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RE: Comment about 'Fractionating Human Intelligence'. Non-existent flaws in the original article and their relation to limitations of the P-FIT model

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ABSTRACT

In this invited response, we first note that contrary to the claims of Haier and colleagues, they were not part of the review process for the article *Fractionating Human Intelligence*. We then expand on the rationale underlying the primary objective of the study, to determine whether the axes of individual differences in cognitive ability reflect the manner in which the human brain is organised into intrinsic functional networks, and contrast the resultant advance with the more limited aims and methods of the P-FIT model. We address the primary objection of Haier and colleagues by demonstrating that the unrotated model is not only a poorly defined psychological construct, but also neurobiologically implausible in the context of the fMRI data. Finally, we discuss how the distinct strengths of behavioural and neuroimaging data may best be combined in order to refine a model that captures the intrinsic architecture of human cognitive abilities.

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Contents

1. Introduction

It would be inappropriate for us to comment on the editorial policies of the journal *Neuron*. However, we respectfully remind the author that he was asked to write an editorial comment ('a Preview'), not to review the paper. The paper had in fact been rigorously peer-reviewed previously by 3 anonymous individuals who were clearly experts in this field of study and was accepted for publication based on their evaluation of changes that we had made in response to their multi-stage reviews. That is to say, the journal followed standards of practise for peer-reviewed

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scientific publishing. The fact that an invited commentary was evaluated by the Editorial team and not considered worthy of publication is not, in our experience, unusual. Nor is it unusual for researchers within the broader scientific community to question novel ideas that challenge mainstream views. However, the authors of this 'Preview' appear to suggest that (in this case) the peer-review process should have been subverted to accommodate a wider range of uninvited reviews, including those of anonymous bloggers. We also note, that it is somewhat irregular to publish personal email communications without first seeking permission.

In our opinion, the decision of the editors at *Neuron* was most likely to have been made because the points outlined by Haier and colleagues related to their having misunderstood the basic aims, methods and results of our original article, which are quite distinct from those of their P-FIT model. Contrary to their claims, there are no substantial methodological 'flaws' or unintentional 'conceptual confusions' in the original article; rather, our analyses focused on addressing a specific and interdependent series of empirical questions. Consequently, we welcome the opportunity to address their comments in more depth here.

2. Point 1: Qualitative differences in the aims and approaches of *Fractionating Human Intelligence* and the P-FIT model

Haier and colleagues state that the aims of *Fractionating Human Intelligence* (Hampshire, Highfield, Parkin, & Owen, 2012) are similar to those of their P-FIT model — citing 'common interests about the importance of combining neuroimaging with psychometrics.' They claim to have 'used imaging analyses to test and constrain the Parietal Frontal Integration Theory (PFIT) of intelligence (Jung & Haier, 2007).' However, the aims of these two lines of research are altogether distinct. Specifically, Haier and colleagues assume that it is impractical to differentiate between the relative validities of an unrotated factor solution, a rotated factor solution and a hierarchical factor solution. They advocate the unrotated factor solution because it 'minimises judgement calls' and go on to seek a neural analogue. However, they also acknowledge that this approach is limited insofar as the first unrotated factor will certainly produce the largest possible heterogeneous mixture of abilities. Furthermore, they favour the method of applying a prior sub-division of the brain according to Brodmann areas (Jung & Haier, 2007); that is, by cytoarchitectonic sub-divisions. It is well established, that the functional subdivisions of the brain do not map well onto the cytoarchitecture at all. In this respect, they take the largest possible mixture of abilities, and map them onto what are known to be mixtures of brain systems. Unsurprisingly, they observe correlations with many Brodmann areas, but these correlations lack cognitive specificity and spatial resolution. This exercise neither attempts to, nor provides, any advance in our understanding of the architecture of human cognition, or relatedly, the structure of individual differences in cognitive abilities. The rationale provided to justify this limitation is that when estimating factor models 'all solutions can be appropriate in some circumstances and not in others, and evaluating any solution is a matter of judgement, just like deciding whether the most useful view of a building in which you need to do some kind of work depends on whether you're going to be sitting at a desk in an office or fixing the roof'. According to this logic, factor models produce transient descriptions that treat the brain like housed mercurial systems that have no consistent structure. They can be changed according to the needs of the psychometrician or the exact set of tests that are being applied. This rationale may be convenient for explaining away the limitations of psychometric methods (such as PCA and hierarchal factor analyses of behavioural individual differences). However, it is clearly flawed on the grounds of neurobiological plausibility; specifically, neuroimaging research has demonstrated that, like the house in Haier and colleagues' analogy, the brain has a highly consistent structure of intrinsically connected functional networks (Cole, Bassett, Power, Braver, & Petersen, in press; Smith et al., 2009).

