Testing the structure of human cognitive ability using evidence obtained from the impact of brain lesions over abilities

John Protzko [a,*], Roberto Colom [b]

[a] *Central Connecticut State University, USA*
[b] *Universidad Autónoma de Madrid, Spain*

ABSTRACT

Here we examine three classes of models regarding the structure of human cognition: common cause models, sampling/network models, and interconnected models. That disparate models can accommodate one of the most globally replicated psychological phenomena—namely, the positive manifold—is an extension of under-determination of theory by data. Statistical fit indices are an insufficient and sometimes intractable method of demarcating between the theories; strict tests and further evidence should be brought to bear on understanding the potential causes of the positive manifold. The cognitive impact of focal cortical lesions allows testing the necessary causal connections predicted by competing models. This evidence shows focal cortical lesions lead to local, not global (across all abilities), deficits. Only models that can accommodate a deficit in a given ability *without* effects on other *covarying* abilities can accommodate focal lesion evidence. After studying how different models pass this test, we suggest bifactor models (class: common cause models) and bond models (class: sampling models) are best supported. In short, competing psychometric models can be informed when their implied causal connections and predictions are tested.

1. Introduction

All cognitive abilities positively covary. People with high performance in one ability tend to show high performance in other abilities, all over the world (Carroll, 1993; Jensen, 1998; Kovacs & Conway, 2019; Spearman, 1904; Tucker-Drob, Braindmaier, & Lindenberger, 2019; Warne & Burningham, 2019). To explain this covariance, causal explanations are required. To understand the relative strength of each explanation, we can test the implications of the models using evidence from neuroscience.

Covariance between two variables (A & B) can occur for any of six reasons: A causes B, B causes A, A and B cause one another, A and B share a common cause, A and B share a collider, and their covariance is spurious (see also Rohrer, 2018). These are not mutually exclusive and multiple can be operative at the same time leading to the covariance. As the positive manifold is not an artifact or spurious (Spearman, 1904; Jensen, 1998, Kovacs & Conway, 2019, Tucker-Drob et al., 2019; cf. Anderson, 2017) nor does it only shows up in some populations (Warne & Burningham, 2019), the reason must involve causal connections in some way. Importantly, all theories about the cause of the positive manifold have testable causal hypotheses that we explore here.

In parallel, neuropsychological research has shown chronic *focal* cortical lesions lead to local, not global (e.g. not across *all* abilities), deficits (Luria, 2012; Ruíz Sánchez de León, Quiroga, & Colom, 2019). This has been shown relying on clinical case studies (Vaidya, Pujara, Petrides, Murray, & Fellows, 2019), as well as analyzing relatively large samples using neuroimaging approaches such as voxel-based lesion symptom mapping (VBLSM; e.g. Barbey et al., 2012; Barbey, Colom, Paul, & Grafman, 2014; Gläscher et al., 2009, 2010).

The purpose here is to bring these two worlds together, cognitive structure of individual differences (psychometric approach) and findings from lesion studies regarding impact over cognitive performance (neuroscience approach) to discuss what bearing lesion evidence has for understanding the causes of the positive manifold and the structure of human intelligence. This will be done through the lens of causality and causal implications of measurement models. We focus our attention on *chronic* lesions because, as underscored by Vaidya et al. (2019) these lesions "can reveal the necessary contributions of damaged brain regions that are not recovered by reorganization and plasticity" (p. 660). This might shed light beyond the usual statistical comparison among candidate models which, as we will see, leads to conceptual dead ends. Finally, we take a falsifiability approach. Instead of searching for

evidence that confirm a theory, we look at the testable causal implications of different theories and see how those testable (yet often une-numerated) predictions play out. To this end, we do not aim to show how certain psychometric models inform neuroscience, as it is entirely possible the causal structure represented by a psychometric model does not reflect brain organization. As psychometric models are causal theories, however, neuroscience can inform the plausibility of the necessarily implied causal effects of different models.

The value for individual differences research is enumerating how model fit can fail to differentiate among theories of the positive manifold. We highlight how neuropsychology is relevant by allowing causal tests of the necessary implications of different theories. Efforts invested to connect individual differences research and neuropsychology might also help to alleviate limitations in both worlds (see McFarland, 2017, 2019).

We show the positive manifold must occur because of some causal reasons. Different explanations have distinguishable implications for what would happen if a local manipulation were applied to only some mental abilities. Focal chronic cortical lesions are one example of such a local manipulation. As these lesions show local instead of global effects, explanations for the positive manifold predicting spreading activation hardly account for available lesion evidence, while explanations that necessarily imply no such transfer between abilities are more plausible.

The article is organized as follows: Section 2 introduces different classes and explanations for the positive manifold. Section 3 presents the problem of trying to differentiate which ones may be more likely. Section 4 introduces the neuropsychology evidence showing chronic focal cortical lesions lead to local not global intellectual deficits. Section 5 describes how the necessarily causal implications of different models accommodate the focal lesion locality effects. Section 6 summarizes key conclusions.

2. Three classes of explanations of the positive manifold

The positive manifold (Table 1) could occur for a number of reasons albeit known explanations usually fall into one of three classes: (1) common cause, (2) interconnected (causal interactions), and (3) sampling models (see Table 2).

We discuss the logic behind these models before exploring their predictions and interrogating brain lesion studies for finding candidate answers beyond the psychometric approach.

2.1. Common cause models

This class posits the positive manifold occurs because on top of measuring local (specific) abilities, each test is also measuring the same underlying general ability. These structures allow for clusters of sub-factors explaining why, for example, verbal abilities correlate more strongly with other verbal abilities (e.g. Jensen, 1998).

Correlated factor models (Fig. 1A) posit sub-factors cause differences in subtest performance, with the clusters of sub-factors covarying with one another (for example: McAuley & White, 2011; Miyake et al., 2000; Neubert, Kretzschmar, Wüstenberg, & Greiff, 2015). These sub-factors could be considered specialized packets of independent abilities (e.g. Fodor, 1983, 1985). The number and nature of these sub-factors has been a matter of intense investigation, both psychometrically (see, for example: Thurstone, 1935; Cattell, 1943; Vernon, 1964; Horn, 1976; Johnson & Bouchard Jr, 2005; McGrew, 2009; Schneider & McGrew, 2018) and in cognitive neuroscience (see, for example, Sporns & Betzel, 2016) but such considerations of number and nature are not of interest to the main goal of the present article. The key point is all sub-factors tend to correlate with one another, necessitating explanation. In correlated factor models, however, there is no attempt to explain this covariation.

In hierarchical models (Fig. 1B), performance on individual tests covary (the positive manifold) but are also clustered together into sub-factors. These are related abilities like verbal ability, visuospatial ability, processing speed—also covary, necessitating a further explanation which, in these models, is that there is a higher-order factor, *g* in this case, *causing* differences in the sub-factors. A misconception is such common-cause variables must be mental processes. It may be the case that the cause of covarying sub-factors is environmental factors (Dickens & Flynn, 2001), neural architecture (e.g. Garlick, 2002; see also Anderson, 2017) or cellular effects (Geary, 2018). If the positive manifold results from shared genes across cognitive processes, for example, it would manifest either because of a common cause (shared genes) or a bifactor model (if residual covariance occurred within cognitive processes, such as verbal abilities correlating together).[1]

Finally, in bifactor models (e.g. Gignac, 2016; Fig. 1C) there are clusters of sub-factors whose intercorrelations are explained by a separate second-order factor, *g*, also present in all of the subtests. All subtests correlate with one another (the positive manifold) because, to some extent, they are all *also* measuring the same underlying ability (*g*). Of particular interest is that correlated factor models show the exact same statistical fit as the equivalent hierarchical model when the number of sub-factors is low (2–3;e.g. Gignac & Kretzschmar, 2017). Because of this equal fit, other considerations must be taken into account for evaluating between these models.

