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How fragile is our intellect? Estimating losses in general intelligence due to both selection and mutation accumulation

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ABSTRACT

Two dysgenic models of declining general intelligence have been proposed. The first posits that since the Industrial Revolution those with low *g* have had a reproductive advantage over those with high *g*. The second posits that relaxed purifying selection against deleterious mutations in modern populations has led to *g* declining due to mutation accumulation. Here, a meta-analytic estimate of the decline due to selection is computed across nine US and UK studies, revealing a loss of .39 points per decade (combined $N = 202{,}924$). By combining findings from a high-precision study of the effects of paternal age on offspring *g* with a study of paternal age and offspring *de novo* mutation numbers, it is proposed that, 70 *de novo* mutations per familial generation should reduce offspring *g* by 2.94 points, or .84 points per decade. Combining the selection and mutation accumulation losses yields a potential overall dysgenic loss of 1.23 points per decade, with upper and lower bound values ranging from 1.92 to .53 points per decade. This estimate is close to those from studies employing the secular slowing of simple reaction time as a potential indicator of declining *g*, consistent with predictions that mutation accumulation may play a role in these findings.

1. Introduction

General intelligence is an adaptation to solving evolutionarily novel and domain general fitness problems, i.e. problems which occur on an irregular rather than predictable basis throughout the course of evolution and which are *complex*, requiring the recruitment and coordination of large numbers of specialized mechanisms in solving them (Geary, 2005; MacDonald, 2013). As a complex adaptation, it has been proposed that general intelligence may be functionally sensitive to the effects of deleterious mutations (Houle, 2000; Miller, 2000a,b). On this basis, it has been argued that deleterious mutations exhibiting small effects accumulate within a population – persisting within genomes for long periods of time before substantially inhibiting fitness, thus giving rise to individual differences in general intelligence (*g*) and other potentially mutation-sensitive traits, such as health and physical attractiveness (Miller, 2000a,b; Penke, Denissen, & Miller, 2007).

Individual differences in *g* have been consistently negatively correlated with fitness outcomes (measured in terms of completed fertility and family size) since the early 20th century in the US and UK (Lynn, 2011; Lynn & van Court, 2004). Using proxies for *g* such as socio-economic status and education, the negative correlation extends back to the early 19th century in many Western countries (Skirbekk, 2008). This pattern of fertility seems to have arisen with declining inter-group competition (Woodley & Figueredo, 2013) in conjunction with rising education (Meisenberg, 2010), the development of effective methods for fertility control, and also with increasingly redistributionistic economies, which promoted fertility amongst those with lower *g*, whilst inhibiting it amongst those with higher *g* (Lynn, 2011).

An alternative, but complimentary theory holds that, as a potentially mutation-sensitive trait, *g* should be decreasing as a result of increasing mutation load stemming from the relaxation of purifying selection against the carriers of deleterious mutations (Crabtree, 2013; Hamilton, 1999; Muller, 1950). Integral to this is the *mutation load paradox* (Kondrashov & Crow, 1993), which results from the observation that the rate at which deleterious mutations accumulate across human generations is sufficiently high that around 88% of the population should fail to reproduce, which is not what is observed (Lesecque, Keightley, & Eyre-Walker, 2012). Based on direct counts in the genomes of children born to fathers of different ages, it has been found that offspring acquire around 70 *de novo* (new) Single Nucleotide Polymorphism (SNP) mutations each generation (35 years; Kong et al., 2012). Fathers bequeath the majority of these mutations to their offspring as new germ-line mutations occur primarily in sperm, owing to higher turnover compared with eggs. Of these 70 *de novo* mutations, around 2.2 are deleterious (Keightley, 2012).

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It has been proposed that relative fitness differentials between individuals in modern populations may solve the paradox (Lesecque et al., 2012), however an overlooked and potentially much more significant factor in resolving paradox is the observation that historically, child mortality may have been the focal point of purifying selection against deleterious mutations, as the generational loss in fitness was extremely high, in line with theoretical expectations (50% in Sweden and France between 1600 and 1700 AD; Cunninghman, 2005), and would furthermore have been disproportionately concentrated amongst those with low socioeconomic status (Clark, 2007; Fagan, 2001), which is a proxy for lower *g* and therefore potentially higher mutation load. Owing to improvements in medicine and nutrition, child mortality has been reduced to around 1% in modern Western populations (Volk & Atkinson, 2008). This would have substantially reduced the strength of the purifying selection operating on these populations, which has in turn enabled potentially deleterious mutations to accumulate across generations largely unchecked (Hamilton, 1999; Kong et al., 2012).

There is currently little in the way of crosstalk between advocates of either the selection or mutation accumulation theories of dysgenics (*cf.* Crabtree, 2013; Lynn, 2011). This oversight may have led to proponents of these theories systematically underestimating the true dysgenic loss in *g*, which will reflect combined losses due to both selection and accumulating mutations. Here a case for this will be made with a meta-analytic estimate of the *g* loss due to selection in two countries (the US and UK), in addition to an attempt to quantify the additional loss due to mutation accumulation by combining the results of high-quality studies investigating offspring *g* and *de novo* mutation counts as a function of paternal age.

