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A life history model of the Lynn–Flynn effect

Michael A. Woodley

University of London, School of Biological Sciences, Royal Holloway, Egham, Surrey TW20 0EX, United Kingdom

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ABSTRACT

A new life history model of the Lynn–Flynn effect is presented based on the idea that life history speed is primarily associated with a trade-off between positive manifold strength (fast life history) and differentiation with respect to abilities (slow life history) rather than individual differences in levels of *g* (which instead function as a fitness indicator and are associated with pleiotropic mutation load). Given that the Lynn–Flynn effect concerns only the non-*g* variance in test scores it is proposed that it is associated with ability differentiation resulting from recent population-level shifts towards slower life history speed as a consequence of the mitigation of sources of environmental unpredictability and harshness, such as pathogen stress and malnourishment. Smaller family sizes are also significant as not only do they result from slower life history speed, but they might also potentiate further life history slowing. Education may also slow life history, furthermore it is involved in the cultivation of specialized patterns of cognitive abilities. Empirical predictions are made in the discussion, which if tested, could present definitive evidence either confirming or refuting the life history model.

1. Introduction

Richard Lynn was the first to bring the phenomenon of massive gains on standardized intelligence tests over time to widespread attention with a study published in *Nature* involving Japanese cohorts (Lynn, 1983). Subsequent research by Flynn (1984, 1987) expanded the scope of the search for these gains and found that they are occurring in a number of industrialized countries at a rate of approximately three points a decade. The effect has been christened the 'Flynn effect' (Herrnstein & Murray, 1994), however Rushton (1999) has argued that the effect should be termed the Lynn–Flynn effect, owing to the equally important contributions of both researchers to its elucidation. Congruent with Rushton's suggestion, the effect shall here be referred to as the Lynn–Flynn effect.

A number of studies have found that the effect manifests itself most strongly on fluid intelligence subcomponents, e.g. non-verbal abilities (Colom, Andres-Pueyo, & Juan-Espinosa, 1998; Emanuelsson, Reuterberg, & Svensson, 1993; Emanuelsson & Svensson, 1990; Flynn, 1987, 1998; Lynn & Hampson, 1986, 1989; Teasdale & Owen, 2000), these 'differential gains' have in turn led to debate concerning whether or not the effect concerns *g* (Colom, Juan-Espinosa, & García, 2001; Flynn, 1999a, 1999b, 2000; Jensen, 1998; Must, Must, & Raudik, 2003; Rushton, 1999, 2000a). A recent study by Rushton and Jensen (2010), has examined this issue in some detail. They found that *g*-loadings and inbreeding depression scores on the 11 subtests of the Wechsler Intelligence Scale for Children correlated either significantly negatively or not at all with secular gains due to the Lynn–Flynn effect, which reinforces the idea that the effect is not *g*-associated. Another relevent finding is that the assumption of factorial invariance with respect to cohorts (a necessary criterion for the invocation of *g* in the Lynn–Flynn effect) is untenable (Wicherts et al., 2004).

The idea that the Lynn–Flynn effect is not occurring on *g* has raised another issue, namely to what extent might the effect simply be an artifact stemming from factors such as heightened test sophistication or even the tendency for tests to loose their *g*-loading as a consequence of training, retesting and general familiarity (Brand, 1987, 1990; Brand, Freshwater, & Dockrell, 1989; Flynn, 1990; Jensen, 1996; Rodgers, 1998; Rushton & Jensen, 2010)? Still, others maintain that the effect is associated with real world increases in intelligence (Howard, 1999, 2001).

In this study, after a brief review of the various major proposed causes of the Lynn–Flynn effect, the recently developed cognitive differentiation–integration effort hypothesis (Woodley, 2010) will be used to propose a new causative model, namely that it results from the operation of environmental factors (such as the diminution of pathogen stress, improvements in nutrition, and education) which have acted to slow the life history speeds of populations in such a way that favors the investment of effort into the development of specific cognitive abilities. The model will be used to (a) account for why the effect appears to be associated exclusively with non-*g* variance, (b) explain the contradiction between secular gains in IQ and losses due to dysgenesis, and (c) address the issue of what the effect actually measures. In the final section the

E-mail address: Michael.Woodley03@gmail.com

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implications of this model will be discussed along with empirical predictions.

2. The causes of the Lynn–Flynn effect

Amongst those who maintain that the effect is associated with real gains in cognitive ability, a variety of causative theories have been proposed. Lynn (1989, 2009) has argued that the principal cause has been improvements in nutrition, as malnourishment especially with respect to micronutrients is associated with inhibited brain development.

General improvements in the quality of education (Husén & Tuijnman, 1991; Teasdale & Owen, 1989; Tuddenham, 1948) have also been proposed as a prospective source of the effect, as has the idea that smaller families permit greater resource consolidation into fewer offspring (Zajonc & Mullally, 1997). Dickens and Flynn (2001) have proposed a social multiplier model, predicated upon the idea that the ambient cognitive 'background' of a society exerts an enhancing effect on IQ through feedback. Wide scale literacy, access to the internet and computer games would be examples of the sorts of social multipliers that once in widespread usage, might raise the mean IQ of a population in such a way that leads to greater demand for yet more cognitively demanding sources of stimulus.