An under-appreciated fact is the bifactor model is often more in line with conceptualizations of *g* than is the hierarchical model. Consider the following: "As long as a task is at least somewhat cognitive in nature, it will be at least a partial measure of individuals' general cognitive ability. Spearman (1927, pp. 197–198) called this the "indifference of the indicator"" (Warne & Burningham, 2019, p. 3). This notion captures the idea that *g* is being *directly* measured in an individual subtest which can only correspond to the bifactor model (due to the presence of causal arrows from *g* directly to the subtest). In hierarchical models, there are no such direct causal pathways from *g* to subtest performance not through a sub-factor.

2.2. Interconnected models

Another class of models of the positive manifold present the explanation in terms of causal interactions. Network models are one such class of explanation (Fig. 2A), where networks of either cognitive abilities or brain regions causally interact to give rise to an emergent process (intelligence; Barbey, 2017; Fig. 2B). Statistically, network models are simply another way of modelling interconnections among variables, with individual cognitive abilities as 'nodes' and correlations between them as 'edges', for example (e.g. Fig. 2A).

As networks are simply another way of modelling the same intercorrelations among objects, for network models to contribute to scientific theory they must identify control or driver nodes (Pósfai, Liu, Slotine, & Barabási, 2013). Driver nodes are the subset of nodes that have causal power—where externally manipulating the node can move the network to a different state—whereas non-driver nodes and their edges are simple covariances due to non-direct causal connections (such as common cause; Barabási, 2016). Thus, from a causal perspective, driver nodes represent those connections that are causal to the behavior of the system.

Statistical networks can be understood as behavioral or neural correlations. During performance on manifold cognitive measures, common neural brain regions, largely in the Parietal and Frontal regions (see Jung & Haier, 2007), are active (see Duncan & Owen, 2000; see also Assem, Glasser, Van Essen, & Duncan, 2020; Duncan, Assem, & Shashidhara, 2020; Kievit et al., 2018). If we ascribe causality to the common regions, then they may either act as driver nodes within the neural network, or the model may be conceptualized more as a hierarchical model (if the direction of causality runs from the common brain regions

---

[1] We thank an anonymous reviewer for pressing us on this point.

Table 1

Correlation matrix for 9 WAIS-IV subtests completed by 1002 individuals representative of the population in Spain. The positive manifold is shown. All correlations are positive regardless of the type of intelligence subtest (verbal, visuospatial, and processing speed in this instance). However, correlations are higher within cognitive domains. Data from the WAIS-IV standardization sample in Spain (Wechsler, de la Guía, & Vallar, 2012).

	SIM	VOC	INF	BD	MAT	PUZ	SS	COD	CAN
Similarities		0.68	0.67	0.59	0.65	0.57	0.60	0.63	0.28
Vocabulary			0.63	0.53	0.60	0.50	0.55	0.59	0.21
Information				0.54	0.59	0.50	0.52	0.56	0.24
Block Design					0.76	0.74	0.72	0.74	0.33
Matrices						0.71	0.72	0.76	0.38
Puzzles							0.66	0.67	0.29
Symbol Search								0.86	0.51
Coding									0.49
Cancelation									

Table 2

Three classes of explanations for the positive manifold.

Model	
Class	Examples
Common Cause	Correlated Factors, Hierarchical, Bifactor
Interconnected	Network, Investment, Mutualism
Sampling	Bonds, POT (Process Overlap Theory)

directly to the recruited abilities (sub-factors)) or as a bifactor model (if the direction of causality runs from the common brain regions to subtest performance, bypassing the sub-factors).

Explanations of the positive manifold may take also the causal interactionist approach. These explanations posit either direct or indirect causal relations among the cognitive abilities themselves. What start as uncorrelated, or minimally correlated abilities, become more heavily correlated over time through causal connection and reinforcement (Cattell, 1963 for Gf-Gc model; see also Anderson, 2005, 2017; and Schweizer & Koch, 2002 for a different 'first cause' starting ability). Thus, causal feedback mechanisms create the positive intercorrelations of sub-factors. In this way, the positive manifold at the subtest level is an artifact of the causal processes that led to the sub-factors becoming correlated. Sub-factors cause performance on their constituent subtests; the positive manifold at the subtest level is an effect of investment at the higher-level.

Another similar model is dynamic mutualism (van der Maas et al., 2006; Kievit et al., 2017; built off the multiplier model by Dickens & Flynn, 2001). This model posits a number of elementary cognitive processes present at birth that are functionally uncorrelated. The elementary processes are, however, *causally* connected over time: "In [dynamic mutualism], all processes of the system are initially undeveloped and uncorrelated. During the development of the system, the dynamical interactions give rise to correlations among the processes of the system." (van der Maas et al., 2006, p. 844). Thus, dynamic mutualism is, of necessity, a causal model positing cognitive abilities cause development in other abilities over time (Kan, van der Maas, & Levine, 2019).

2.3. Sampling models

Sampling models posit the positive manifold occurs from a large number of elementary cognitive processes. These processes are not as broad as, say, 'Verbal Ability' but often much narrower (e.g. Bartholomew, Deary, & Lawn, 2009; Detterman, 1987; Thomson, 1916), as small as individual neurons or nerve cells (Thomson, 1951). When a given subtest is administered, the process of solving that test recruits a large, but *incomplete*, number of possible bonds. When a separate subtest ostensibly measuring a different process is administered, different bonds are excited to solve the items.

Crucially, a subset of the bonds excited between the two subtests overlap. In this way, performance on the tests covary because they involve shared cognitive processes/bonds (Fig. 3A).
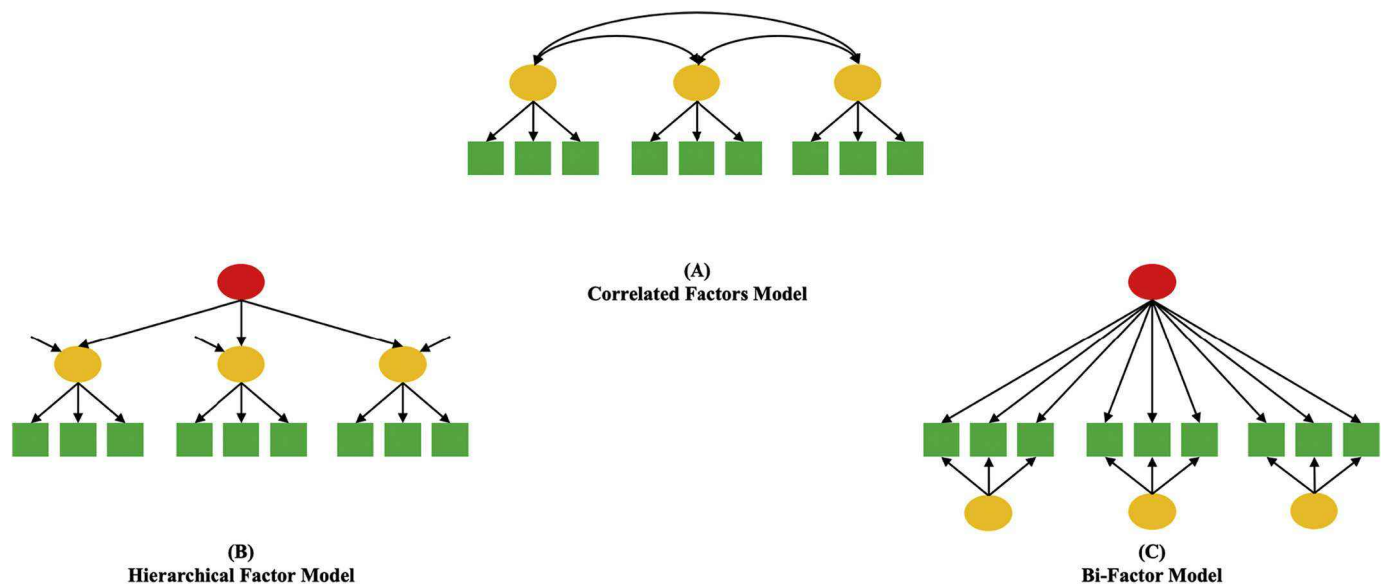
Fig. 1. Examples of common cause models: (A) correlated factors model; (B) hierarchical model; (C) bifactor model. Green squares represent manifest variables, yellow circles represent sub-factors, red circles represent *g*. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
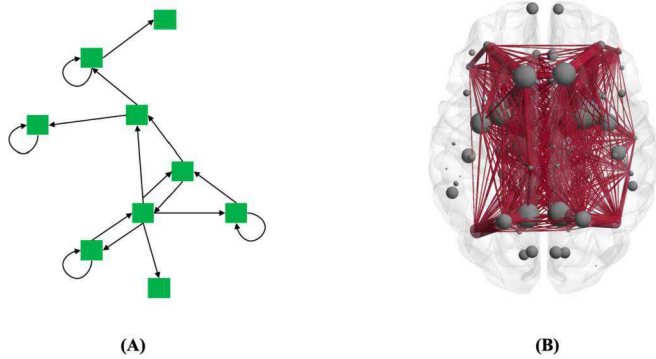
Fig. 2. Examples of interconnected models. (A) The model on the left represents a network-type connection where abilities (modelled as manifest variables) are causally connected. The three nodes connected by double arrows, as well as the node in the bottom right can causally connect to every other node in the network. (B) The figure on the right represents such an approach as applied to brain systems.