By contrast, in *Fractionating Human Intelligence* we propose that the commonly reported intrinsic structure of the human brain underpins distinct axes of human cognitive abilities. This perspective is supported by a rapidly growing neuroimaging literature, which has repeatedly reported highly similar functional brain networks to those in our original article across a wide range of task contexts (Laird et al., 2011). Indeed we have observed the same functional sub-divisions across diverse task manipulations, when contrasting clinical populations (Hampshire, Duncan, & Owen, 2007; Hampshire & Owen, 2006, 2010; Hampshire, Thompson, Duncan, & Owen, 2011), and they are even evident in the fluctuations in functional activations during rest (Dosenbach et al., 2006). That is, the brain is highly structured and has consistent intrinsic functional connectivity patterns. Thus, whilst the structure of cognition undoubtedly has different levels of resolution and, dependent on the perspective offered by the exact choice of paradigms, may appear to differ in a behavioural factor analysis, the intrinsic structure of neural systems that support cognition is relatively consistent. Deriving a behavioural factor model that captures this intrinsic structure is of great practical application, because we know that these functionally distinct sub-regions of the brain are sensitive to different disorders, genotypes and pharmacological interventions. Consequently, a neurobiologically grounded factor model has the potential to provide differential markers of the functioning of these systems and could be used to provide a common framework for sub-classifying clinical populations, benchmarking experimental therapeutics, and translating insights across traditional clinical domains. A single mixed measure of multiple systems with ambiguous orientation as advocated by Haier and colleagues would not be at all well suited for this purpose. Indeed, as is evident from their comments, deriving a neurobiologically grounded multifactorial model of cognitive abilities is a major challenge, and one that they would not have attempted to address. In *Fractionating Human Intelligence*, we sought to derive this model by combing the relative strengths and weaknesses of neuroimaging and behavioural data. We argue that these types of data are complementary as they provide different types of information that relate to the same underlying cognitive systems. In terms of Haier and colleagues' house analogy, these methods provide alternative perspectives that when combined provide a more accurate picture of the intrinsic architecture of human cognition.

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3. Point 2: No 'conceptual confusion' when comparing factor models

Haier and colleagues claim that the comparison of factor models from behavioural and neuroimaging data was 'conceptually confused' because only the former focused on individual differences measures. They suggest that to make such a comparison at all is 'questionable'. On the contrary, whether different components of cognitive ability have analogs in distinct brain networks is an empirical question that was central to our hypothesis; consequently we tested it. This question was critical, because as Haier and colleagues rightly point out 'there is no logical reason to conclude that g is not unitary because two or more brain networks may be involved'. Indeed we did not suggest otherwise, because even if intelligence relates to multiple functionally distinct brain networks, the capacities of those networks could be highly correlated. We referred to this possibility in terms of spatially 'diffuse factors', or in the words of Haier and colleagues, factors that may be 'invisible to the fMRI technique'. Critically, if individual differences in ability were driven *entirely* by spatially diffuse factors, then it would be highly unlikely that the task-network loadings and the task-behavioural component loadings would correlate. That is, the functional sub-divisions observed within MD cortex would not be evident within the behavioural individual differences analysis. Consequently, we tested the hypothesis that there was a relationship between the neuroimaging and behavioural factor models using a rigorous combination of correlation analyses and permutation modelling. The rotated task-component loadings were strongly and significantly correlated. Furthermore, the task-network loadings from the neuroimaging data provided a better predictor of the behavioural data than all 1000 random permutations. These results were exceedingly unlikely to have occurred due to chance. In the original article, we proposed that this relationship could allow the maximum contribution of diffuse factors to 'g' to be estimated, a suggestion that we will revisit in Point 5. We also note that this relationship provides relatively unambiguous evidence in support of our selection of rotated behavioural factor orientations.

