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The secular decline in general intelligence from decreasing developmental stability: Theoretical and empirical considerations

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ABSTRACT

The *g*-loss predicted based on genetic selection is smaller than that observed across various ratio-scale measures of cognitive ability. The difference may result in part from the accumulation of deleterious mutations across generations, reducing *g* via their effects on developmental stability/fitness. Previously published secular trend data on a developmental stability measure, craniofacial fluctuating asymmetry (FA) size, for white US males and females covering 14 and 15 decades respectively, are re-analysed. When the secular increases in FA size are rescaled as declines in latent developmental stability, and multiplied by the validity and reliability adjusted developmental stability-*g* correlation, *g*-losses of −.16 points per decade are predicted for the males, females and the combined sample. Predicted fitness losses due to mutation accumulation may account for 30% of the generational decline (−.05 points per decade), indicating only a small role for mutations in secular *g*-loss. The remaining 70% (−.11 points per decade) may result from developmental stability disrupting environmental change, such as increased exposure to pollutants. Adding these to the *g*-loss due to selection (re-estimated at −.54 points per decade) yields a combined decadal loss of −.70 points. Additional adjustments for replacement migration and the generation length-*g* interaction yield a larger magnitude decadal *g*-loss of −1.25 points.

1. Introduction

The loss in general intelligence (*g*) due to genetic selection has been estimated at −.39 points per decade in the UK and US (Woodley of Menie, 2015). Studies investigating secular declines in ratio-scale measures (i.e. measures with a true zero) of cognitive ability, such as simple reaction time (Woodley of Menie, te Nijenhuis & Murphy, 2015), backwards digit span (Woodley of Menie & Fernandes, 2015), colour acuity (Woodley of Menie & Fernandes, 2016) and spatial perception (Pietschnig & Gittler, 2015) indicate much larger losses (unweighted average of −2.3 points per decade). Understanding the causes of this disparity is an important and open research question.

1.1. Mutation accumulation as a possible contributor

Aging fathers are a major source of common and harmful *de novo* (new) mutations in human populations (Kong, Frigge, Masson, et al., 2012). Child mortality and hypergamy (i.e. "marrying up") may historically have been the principal sources of purifying selection against these *de novo* mutations. In the West, child mortality dropped from >50% in the Middle-Ages to <1% in the 20th century (Volk & Atkinson, 2008), furthermore traits believed to signal genetic quality, such as physical attractiveness, no longer confer a fitness advantage under conditions where modern birth-control is practiced (Pflüger, Oberzaucher, Katina, Holzleitner & Grammer, 2012). These changes may have permitted harmful mutations to accumulate across generations, reducing the evolutionary fitness, or genetic quality of modern populations (Crow, 1997; Hamilton, 1999; Lynch, 2010; Muller, 1950).

Fitness Indicators Theory (Miller, 2000) predicts that *g*, as a strong indicator of underlying genetic quality, should be very sensitive to the effects of deleterious mutations. Therefore the build-up of these mutations over time might reduce *g* independently of the effects of genetic selection.

The theory also predicts that because of age-related increases in the numbers of germ-line *de novo* mutations, older fathers should produce less intelligent offspring (Arslan, Penke, Johnson, Iacono, & McGue, 2014). The *g* lost by the offspring due to paternal age could therefore be used as a proxy for the between-generation loss — for example, if each decade of paternal age reduces offspring *g* by one IQ point, then the offspring of fathers aged 35 are on average 3.5 IQ points less intelligent than their fathers (i.e. .1 * 3.5 decades). Assuming that those mutations are not purified, these losses should compound across generations (Woodley of Menie, 2015).

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Recently, Arslan et al. (2014) found no effect of paternal age on offspring *g* after controlling for parental *g*, education and birth order, however a small but significant decrease in offspring *g* of -.84 points per decade of paternal age was noted prior to controlling for birth order. On the basis that collinearity between paternal age and birth order may have destabilized a theoretically sound but small-magnitude effect (Myrskylä, Silventoinen, Tynelius & Rasmussen, 2013, p.6), Woodley of Menie (2015) utilized this unadjusted value as a proxy for the effects of accumulating *de novo* mutations on population *g*, yielding a combined loss of -1.23 points per decade when added to the loss due to selection. Another study (D'Onofrio et al., 2014) however replicated the null result in a much larger sample ($N > 500{,}000$), which substantially increases the robustness of this finding. These results therefore compellingly indicate that, contrary to expectations, there is no association between paternal age and offspring *g*. This in turn invalidates the approach developed in Woodley of Menie (2015) for estimating the impact of mutation accumulation on *g*.

One explanation for the lack of a paternal age effect is that in the normal range, the development of *g* might be buffered against the direct effects of deleterious *de novo* mutations, which results in high levels of *canalization* (Arslan & Penke, 2015a; Penke & Jokela, 2016) — this being the capacity to reliably develop a trait despite the presence of environmental and genetic interference (Nijhout & Davidowitz, 2003). This model suggests only a small role for *de novo* mutations in the maintenance of *g* variance, and is the opposite of what is predicted by the Fitness Indicators Theory (Arslan & Penke, 2015b; Penke & Jokela, 2016). These findings and new theoretical developments mean that the relationship between generational increases in the population burden of harmful mutations and *g* must be reconsidered.