As shown in Fig. 3B, elementary processes (yellow circles) differentially cause performance on individual subtests. These subtests are then combined into component scores. These components replace sub-factors in that they are observed (not latent) and caused by (not causing) performance on the subtests. Overall scores are mostly additive combinations of these components, mirroring the actual process of scoring IQ assessment batteries (e.g. Lichtenberger & Kaufman, 2009; Roid & Barram, 2004; Süß & Beauducel, 2015).

The Process Overlap Theory is a complex extension of the sampling model (POT; Kovacs & Conway, 2016b, 2016a, 2019). When taking a cognitive task, demands activate relevant processes. These elementary processes are more concentrated within traditional sub-factors instead of every test randomly sampling the same large number of distributed processes (in the Bonds model) and represent cognitive operations more complex than single neuron behavior (Fig. 3C). Second, a *g*-factor is *caused* by the underlying sub-factors which corral the elementary processes. Literally, *g* emerges from the floors below: "the positive manifold is an emergent property and, consequently, it translates to a formative model with regard to the general factor…the result of the specific patterns in which items response processes overlap…a result of how processes overlap to produce cognitive activity required by mental tests" (Kovacs & Conway, 2016a, pp. 162–171). Thus, IQ or *g* can then be conceptualized as formative things, i.e. cognitive indices. Third, the

combination of mental processes is multiplicative, not additive as in the bonds model (Kovacs & Conway, 2016b). Finally, performance on the sub-factors are constrained through an Executive Function (EF) filter and its basic component processes (updating, shifting, and inhibition; Miyake et al., 2000) (shown in the Gf circle of Fig. 3C). This means that the intercorrelations of sub-tests is different for people with different EF levels. Low EF levels constrain the performance of sub-factors to be equal, so sub-factor differences are more equal and a higher *observed* positive manifold appears. High EF levels do not constrain the performance and the positive manifold may be weaker as some sub-factors may be high or low in individuals (see Anderson, 1992 for a similar gating mechanism giving rise to covariance through processing speed). This observed pattern of results is referred to as cognitive differentiation (Abad, Colom, Juan-Espinosa, & García, 2003; Molenaar, Dolan, Wicherts, & van der Maas, 2010).

3. Which model is more likely?

After seeing the classes of explanations for the positive manifold and some examples of those classes, the next question naturally emerges: 'Which one is more likely?'. Historically, in the context of hierarchical models, when determining among competing models, the same data is fit to different models (different sub-factors) and measures of global statistical fit are calculated (e.g. CFI, RMSEA, χ^2, BIC). Then, the model with the 'best' global fit is retained and deemed the frontrunner (see Johnson & Bouchard Jr, 2005 for a paradigmatic example).

This approach, however, has a number of shortcomings. The most pertinent is global-model-fit comparisons work best for nested models. Therefore, testing non-nested models of different structure using global fit statistics becomes difficult (though certainly not impossible; see Merkle, You, & Preacher, 2016). Second, different models on the same data may show nearly the same global fit, leaving the outcome of such comparisons to tenuous principles such as 'parsimony'. Third, global model statistical fit is an insufficient method of theory comparison. As each model and (especially) class of models represent different scientific theories with their own unique testable hypotheses, simple fit to data alone cannot demarcate among them.

3.1. Model equivalence/underdetermination

In the world of global statistical model fit comparisons, a situation often arises where two models yield identical global fit statistics. This result is referred to as model equivalence, but this statistical version is simply a subtype of a much larger scientific problem referred to as 'underdetermination of theory by data'. In short, different theories can equally account for any data. Model equivalence is a problem not only when comparing multiple models within the same 'class' but especially across classes (see also Lee & Hershberger, 1990).

Take the following from the attempts to explain the positive manifold as an example. Correlated factor models yield identical global fit statistics with the same model expressed as a hierarchical factor model with usual 2–3 sub-factors (Gignac & Kretzschmar, 2017). Thomson's Bonds model also provide identical fit to the same data as a hierarchical model (Bartholomew et al., 2009). By extension, the Bonds model is therefore statistically equivalent with correlated factor models as well. Furthermore, bifactor models have been shown to generally fit better than hierarchical models (Gignac, 2016), meaning they also fit better than correlated factor models and fit better than Bonds models and any other equivalent model. At the time of this writing, it is unclear whether other sampling or interconnection models would also provide equivalent global model fit, but it would be unsurprising if such models were capable of doing so. Thus, especially across non-nested models, comparisons of global model fit on the same data are insufficient for determining which class of models or even specific models of the positive manifold is 'correct' as they are often equivalent.

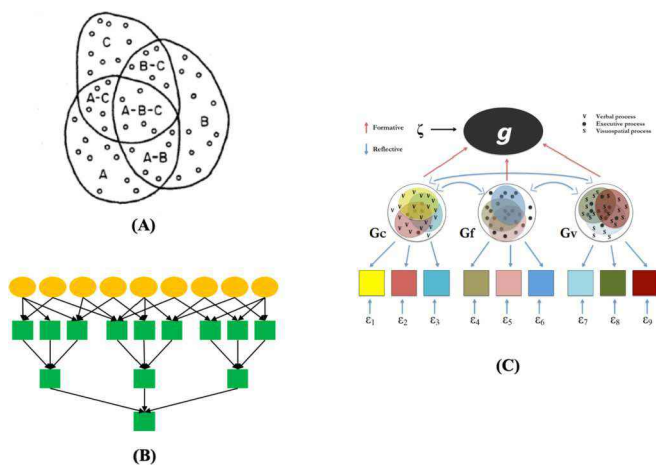All models are incomplete representations of reality. Hierarchical

Fig. 3. Sampling models: (A) Classic Thomson's model; (B) Bonds model of intelligence as seen in clinical intelligence testing; (C) Process Overlap Theory (from Kovacs & Conway, 2016a) Gc = Crystallized Intelligence, Gf = Fluid Intelligence, Gv = Visuospatial Intelligence.

models cannot easily accommodate different developmental patterns (e.g. Heckman, 2006; Kievit et al., 2017). Bifactor models can appear to falsely fit data because of problems with heterogeneity in the population (Raykov, Marcoulides, Menold, & Harrison, 2018). Sampling and Network models have difficulty accounting for the failure of transfer from increasing one cognitive variable not altering the behavior of another (Detterman & Sternberg, 1993; Protzko, 2017; Protzko & Bailey, *Under Review*). Causal Interactionist models have difficulty accounting for the fact that raising intelligence at one point does not cause an increase or stable effects at later ages (Protzko, 2015, 2016; Bailey et al., 2017).

