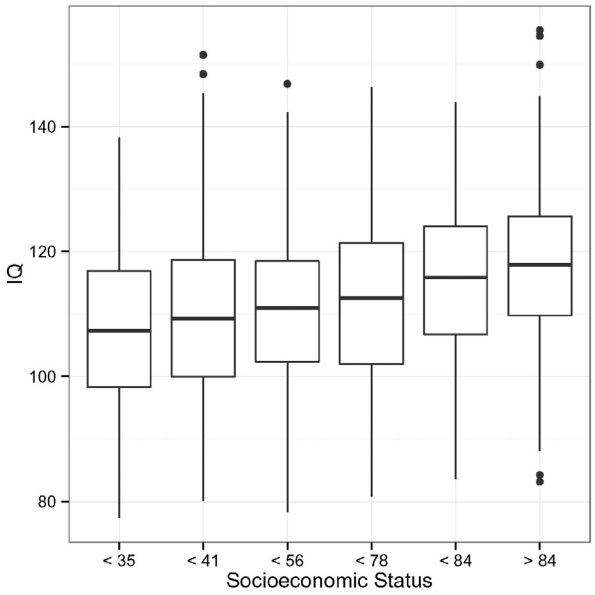

Fig. 3. Box plot of intelligence by Socioeconomic Status quantile, controlling for non-independence of twins in a pair.

Table 1
Standardized parameter estimates (and 95% confidence intervals) for the full g × SES model of adolescent IQ (significant effects in **bold**).

Parameter	Lower bound	Estimate	Upper bound
Additive genetic (A)	**0.794**	**0.863**	**0.904**
Shared environment (C)	−0.340	0.080	0.340
Unique environment (E)	**0.387**	**0.412**	**0.441**
A × SES moderation	−0.052	0.000	0.056
C × SES moderation	−0.254	−0.028	0.254
E × SES moderation	−0.053	−0.025	0.002
Mean	−0.160	−0.079	0.002
Linear means moderation	**0.222**	**0.271**	**0.320**
Quadratic means moderation	−0.003	0.063	0.129

Note: Parameters standardized with respect to the variances of A, C, and E but not with respect to the variance of the product term itself (Marsh, Wen, & Hau, 2004).

Table 2
Fit Statistics for Full G × SES and nested sub-models.

Model	ep	AIC	Δ−2LL	Δdf	p-value
1: Fully moderated model	9	7408.8	–	–	–
2: Drop A × SES interaction	8	7407.2	0.393	1	0.53
3: Drop A, C, & E × SES interactions	6	7405.0	2.194	3	0.53
4: Moderation, but no C main effect	8	7407.4	0.543	1	0.46
5: AE model (No C, no moderation)*	**5**	**7404.4**	**3.572**	**4**	**0.47**
6: Drop A from model 5	4	7702.0	303.19	5	2.07×10^{-63}

Note. *ep* = number of estimated parameters; *AIC* = Akaike Information Criterion; Δ−2*LL* = change in −2 × log-likelihood; Δ*df* = change in degrees of freedom. All comparisons are with respect to model 1: smaller p-values indicate worse fit.
* Model 5 is preferred according to AIC.

next tested the hypothesis that all interaction effects could be dropped (Table 2, model 3). Setting all three moderation effects to zero had negligible effect on model fit with respect to the full model ($\chi^2(3) = 2.194$, $p = 0.53$), reducing the model to one of simple main effects only. This model could be further reduced by setting the shared environmental effect (C) to zero — again, with negligible loss of fit with respect to the full model (see Table 2, model 5). This change was also insignificant on a 1 degree of freedom test against model 3 ($\chi^2(1) = 3.57$, $p = 0.47$), suggesting that familial variation in adolescent IQ in this population is well accounted for by additive genetics (attempting to drop the additive effect of genes from the caused a significant loss of fit ($\chi^2(5) = 303.2$, $p = <.001$) with unique environmental effects including measurement error accounting for the remaining variance. In this final model (model 5 in Table 2), the genetic variance was 0.83, (95% CI: .79, .86), with unique environment variance estimated at 0.16 (95% CI .14, .20).

4. Discussion

We tested for evidence of gene × SES interactions in a large sample of adolescent twins with a reliable measure of IQ and a widely validated indicator of parental SES. No significant support for interactions, whether genetic or environmental, was found. Whereas deprivation is observed to correlate with reductions in variance in ability (Kennedy, Vanderiet, & White, 1963), at least in this sample, family status does not appear to be a significant moderator of Australian adolescent's cognitive development.