4. Point 3: Relative strength of neuroimaging data

One of the core objections of Haier and colleagues is that we used 'factor analysis on brain image voxels to find clusters interpreted as brain networks' but that 'the factor definitions are arbitrary, as the factors can be rotated in many ways'. They propose that 'the unrotated factor, for example, could well be a neuro-g and it is important to consider that alternative interpretation'. In fact, we applied independent component analysis for the bulk of our comparisons between behavioural and brain imaging data, a method that is based on more powerful assumptions and that is used widely throughout the neuroimaging literature. It is also notable, that they do not dispute the fact that an unrotated factor solution would provide the largest possible mixture of functional networks. Moreover, and as discussed above, this mixture would be of no use for the purposes of probing the intrinsic structure of cognitive abilities. In fact, in terms of common structure in the behavioural and neuroimaging data, it should be noted that the first unrotated component from the MD cortex mask had no significant correlation with the first unrotated components from the individual differences data ($r = 0.17$, $p > 0.6$); thus, this unrotated spatial component provides a poor candidate for neuro-g. More importantly, Haier and colleagues have overlooked the fact that neuroimaging data have quite distinct properties relative to behavioural individual differences data. We argue that these distinct properties allow the question of factor orientations to be further disambiguated — a point that we covered briefly in the discussion section of the original article and will be expanded upon below.

When analysing behavioural individual differences data, cognitive abilities are, for all intents and purposes, randomly mixed across individuals. Consequently, the underlying factors/abilities may only be estimated indirectly using the factor analysis. This is a poorly posed problem because there are a vast number of potential orientations that can be applied to such data and as observed by Haier and colleagues, deciding which one to use is a 'judgemental decision'. This is not the case with neuroimaging data because, as opposed to being randomly mixed across space, cognitive systems are *organised* into distributed networks that consist of spatially distinct and often anatomically non-contiguous cortical regions. Multiple Demand Cortex (MD) for example, is composed of a number of distinct sub-regions. These include anterior insula/inferior frontal opercula (AIFO), inferior frontal sulci (IFS), and the inferior parietal cortices (IPC). The AIFO are spatially quite distant from the IFS and are anatomically non-contiguous with IPC. Furthermore, with imaging data one can measure task vs. rest, a contrast for which, to our knowledge, there is no equivalent in behavioural individual differences data. Consequently, it is possible to derive a reasonably direct measure of the relative levels to which one or other sub-region is recruited during performance of a task simply by comparing their mean activation levels during task vs. rest.

These properties can be used to confirm the orientation of the factor model in an unambiguous manner. For example, one can ask whether the functional-anatomical sub-regions from which MD cortex is composed form part of a single, multiple or higher order functional network simply by examining the strengths of the correlations in their activation levels across tasks relative to rest. As can be seen in Table 1, two fully dissociable networks are evident in our data set when analysed in this manner. One network includes the AIFO bilaterally, the other the IFS and IPS bilaterally. There are strong correlations between all regions within each network, in several cases approaching unity, and no significant correlations between networks, in many cases approaching 0. Notably, when task–rest activations are analysed in this manner, there is no requirement for rotation of factors (although it is important to consider that there may be some spatial overlap). Furthermore, and as reported in the original article, when we examined task–rest contrasts using ROIs centred within the ICA networks, we observed that in some task contexts one ROI was highly active whilst there was little activation in the other relative to rest, in another context the opposite pattern was observed, and in others they were strongly co-recruited. This pattern of results conforms to the qualitative definition of independent cognitive systems (Henson, 2006), yet the first

