

In press, *Behavioral and Brain Sciences*.

Negative results are needed to show the specific value of a cultural explanation for *g*: A commentary on Burkart et al.

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Abstract

The authors suggest that social learning can explain the cognitive positive manifold for social animals including humans. We caution that simpler explanations of positive trait intercorrelations exist, such as genetic load. To test the suggested explanation's specificity, we also need to examine non-social species and traits such as health that are distal to cognitive abilities.

Commentary

We commend the authors for writing a clear review of the available data on intra-species *g* and inter-species *G*. Although data on individual differences on non-human animals are scarce, mapping out the potentially far-ranging implications will hopefully encourage more high-quality non-human individual differences research. Their effort to establish clearer and cross-species terms in the modularity debate and to highlight the existence of secondary modules are very welcome. Together with the acknowledgement, as nicely laid out by the authors, that evolution is a tinkerer, not an engineer (Jacob, 1977) we think these efforts will lead to progress in the understanding of the evolution and genetics of general intelligence.

We agree that integration between sub-disciplines currently focused either on universals or individual differences (e.g. evolutionary psychology and behavior genetics) is necessary (Arslan & Penke, 2015; Penke, Denissen, & Miller, 2007) and hope warnings about the lack of generalizability in non-human animal cognition research will be heeded (Arden, Bensky, & Adams, 2016; Thornton & Lukas, 2012). On the other hand, we are unsure whether the effort to connect the appearance of *psychometric g* to the presence of *general intelligence* in a species succeeds. This leads us to examine what could cause *g* in the absence of a core intelligence trait.

We agree with the authors that psychometric *g* and domain-general intelligence should not be thoughtlessly equated (Penke et al., 2011). While a species whose individuals vary in a core domain-

general intelligence ability should exhibit a psychometric g , finding a psychometric g does not imply that variation in a core ability causes it. If g were caused by a core ability, then training this ability should show transfer effects to distal cognitive abilities. The authors cite such training studies, but acknowledge controversy about bias and methods (see also Colom et al., 2013; Redick, 2015). Noack, Lövdén, and Schmiedek (2014) conclude the existing literature cannot establish such latent transfer effects. Claims of bilingual advantage have been similarly contested (Paap, Johnson, & Sawi, 2015). If training of purported core abilities such as executive functions does not increase latent g , the case for core abilities causing the positive manifold weakens considerably. Moreover, positive correlations have not only been found among cognitive abilities but also between cognitive abilities and other fitness-related traits such as health, psychopathology and height (Arden et al., 2016). Hagenars et al. (2016) showed molecular evidence that a shared genetic aetiology underlies the phenotypic associations between health and intelligence.

Reasonable, less cognition-specific explanations have been put forward to explain such positive manifolds. First among them is probably genetic load (Hill et al., in press; Penke et al., 2007). Individuals vary in the number of deleterious genetic mutations they carry. Depending on where they occur, such wrong turns on the genotype-phenotype map could affect the integrity and condition of the whole organism, its brain, or more specific abilities. If many of the variants affecting cognitive abilities are pleiotropic (be that because they affect early development steps or because they disrupt frequently re-used genetic patterns), then we would also expect positive correlations between cognitive abilities and other fitness-related individual differences (Deary, 2012; Houle, 2000). Although causal inference from genetic correlations is hard (Johnson, Penke, & Spinath, 2011; Solovieff, Cotsapas, Lee, Purcell, & Smoller, 2013), we ought to consider the possibility that biological pleiotropy, not just health behavior, explains associations between intelligence and health. For example, evolutionarily conserved genomic regions are strongly enriched for genetic variants affecting intelligence (Hill et al., in press). We think this can explain some of the g phenomenon. An explanation based on genetic load can even explain correlations between abilities resulting from primary modules. Other sources of individual differences such as stochastic events in early development can take an explanatory role similar to genetic load, by affecting early developmental steps and pervasive building blocks of the organism (Deary, 2012).

Hence, our null hypothesis should not be complete independence of cognitive abilities, even if we knew they were primary modules. Some intercorrelation should be expected. The expected *degree* of intercorrelation depends on many unknowns, among them the degree of pleiotropy, the mutational target size, metabolic costs and ontogeny of cognitive abilities.

With the non-human data available so far, we see a gap in the authors' case: the absence of clear negative results in the search for g/G . The authors report no taxon where the search for the g factor was conducted with sufficient power and appropriate methods but still failed. We suggest that only after also gathering data from less social species can we ask whether e.g. social learning increases correlations between cognitive abilities. And only when correlations with non-cognitive abilities are compared can we ask what explains the increased correlation between cognitive abilities. As the authors point out, non-human research can help test explanations for g with designs infeasible in humans, such as cross-fostering experiments. This extends to genetic load. Both using genetically uniform strains and mutation accumulation lines (although these are already extremely time-consuming in micro-organisms) could help clarify the involvement of genetic load.

We lack the space to fully address alternative explanations for interspecies G, but hope to also see joint phylogenetic tree analyses of sociality and variance explained by *g*. To be able to test this, recommendations for increased sample sizes in such studies should be followed (Thornton & Lukas, 2012). Differential measurement error across subtests and species has to be modelled and corrected for, not just used to explain negative findings.

In conclusion, we would add the following to the authors' call for research: we need individual differences data along the whole gradient of sociality, including even e.g. octopuses, and studies should also examine more distal traits such as health and size. Then, the specific added value of the proposed model can be tested. We want to echo this and previous calls (Arden et al., 2016; Thornton & Lukas, 2012) for more individual differences research on non-human animal cognition. We hope for more stimulating evolutionary theorizing on individual differences, as in this target article.

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