search strategy, I located 288 primary studies, published in 1973–2002, which reported mean MWT scores for 527 groups of German, Austrian, and Swiss study participants (healthy adults as well as patient samples), totaling nearly 29,000 subjects. This large-scale meta-analysis of unrepresentative samples yielded an ΔIQ estimate of 2.61 for the gC measure MWT. This figure is comparable with the finding from the Austrian psychiatric patient sample and further nicely dovetails with extant evidence from population-based studies. Flynn (1984) originally arrived at a ΔIQ estimate of about 3 (USA, 1932–1978), which was later updated to about 2.5 (USA, 1972–1995 [Flynn 1998c]). A reanalysis of the extant international evidence by Storfer (1990, p. 439) suggests that ΔIQ was about 3.75 during the first quarter of the twentieth century, about 2.5 for the subsequent decades until about the mid-1960s, and probably less since then.

To summarize, Blair's claim of a gF–gC dissociation supposedly seen in the Lynn–Flynn effect (in order to support his gF" concept) is neither supported by the empirical record in this area nor by the new findings presented here. We are all well advised not to devote ourselves to phlogiston theories of human intelligence.

How relevant are fluid cognition and general intelligence? A developmental neuroscientist's perspective on a new model

Marko Wilke

Department of Pediatric Neurology and Developmental Medicine, Children's Hospital, University of Tübingen, 72076 Tübingen, Germany.

Marko.Wilke@med.uni-tuebingen.de

Abstract: Blair boldly proposes a model integrating different aspects of intelligence. Its real-life value can be put to the test by using programs designed to develop children's abilities in areas predicted to be crucial for minimizing adverse outcome. Until support from such programs is available, the model is an interesting hypothesis, albeit with remarkable possible repercussions. As such, it seems worthy of further development.

In his target article, Blair provides a comprehensive model for identifying and describing different aspects of intelligence (broadly defined), including the neurobiological underpinnings. As with many models proposed, a developmental neuroscientist is tempted to ask: So what? Numerous models are out there, aiming to describe and explain the multitude of observations regarding "intelligence" both in impaired and unimpaired subjects. What makes this work stand out is the direct applicability of the concept and, even better, the fact that we are liable to put it to the test both clinically and in neuroscience research. Clinically, those working with children from disadvantaged backgrounds or with children showing mental retardation can direct their attention towards developing programs aiming to influence the specific aspects of fluid cognition that Blair hypothesizes to be central in determining later outcome, as measured by as yet inappropriate tests. For neuroscience research, a number of directions seem to suggest themselves as to how the pertained distinction of fluid and general intelligence could be disentangled, for example, by using modern neuroimaging methods. As it is, the target article describes a bold new concept, thoroughly doing away with the monolithic idea of *g*-and-nothing-else. As such, it is likely to draw criticism from "proponents of the old order," and probably rightly so. However, programs designed to test the concept can (and, hopefully, will) be developed that enable supporting the concept with not only theoretical neuroscience data (such as functional magnetic resonance imaging [fMRI]) but, ideally, with the very practical and highly important result of children simply doing better in life. If this were the case, Blair must be commended for boldly going down this road. If not, then it will be just another model, with not much relevance for clinicians' daily work.

There are drawbacks, of course. What about the role of the thalamus and the cerebellum, both of which have been considered cornerstones for the cognitive impairment seen not only in schizophrenia (Clinton & Meador-Woodruff 2004; Rapoport et al. 2000; Schultz & Andreasen 1999)? Considering that the thalamus was classically used to define prefrontal cortex as the projection area of the mediodorsal thalamic nucleus, should it not be expected to play some kind of role, as a gatekeeper or in some other form, hitherto unknown? In our study on gray matter correlations with a broad measure of intelligence, the thalamus was implicated in these correlations in a connectivity analysis, as was the medial temporal lobe (Wilke et al. 2003). Interestingly, the correlation of global gray matter and IQ (as assessed by the Wechsler batteries and thus reflecting mainly general intelligence) only develops during childhood, perhaps lending support to the notion of fluid skills playing a larger role in early childhood. Also, if there is a dissociation of fluid skills and general intelligence in adults in a way that only fluid skills are affected, should there not also be a model for an isolated decrease in general intelligence which could shed additional light on the issues? Finally, could the differential effects of prefrontal cortex lesions in the neonatal period and in adulthood not also be seen as simply being an indication of the generally larger cortical plasticity in children? I am sure others will come up with more, and more serious, issues this model has to accommodate, and this process will be interesting to follow.

Still, it also seems interesting to complement this work with two timely studies published recently. In one fMRI study, Breitenstein et al. (2005) distinguished good learners from bad learners by the amount of hippocampal activation. This is all the more interesting as all subjects were healthy adults, indicating that, employing the right kind of paradigm and using performance data as a guide, it may be possible even in healthy subjects to tease out the different aspects of cognition described by Blair. Even more interesting and lending strong support for one of the main theses of the target article is the study by Heinz et al. (2005). Here, subjects with three genetically defined variants of a serotonin-transporter system were investigated by using fMRI and applying the concept of functional connectivity. This serotonin transporter is believed to play a crucial role in a subject's liability to develop major depression. It could be demonstrated that the strength of the coupling between the amygdala and the ventromedial prefrontal cortex is a function of the genetic variant of the subject. Therefore, a genetic influence on behavior via the pathway that plays a crucial role in Blair's model of cognition-emotion reciprocity is suggested. This adds evidence for a genetic contribution to or modulation of the putative environmental influence that Blair hypothesizes, which (by virtue of lending support to the mechanism in itself) further strengthens the point made about this link.

Overall, I believe this to be a very interesting model which accommodates a number of observations and lends itself to rigorous testing. As it is, however, its virtues, beyond explaining the observed, can be assessed only in years to come, following extensive discussions of the pros and cons. It is as yet too early to decide, but for the sake of children possibly profiting from a more targeted approach to support, I wish the model well.

Can fluid and general intelligence be differentiated in an older adult population?

Nancy A. Zook[a] and Deana B. Davalos[b]

[a]Department of Psychology, Purchase College, State University of New York, Purchase, NY 10577; [b]Department of Psychiatry, University of Colorado Health Sciences Center, Denver, CO 80262.

nancy.zook@purchase.edu **Deana.Davalos@UCHSC.edu**

https://doi.org/10.1017/S0140525X06399037 Published online by Cambridge University Press