The lack of evidence for significant moderation of gene expression in this sample provides additional insight into the circumstances under which gene interactions with SES lead to differences in intelligence. These data indicate that parental SES is not an obligate moderator of genetic variance in IQ. Along with other results (Hanscombe et al., 2012; Hart et al., 2013), the findings thus constrain and inform bioecological models which suggest a monotonic increase in heritability across varied environmental resources (Bronfenbrenner & Ceci, 1994).

US childhood and adult studies have largely (but not exclusively) supported the bioecological model of intelligence, where, unlike disorders following a diathesis stress (Gottesman & Shields, 1982) model, rich environmental support maximizes (rather than minimizes) genetic effects. This is compatible with observations of increasing heritability and decoupling of children's attainment from parental status with increasing access to education (Heath et al., 1985) as well as with the finding that environmental factors such as higher teacher quality not only raise scores of all children in the classroom, but also amplify genetic individual differences, for instance on reading (Taylor, Roehrig, Soden Hensler, Connor, & Schatschneider, 2010). Here, we not only saw no evidence of gene × SES interaction, but also (unlike previous studies (Hanscombe et al., 2012; Tucker-Drob et al., 2011; Turkheimer et al., 2003), we did not find evidence for significant between-family × SES interaction. Between-family interactions wash out by adolescence (Harden et al., 2007). A mechanism for this "washing out" is required.

The implications for the bioecological model bear some examination. The bioecological model is compatible with both the finding of enduring g × SES effects on IQ heritability in the US, *and* with the result that UK (Hanscombe et al., 2012) and (present data) Australian subjects show no such effects — indeed, the bioecological model uses high heritability precisely as an index of optimal environmental provisioning (Bronfenbrenner & Ceci, 1994). The mechanisms leading to these differences, however, need to be made explicit in the model.

It would seem improbable that 21st century Australia should provide exactly the social resource required for all individuals to reach the maximum possible IQ under any conceivable regime. The present

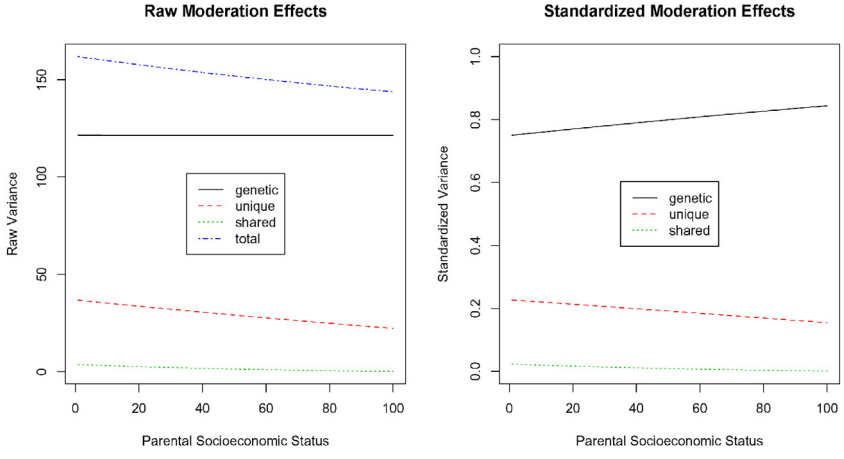

Fig. 4. Saturated models of raw (left panel) and standardized (right panel) variance in intelligence at each level of parental SES. Note: No moderated effects differed significantly from zero (horizontal). "genetic" = variance due to additive genetic effects; "shared" = shared environmental variance; "unique" = unique environmental variance; "total" = total unstandardized variance.

results, however, do not suggest that. Instead, they indicate that the currently available resources are not distributed unevenly among different SES groups in ways that impact on adolescent IQ development. It is likely that especially for the most gifted, further "resources" would see widening of the gaps, and even greater maximal IQ. We place resources in quotes, as it is unclear what it is among US families that creates SES-linked effects on heritability, and what it might be that in Australia decouples this factor or factors from SES.