A relevant example from the history of intelligence research involves whether a single cognitive process or multiple cognitive processes could explain a common cause for the positive manifold. One analysis showed separate independent processes could better explain the positive manifold than a common cause (Kranzler & Jensen, 1991). A re-analysis of the same dataset, however, showed the opposite conclusion (Carroll, 1991). The method of inquiry, factor analysis in this example, was unable to differentiate the two competing accounts.

So, if the positive manifold can be accommodated by numerous, even potentially contradictory, yet statistically identically fitting, theories (enumerated here and future unknown theories; e.g. building on Schmiedek et al., 2019) that are all incomplete—what can be done to determine which theory(ies) is(are) more likely? This is where theories have to do more than just explain existing data. They must provide novel testable hypotheses. This latter requirement to generate testable predictions and not just account for data is the very foundation of the aversion to overfitting models. Models that are overfit to data *necessarily* provide better fit, yet we often reject them because the predictions they offer are viewed as unlikely to occur. For demarcating between explanations for the positive manifold it is crucial to move beyond statistical modelling and fit indices. The question is not 'how can we best *measure* future evidence and the positive manifold', but instead, *what testable predictions do our measurement models make?* It is such hypotheses that we will connect below with the research on brain lesion studies.

3.2. Measurement models are causal

The paths in measurement models are directed causal paths. The basic justification of factor models is grounded in causality (see Borsboom, 2005; Borsboom, Mellenbergh, & Van Heerden, 2003; Pearl, 2009). In short, variation in performance on a series of items or subtests cannot be attributed to a latent ability unless the latent ability is considered to cause differences in the behavior of interest.

This was not always the case, however. Older views of latent factors as 'measurement' rested on the assumptions of operationalism, that the definition of a construct was in what it measured; or logical positivism, that measurement is a placeholder or 'promissory note' (c.f. Borsboom, 2005) or a 'working reference frame' (Cronbach & Meehl, 1955). Thus, any concept of measurement was operationalized as behaviors (in this case, test performance). Such views have long been rejected as being unsustainable as ways to conceptualize measurement (in psychological research, see, for example, Borsboom, Cramer, Kievit, Scholten, & Franić, 2009; Maul, Irribarra, & Wilson, 2016). The justification of claims of latent variable measurement at present rests on these causal assumptions; a latent variable causes performance in the reflective behaviors or items (Borsboom, 2005; Hausman & Woodward, 1999).

This causal implications of theories are what can allow to evaluate among theories of the positive manifold. A hierarchical model of intelligence, as shown in Fig. 1B, makes the explicit causal hypothesis that a change to *g*, the higher order factor, will *cause* a change in sub-factor performance and subsequently a change in subtest performance (see Protzko, 2017, for an elaboration on this argument). This causal effect will be proportional to the loading of *g* onto the sub-factors and the sub-factors onto the subtests (see the proportionality constraint; Gignac, 2016). Contrasting this with the bifactor model in Fig. 1C, a change in *g*

will cause a change in performance on each of the subtests (to different degrees) *but no change in common performance summarized by the sub-factors* (this explicitly modelled in the absence of causal connections from *g* to the sub-factors). These causal paths allow for testing different measurement models against one another in stricter ways than model fit statistics. The evidence becomes weaker when the paths in the measurement model are correlational and not causal. Dynamic causal models like mutualism, even without reference to latent variables, make the explicit assumption that alterations in one variable will cause unfolding changes in other variables through development.

3.3. Causes, not covariance

To summarize before moving on, here we take the stance that any covariance observed between two variables (A & B) occur for one or more of six reasons: 1) A causes B; 2) B causes A; 3) A and B cause one another in reciprocal interactions; 4) A & B share a common cause; 5) A & B share a collider; 6) The covariance is spurious. In possibilities 1–4, covariance is subsumed under causality. Possibility 5, colliders, occurs when two variables are uncorrelated in the population, but both share a common effect in a sampled subpopulation. An example of colliders would be: suppose having a pleasant singing voice is unrelated to memory for text in the population. Yet having a pleasant singing voice and good memory for text both cause an increase in the likelihood of being a stage actor. If one were to do a study using only musical theater actors, one may see a spurious covariance between singing voice quality and memory for text (see also Berkson, 1946; see also Anderson, 2017 for a different theoretical take in intelligence). Regarding colliders or spurious covariation, enough research has been done around the world in dozens of disparate societies since the early 20th century to refute collider bias or spuriousness (Warne & Burningham, 2019). Thus, the positive manifold *must* occur because of causal connections (the first four reasons). We call this the causal necessity argument.

This eliminates the correlated factors model (Fig. 1A) from consideration unless the covariance between sub-factors is turned into causal connections (where one sub-factor causes another sub-factor either directly, indirectly, or in a reciprocal interaction). This would mean the correlated factor model decomposes into either a network model (for cross-sectional theories) or a type of causal interactionist model (for longitudinal theories) or a hierarchical model (for sharing a common cause).

As any explanation for the positive manifold must decompose into a model with causal connections (by the causal necessity argument), those causal connections provide the opportunity to put statistical measurement models to experimental tests. This 'experimental psychometrics' (Protzko, 2017) allows for testing between measurement models.

This leaves us at the following point: What is needed is supplementary causal evidence providing tests of the (necessarily) causal connections implied in any adequate explanation of the positive manifold. Again, this is not a function of how to model the positive manifold, but of taking the measurement models at face value, observing what necessarily causal testable predictions each of those models make (as qualitative prediction, not measurement model fit), and comparing observed evidence of those implications. If a theory makes testable causal predictions that are not borne out, this counts as evidence against the theory (in a falsificationist framework of evidence).

3.4. Causation through manipulation

One way to obtain evidence for causality is through manipulability. In short, X has an unconditional causal effect on Y if manipulations to X lead to changes in Y (Woodward, 2003). If a manipulation of X does not alter Y, it may be inferred that no unconditional causal effect exists between X and Y. This conception of causal evidence is important and relevant to the discussion here because, as we will see, if an effect (e.g. focal and chronic cortical lesion) affects A but not B, yet A and B covary

and must be causally connected (via the causal necessity argument), then A cannot be an unconditional cause of B, nor can the manipulation have altered any common cause of A and B. Any explanation of the positive manifold that implies an unconditional cause between A and B would thus be evidenced against.

Unlike work looking at manipulations such as cognitive training, drugs (e.g. Schubert et al., 2018) or early environmental interventions as a way to increase a given ability (see Protzko, 2017; Protzko & Bailey, *Under Review*), manipulability can easily work when *reducing* a local ability. The effects of focal chronic cortical lesions follow a consistent and somewhat predictable pattern of effects. Focal chronic lesions to the cortex lead to local instead of global cognitive deficits. Here global means across all cognitive abilities. We next review some of the available evidence as it might help to provide clarity to the testing among causal explanations for the positive manifold.

4. Brain lesion mapping and cognitive effects

Structural and functional brain imaging studies based on the individual differences approach report correlations between variations in brain features and cognitive differences usually measured by standardized tests (Basten, Hilger, & Fieback, 2015; Colom, 2014; Colom, Karama, Jung, & Haier, 2010; Colom & Thompson, 2011; Dubois & Adolphs, 2016; Haier, 2017). The obtained outcomes are correlations and do not tell if the identified brain regions are *causally* involved in the cognitive differences. Brain lesion studies, however, can tell whether a manipulation to brain region X leads to an impairment in cognitive function Y supporting the measured differences (Barbey et al., 2012, 2014; Gläscher et al., 2009, 2010).

