power of *g* would likely be attributable to gF′. However, while such an end point would perhaps seem a blow to proponents of psychometric *g*, it would really represent a considerable advance for cognitive psychology and developmental neuroscience. Studies demonstrating influences on fluid cognitive functioning that are distinct from *g* provide an important source of information for the study of relations between mental ability and behavior. In particular, developmental neuroscience work on fluid cognition provides a valid neural architecture for clearly defined cognitive functions and processes that would seem to have brought the study of intelligence very, very close to a neurobiologically grounded explanation for individual differences in *g*. Such a unification of psychometric, componential, and neuroscientific approaches to the study of intelligence has for long been desired, and research relating prefrontal corticolimbic circuitry to working memory and to psychometric *g* would seem to have come as close as possible to filling the bill for the identification of a neural basis for general intelligence (e.g., Duncan et al. 2000). In fact, were it not for the dissociation data outlined in this target article, one might really craft a compelling story regarding individual differences in the neurobiology of the prefrontal cortex and general intelligence. However, it would seem that such an explanation would really amount to nothing more than a crafty story and that *g* remains as inscrutable as ever.

Open Peer Commentary

What we need is better theory, not more data

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Abstract: Although I find Blair's case for arguing for the distinction between fluid cognitive functions and general intelligence less than compelling, I believe him. However, I also believe that what is required next is a theory of both general intelligence and fluid cognitive functions that articulates the distinction. In the absence of this, more data, particularly of the neuroscience variety, is likely to stall rather than advance progress.

I was pretty much on board with the first third of Blair's target article. He does a good job of summarizing a body of research that at least opens the possibility that there is a case for dissociating "fluid cognitive functions" from psychometric *g*. However, the case is not overwhelming, and the road that Blair subsequently takes – to argue that the structure of the brain supports the dissociation – if anything, weakens his case. What the article cries out for is the development of a theory that justifies the dissociation. Such a theory would say something like this: Psychometric *g* maps onto cognitive parameters "a, b, c," whereas fluid cognitive functions map onto a different set ("x, y, z"). In the absence of such a theory we are thrown back on the state of the evidence – which is actually rather poor. Let me unpack this a bit.

Nearly all the evidence in favour of a dissociation between psychometric *g* and fluid cognitive functions boils down to their less than perfect correlation. I am prepared to suspend disbelief that the psychometric measures of *g* really measure *g* (as theoretically unhelpful as that is) because there is a hundred years of convention to go by. But what of the measures of fluid cognitive functions? The first problem is that they are psychometrically much more unreliable. It is little wonder that many studies find that fluid cognitive functions are not perfectly correlated with measures of *g*. "Big deal," I hear my psychometrician friends say, "it is just another example of how cognitive/experimental/neuro/developmental psychologists do not know how to develop useful tests – when they get good at it they will find the correlations high enough to support the case that fluid cognitive functions and *g* are indistinguishable." But this brings me to the more fundamental problem. How do we know that they are measures of fluid cognitive functions, in the absence of a theory of what those functions are? For example, what does a Stroop task, in its many instantiations, measure (if you care to, substitute your favourite "frontal" task)? Is it a measure of (a) resistance to interference or (b) task switching, or (c) working memory capacity, or maybe even (d) speed of processing? The unhelpful answer is, very likely, all of them. But even leaving that aside, each of these constructs are themselves nearly always free-floating in current research. The constructs usually amount to nothing much more than the operationalization of performance on some tasks and are the subject of a "theory" that contains but one reference – and that is itself (a theory of speed of processing, a theory of working memory capacity, or whatever). I suspect that few other commentators will address this issue. Like the blind spot on our collective retinae, we have become so used to it that it is noticed only by those who specially look out for it. Rarely are such constructs pitted against each other for their explanatory value, and almost never do they feature in a wider theory of the structure of the mind/brain.