Another theory is that heterosis (hybrid vigor) might be associated with gains in IQ (Mingroni, 2007). Recently pathogen stress has been found to be a significant predictor of cross-national variation in cognitive ability. An implication of this is that pathogens attenuate the development of full IQ by commandeering bioenergetic resources ordinarily reserved for brain development into their own growth and fitness. Measures aimed at eradicating pathogens or mitigating their effects at cross-national scales might therefore be behind the Lynn–Flynn effect (Eppig, Fincher, & Thornhill, 2010).

Which of these theories are plausible? As the Lynn–Flynn effect does not concern *g*, heterosis can be ruled out as a significant contributor owing to the fact that inbreeding depression on test scores and other genetic effects correlate with the *g*-loadings of tests, unlike secular gains (Rushton, 1999, 2000a; Rushton & Jensen, 2010). The social multiplier model suffers from a number of apparent shortcomings. The model has been interpreted as requiring large-scale changes in the historical variance of IQ despite the fact that there is no evidence for such changes (Rowe & Rodgers, 2002). Also there is no indication given by the model as to the time scales over which social multipliers might exert their effects, which in turn impacts its predictive power. Do they operate across the life span of individuals for example or over larger population scales (Leohlin, 2002; *cf* Dickens & Flynn, 2002)?

The pathogen stress, nutrition, family structure and education models do seem to be significantly involved in real world observations of the Lynn–Flynn effect (e.g. Daley, Whaley, Sigman, Espinosa, & Neumann, 2003), thus presenting plausible reasons as to why cognitive abilities might have risen over time. In the subsequent sections, it will be shown how these are complimentary with respect to the novel hypothesis presented here.

3. The cognitive differentiation–integration effort hypothesis

In human populations it has been found that measures of life history (the fundamental pattern of bioenergetic resource allocation and tradeoffs) combine to give rise to a latent factor termed *K*. This dimension captures individual differences in preferences towards the development of a fast life history, characterized by high mating effort versus a slow life history, characterized by high somatic effort (resources allocated to growth, repair, maintenance and learning) and parenting effort. (Figueredo, Vásquez, Brumbach, & Schneider, 2004).

Rushton (1985, 2000b) predicted that general intelligence forms part of the human life history matrix, with those oriented towards slow life histories possessing the highest *g* and those oriented towards fast life histories possessing the lowest. Research at the individual differences level has failed to find consistent support for this however, with correlations between *K* and *g* typically being both very low magnitude and non-significant (Gladden, Figueredo, & Jacobs, 2008; Sefcek, 2007; Sefcek & Figueredo, 2010).

The cognitive differentiation–integration effort (henceforth CD–IE) hypothesis is a model of cognitive development conceived as a theoretical explanation for how cognitive abilities and life history might be connected. The CD–IE hypothesis is predicated upon the idea that there are two largely independent sources of genetic variance in general intelligence – one source corresponds to Miller's (2000a, 2000b) theory that the basic positive manifold arises through the action of pleiotropic mutations, and that individual differences in levels of *g* function as a fitness indicator in sexual selection. The other source is associated with what could be termed 'horizontal' variance in general intelligence characterized by a life history trade-off between positive manifold strength and the development of separate abilities. This manifests its self in ability differentiation/integration effects such as Spearaman's Law of Diminishing Returns. The tradeoff concerns two hypothetical types of effort – cognitive integration effort (CIE), associated with a strengthening of the manifold via the equal investment of bioenergetic resources (such as time, calories and cognitive real estate) into diverse abilities, and cognitive differentiation effort (CDE), associated with a weakening of the manifold via the unequal investment of resources into individual abilities. This tradeoff is independent of the 'genetic *g*' detectable through the Jensen effect on the heritability of *g*, which appears to be largely impervious to differentiation/integration effects (Jensen, 2003). Variation in manifold strength in this model results from either equal or unequal investments of resources into abilities. CIE is associated with fast life history speed and may constitute a form of mating effort as by strengthening the manifold it may give rise to a heightened capacity to cope with situations requiring the coordination of diverse abilities, such as those that might be encountered in the short term mating market; CDE is associated with slow life history and may constitute a form of somatic effort as slower development permits the cultivation of specific abilities which gives rise to a capacity for ecological specialization in response to competition. Greater parenting effort may also result in the selective reinforcement of specific patterns of ability cultivation amongst offspring (Woodley, 2010).

Evidence for the CD–IE hypothesis comes from the observation that slower life history individuals such as those with autistic-like personalities exhibit less evenly balanced abilities (with a bias towards visuospatial ability) than faster life history individuals (Del Giudice, Angeleri, Brizio & Elena, 2010). This suggests that cognitive generalism vs. specialism might be a component of the human life history matrix. Thus far the hypothesis has not been comprehensively tested at the individual differences scale.

Fig. 1 illustrates the simplified hierarchical relationship between sources of genetic variance unique to general fitness (*F*) and life history (*K*), along with their connections to *g* and the CD-IE tradeoff. *F* and *K* are assumed to be modestly positively correlated (Sefcek & Figueredo, 2010), however there is much variance that is exclusive to each latent trait, indicating the operation of separate genetic pathways. It is also assumed that many environmental factors would simultaneously influence both the level and composition of general intelligence through their shared developmental effects on general fitness and life history. The degree to which an environmental factor could affect the level of *g*