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unrotated component would take the greatest possible linear mixture of the two. Indeed, as discussed above, the AIFO and IFS networks are one of the most consistent features of the cognitive neuroscience literature. Few researchers would consider them to be part of the same functional network. Thus, the unrotated factor model that is advocated by Haier and colleagues not only lacks cognitive specificity, it also provides a far less accurate match for the finer resolution information present in the behavioural factor model and most importantly, it lacks any neurobiological plausibility.

5. Point 4: The complicated problem of analysing individual differences in neuroimaging data

As discussed above, Haier and colleagues have overlooked the fact that neuroimaging data is better suited to deriving a mixing matrix; that is, a matrix that describes the levels to which distinct cognitive systems are recruited during the performances of different tasks. They suggest applying the same behavioural individual differences approach when examining the neuroimaging data. However, this suggestion reveals a clear inconsistency in their thinking. Specifically, they state that only limited insights can be gained by examining the individual differences that they suggest should have been the focus of the analyses. In fact our choice of analysis approach was quite deliberate, because the levels to which networks are typically recruited may be estimated very reliably with a relatively small set of individuals; a fact that is demonstrated by the analyses in which we included each individual's voxelwise data as a discrete set of 12 columns in the PCA. This approach, did not constrain the same tasks to load on the same components across individuals, yet the variance in task-component loadings was surprisingly small as demonstrated in Fig. 1. Thus, our data provided a demonstrably reliable estimate of the relative levels to which each network was recruited during performance of the 12 tasks when analysed at the group level. By contrast, individual differences analyses of these functional networks, whilst certainly being of great interest (and the focus of both our past and ongoing research) are far more complex and unreliable. Indeed, whilst the question of whether there is a higher order network or ability is poorly posed for factor analysis of individual differences in behavioural data, we would argue that it is even more so for analysis of individual differences in neuroimaging data. Consequently, any attempt to determine the contributions to 'g' of spatially diffuse factors in this manner would be altogether misguided because it would provide an amplified variant of the single order vs. higher order ambiguity.

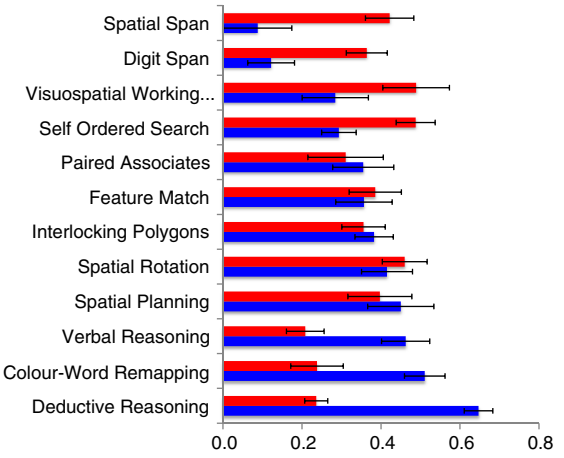

Fig. 1. Reliability of task-component loadings across individuals. When each individual's voxel-wise data were included in the PCA as a set of 12 columns, a method that constrains the same voxels to load onto the same components across individuals; the task-component loadings, which were free to vary across individuals, were observed to be very reliable. Error bars represent SEM.

Neuroimaging data is noisy, and that noise often correlates across spatial locations within the brain. These correlations in turn produce spatial components that do not have a neural origin (e.g. due to head movements, temporal drifts in global signal to noise ratios and spiking artefacts) Haier and colleagues appear to be unaware of this issue, as they suggest that the analysis should have focused on the observation of more than 2 components in three individuals. These additional components are almost certainly noise-related and are unlikely, therefore, to contribute to intelligence. Indeed, the three individuals with more than 2 significant components did not differ markedly from the rest of the cohort in terms of performance. Haier and colleagues might focus on such differences, but they would almost certainly be attempting to interpret movement related noise artefacts. The fact that global brain activation magnitudes and connectivity measures vary across individuals for a range of reasons that do not have a neural origin, means that any second order factor derived from individual differences measures of brain imaging data would be greatly inflated.