Sensing that the distinction, if it is real, might be important, Blair then takes two steps. One is a look for corroboration in neuroscience data, and the other is to argue for a new set of measurements of this distinctive construct (fluid cognitive functions). This reminds me of the very strategy that Arthur Jensen has used in his advocacy of psychometric *g* itself (see Anderson [2000], Barrett [2000]; and Jensen [2000a; 2000b] for a discussion). Without knowing what it is that we are looking for, we can either make little sense of some arbitrary data (e.g., positive correlations between IQ and gray matter – how exactly does this speak to the dissociation?), or we resort to forgetting what psychometric *g* is supposed to be about. For example, the claim that data from studies of the amygdala, or whatever, show that emotion and stress are important determinants of fluid cognitive functions is relevant only to the dissociation of fluid cognitive functions and psychometric *g* if we are discussing the presumed cognitive overlap – for by definition there is no emotion or stress content to psychometric *g*. Consequently, this line of evidence and reasoning renders the dissociation vacuous. Further, although new measures of a different construct are a necessary step for science, the new measures that Blair wants to develop seem to be alternative predictors of various real-life behaviours. Therein lies fool's gold – psychometric *g* has already cornered the market.

I do believe Blair's central claim (that fluid cognitive functions show some independence of *g*), but I believe this because I have a theory of cognitive functioning that says it is so (see Anderson 2001). Briefly, this theory says that there are two dimensions to *g* – one related to individual differences in IQ and dependent on variation in speed of processing, and the other developmental, related to mental age and dependent on the maturation of modular functions, some of which are intrinsically related to "executive functioning" (see Anderson 2005). So I find myself in general agreement with Blair's manifesto and in wholehearted agreement that evidence from psychopathology, neuropsychology (where the studies are driven by theory-based hypotheses), and in particular the study of atypical

and typical development, will be crucial for scientific advance. I also agree with the spirit of his final quote. Were it not for the dissociation of fluid cognitive functions and psychometric *g*, there is a compelling story that relates functioning of the prefrontal cortex and general intelligence, but that "such an explanation would really amount to nothing more than a crafty story and that *g* remains as inscrutable as ever" (target article, sect. 8.1, last para.). Trouble is, it is not the *evidence* cited in this review that illuminates *g*, but a theory that says just how *g* and fluid cognitive functions are different.

Heterogeneity in fluid cognition and some neural underpinnings

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Abstract: In agreement with Blair, I favor the idea of dissociative patterns in cognitive performance, even more when it comes to development. However, such dissociations are present not only between fluid cognition and general intelligence, but also within fluid cognition itself. Heterogeneity of executive attention, even when indexed with a single paradigm, is further discussed in relation to anterior cingulate cortex.

Blair's target article raises a critical issue: What should be the key area for diagnosis and intervention in cognitive functioning – particularly when addressing early stages of development? Can the assumption of one and only *g* factor underlying mental abilities really be translated into valuable operational tools, as proposed by traditional psychometrics? The main thesis sustained by the author offers a symmetrically opposed answer – there are patterns in cognitive performance delineating not one, but many factors – which offers itself undoubtedly as a good candidate for assessing individual differences and also for targeting interventions tailored to each individual's needs.

Yet, there must be some caution in treating fluid cognition unitarily – or more specifically, what Blair calls "*g*F'" (that part of fluid cognition independent of *g*). The conceptual interchangeability of fluid cognitive functioning with working memory and executive function(s) as if they are overarching the same construct is useful in contrast to a generic general intelligence, but not really valid when one is trying to clarify the true nature of fluid cognition.

In agreement with the author, I favor the idea of dissociative patterns in cognitive performance, even more so when it comes to development, considering that, as Blair acknowledges, diverse aspects of cognition develop rapidly – but unequally – in early life. During development, dissociations of cognitive functions actually seem to be the rule rather than the exception.

However, I argue that such dissociations are present not only between fluid cognition and general intelligence, but also within fluid cognition itself. The equivalent term for fluid cognition executive function(s) is recognized by many authors as an umbrella concept, encompassing at least working memory, inhibition/inhibitory control/executive attention and flexibility/set shifting (see Miyake et al. 2000).