Understanding the specific inputs that maximize intellectual development is clearly of importance, as, once identified, these may potentially be decoupled from parental SES and explicitly raised, perhaps cost-effectively. In seeking explanations for the lack of gene × SES interactions on IQ in the UK, Netherlands, and, now, in Australia, one answer may lie in school provision. The finding that attending pre-school reduces SES-linked achievement differentials suggests that school provides the substantive environment underlying differences in the expression of genetic potential in achieved intellectual capacity (Tucker-Drob, 2012). It is possible that, in comparison to the US, aspects of the funding or management of Australian education act to provide educational environments more similar to those that, in the US also, act to reduce SES interactions on heritability (Hart et al., 2013). The nature of these factors is currently, however, unclear. Significant variance in E, which is recorded universally, suggest that there are existing factors, unmeasured here, which might be harnessed to further raise mean IQ among westernized nations, with consequent increases in correlates of IQ, such as wealth, health, and civil growth (Deary, 2012; Murray, 2003).

Candidate environmentally-mediated influences include macro features such as pre-school attendance (Tucker-Drob, 2012). The effective activities of school include factors as prosaic as time spent in systematic study (Brinch & Galloway, 2012), to factors of teacher quality (Taylor et al., 2010), and achievement reading and in mathematics as enablers of increased general ability (Ritchie & Bates, 2013). Tests of these factors in genetically informative designs would be valuable — environmental effects on means may have different origins, perhaps not affecting latent general ability in all cases (Ritchie, Bates, & Deary, 2015; Ritchie, Bates, Der, Starr, & Deary, 2013).

This study is not without limitations. Studies should be combined where possible afford a well-powered meta-analytic analysis of studies. What is desired ultimately is a mechanism for the moderation of the effects of genes. Such a mechanism must be a change at a level invisible to the present analyses, for instance expression-changes in specific genes linked to learning (Nithianantharajah et al., 2013). Neither can we specify the environmental factors. We have discussed learning opportunities, but physical factors such as infection may also be moderators. It does not follow from a lack of moderation that IQ could not be further raised in some children — simply that such IQ-maximizing environments are sufficiently rare that they escape detection here. Given the importance of maximizing cognitive development, testing these possibilities in diverse, genetically-informative cohorts should be a priority (T. C. Bates & Lewis, 2012).

Acknowledgments

We thank the twins and their families for participation; Marlene Grace and Ann Eldridge for phenotype collection; Kerrie McAloney for study coordination; and David Smyth, Harry Beeby, and Daniel Park for IT support. Data collection was supported by the Australian Research Council (A7960034, A79906588, A79801419, DP0212016, DP0664638, DP1093900).

References

Bates, D., Maechler, M., & Bolker, B. (2011). lme4: Linear mixed-effects models using S4 classes.R package version 0.999375–42. (Retrieved from http://CRAN.R-project.org/package=lme4s2011).

Bates, T.C., & Lewis, G.J. (2012). Towards a genetically informed approach in the social sciences: Strengths and an opportunity. *Personality and Individual Differences*, *53*(4), 374–380. http://dx.doi.org/10.1016/j.paid.2012.03.002.

Bates, T.C., Lewis, G.J., & Weiss, A. (2013). Childhood socioeconomic status amplifies genetic effects on adult intelligence. *Psychological Science*, *24*(10), 2111–2116. http://dx.doi.org/10.1177/0956797613488394.

Bates, T.C., Neale, M.C., & Maes, H.H. (2016). umx: A library for Structural Equation and Twin Modelling in R. *Journal of Statistical Software* (Retrieved from https://github.com/tbates/umx, under review).

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., ... Fox, J. (2011). OpenMx: An open source extended structural equation modeling framework. *Psychometrika*, *76*(2), 306–317. http://dx.doi.org/10.1007/s11336-010-9200-6.

Brinch, C.N., & Galloway, T.A. (2012). Schooling in adolescence raises IQ scores. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(2), 425–430. http://dx.doi.org/10.1073/pnas.1106077109.

Bronfenbrenner, U., & Ceci, S.J. (1994). Nature–nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*, *101*(4), 568–586. http://dx.doi.org/10.1037/0033-295X.101.4.568.

Ceci, S.J., & Papierno, P.B. (2005). The rhetoric and reality of gap closing: When the "have-nots" gain but the "haves" gain even more. *American Psychologist*, *60*(2), 149–160. http://dx.doi.org/10.1037/0003-066X.60.2.149.

Deary, I.J. (2012). Intelligence. *Annual Review of Psychology*, *63*, 453–482. http://dx.doi.org/10.1146/annurev-psych-120710-100353.

Deary, I.J., Spinath, F.M., & Bates, T.C. (2006). Genetics of intelligence. *European Journal of Human Genetics*, *14*(6), 690–700. http://dx.doi.org/10.1038/Sj.Ejhg.5201588.