Table 1
Correlations between ROI activation levels across tasks show two dissociable networks.

		Right AIFO	Right IFS	Right IPS	Left AIFO	Left IFS
Right AIFO	Pearson correlation Sig. (2-tailed)					
Right IFS	Pearson correlation Sig. (2-tailed)	0.058 0.859				
Right IPS	Pearson correlation Sig. (2-tailed)	−0.18 0.576	**.897**** **0**			
Left AIFO	Pearson correlation Sig. (2-tailed)	**.912**** **0**	0.097 0.764	−0.174 0.588		
Left IFS	Pearson correlation Sig. (2-tailed)	−0.211 0.509	**.729**** **0.007**	**.589*** **0.044**	0 0.999	
Left IPS	Pearson correlation Sig. (2-tailed)	−0.238 0.456	**.724**** **0.008**	**.608*** **0.036**	−0.038 0.908	**.978**** **0**

Significant correlations highlighted in bold. * $p < 0.05$; ** $p < 0.005$.

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This issue is exacerbated further by the fact that the relationship between individual differences in brain activation magnitudes (or structure) and abilities is complex, non-linear and bidirectional in terms of causality. In this respect we agree with Haier and colleagues when they state that 'the brain is a complex electro-chemical organ, with both temporal and spatial characteristics that function in small world networks' and that this 'likely limits the ability of fMRI (or any single neuroimaging modality for that matter) to detect any general characteristic underlying cognition that might be defined as g.' Consider then, that the capacities of the functional networks that we observed will undoubtedly relate to any number of biological variables. For example, many studies have demonstrated that the brain regions that support a cognitive process are often *more active* in those individuals who have a deficit in that process; for example, individuals who have suffered multiple concussions (Hampshire, MacDonald, & Owen, 2013) or who are in the early stages of dementia (Quiroz et al., 2010). Conversely, many other studies have reported that the brain regions that support a cognitive process are *less active* in individuals who have a deficit in those processes; for example, individuals with obsessive–compulsive disorder (Chamberlain et al., 2008) and attentional deficit hyperactivity disorder (Rubia et al., 1999) or who suffer from pathological gambling (Grant et al., 2013). Moreover, environmental factors affect both brain structure and function significantly across short and long time frames (Kempton, et al., 2011; Maguire et al., 1999). Thus, it is not simply the case that the relationship between individual differences in neural activity and cognitive ability is non-linear; in fact, the direction of that relationship can be inverted dependent on the underlying neurobiological difference and the direction of causality is ambiguous. As discussed above, correlating individual differences in measures of an unrotated 'g' component with individual differences in metrics of brain structure or function provides no information about the intrinsic structure of human cognitive abilities. Nor does it provide any insights into the question of whether 'g' has a basis in diffuse neural factors, a specific functional network, or the limitations of behavioural factor analysis methods. For these reasons we did not attempt to measure individual differences in network capacities using neuroimaging; instead we constructed simulations of individual differences based on the statistically robust task-network loadings from the group-level neuroimaging data and assumed that the capacities of those cognitive systems had an approximately Gaussian distribution. This is an approximation, but it is based on a wealth of scientific support.