4.1. Local instead of global deficits

The basic maxim of focal chronic cortical lesions leads to local instead of global deficits has been realized since the beginning of lesion research. Research on the effects of lesions on cognitive functions started small with single case studies (e.g. H.M. (Dittrich, 2017; Scoville & Milner, 1957), and Phineas Gage (e.g. Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Van Horn et al., 2012) are well-known examples). As the field progressed, however, research began to incorporate larger samples, more circumscribed lesions, and comparison (control) individuals. Often, these latter individuals would be patients living similar lives (e.g. in the same mental hospital for non-lesion reasons) or patients with focal lesions in different regions (see Price, 2018 for the argument from dissociation).

Much of the work looking at the behavioral spread of focal damage restricts itself to tasks that are relevant to the damaged area. For example, in studies of patients with right cortical lesion damage to white matter tracts, certain lesions are shown to alter spatial neglect while other regions to object-centered neglect (e.g. Vaessen, Saj, Lovblad, Gschwind, & Vuilleumier, 2016). Yet measures of verbal ability or global processing speed are often not measured in these studies. Again, the simple reason is likely why bother to test verbal ability deficits to right cortical damage when most verbal regions are located in the left hemisphere. Such evidence cannot be used for our purposes here because the very point is to understand not just *what* is affected by damage to region X, but more specifically what *is not affected* yet covaries with the abilities affected by region X which must be causally related in some way. Thus, we must restrict the literature search to studies involving not just focal lesions (instead of lesions grouped together by entire hemispheres, see Theiling, Petermann, & Daseking, 2013 for example) and cognitive measurement, but studies investigating a breadth of abilities not a priori believed to be affected (dissociation).

From this literature, we see the same pattern of focal chronic cortical lesions leading to local, not global deficits. Lesions to the frontal or prefrontal cortex leads to deficits in working memory ability, as well as in the ability to plan–out a sequence of moves (McFie, 1961); spatial

short-term memory, however, is completely unaffected compared to age and pre-morbid IQ matched controls (Owen, Downes, Sahakian, Polkey, & Robbins, 1990) or patients with posterior lesions (Duncan, Burgess, & Emslie, 1995). Yet, via the positive manifold, short-term memory strongly covaries with working memory and sequencing (Colom, Shih, Flores-Mendoza, & Quiroga, 2006; Unsworth & Engle, 2007)—and via the causal necessity argument, they must be causally related in some way. Thus, from prefrontal cortical lesions studies it cannot be the case that working memory or sequencing causes short-term memory. This is because exogenous manipulations to working memory and sequencing lead to no change in short-term memory.

Narrowing the size of the lesion, damage to the dorsolateral prefrontal cortex leads to deficits in executive function (EF). However, crystallized knowledge, the bulk of information already stored, shows no such deficit compared to children with either focal lesions in other areas or psychiatric disorders (Filley, Young, Reardon, & Wilkening, 1999). Yet EFs covary with crystallized knowledge (Friedman et al., 2006) and must be causally related in some way. Thus, from dorsolateral prefrontal cortical lesion studies, EF likely are not causal to crystallized knowledge as manipulations to EF lead to no change in crystallized knowledge.

Narrowing still, lesions to the caudate nucleus lead to deficits in problem solving ability and short-term memory (Mendez, Adams, & Lewandowski, 1989). However, compared to a control group, caudate lesions have no effect on subtest performance based on verbal ability at least two months later. Thus, if verbal ability and problem-solving ability/short-term memory covary, must be causally related in some way, yet manipulations to problem solving ability and short-term memory lead to no changes in verbal ability over the span of two months, problem solving ability and short-term memory do not cause verbal ability on a relatively short timescale.

The work explored here compares patients with focal chronic lesions to controls of some sort. In some instances, this involves measuring pre-morbid IQ via vocabulary or reading tests such as the National Adult Reading test (Blair & Spreen, 1989; Nelson & Willison, 1991). Implicit in the use of such measures is that reading ability is not affected by frontal lobe damage. One cannot accurately measure pre-morbid IQ via reading ability if reading ability is reduced by frontal lobe damage. Therefore, either the use of such measures to approximate pre-morbid IQ is inherently flawed, or such measures are valid predictors but unaffected by many focal lesions, adding to the evidence that not all covarying abilities are causally affected by focal lesions. Narrower investigations, involving damage to smaller and smaller regions, comparing voxel to voxel across large numbers of participants, can lead to even stronger evidence, as we see next.

### 4.2. Voxel-based lesion symptom mapping (VBLSM)

VBLSM compares the psychological features of interest of patients with a chronic lesion at a given voxel with patients without a lesion at that voxel (Fig. 4). Unlike functional neuroimaging studies –which usually rely on the brain metabolic demands and provide a correlational association between brain and psychological signals—VBLSM can identify regions playing a possibly causal role, based on mapping which damaged voxel is associated with cognitive impairments. This involves studies in which a typical psychometric/neuropsychological test battery are given to large groups of patients with lesions in different regions across the brain.

VBLSM relates patients' psychological scores to their individual lesion pattern. Thus, for instance, after analyzing 241 patients with single, focal, stable, and chronic brain lesions, lesions within a circumscribed set of areas of the frontal and parietal left hemisphere were related to deficits in general intelligence (*g*) scores extracted from a nine-subtest battery (Fig. 5) (Gläscher et al., 2010).

Instead of focusing on *g*, VBLSM can be used to identify lesions in which specific voxels correspond to deficits either in subtest or subfactor abilities. In this regard, visuospatial skills (Picture Arrangement,
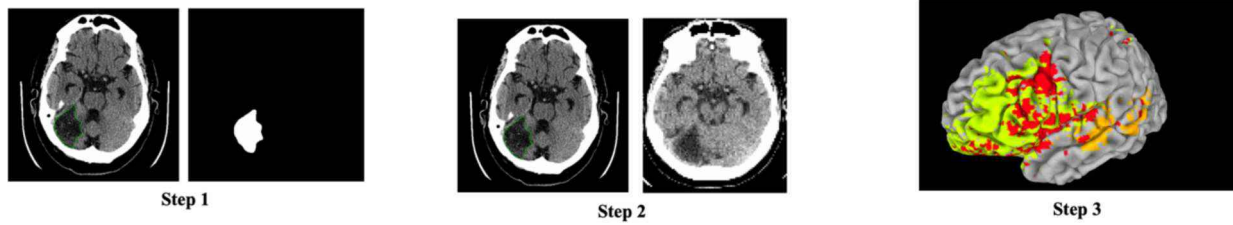
Fig. 4. Summary of steps for computing VBLSM: (1) Creation of masks, using the original/native registration, for identifying the damaged area; (2) Normalization of the native image with respect to a reference group to improve comparability across individual brains, (3) Application of the masks created in step 1 and normalized in step 2 to the psychological outcomes of interest. This allows the visualization of which damaged regions do have impact over the measured psychological variables. The figure at the far right shows an example using data from the Barbey et al.'s (2012) large-scale lesion study.
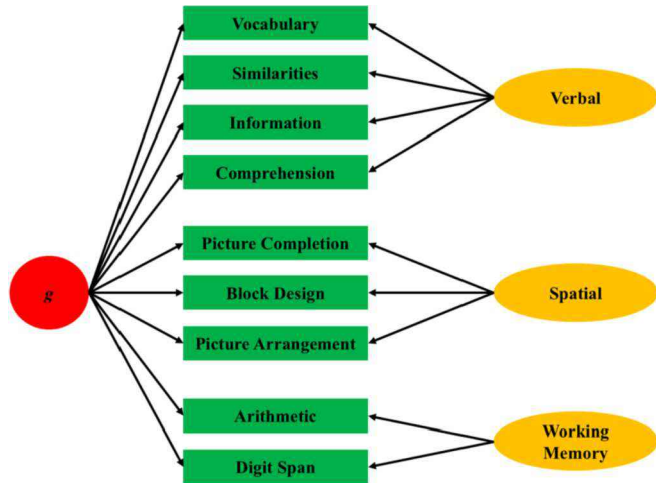
Fig. 5. Measurement model including 9 subtests from the Wechsler battery completed by the patients in the Gläscher et al.'s (2010) study. The factor structure obtained for the patients replicated the observed with the general population.