2. Estimating selection against *g*

Throughout the 20th century, numerous researchers have attempted to utilize the size of the selection effect against IQ as measured by the strength of the negative correlation between IQ and individual-level fitness outcomes (typically sibship number or completed fertility; i.e. fertility measured at the end of reproductive life) to estimate the hypothetical decline in IQ (Lynn, 2011). The Breeder's equation (Fisher, 1929) is frequently employed in studies of this kind:

$$R = S * h^2$$

In this equation, *S* constitutes the size of the selection pressure operating on IQ transformed into a *phenotypic* change (i.e. the degree to which the trait will change over a generation assuming no biological regression to the mean, or perfect heritability). h^2 represents the additive heritability of IQ. The product of these two terms gives us the expected responsiveness to selection, or *R*, which in terms of IQ is scaled as a change in 'genotypic IQ', or the degree to which the underlying genetic potential for a certain level of IQ should decline per generation (Lynn, 2011).

Here, an attempt will be made to generate an aggregate of the loss in heritable *g* due to selection via meta-analytic treatment of studies reporting predicted declines in genotypic IQ based on the magnitude of selection.

2.1. Inclusion rules

Thus far, nine studies have attempted to estimate the IQ decline due to selection in US and UK samples. Although there are studies reporting potential genotypic IQ declines in other countries, such as Libya (Abdalgadr Al-Shahomee, Lynn, & El-ghmary Abdalla, 2013), Taiwan (Chen, Chen, Liao, & Chen, 2013) and Sudan (Khaleefa, 2010), only the nine studies from the US and UK will here be used in generating an aggregate of *g* loss, as these countries are closely matched in terms of important bio-cultural dimensions.

2.2. Validity generalization

Prior to computing the aggregate, all decline estimates must first be corrected for error sources. This is achieved using *validity generalization*, which involves identifying and correcting sources of error in studies (Hunter & Schmidt, 2004).

The final estimates are rescaled in terms of '*heritable g*' – i.e. the decline in the heritable variance component of *g* (Woodley & Figueredo, 2013), rather than simply 'genotypic IQ'. Five error sources are identified and corrected:

(I) All generational decline estimates are rescaled using a standard familial generational length of 3.5 decades (i.e. Helgason, Hrafnkelsson, Gulcher, Ward & Stefánsson, 2003), from which decadal declines can be computed. Inconsistent conceptualizations of generational length between studies are also a source of heterogeneity amongst decline estimates.

(II) All estimates are rescaled using a standardized h^2 value for *g*. The value of .86 from the study of Panizzon et al. (2014) is employed, as they provide a high-precision and direct estimate of the heritability of *g*. As subtest heritabilities and sensitivity to dysgenic selection both rise with *g* loading (Kan, Wicherts, Dolan, & van der Maas, 2013; Peach, Lyerly, & Reeve, 2014; Rushton & Jensen, 2010; Woodley & Meisenberg, 2013), decline estimates based on low values of h^2 derived from fullscale IQ will attenuate the loss due to dysgenics. Inconsistent heritability values are also a source of heterogeneity amongst decline estimates.

(III) A correction is implemented based on whether parameter *S* was determined on the basis of the IQ/sibship correlation. Van Court and Bean (1985) found that these correlation magnitudes were larger than those based on the IQ/completed fertility relationship when estimated using the same large sample of the US population (−.31 vs −.18). Decline estimates based on sibship may be inflated by within-family sources of IQ variance that are independent of dysgenics (e.g. Zajonc & Sulloway, 2007). These are corrected downwards via multiplication by the quotient of the two correlations i.e. .58 (.18/.31), using van Court and Bean (1985) as the reference study for this correction (Hunter & Schmidt, 2004, pp. 47–49).

(IV) A correction for the reliability of the IQ measures is made to the estimates. As reliability data are not available for the samples, coefficients will instead be approximated by simulation. Shevlin, Miles, Davies, and Walker (2000) employ a Monte Carlo simulation to determine the impact of *N* on Cronbach's α for a simulated six-indicator latent variable. Assuming a mean factor loading of .7 (consistent with *g* accounting for approximately 50% of the variance in IQ on average; Brand, 1996) and the maximum level of correlated error, the α coefficients in Shevlin et al.'s Table 1 (p. 232) correlate significantly at .91 with the four values of *N*. To obtain simulated α coefficients by *N*, the following formula is used: $\alpha = .00002 * N + .8769$. For values of $N > 6155$, the simulated coefficients exceed 1, therefore a value of 1 is employed in these cases.

(V) A correction for psychometric validity (i.e. the degree to which IQ batteries imperfectly measure the construct *g*) is made to the estimates by dividing them by .9. This value is derived from Jensen (1998, p. 383).

2.3. Results

Table 1 presents nine studies and 10 effect sizes in which declining IQs have been computed on the basis of the negative correlation between IQ and fitness outcomes, along with their original h^2 or parent–child similarity coefficients. Also presented are the