6. Point 5: Combining neuroimaging and behavioural data can provide novel insights into the basis of higher order 'g'

As observed above, there was a significant relationship between the rotated task-behavioural component loadings and the task-functional network loadings. This relationship allowed us to take an approximate gauge of the likely basis of the second order 'g' factor that can be generated by applying oblique rotation to the first order behavioural components. More specifically, we argued that the neural basis of 'g' could have one of three distinct types of source. (1) A dominant higher order functional network, (for example if the activation levels of the AIFO and IFS networks were highly correlated across tasks). (2) The tendency for tasks to be co-recruited, but to independent (uncorrelated) levels across tasks. This is essentially the same as blended models from the classic literature. (3) Diffuse factors that cause the capacities of dissociable cognitive systems to be correlated and that are not captured by fMRI.

Consider that the neuroimaging data provides a relatively unambiguous measure of the levels to which our 12 tasks co-recruit multiple functionally dissociable networks within

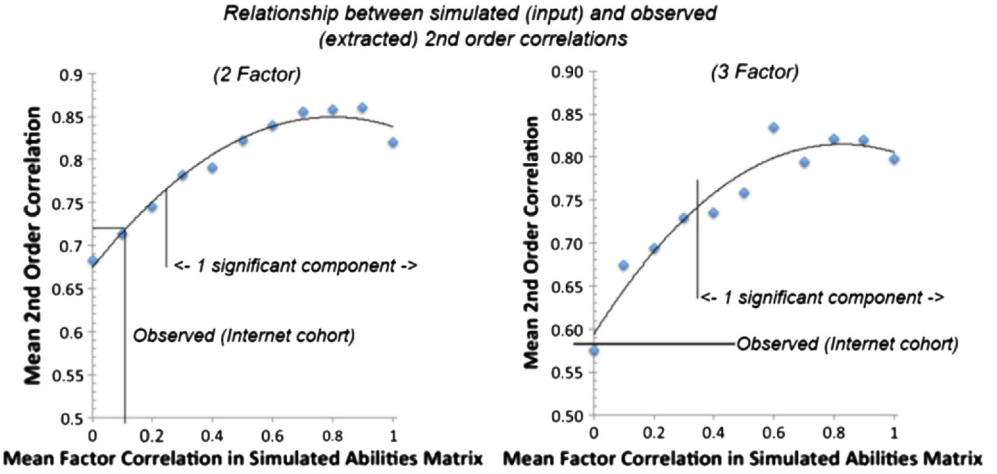

Fig. 2. The relationship between the mean correlations in simulated network capacities (input) and the mean correlations between the latent variables (extracted) when applying Principal Axis Factoring with oblique Promax rotation (SPSS 21 — all setting at default). Simulations of behavioural individual differences were generated using task-network loadings from the neuroimaging data as the mixing matrix and assuming Gaussian distributions of abilities. Increasing the correlations between the underlying factors generated greater correlations between the extracted components when orthogonal rotation was applied. The cross component correlations observed in the real behavioural cohort intersected this function at close to 0 in both the 2- and the 3-factor simulations. Furthermore, when factors correlated at above 0.35, there was only 1 significant component. These results suggest that the higher order 'g' component observed in the behavioural data is primarily a consequence of functional brain networks being co-recruited across multiple task contexts.

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the brain (blending/task mixing). We can effectively rule out (a) because, as discussed above, the networks are spatially distinct and their activation levels relative to rest are qualitatively independent (in fact the mixing matrix from the spatial ICA suggests that the correlation between the activation levels of networks across tasks is somewhat negative). Then there is a range of possibilities regarding the relationship between these networks and the structure of individual differences in intelligence. At one far end of the range, there could be no relationship at all. For example, the capacities of the networks could be at unity due to diffuse factors, or some other cognitive system that is not visible to fMRI could contribute heavily to performances. We know that we are not close to this end of the spectrum because there is a strong and statistically significant conformity between the behavioural and neuroimaging factor models. At the other end of the range, the functional networks could have completely independent capacities, and there could be no additional unmeasured general cognitive system. Fig. 2 captures 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 (simulated abilities) by the observed matrix of task-network activations, then adding Gaussian noise scaled by the observed behavioural communalities and a noise level constant. 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 real behavioural cohort by adjusting the noise constant.