Block Design, and Picture Completion) seem vulnerable to damage in areas of the right hemisphere. Damage to these regions cause deficits in visuospatial skills, but not deficits in other cognitive abilities. As shown in Fig. 6, the lesion map for similarities (figuring out how two words are related, such as how are chop and carve related?) shows remarkable

overlap with the lesion map for *g*, whereas the lesion map for block design (manipulating colored blocks to match a given drawing) shows meager overlap with the lesion map for *g*. Interestingly, however, the psychometric *g* loadings of similarities and block design are almost identical (0.61 and 0.57, respectively). Furthermore, given the positive manifold, similarities and block design performance is correlated—and given the causal necessity argument, any explanation must be causal in nature.

Overall, lesions in one brain region cause local deficits. This point becomes important to understand the causal connections implied by the measurement models depicted in Figs. 1, 2 & 3, considered in psychometric research, when understanding the structure of intelligence and ultimately more and less likely reasons for the positive manifold.

5. What do lesion studies tell about the positive manifold and the structure of human cognition?

Now we can assemble the pieces and see what evidence lesion studies might play in the psychometric structure of cognitive abilities. Again, the question is not how to measure the effects of lesions from a modelling framework, but what causal and testable predictions different models for the positive manifold necessarily make. The pattern from the lesion evidence reviewed above is indeed focal chronic cortical damage leads to local cognitive effects. It is important to point out that there is no a priori reason for this to be the case. If the brain were an entire causal network where every node was a driver node, any lesion anywhere could impact the whole system. Luckily for human functioning, this is not the case. The brain does not follow a simple 'all nodes are causal' perspective. One of the main benefits of a network with non-
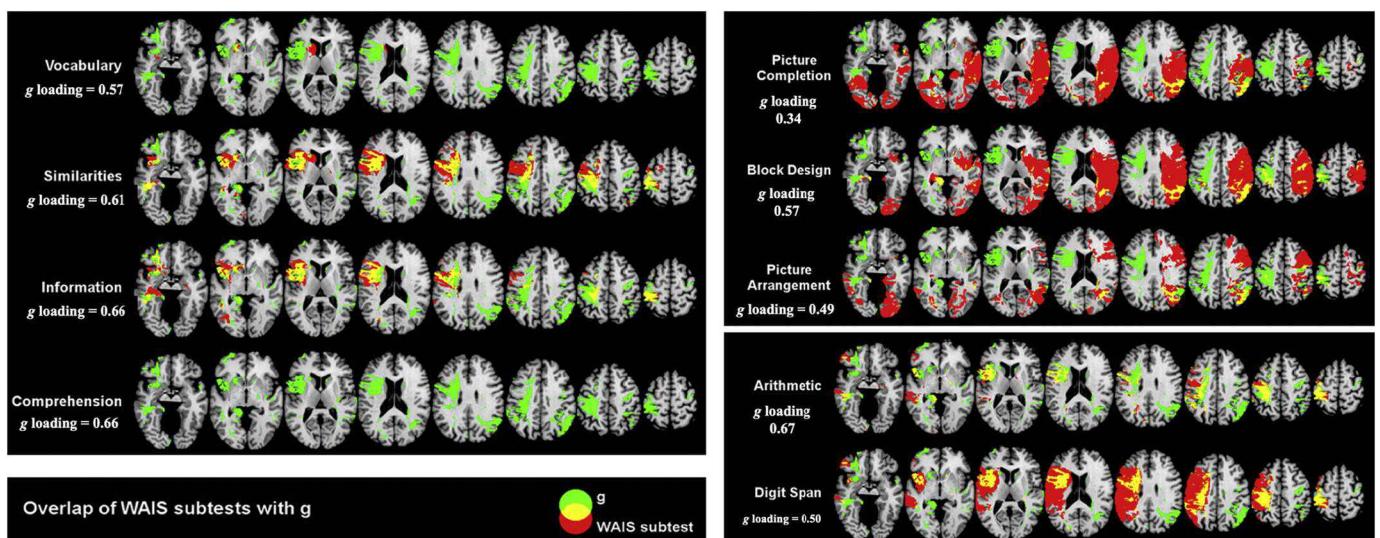
Fig. 6. Overlap between each of the nine subtests from the Wechsler battery (WAIS) completed by the patients and the lesion map obtained for the general factor of intelligence (*g*). The psychometric *g* loading computed for each subtest is also shown (adapted from Gläscher et al., 2010).

driver nodes over a centralized system is precisely its robustness (in a systemic sense) to disruptions (Santarnecchi, Rossi, & Rossi, 2015; Santonja et al., 2021).

Some important points to consider before we combine the two strands. First, cortical lesions can damage multiple psychological functions. Furthermore, lesions can be large and distributed, challenging the definition of 'focal'. Per the evidence provided above of increasingly focal lesions, while multiple processes are impaired, not all covarying (and necessarily causally connected in some way) abilities are impaired. This was shown not only in the work using VBLSM, but also the work on caudate nucleus damage impairing problem solving and short-term memory but not verbal ability; as well as dlPFC lesions causing a deficit in EF, but not in crystallized knowledge (all compared to controls). It is the dissociation of lesion effects that are relevant here.

Second, lesion studies are complex, especially when combined with the inherent degeneracy of the brain (many cortical regions may be required for one function, one region may confer many functions, and so forth). Yet this complexity is not a concern for the arguments here. The central feature is investigating causality through manipulability (Woodward, 2003). If an ability such as EF can be manipulated and it causes changes in other abilities, then we have good reason to believe that EF is causally related to those other abilities on the timescale under investigation. If we manipulate EF and no change is seen in crystallized knowledge (Gc), for example, we may discard the idea that EF causes Gc on an immediate timescale. If we manipulate EF and a change is seen in *later* Gc, it may help establish a developmental causal connection (see Section 5.4 below for an expounding on this developmental causality).

As a reminder, a psychometric model like that found in Fig. 1B makes the explicit causal prediction that a manipulation to one sub-factor (make it EF) will cause changes in subtests only causally reflective of that sub-factor. Furthermore, there will be no causal effect 'upwards' to *g*, nor 'across' to other sub-factors (call one crystallized knowledge). This is explicitly modelled in the hierarchical structure by the presence and (crucially) the absence of causal pathways between the variables. To test the causal predictions of such a model, we can manipulate the sub-factor EF (through any means) and see whether there is predicted *absence* of changes in crystallized knowledge (see Protzko, 2017 for an elaboration and examples).

Thus, it may appear odd that there is no need to understand the specific neural-to-psychological processes of the specific brain regions under question (cf. Krakauer, Ghazanfar, Gomez-Marin, MacIver, & Poeppel, 2017). The central argument (a form of black-box causation) again laid out is as follows: (a) Psychometric models of the positive manifold are causal models. (b) Causal models entail necessary testable causal predictions. (c) The best test of competing models is to manipulate an element of the model and see if the necessarily entailed results play out. (d) Focal lesions represent such a valid manipulation. And (e) as long as the lesion does not also lesion regions responsible for other cognitive abilities (e.g. focal), the dissociation of psychological effects tests the entailed predictions of the different psychometric models. This is how neuroscience can inform psychometrics, by providing tests to the causal implications of the psychometric models.

To develop the key argument, take as an example a focal lesion to the right inferior frontal cortex (rIFC). Based on neuroanatomical studies, we would expect this focal damage leads to local effects only. Indeed, such damage has been shown to affect the inhibition component of EF, but little else (Aron, Robbins, & Poldrack, 2004; Swick, Ashley, & Turken, 2008). Thus, lesions to this region represent a manipulation to inhibitory control. We can then look at what different psychometric theories predict will happen when there is a manipulation to inhibitory control (or EF as a standalone sub-factor). We expect to observe no deficits to crystallized knowledge or processing speed, or verbal abilities etc. as determined by the pattern of frontal evidence reviewed above. Furthermore, this damage lies outside of the left frontal pole, and as such does not show deficits in a higher-order *g*-factor (if necessitated by theory; Gläscher et al., 2010). With this pattern of responses in hand as

an example, we can explore the causal connections necessitated in the proposed models to explain the positive manifold. Importantly, our argument is not restricted to the rIFC and is informed by the whole pattern of lesion studies reviewed above. We only present this as an illustrative example.