Gottesman, I.I., & Shields, J. (1982). *Schizophrenia, the epigenetic puzzle*. Cambridge: Cambridge University Press.

Grant, M.D., Kremen, W.S., Jacobson, K.C., Franz, C., Xian, H., Eisen, S.A., ... Lyons, M.J. (2010). Does parental education have a moderating effect on the genetic and environmental influences of general cognitive ability in early adulthood? *Behavior Genetics*, *40*(4), 438–446. http://dx.doi.org/10.1007/s10519-010-9351-3.

Hanscombe, K.B., Trzaskowski, M., Haworth, C.M., Davis, O.S., Dale, P.S., & Plomin, R. (2012). Socioeconomic status (SES) and Children's Intelligence (IQ): In a UK-representative sample SES moderates the environmental, not genetic, effect on IQ. *PloS One*, *7*(2), e30320. http://dx.doi.org/10.1371/journal.pone.0030320.

Harden, K.P., Turkheimer, E., & Loehlin, J.C. (2007). Genotype by environment interaction in adolescents' cognitive aptitude. *Behavior Genetics*, *37*(2), 273–283. http://dx.doi.org/10.1007/S10519-006-91134.

Hart, S.A., Soden, B., Johnson, W., Schatschneider, C., & Taylor, J. (2013). Expanding the environment: Gene × school-level SES interaction on reading comprehension. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*. http://dx.doi.org/10.1111/jcpp.12083.

Haworth, C.M., Wright, M.J., Luciano, M., Martin, N.G., de Geus, E.J., van Beijsterveldt, C.E., ... Plomin, R. (2010). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry*, *15*(11), 1112–1120. http://dx.doi.org/10.1038/mp.2009.55.

Heath, A.C., Berg, K., Eaves, L.J., Solaas, M.H., Corey, L.A., Sundet, J., ... Nance, W.E. (1985). Education policy and the heritability of educational attainment. *Nature*, *314*(6013), 734–736 (Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/4039415).

Jackson, D.N. (1984). *Multidimensional aptitude battery manual. Vol. 1*, Port Huron, MI: Research Psychologists Press.

Jackson, D.N. (1998). *Multidimensional aptitude battery II*. Port Huron, MI: Sigma Assessment Systems.

Kennedy, W.A., Vanderiet, V., & White, J.C. (1963). A normative sample of intelligence and achievement of negro elementary-school children in the southeastern United-States. *Monographs of the Society for Research in Child Development*, *28*(6), 1–112 (Retrieved from <Go to ISI>://A1963CFZ5800001).

Marsh, H.W., Wen, Z., & Hau, K.T. (2004). Structural equation models of latent interactions: Evaluation of alternative estimation strategies and indicator construction. *Psychological Methods*, *9*(3), 275–300. http://dx.doi.org/10.1037/1082-989X.9.3.275.

McGue, M., & Bouchard, T.J., Jr. (1984). Adjustment of twin data for the effects of age and sex. *Behavior Genetics*, *14*(4), 325–343 (Retrieved from http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=6542356).

McMillan, J., Beavis, A., & Jones, F.L. (2009). A new socioeconomic index for Australia. *Journal of Sociology*, *45*(2), 123–149. http://dx.doi.org/10.1177/1440783309103342.

Medland, S.E., Nyholt, D.R., Painter, J.N., McEvoy, B.P., McRae, A.F., Zhu, G., ... Martin, N.G. (2009). Common variants in the trichohyalin gene are associated with straight hair in Europeans. *American Journal of Human Genetics*, *85*(5), 750–755. http://dx.doi.org/10.1016/j.ajhg.2009.10.009.

Murray, C. (2003). *Human accomplishment: The pursuit of excellence in the arts and sciences, 800 B.C. to 1950*. New York: Harper Collins.

Neale, M.C., & Maes, H.H. (1996). *Methodology for genetics studies of twins and families (6th ed.)*. Dordrecht, The Netherlands: Kluwer.

Neale, M.C., Hunter, M.D., Pritikin, J.N., Zahery, M., Brick, T.R., Kirkpatrick, R.M., ... Boker, S.M. (2015). OpenMx 2.0: Extended structural equation and statistical modeling. *Psychometrika*. http://dx.doi.org/10.1007/s11336–014-9435-8.

