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Short Communication

Brief response to Ashton and colleagues regarding *Fractionating Human Intelligence*Adam Hampshire<sup>c,\*</sup>, Beth Parkin<sup>a</sup>, Roger Highfield<sup>b</sup>, Adrian M. Owen<sup>a</sup><sup>a</sup>The Brain and Mind Institute, The Natural Sciences Centre, Department of Psychology, The University of Western Ontario, London, ON N6A 5B7, Canada<sup>b</sup>Science Museum, Exhibition Road, London SW72DD, UK<sup>c</sup>Cognitive, Computational and Clinical Neuroimaging Lab, Imperial College London, UK

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## ABSTRACT

Ashton and colleagues concede in their response (Ashton, Lee, & Visser, in this issue), that neuroimaging methods provide a relatively unambiguous measure of the levels to which cognitive tasks co-recruit different functional brain networks (task mixing). It is also evident from their response that they now accept that task mixing differs from the blended models of the classic literature. However, they still have not grasped how the neuroimaging data can help to constrain models of the neural basis of higher order 'g'. Specifically, they claim that our analyses are invalid as we assume that functional networks have uncorrelated capacities. They use the simple analogy of a set of exercises that recruit multiple muscle groups to varying extents and highlight the fact that individual differences in strength may correlate across muscle groups. Contrary to their claim, we did not assume in the original article (Hampshire, Highfield, Parkin, & Owen, 2012) that functional networks had uncorrelated capacities; instead, the analyses were specifically designed to estimate the scale of those correlations, which we referred to as spatially 'diffuse' factors.

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## 1. Introduction

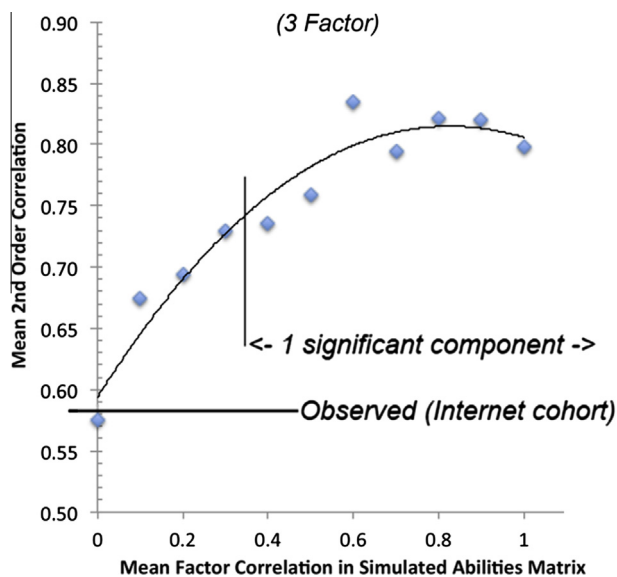
Ashton and colleagues concede in their response (Ashton, Lee, & Visser, in this issue), that neuroimaging methods provide a relatively unambiguous measure of the levels to which cognitive tasks co-recruit different functional brain networks (task mixing). It is also evident from their response that they now accept that task mixing differs from the blended models of the classic literature. However, they still have not grasped how the neuroimaging data can help to constrain models of the neural basis of higher order 'g'. Specifically, they claim that our analyses are invalid as we assume that functional networks have uncorrelated capacities. They use the simple analogy of a set of exercises that recruit multiple muscle groups to varying extents and highlight the fact that individual differences in strength may correlate across muscle groups. Contrary to their claim, we did not assume in the original article (Hampshire, Highfield, Parkin, & Owen, 2012) that functional networks had uncorrelated capacities; instead, the analyses were specifically designed to estimate the scale of those correlations, which we referred to as spatially 'diffuse' factors. This misunderstanding is surprising, because it was clearly stated in the original article that determining "if the capacities of the MDwm and MDr networks

were influenced by some diffuse factor like conductance speed or plasticity" was the primary motivation for running the reported simulations.

Consider, that the neuroimaging analyses provide a relatively unambiguous measure of the extent to which tasks recruit functional brain networks (as conceded by Ashton and colleagues) and that there is a range of possibilities regarding the underlying correlations between the capacities of those networks. At one end of the range, individual differences in the capacities of the networks could be so highly correlated as to be at unity. We know that this is not the case because the task-component loadings would not correspond significantly across imaging and behavioural data sets at this extreme. As stated in the original article, this significant relationship between imaging and behavioural factor models allows for a novel test of how correlated the capacities of the systems are likely to be. Figure 1 clarifies this relationship in the context of the original data. Simulations of individual differences were generated in which the capacities of the underlying networks were correlated to varying levels via loadings on a simulated higher order 'g' factor. As per the original article, simulated performance matrices were generated by multiplying the matrices of simulated network capacities (abilities) by the observed matrix of task-network activations, then adding Gaussian noise scaled by the observed behavioural communalities and a noise level constant. In keeping with Ashton and colleagues' comment regarding latent

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**Fig. 1.** The relationship between the mean correlations in simulated network capacities (input) and the mean correlations between the first 3 latent variables (extracted) when applying Principal Axis Factoring with oblique Promax rotation (SPSS 21 -all setting at default).

variables, here, Principal Axis Factoring was conducted on each simulated performance matrix and Promax oblique rotation was applied to calculate second order correlations. The total variance explained by the first 3 latent variables was held constant at the level observed in the behavioural cohort by adjusting the noise constant.

As can be seen from Fig. 1, the *smallest possible* set of second order correlations is generated when the capacities of the networks are assumed to be uncorrelated. Simply put, the greater the correlation between the network capacities that are input to the simulation, the stronger the correlation between the obliquely orientated latent variables that are extracted (note - adding a fourth general network to the simulation has the same effect as increasing the cross-network correlation in this manner). Plotting

the mean second order correlations from the equivalent factor analysis of the real behavioural data provides an estimate of the level to which network capacities are likely to be correlated. Here, this value intersects the simulated curve remarkably close to 0 on the  $x$ -axis. Thus, as outlined in the original article, when the tendency for tasks to co-recruit multiple independent functional brain networks is considered (task mixing), the results of the behavioural factor model support the view that the capacities of those networks are independent. A secondary but notable point is that *at this level of mixing*, only 1 significant latent variable (using the Kaiser convention) would be observed if correlations in network capacity are  $>0.3$ ; however, three significant variables are evident in the real behavioural data. Thus, the conformity between imaging and behavioural factor models, the placement of the behavioural higher order correlations on the simulated curve, and the number of significant latent variables, provide converging evidence in support of the view that the functional network capacities are largely independent.

Contrary to the suggestion of Ashton and colleagues, no neuroimaging method can accurately measure the capacity of a functional brain network; consequently, that approach for estimating 'g' is entirely intractable. Similarly, there is no method for deriving unambiguous hierarchical factor structure from individual differences in the performances of blended cognitive tasks; thus, and as history has proven, the purely behavioural approach for estimating 'g' is also intractable. However, by combining measures of network mixing from neuroimaging data with measures of individual differences from behavioural data, it is possible to take an approximate gauge of the neural basis of 'g'. As pointed out by Ashton and colleagues if our results are accurate, then they support the hypothesis that the tendency for tasks to recruit multiple networks generates the illusion of a higher order latent variable 'g'.

## References

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