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# The eclipse of heritability and the foundations of intelligence

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A B S T R A C T

It is well known that theory in human cognitive ability or 'intelligence' is not well developed, especially with regard to sources of trait variation. Roots of theory have been sought in biology, and it is now widely accepted, on the basis of twin studies, and statistical analysis of variance, that at least half of the normal trait variation can be attributed to genetic variation, a correlation known as the trait 'heritability'. Since the 1990s, methods in molecular biology have been adopted to go 'beyond' this mere statistical attribution to the identification of individual genes responsible for trait variation. More than a decade of intense effort, however, has failed to produce unambiguous, replicable findings; explanations for the 'missing heritability' are now being demanded; and calls for new perspectives on the roles of genes and environments in development and trait variation are being demanded. Here, I propose a dynamic systems perspective indicating how the processes in which heritability becomes missing are the very ones that provide the roots of new intelligence theory.

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## 1. Introduction: promise and disappointment

The cognitive ability of humans is probably the most complex of functions ever evolved: describing and characterizing it, therefore, has been especially difficult. Dominant IQ theory has relied mainly on correlations among test scores, and associations with other supposed criteria like school achievement, to arrive at models based on 'strength' or 'capacity' metaphors of a pervasive mental power, referred to as '*g*'. But a generally accepted theory of what that power consists of, what actually varies, and how, is still not available after more than a century of scientific inquiry. Accordingly, as Deary puts it, 'There is no such thing as a theory of human intelligence differences – not in the way that grown-up sciences like physics or chemistry have theories' (Deary, 2001, p. ix).

One prominent tendency has been to seek sources of variation in biology. This has been pursued by adopting ANOVA methods to partition phenotypic variance statistically into genetic and environmental components. These methods were originally devised in the 1920s for agricultural programs, where the size of the genetic component, the 'heritability', could indicate the likely fruitfulness of selective breeding. The approach was imported into psychology by Burt (1958) using IQ correlations of twins. Since then, results of a number of twin studies of cognitive ability have been taken to confirm the earlier estimates of a sizeable heritability of between 0.5 and 0.8, meaning that 50–80% of the variance in cognitive ability is genetic in origin.

Obviously, the approach of partitioning variance neither identifies specific genes nor describes pathways of development of trait variation, so does not, of itself, advance theory. Over the last two decades, however, remarkable advances in molecular biology have promised a leap 'beyond heritability' to identification of the specific genes associated with trait variation. Teams of psychologists and molecular biologists have scanned subjects' DNA at the single nucleotide level (as single nucleotide polymorphisms, or SNPs) in genome-wide association studies (GWAS) and attempted to correlate allelic variation with

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variation in IQ scores. The apparently high heritability (genetic variation) derived from twin studies had already suggested that GWAS would reveal numerous genes of substantial effect (e.g. Nuffield Council on Bioethics, 2002). Early results seemed encouraging, so that by 1998 newspapers were reporting worldwide that 'genes for intelligence' had been found (e.g. May 14th 1998, the New York Times).

Early promise, though, has not been sustained in subsequent research. In the area of cognitive ability, as in other better-defined traits, and categorical disease states, identified associations have been few, accounting for only a tiny proportion (<5%) of the putative heritability, and failures to replicate have been widespread (Cirulli et al., 2010; Eichler et al., 2010; Plomin, 2012). Typical current conclusions are that 'Molecular genetic studies have yet to identify reliably reproducible contributions from individual genes' (Deary, Penke, & Johnson, 2010, p. 201), and 'Despite our large sample size and three-stage design, the genes associated with childhood *g* remain tantalizingly beyond our current reach' (Davis et al., 2010, p. 759; for recent review see Turkheimer, 2011).