Nithianantharajah, J., Komiyama, N.H., McKechanie, A., Johnstone, M., Blackwood, D.H., Clair, D.S., ... Grant, S.G. (2013). Synaptic scaffold evolution generated components of vertebrate cognitive complexity. *Nature Neuroscience*, *16*(1), 16–24. http://dx.doi.org/10.1038/nn.3276.

Purcell, S. (2002). Variance components models for gene–environment interaction in twin analysis. *Twin Research*, *5*(6), 554–571 (Retrieved from http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=12573187).

R Core Team (2014). *R: A language and environment for statistical computing (version 3.0.1)*. Vienna, Austria: R Foundation for Statistical Computing (Retrieved from http://www.R-project.org).

Ritchie, S.J., & Bates, T.C. (2013). Enduring links from childhood mathematics and reading achievement to adult socioeconomic status. *Psychological Science*, *24*(7), 1301–1308. http://dx.doi.org/10.1177/0956797612466268.

Ritchie, S.J., Bates, T.C., & Deary, I.J. (2015). Is education associated with improvements in general Cognitive ability, or in specific skills? *Developmental Psychology*, *51*(5), 573–582. http://dx.doi.org/10.1037/a0038981.

Ritchie, S.J., Bates, T.C., Der, G., Starr, J.M., & Deary, I.J. (2013). Education is associated with higher later life IQ scores, but not with faster cognitive processing speed. *Psychology and Aging*, *28*(2), 515–521. http://dx.doi.org/10.1037/a0030820.

Rowe, D.C., Jacobson, K.C., & Van den Oord, E.J. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as moderator. *Child Development*, *70*(5), 1151–1162 (Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10546338).

Sackett, P.R., Kuncel, N.R., Beatty, A.S., Rigdon, J.L., Shen, W., & Kiger, T.B. (2012). The role of socioeconomic status in SAT-grade relationships and in college admissions decisions. *Psychological Science*, *23*(9), 1000–1007. http://dx.doi.org/10.1177/0956797612438732.

van der Sluis, S., Posthuma, D., & Dolan, C.V. (2012). A note on false positives and power in G × E modelling of twin data. *Behavior Genetics*, *42*(1), 170–186. http://dx.doi.org/10.1007/s10519-011-9480-3.

van der Sluis, S., Willemsen, G., de Geus, E.J., Boomsma, D.I., & Posthuma, D. (2008). Gene–environment interaction in adults' IQ scores: Measures of past and present environment. *Behavior Genetics*, *38*(4), 348–360. http://dx.doi.org/10.1007/s10519-008-9212-5.

Soden-Hensler, B. (2012). *An examination of gene × socioeconomic status interactions for reading achievement*. (Ph.D.) Florida: Florida State university (Retrieved from diginole.lib.fsu.edu/etd (5438)).

Stanovich, K.E. (1986). Matthew effects in reading: Some consequences of individual differences in the acquisition of literacy. *Reading Research Quarterly*, *21*, 360–406.

Taylor, J., Roehrig, A.D., Soden Hensler, B., Connor, C.M., & Schatschneider, C. (2010). Teacher quality moderates the genetic effects on early reading. *Science*, *328*(5977), 512–514. http://dx.doi.org/10.1126/science.1186149.

Tucker-Drob, E.M. (2012). Preschools reduce early academic-achievement gaps: A longitudinal twin approach. *Psychological Science*, *23*(3), 310–319. http://dx.doi.org/10.1177/0956797611426728.

Tucker-Drob, E.M., & Bates, T.C. (2015). Large cross-national differences in gene × socioeconomic status interaction on intelligence. *Psychological Science*. http://dx.doi.org/10.1177/0956797615612727.

Tucker-Drob, E.M., Briley, D., & Harden, K.P. (2013). Genetic and environmental influences on cognition across development and context. *Current Directions in Psychological Science*, *22*(5), 349–355. http://dx.doi.org/10.1177/0963721413485087.

Tucker-Drob, E.M., Rhemtulla, M., Harden, K.P., Turkheimer, E., & Fask, D. (2011). Emergence of a gene × socioeconomic status interaction on infant mental ability between 10 months and 2 years. *Psychological Science*, *22*(1), 125–133. http://dx.doi.org/10.1177/0956797610392926.

Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I.I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14*(6), 623–628 (Retrieved from <Go to ISI>://000186544100015).

Wright, M.J., & Martin, N.G. (2004). The Brisbane adolescent twin study: Outline of study methods and research projects. *Australian Journal of Psychology*, *56*(2), 65–78.