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A watershed model of individual differences in fluid intelligence

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ABSTRACT

Fluid intelligence is a crucial cognitive ability that predicts key life outcomes across the lifespan. Strong empirical links exist between fluid intelligence and processing speed on the one hand, and white matter integrity and processing speed on the other. We propose a watershed model that integrates these three explanatory levels in a principled manner in a single statistical model, with processing speed and white matter figuring as intermediate endophenotypes. We fit this model in a large (N=555) adult lifespan cohort from the Cambridge Centre for Ageing and Neuroscience (Cam-CAN) using multiple measures of processing speed, white matter health and fluid intelligence. The model fit the data well, outperforming competing models and providing evidence for a many-to-one mapping between white matter integrity, processing speed and fluid intelligence. The model can be naturally extended to integrate other cognitive domains, endophenotypes and genotypes.

1. Introduction

Fluid intelligence, or fluid reasoning, is a core feature of human cognition. It refers to the ability to solve novel, abstract problems that do not depend on task-specific knowledge (Blair, 2006; Carroll, 1993; Deary, 2012; Horn and Cattell, 1966). In contrast to crystallised intelligence, which continues to improve across most of the lifespan, fluid intelligence shows strong age-related declines (Horn and Cattell, 1966; Salthouse, 2009). Understanding the causes of this decline is important for healthy ageing, as preserved fluid intelligence is strongly associated with independent day-to-day functioning (Tucker-Drob, 2011; Willis and Schaie, 1986), and is inversely related to mortality risk (Aichele et al., 2015). At the other end of the lifespan, low fluid intelligence in adolescence predicts poor outcome in later life (Huepe et al., 2011) and is a risk factor for psychopathologies such as schizophrenia (Blair, 2006; Snitz et al., 2006). However, our understanding of how this crucial cognitive ability relates to broader, mechanistic frameworks of cognition and the brain is limited. A promising line of research focuses on the relationships between fluid intelligence, processing speed and white matter organisation. Although intriguing, these empirical relationships are often interpreted in isolation, relating fluid reasoning to processing speed (e.g. Sheppard and Vernon, 2008), processing speed to white matter (e.g. Penke et al., 2010), or fluid intelligence to white matter (e.g. Haász et al., 2013), but never the three together. One unresolved question is therefore whether fluid intelligence, processing speed and white matter can be thought of as part of a single, hierarchical system.

Here, we propose a statistical framework to examine this question, developed by formalizing a conceptual model taken from the literature on psychopathological constructs and their causes. This so-called 'watershed model' (Cannon and Keller, 2006) uses the metaphor of a river system to illustrate how complex behavioural traits can be seen as the downstream consequence of many small upstream (e.g., neural/genetic) contributions. From this perspective, the relationship between fluid intelligence (hereafter FI), processing speed (PS) and white matter (WM) is hierarchical, such that WM influences PS, which in turn affects performance on tests of FI. We show that this model naturally accommodates a wide and disparate range of empirical findings, integrates a series of relatively well-established findings into a single larger model, and, most importantly, can be formally tested using Structural Equation Modelling (SEM). We derive a variety of statistical predictions that follow from the watershed model, and use SEM to test these predictions empirically in a large (N=555), population-

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based sample of ageing adults (18–87 years, Cam-CAN). First, we examine the empirical evidence concerning FI, PS and WM.

2. Processing speed, fluid intelligence and white matter

Processing speed refers to the general speed with which mental computations are performed. It has been considered a central feature of higher cognitive functioning since the development of the first formalized models of (fluid) intelligence (Salthouse, 1982; Spearman, 1927). It shows comparatively steep age-related declines, similar to or even stronger than FI (Horn and Cattell, 1966; Salthouse, 2000; Schaie, 1994), including in longitudinal samples (Deary and Der, 2005). Processing speed is a broad concept that has can be measured in a variety of ways (Salthouse, 2000). One common approach is to use a set of tasks with strict time-limits, and consider the shared variance across those tasks to reflect an individuals' ability to perform cognitive tasks under time pressure (Babcock et al., 1997). Even purely physiological measures have been considered, such as the latency of neural evoked responses (Salthouse, 2000, Schubert et al., 2015). Other possibilities include the parameters estimated from response time distributions in a single task, such as the mean, standard deviation and exponential for an ex-gaussian distribution, or parameters such as drift rate and boundary separation in diffusion models (Matzke and Wagenmakers, 2009; Ratcliff et al., 2016). Here, we focus on the most basic and simple notions of processing speed, sometimes called psychomotor speed, namely the mean and standard deviation of RT distributions for simple tasks.