Inhibitory control/executive attention is an essential construct for both developmental and adult studies. It has a reversed U trajectory, being low in children, high in adults, and low again in elderly people. It has been linked to the developmental progression and further regression of prefrontal/medial frontal structures, and it is thought to be involved in the acquirement of mentalizing abilities. Yet it has proved to be a composite, both theoretically and methodologically (e.g., see the aggregate battery scores proposed by Carlson & Moses 2001). Because many tasks that are claimed to index inhibitory control have additional requirements, they end up measuring other variables, as well; therefore, I suggest that it is rather difficult, but maybe computationally "cleaner," to choose computationally (neurally) well-defined tasks when trying to assess and explain inhibitory control, rather than more complex ecologically valid tasks (like many neuropsychological tasks).

I chose the spatial conflict task (Gerardi-Caulton 2000), a modified version of the Simon task, commonly used to measure conflict resolution in adults (Simon & Bernbaum 1990) and having a neural correlate at the level of the anterior cingulate cortex (ACC), as shown by functional magnetic resonance imaging (fMRI) (Fan et al. 2003). My computerized version consisted of presenting two visual stimuli (e.g., a teddy bear and an apple), either on the right or on the left of the screen, with the subject being instructed to respond according to the identity of the stimulus while ignoring the relation between the location of the image and the location of the appropriate response key. Children (2–7 years of age), typically developed, were tested under three experimental conditions: spatial conflict without any other requirement (similar to the adult version of the task), spatial conflict plus working memory load (the subject having to remember which stimulus was assigned to each response key), and spatial conflict plus reward (each correct response being followed by animation of the stimulus). My surprising results support the heterogeneity of executive attention and the presence of distinctive intra-individual patterns, since I found no correlation between incongruent reaction times (RTs) in the three conditions and no correlation between conflict rates (incongruent minus congruent RTs), the longest RTs being present in the reward condition (Benga 2004).

In neural terms, these results could be related either to the involvement of different brain circuits of the prefrontal-limbic network – proposed also by Blair as subserving fluid cognition – the ACC having only the role of conflict monitoring in each, or to different divisions (e.g., dorsal versus ventral [see Bush et al. 2000]) of the ACC involved in different tasks. Although adult neuroimaging studies have shown the activation of the dorsal ACC (thought to be mediated by the dopaminergic system) for spatial conflict tasks without additional requirements, I suggest the involvement of ventral ACC structures, mediated by an opioid system, in reward-related spatial conflict task. (I propose two different biochemical underpinnings to the ACC divisions, inspired by the two biochemical systems described by Luciana 2001.)

ACC divisions have often been explored in terms of their critical role in regulatory behaviors and cognition-emotion interaction, which is also emphasized by Blair. Moreover, they can be integrated in the larger framework proposed here: the amenability of fluid cognition to experience.

An opioid mediation of the ventral ACC could explain, in this line of thought, its vulnerability toward early disruptions of attachment (see Panksepp [2003] for linking attachment to opioids). I have suggested previously (Benga 2001) that dysfunctions in maternal contingency – leading to alterations in attachment – have disturbing, long-lasting effects upon the ACC, and they could explain why institutionalized children show later in life coupled deficits in executive function and social/emotional behavior (Gunnar 2001; O'Connor et al. 1999). According to the ontogenetic scenario suggested by Posner and Rothbart (1998; 2000), in the second half of the first year of life, ACC comes into function, being initially the center of emotional control and later of cognitive control. The correct maturation and functioning of the ACC might depend on contingent external input, offered by a constant caregiver. Animal models (Mathew et al. 2003) link early disruptions of maternal contingency to later biochemical modifications in the ACC: the decrease in the NAA/Cr indicating a decrease of neuronal viability, and the Glx/Cr ratio suggesting the activation of the hypothalamic-pituitary-adrenal axis.