Most explanations for the missing heritability point to the likely large numbers of loci, each with small effects, requiring huge sample sizes for replicable associations to emerge (Plomin & Davis, 2009). But there are other possible explanations for the missing heritability. One of these is that, since reliable GWAS demand precision standards which IQ measures (especially the estimates and surrogates usually used in twin studies) may not meet, correlations will be labile. Another possibility is that the high heritability reported for IQ doesn't actually exist. There are major misgivings about the assumptions of the twin method on which most GWAS are predicated (Joseph, 2010; Richardson & Norgate, 2005). Attention has been increasingly drawn to gene–gene and gene–environment interactions that can create 'phantom' heritabilities (Zuk, Hechter, Sunyaeva, & Lander, 2012). And, of course – especially in a field in which false positives are so common – we have to remember the old adage about correlations not necessarily being causes.

However, there may be more fundamental reasons for the elusiveness of those associations. As Turkheimer (2011) notes, the search for independent genetic causes (as with earlier searches for environmental causes) of variation have created new questions and that '(s)ome new paradigm, unglimpsed at present, will be required before meaningful progress can be made' (p. 600). Here it is argued that the new paradigm has been available, at least in outline, for some time, and is further revealed in the very molecular biology that, while aiming to discover genes for intelligence, now deems such genes to be 'missing'.

## 2. An 'omics' perspective

The basic psychometric and behaviour genetic model of IQ variation is based on an additive (independent factors) model of genes and environments. It first assumes that intelligence is a simple quantitative trait – a 'speed' or 'power' entity like body strength or running speed – and is suitably measured by IQ tests (see Michell, 2013, on the dangers of assuming such constructs). Next it is assumed that genes have independent agency, the variant alleles exhibiting greater or lesser 'strength'. These contribute to phenotypic differences independently and in equal increments. It is further assumed that different environmental factors exert their effects on trait variation in the same independent way. The trait score is then a sum of those independent gene and environmental factors, with no other variation-creating factors or processes involved. The twin method and GWAS are predicated on these assumptions. In some accounts some degree of 'interaction' may occur to attenuate this additive model, creating some deviation from the expected value, but this effect is usually assumed to be quite small. Thus the 'narrow sense' heritability usually referred to in this field is an estimate of the proportion of trait variance that can be attributed to additive genetic effects.

The results of the molecular biological and genomic research have, over the last few years, become increasingly at odds with that model. They show that few genes (or, indeed, environments) are utilized in development, and create trait variation, in that simple additive fashion. In the process, though, the same results are revealing remarkably intelligent systems, even at the level of gene usage and cell biology, suggesting new unifying concepts of intelligence. Such intelligent systems are now deemed to be important because of the previously unsuspected complexity of the environment. The standard behaviour genetic model proposes development as a kind of assembly into randomly varying outcomes that are only adventitiously adaptive. But, even for the single cell, the environment is continuously changing in its deeper form and structure.

For example, the developing cell in a multicellular organism is faced with the changing surround of other cells, and 'storms' of signals from them, and from elsewhere: they must undergo frequent reprogramming, change of identity, location, and so on, to contribute to the developing whole. Although (as with genes) we have tended to idealize environments as arrays of independent factors, as simple cues for predetermined sets of responses, we now know there is much more structure-for-predictability in complex changing environments. For example, pairwise correlations can be further associated with (conditioned by) a third variable, and so on to higher dimensions two-way, three-way and higher interactions: i.e. multivariate dependencies. When the dimensions are changing in time and space (spatiotemporal), and are nonlinear, information of great depth, but predictability, is possible.

Indeed, wherever researchers have looked, they have found deeper, dynamic structure in environments, that can render complex environments predictable (Becks, Hilker, Malchow, Jürgens, & Arndt, 2005). Such information-in-structure is theoretically measurable in terms of 'mutual information' derived from Shannon entropy, which generalizes naturally to higher order dependencies among variables (Prokopenko, Boschetti, & Ryan, 2009; Slonim, Atwal, Tkačik, & Bialek, 2005). These measures correspond with intuitive notions of underlying structure in systems. Since Darwin, observers have marvelled at the anatomical complexities wrought by natural selection in organs like

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