The empirical association between PS and FI is one of the most robust findings in psychology (Sheppard and Vernon, 2008). This association holds across the lifespan (Salthouse, 1994), in both healthy elderly (Ritchie et al., 2014) and in the extremes of mental retardation (e.g. Kail, 1992). Longitudinal studies of either end of the lifespan show similar patterns. Dougherty and Haith (1997) showed that infant reaction time at 3.5 months predicts IQ several years later, and Fry and Hale (1996) showed in 214 children and adolescents how longitudinal changes in processing speed mediated changes in fluid intelligence and working memory. At the other end of the lifespan, declines in PS and FI show considerable correlations in old age, with estimates ranging from 0.53 (Zimprich and Martin, 2002) to 0.78 (Ritchie et al., 2014). Similarly, a large longitudinal cohort study (Ghisletta et al., 2012) showed that a considerable portion of within-subject age-related decline was shared between FI and PS. Although few studies have explicitly examined the temporal ordering of developmental changes, those that do generally find that declines in PS affect declines in FI and related cognitive abilities. For instance, Kail (2007) examined 185 children (age 8–13) tested twice on multiple outcomes, and found that the best mediation model described a developmental cascade, wherein improvements in processing speed affected working memory which in turn enhanced reasoning. In older adults, Robitaille et al. (2013) showed in two separate cohorts that within-subject declines in processing speed mediated within-subject declines in multiple cognitive domains, including fluid reasoning. Finally, Finkel et al. (2007) used bivariate latent change score models in older adults to show that processing speed was a leading indicator of cognitive changes, including in abstract reasoning tasks. Together, these behavioural findings suggest a strong relationship between processing speed and fluid reasoning ability.

The most common metric of PS is the central tendency, such as the mean or median, of RTs on a simple reaction time task. However, individual differences in the *variability* of RTs also relate to fluid reasoning ability (Rabbitt, 1993), such that less variable responses are associated with higher scores on fluid reasoning tasks. This 'cognitive consistency' in RTs has been shown to predict cognitive performance in elderly subjects beyond mean RT (MacDonald et al., 2009). Both the central tendency and variability of PS predict all-cause mortality (Batterham et al., 2014; Hagger-Johnson et al., 2014), supporting the idea that both are important and independent components of PS. The role of variability can be observed even on the purely neural level: A study using EEG in young adults (Euler et al., 2015) found evidence for the role of variability of neural responses, such that individuals with more stable (less variable) responses to novel stimuli tended to have higher fluid reasoning ability.

Recent work suggests that the proper conceptualisation of the relation between PS and FI is as a causal factor (e.g., Kail, 2000; Rindermann and Neubauer, 2004; Robitaille et al., 2013). The most influential causal account comes from Salthouse (1996), who suggested at least two mechanisms by which PS affects cognitive performance, namely the *limited time mechanism* and the *simultaneity mechanism*. The former suggests that in any timed task, slower speed of processing simply precludes the timely completion of cognitive operations, leading to poorer scores; the latter suggests that high PS is necessary to juggle mental representations simultaneously, in order to perform complex cognitive operations (see Burzynska et al., 2013, for neuroimaging evidence for this claim). More recent work (Schubert et al., 2015) used drift-diffusion and EEG modelling to show that there are multiple components to processing speed, and that these components play different causal roles in different cognitive tasks. In summary, nearly all of the papers reviewed above, either explicitly or implicitly, consider PS to be a 'lower', or more fundamental, mental process that is not identical to FI itself (see also Schubert et al., 2015). We can also go further down this presumed causal hierarchy to understand the possible determinants of PS. One such candidate is the structural organisation of white matter tracts.

Among the most influential studies showing the importance of white matter organisation are two papers by Penke and colleagues, who showed that the first principal component of fractional anisotropy (FA, a measure of white matter organisation) predicted both information processing speed (Penke et al., 2010) as well as general intelligence (Penke et al., 2012). Further work has shown that decreased WM organisation has been associated with decreased PS both in healthy adults (Tuch et al., 2005; Penke et al., 2010) and in individuals suffering from clinical conditions associated with WM loss such as Multiple Sclerosis (Kail, 1997, 1998; Roosendaal et al., 2009; Segura et al., 2010; see Bennett and Madden, 2014, for a review). However, in a sample of 90 older adults, Yang et al. (2014) did not find strong associations between white matter organisation and reaction time components derived from a diffusion model. WM organisation has also been associated with the variability of RTs in children (Tamnes et al., 2012), in healthy controls and preclinical Alzheimer's dementia (Jackson et al., 2012), and decline in WM has been proposed as a key cause of age-related changes in cognition (O'Sullivan et al., 2001). This relationship between WM and performance variability has been found to strengthen with age (Fjell et al., 2011; Laukka et al., 2013; Lövdén et al., 2013b). Other studies have found direct relationships between WM measures and FI (Haász et al., 2013; Kievit et al., 2014) and specific neural (including white matter) structural correlates of intra-individual variability (MacDonald et al., 2009, 2006). Similarly, lesions in WM predict age-related declines in mental speed (Rabbitt et al., 2007a). Assessing a broad set of cognitive and neural markers in a large, age-heterogeneous cohort, Hedden et al. (2014, p. 1) conclude that 'The largest relationships linked FA and striatum volume to processing speed and executive function'.

A critical link in our model is the behavioural consequence of the microstructural properties evident in the white matter