As can be seen from Fig. 2, the *smallest possible* set of second order correlations is generated when the capacities of the networks are assumed to be uncorrelated. 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. 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 curve simulated based on the neuroimaging data remarkably close to 0 on the x-axis. The same is the case when repeating the simulation with just the first two (MD) networks. Thus, when the tendency for tasks to co-recruit multiple independent functional brain networks is considered, the results of the behavioural factor model support the view that the capacities of those networks are largely independent.

A secondary but important 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 >0.35; however, three significant variables are evident in the real behavioural data. 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. We note that adding an extra 'general' network has the same effect as increasing the correlations, as does lowering the overall baseline relative to which the task-network loadings are calculated.

We re-emphasise that the above analyses are intended to provide an *approximation* based on the (widely held) assumption that the capacities of cognitive systems tend to follow a Gaussian distribution across individuals. This distribution of capacities across individuals could have any number of biological contributors and we did not attempt to measure them with neuroimaging in the context of this analysis for the reasons outlined in Point 4. These simulations clearly demonstrate how the 'g' factor that is calculated by hierarchical factor analysis is at the very least, greatly inflated due to blending/task mixing. They also provide a strong indication that diffuse factors are likely to make a relatively small contribution to 'g'.

7. Summary

Contrary to the claims of Haier and colleagues, their P-FIT model and the 3-factor model reported in *Fractionating Human Intelligence* pursue quite distinct hypotheses. Most notably, our hypothesis considers that there is a 'ground truth' structure to human cognitive abilities that has a basis in intrinsic functional networks and seeks to derive that structure by combining the relative strengths and weaknesses of behavioural and neuroimaging data. Specifically, we propose that the issue of ambiguous behavioural factor orientations is rendered more tractable in the context of group-level neuroimaging data, due to the manner in which cognitive systems are organised, as opposed to randomly mixed across the brain. Conversely, we note that individual differences analyses of brain imaging data are complex and plagued by the issues of global noise related components that have a non-neural and therefore, non-intelligence related origin. When combined, neuroimaging and behavioural data may be used to determine whether behavioural components correspond to the contributions of spatially distinct functional brain networks and to constrain their orientations accordingly. They may also be used to estimate the relative scale of the contributions of factors that are not captured by the neuroimaging model but that do contribute to behavioural individual differences.

In contrast, and as demonstrated by the comments of Haier and colleagues, P-FIT considers the derivation of an intrinsic model of human cognition to be intractable; indeed, they appear to argue that there is no point in identifying such intrinsic structure, although their analogy suggests that they may believe that it exists. In this respect, P-FIT treats neuroimaging as a simple exercise in phrenology, whereby a poorly defined cognitive construct, consisting of an arbitrary and heterogeneous mixture of abilities, is mapped onto a general set of locations in the brain, with no consideration of the underlying functional organisation of the cognitive systems housed therein. This approach fails to provide any insights into the intrinsic architecture of human intelligence and has limited application for providing behavioural markers of the functional brain networks that support distinct aspects of cognition.

One might argue that the exact numbers and orientations derived in *Fractionating Human Intelligence* will change somewhat in a larger neuroimaging sample, or that the model

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may vary significantly dependent on the exact population sub-sample or imaging method that is applied. We do not believe that such variations will be of particularly great magnitude because the observed networks are such a consistent feature across individuals and across the neuroimaging literature as a whole. More likely, it will be the case that additional axes of behavioural ability, which were not dissociable in the context of our battery of tests, will be related to additional functional networks within the brain. Similarly, the components from which these networks are composed and their interactions within the temporal domain will likely relate to a finer resolution set of factors. Nonetheless, these are empirical question for future research, which we are actively pursuing at present. Either way, if we are ever going to understand the neurobiological basis of general intelligence, it will be necessary to leverage the relative strengths and weaknesses of both behavioural and neuroimaging data, as opposed to simply imposing the limitations of one upon the other.