5.1. Hierarchical and bifactor models

In a hierarchical model (Fig. 1B), rIFC damage would correspond to a deficit in a sub-factor. We would therefore expect to see performance deficits on any subtest that is reflective of the sub-factor (due to the presence of causal arrows to the manifest variables). We would *not*, however, expect to see any deficits in unrelated sub-factors (as there is no causal connection between them). As well, we would not expect to see any deficit in *g*, as there is no 'upwards' causation to *g* modelled. As the lesion only affects EF/inhibitory control, but not *g* or other abilities in our example, this pattern of results is therefore indirect evidence in favor of a hierarchical model of intelligence, as the patterns of effects from such a lesion matches the expected causal pathways and their determined effects.

Bifactor models (Fig. 1C) are able to accommodate the example but do so in a very interesting way. The rIFC lesion would correspond to a deficit in a sub-factor which would then manifest as deficits in performance on only those tasks reflective of this factor. As in the hierarchical model, there is no expected deficits in effects on other sub-factors or *g*. So far, the bifactor model is indirectly supported by the pattern of deficits from lesion studies. For manifest performance on a cognitive test in the bifactor model, however, there is also a direct influence of *g* on the test, on top of the causal effect from the sub-factor. It can be the case, therefore, that damage to the sub-factor can lead to *undetectable* deficits in a causally reflective subtest scores because of the superior performance of *g*. This pattern of results is a well-known phenomenon in the neuroscience literature referred to as the cognitive reserve hypothesis. People with higher levels of general intelligence are more likely to 'adapt' or 'hide' their deficits caused by lesions or cognitive deterioration (Katzman et al., 1988, Santarnecchi et al., 2015; Santonja et al., 2021). This is a place a bifactor model is better able to explain effects than a hierarchical factor model. In the hierarchical factor model, there is no connection to test performance from *g* that does not go through the damaged sub-factor. Therefore, there is no causal path available for cognitive reserve to increase the performance on the test. In the bifactor model, however, there is such a causal direct path from *g* to test performance. Only the bifactor model contains a direct path from a theorized *g* to specific subtest performance (see Gignac, 2016). The direct path onto manifest performance not through a damaged sub-factor is what is required for cognitive reserve to operate. Therefore, a bifactor model is able to explain an aspect of lesion evidence that the hierarchical model cannot. While both can accommodate the causal predictions based on lesion evidence, bifactor models are better able to accommodate additional neuropsychological findings.

5.2. Correlated factors and network models

The correlated factor model cannot explain the positive manifold as the correlated links must, by necessity, be causal. As noted above, if we make the links causal, we can treat them the same way we would address a network model (Fig. 2B; see Epskamp, Rhemtulla, & Borsboom, 2016 for an example). In a network model, the paths between sub-factors are causal. Therefore, lesion to the rIFC, using the working example, would be predicted to *cause deficits in other—causally connected variables*. But this is not what is observed on a short timescale (see Section 5.4 below for evidence on developmental timescales). Thus, the pattern of lesion studies is indirect evidence *against* a causal network model of the positive manifold. The way to bypass such disconfirming evidence would be to remove the connections between the damaged node and other unaffected-by-the-example-lesion nodes. As inhibition covaries with, for

example, vocabulary (via the positive manifold), such a model would therefore be unable to explain such covariance without resorting to a common-cause model for why inhibitory control and vocabulary covary, yet EF does not cause vocabulary.

Therefore, cross-sectional network models are unable to accommodate lesion evidence as the connections between covarying variables are unlikely to be causal (due to a lack of spreading loss from a focal lesion to all other abilities). As to date there has been no focal cortical brain region where manipulations cause diffuse effects, we cannot as yet claim any of the nodes are driver nodes. It may be the case in the future some driver node is identified where minor manipulations cause changes in all covarying abilities. But these connections so far, however, cannot submit such connections as covarying but not causal via the causal necessity argument.

Another implication of network models is the common Frontal and Parietal brain regions active across tasks (e.g. Duncan & Owen, 2000). Common brain regions across tasks being activated across multiple tasks, with lesions to those areas not causing diffuse performance deficits, might appear to present a paradox. We think, however, this more appropriately stands as evidence either against the causal role these regions play as driver nodes in a network model, or evidence that the regions act more in a bifactor model with cognitive reserve described earlier able to 'pick up the slack'. Future research will help tease these implications out.

5.3. Sampling models

Bonds models are able to accommodate the pattern of lesion effects, although to do so they must engage in semicircular reasoning. In the bonds model, there is no covariance whatsoever between the bonds outside of being sampled by a given subtest. Therefore, if one were able to create the ideal test that only samples one bond (the key research goal of J. P. Guilford, 1967; theorized to be impossible by Lumsden, 1976 and Detterman, 1987), test performance would be uncorrelated in the population with another test that did not sample the isolate bond. Bonds can be as small as an individual neuron in Thompson's bonds model (Thomson, 1951), and the pattern of lesion studies are at the moment far too global and not granular enough to test such a prediction.

Assuming we could, however, the following prediction would be made (Fig. 3B). Suppose damage to the rIFC was exceptionally small and located on one bond. We would only see manifest deficits on cognitive tests that are causally reflective of it and no other tests. As this is the pattern of results observed in lesion studies, it is indirect evidence in favor of bonds models. The only hesitation should be that in the absence of causal and testable pathways in bonds models, the predictions become 'lesions only affect what they affect and not affect what they do not affect' which is an unimpressive truism. If such theoretical bonds exist, some bonds may surely affect more tasks than others, so when it comes to the effects of focal brain damage, the effect can be restricted to just a small number of tasks, or more widespread. Thus, bonds models are indirectly supported by the lesion evidence.

The POT model (Fig. 3C), a complex variant of sampling models, provides more testable predictions. Using the example, damage to the rIFC would provide a local deficit in a specific process (inhibitory control). That decrement would lead to a strong constraining of additional processes. Meaning, damaging the rIFC would lead to two processes unrelated to inhibitory control, such as reaction time and vocabulary, to become more highly correlated (as they would be constrained via the EF gating filter). Covariance, however, cannot be increased without performance on the abilities also being altered. Therefore, damage to inhibitory control would have to transfer to other abilities, even if the decrement performance was not due to a loss of those abilities but a constrained gating mechanism. One aspect in favor of POT is it may partially account for the cognitive reserve hypothesis as long as it is restricted to within-domains. Part of the POT model involves a within-domain compensatory mechanism. Thus, within a domain very local

damage may be hidden by heightened undamaged processes within that same domain. What POT explicitly does not allow, however, would be cross-domain compensation (e.g. mental rotation deficit compensated by *g* or verbal ability or any cross-domain process). Therefore, future work and refinements to POT would require taking these issues into account.

5.4. Causal interaction models

Causal interaction models are also unable to accommodate the pattern of effects seen in lesion studies. Unlike other models which aim to explain immediate covariance, these developmental models require longer timescales to evaluate causal implications. For example, in Cattell's Investment model, such local damage as to the rIFC would be too 'small' to provide indirect evidence, as the pattern of results would unlikely lead to an overall deficit in fluid abilities (as the pattern of effects are more local). Therefore, in the example of damage to the right inferior cortex, the evidence is neutral. If we instead increase lesion size to the prefrontal cortex, causing a resultant decrease in fluid intelligence at the earliest ages, we can see how the effects play out.