1.2. Developmental stability and g

Small to modest associations exist between *g* and measures of *developmental stability* — this being an index of the sensitivity of a trait to developmental disturbance stemming from deleterious mutations and environments (Nijhout & Davidowitz, 2003). It has been theorized that developmental stability is very closely aligned with fitness, as traits that are sensitive to the effects of deleterious mutations may serve as honest indicators of underlying genetic quality in sexual selection (Penke, Denissen & Miller, 2007).

The existence of small to modest associations between *g* and indicators of developmental stability suggest that even though mutations might not have strong direct effects on *g*[1], by reducing developmental stability they may nonetheless *antagonize* (i.e. interfere with) the canalization of *g*.

One very general indicator of developmental stability, widely studied in biology and psychology, is *fluctuating asymmetry* (FA; van Valen, 1962), which indexes the degree to which an organism develops on a bilaterally symmetric basis (i.e. in terms of the equality of the left and right sides) and is believed to relate to the effects of deleterious mutations and environmental stressors (i.e. pathogens and poor nutrition) interfering with gene expression in development (Gangestad & Thornhill, 1999). Lower developmental stability reflects in greater degrees of FA, which can be measured via the degree of random fluctuation in the positioning of bilateral markers (such as eyes, ears, teeth, bones, and other bodily markers). Several different measures of FA established using different bodily markers have been found to correlate, suggesting that this measure taps a latent *g*-like *general factor of developmental stability* (Furlow, Armijo-Prewitt, Gangestad, & Thornhill, 1997). The range restricted and reliability-corrected phenotypic correlation between FA and IQ has been established in meta-analysis at −.16 (Banks, Batchelor, & McDaniel, 2010). Analysis involving the method of correlated vectors furthermore reveals strong positive correlations between the *g* loading of ability measures and their correlations with FA (Prokosch, Yeo, & Miller, 2005), which indicates that the strongest association between developmental stability and cognitive ability is at the level of *g*.

The idea that mutations have primarily indirect effects on *g* that are *moderated* by their effects on developmental stability makes sense of the apparent lack of a direct effect of paternal age (as a proxy for *de novo* mutations) on offspring *g*. Direct paternal age effects on offspring developmental stability are expected however and indeed, an effect of this on offspring facial attractiveness (a proxy for FA) has been identified (Huber & Fieder, 2014).

Mutation accumulation across multiple generations should also produce secular declines in indicators of developmental stability such as FA, which in turn may reduce *g* over generations via the effect of 'antagonized' canalization.

Increased exposure to xenobiotic substances (i.e. synthetic or naturally occurring chemical pollutants present in the environment to an abnormal degree) can also disturb developmental stability and may in turn reduce *g* (Demeneix, 2014), especially if exposure occurs very early in development (Debes, Weihe & Grandjean, 2015). Thus there are two distinct pathways through which reduced developmental stability might suppress *g* — accumulating mutations reducing genetic quality and xenobiotic exposure reducing phenotypic quality.

1.3. Secular trends in FA: Kimmerle and Jantz (2006)

Kimmerle and Jantz (2006) have conducted an analysis of secular trends in craniofacial FA using crania sourced from various national collections in the US, spanning the birth years 1820–1829, to 1980–1989. Two different types of FA were estimated: shape and size. In estimating shape FA, a centroid (a geometric object comprised of several markers) constructed out of seven craniofacial markers from the right side of the skull, was superimposed onto one constructed utilizing the same markers from the left side of the skull. Differences in alignment yielded a measure of the degree of shape FA. Size FA was computed by simply subtracting the size of the right centroid from the left one. The FA estimates were controlled for the presence of directional asymmetry (i.e. consistent bias in the asymmetry towards one or the other side), where it was found to be present.

The skulls were grouped based on sex and race (white and black). The analysis of secular trends was conducted by regressing the aggregated decadal FA values against birth year using *N*-weighted polynomial regression (both linear and quadratic terms were computed). A significant positive association between degree of shape FA and year of birth was found among the black female sample, however in the other groups there were no temporal trends. These researchers nevertheless note that: "*there is a slight trend (though nonsignificant association) for fluctuating size asymmetry to increase over time*" (p. 257). They also report that: "*Due to the overall tendency toward low r^2 values, a cursory attempt was made to determine whether those individuals with the highest levels of fluctuating asymmetry, particularly during the early 19th century, shared any common life history factors. The cause of death, age at death, geographic location of birth, and even the collection in which the skeletal remains are housed were compared. To date, the only patterns observed are those for birth year.*" (p. 260).

As was mentioned previously, FA should in part reflect phenotypic condition, stemming from environmental factors such as the influences of parasites, malnutrition and other sources of ecological stress, in addition to the underlying burden of deleterious mutations (Gangestad & Thornhill, 1999). This hypothesis led Kimmerle and Jantz (2006) to anticipate that the direction of the correlation between birth year and FA should be negative rather than positive, given improvements in health and nutrition in the US throughout the 19th and 20th centuries. Furthermore, both contemporary and

[1] Consistent with this, molecular data indicate only small deleterious effects of rare, protein altering point mutations on *g*, visible only when a very large range of ability-levels are compared (Spain, Pedroso, Kadeva et al. 2015).