Appendix A. Secondary points

Haier and colleagues make a number of less substantial comments, many of which are dealt within the original article. They state that 'Habituation to repeated stimuli during testing depresses activations and subtraction of activation from a resting baseline may not be optimal for finding a common general brain factor involving intelligent performance.' However, we explicitly examined the effects of task familiarity (which as we report elsewhere can be pronounced in some study designs (Erika-Florence, Leech, & Hampshire, in press)) and, as reported in the original article, observed no significant effects. This is likely to be because all of our tests were brief and demanding. No doubt pronounced learning effects would have been evident over a longer time frame.

They also request further details about the whole brain analysis and imply that we should have focused on the P-FIT regions defined by Haier and colleagues as opposed to Multiple Demand Cortex as defined by Duncan et al. There have been many studies that demonstrate that multiple brain regions are critical for intelligence. These typically vary around a frontoparietal theme, with MD regions forming the strongest candidate (Duncan, 2001, 2005, 2006; Duncan & Owen, 2000; Duncan et al., 2000; Woolgar et al., 2010). Consequently, focusing on the MD brain regions that are known to relate to general intelligence is an appropriate, hypothesis-driven strategy that was uncontested by any of the formal Reviewers of the paper. MD was preferred in part because it is a functionally defined mask; we note that P-FIT appears to utilise the cytoarchitectonic parcellation of the brain provided by Brodmann areas. As discussed in the main response, it is well established that functional subdivisions within the brain do not map at all well onto cytoarchitecture. We also note that whole brain analysis in this case would be somewhat inappropriate for our purposes, because it would include systems that are known to be involved in other domains that are not considered akin to intelligence, but that would be recruited during performance of all 12 tasks; for example, motor control and early visual processing. In any case, the original article reported analyses conducted with ROIs at a wide range of different scales — as requested by one of the formal Reviewers. These analyses demonstrated that the factor model was robust against the exact selection of ROIs, which is un-surprising given the reliable nature of blind source localization methods when presented with different sub-sets of the same data. Nonetheless, the first 3 PCA components from an analysis of all active voxels within the brain is reported in Table 2 and can be seen to conform very closely with the task-network loadings from the behavioural cohort (all $r > 0.8$, $p < 0.001$). We note that by contrast, the first unrotated components from the behavioural and whole brain analyses do not correlate significantly ($r = 0.38$, $p = 0.218$). Consequently, the unrotated factor from the neuroimaging data provides a poorer candidate for neuro-g.

Finally, Haier and colleagues argue that a number of relevant studies (including P-FIT) were not referenced in our original article. However, *Fractionating Human Intelligence* was a research article, not a review. The intelligence literature is vast and stretches back more than 100 years. We selected just those articles that we considered to be most relevant to the questions addressed, but concede that many other relevant articles could have been cited.

Table 2
PCA of the whole brain.

	Rotated 3 factor solutions					
	Behavioural			FMRI		
	1 (STM)	2 (reasoning)	3 (verbal)	2 (STM)	1 (reasoning)	3 (verbal)
Spatial span	0.69	0.22	0.05	0.77	0.40	0.27
Digit span	0.26	−0.20	0.71	0.45	0.09	0.83
Spatial rotation	0.14	0.66	0.08	0.49	0.84	0.16
Feature match	0.15	0.57	0.22	0.37	0.87	0.18
Verbal reasoning	0.05	0.33	0.66	0.09	0.78	0.55
Visuospatial working memory	0.69	0.21	0.07	0.78	0.48	0.28
Paired associates	0.58	−0.04	0.25	0.58	0.61	0.41
Spatial planning	0.41	0.45	0.00	0.63	0.61	0.33
Deductive reasoning	0.19	0.52	−0.14	0.36	0.89	0.12
Colour word remapping	0.22	0.35	0.51	0.30	0.48	0.77
Self ordered search	0.62	0.16	0.16	0.78	0.25	0.49
Interlocking polygons	0.00	0.54	0.30	0.37	0.77	0.33

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