Investment models, where fluid abilities are 'invested' into verbal abilities, make the prediction that manipulations to early fluid abilities will manifest as long-term changes in both verbal and fluid abilities. Dynamic mutualism (van der Maas et al., 2006) gives no preference to one ability over others as the 'driver' of cognitive development and instead each ability is invested causally into all others. Thus, any manipulation to one ability will be reflected in long-term changes in all abilities. Therefore, the simple case of childhood cortical lesions to right frontal regions causing a decrement in fluid abilities, under both investment and mutualistic models, will manifest as long-term decrements in verbal abilities under these models.

Although there is a large literature on focal cortical lesions in childhood, the developmental subset of the literature relevant here is exceedingly small. Four requirements are needed to be filled to properly test the causal implications of such developmental models: 1) Focal cortical lesions need to occur in childhood; 2) to test developmental models, long-term follow-up years after the early lesion must be investigated; 3) To test the manipulation-induced causal assumptions, follow-up measurements must break down the outcomes by focal lesion site; and 4) follow-up measurements must include a range of dissociable intelligence outcomes (e.g. not just Full-Scale IQ or local abilities only). To our knowledge and through an extensive search, no study to date has reported results properly fulfilling all four categories. Thus, we describe results relevant to three of the four requirements. Studies with only two or less of the four (e.g. McFie, 1961; Woods & Teuber, 1978; Woods, 1980; Aram & Ekelman, 1986; Riva & Cazzaniga, 1986; Levine, Huttenlocher, Banich, & Duda, 1987; Nass, Peterson, & Koch, 1989; Aram & Eisele, 1994; Raz et al., 1994; Muter, Taylor, & Vargha-Khadem, 1997; Filley et al., 1999; Anderson, Catroppa, Rosenfeld, Haritou, & Morse, 2000; Jacobs and Anderson., 2002; Pavlovic et al., 2006; Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2007; Jacobs, Harvey, & Anderson, 2007; Duval et al., 2008; Long et al., 2011; Gingras & Braun, 2018) do not contain enough evidence for our purposes here. Furthermore, studies without lesions but instead looking at IQ changes over time in those with endogenous diagnoses, also do not contain the level of causality for the purposes aimed here (e.g. Felton, Naylor, & Wood, 1990; see also Ackerman, Weir, Holloway, & Dykman, 1995).

5.4.1. Long-term outcomes reported by focal lesions site with dissociable intelligence data not in children

McFie (1960) compared 206 individuals with focal lesions to one of six regions: left or right frontal, temporal, or parietal regions when they were middle-aged. The comparison group in this study was the age-matched subtest scores from the norming sample of the WAIS. In this study patients with right frontal lesions showed worse performance on reasoning and processing speed measures such as picture arrangement

and digit-symbol substitution, but showed absolutely no deficits in vocabulary or other verbal tasks such as similarities. Thus, a manipulation can alter some abilities that continue to show up decades later, but not others. This suggests there cannot be a direct or indirect causal connection between all abilities. It is important to point out, however, that this study excluded participants who had sustained the injury at birth or in early childhood. It is therefore not as clear the specific age of lesion onset interacting with age. This is particularly important as age of lesion onset has implications for intellectual development (discussed below). Furthermore, the age of lesion was not reported, so the full implications of developmental models cannot be assessed from this data alone.

Another study looked at the impact of focal traumatic head wounds in World War II vets 30 years after the lesion, compared with vets who sustained peripheral nerve injury (Corkin, Rosen, Sullivan, & Clegg, 1989). Looking just at the vocabulary subtest, damage to all locations led to a developmentally exacerbated decline in vocabulary except for damage to the right parietal lobe. Damage to the right parietal lobe, however, did lead to developmental deficits in perceptual field dependence and spatial tests such as block design. Thus, again, focal damage can lead to developmental deficits in some abilities but not others, indicating that a manipulation can alter some abilities (spatial ability) but not others (vocabulary) in adults. Therefore, if causal interactionist models apply to intellectual development throughout the lifespan, this evidence would stand as counterevidence to the models implied effects. If the causal interactions only apply to childhood, this evidence is neutral to the models.

5.4.2. Long-term outcomes from focal childhood lesions with dissociable intelligence data not reported by lesion location

Westmacott, Askalan, Macgregor, Anderson, and Deveber (2010) looked at 145 children who suffered a unilateral ischemic stroke, with follow-up tests done between 1 and 8 years later with dissociable intelligence data. Unfortunately, the results were not broken down by site of focal lesion on verbal vs. performance IQ deficits. Results did show, however, that intellectual outcomes were *worst* for children suffering lesions perinatally than in middle childhood through adolescence. Therefore, there are likely developmental effects, but the relevance to certain structures of the positive manifold are unclear.

Montour-Proulx et al. (2004) investigated 257 children with cortical lesions to only one region (coded as frontal, parietal, temporal, or occipital) sustained when they were 5 years old and tested them when they were 13 years old. Although this study found that lesions sustained earlier in life lead to worse intellectual outcomes, there was no breakdown of the dissociable intelligence data by brain region.

Hajek et al. (2014) looked at 36 children following arterial ischemic strokes compared to 15 children with asthma. While the children suffered lesions as early as perinatally and had follow-up assessments on average 5 years later, the data was not broken down by lesion location on the dissociable intelligence data. Measures like inhibitory control were particularly affected, but without the data broken down by lesion location this cannot be interpreted for our purposes here.

Braun et al. (2002) looked at 357 adolescents (mean age 17yo) who suffered a unilateral focal lesion 12 years prior. Unfortunately, the results were not broken down by lesion site beyond left/right hemisphere despite having dissociable intelligence data.

5.4.3. Long-term outcomes from non-focal childhood lesions with dissociable intelligence data reported by lesion location

Westmacott, MacGregor, Askalan, and deVeber (2009) investigated the effects of unilateral lesions in 3.5–6 year-olds ($N = 26$) three to six years after their ischemic strokes. While the data provided dissociable verbal and performance IQ measures, from early childhood lesions with adequate follow-up, the breadth of the lesions were too wide to be classified as focal (on average covering multiple cortical lobes). No child had a focal right frontal lesion, only one child had a focal frontal lesion

(to the left side) showing significant deficits in all intellectual abilities except for perceptual reasoning/organization at age 10. Thus, while close, the absence of focal-ness of the lesions in this study corrupt the manipulability argument as it cannot be clear what else is affected by the diffuse lesions.

5.4.4. All four requirements combined?

As noted, no single study contains all four of the required components to adequately test certain developmental models (early focal lesions, long-term follow-up, dissociable intelligence data, and reported by lesion site). Perhaps the closest comes from work showing lesions to the right prefrontal cortex in 2–3 year-olds led to deficits in sustained attention when they were around 11 years old, with no concomitant deficit in processing speed (measured by the Trails Making Test, version A; Anderson, Jacobs, & Harvey, 2005). Unfortunately, the results of the study do not report right prefrontal *only* versus left prefrontal (with children with diffuse lesions being included in both groups) nor does it contain data on functionally unrelated yet covarying abilities (like vocabulary).

There is a case study of an 11-year old with a pre-morbid IQ of 126 in gifted programs who suffered frontal lobe contusions (left greater than right). Eight weeks after the injury verbal IQ was similarly high (VIQ = 119) but performance IQ severely limited (PIQ = 60; Williams & Mateer, 1992). Two and four years after the injury, verbal abilities had remained unimpaired while inhibitory control and attention were significantly impaired. While only a case study, this shows that a manipulation (frontal damage) can cause changes to a subset of abilities but not others. This cannot be the case if all cognitive abilities are causally interrelated over development (as directly implied by mutualism accounts). More evidence, however, is needed.

The only other study coming close to what we are looking for is a study of 30 unilateral lesioned 10-year-olds who were studied when they were 20 years old. Although the data was not broken down by lesion site and there was no expansive intellectual measurement, one interesting dissociation emerged. EF was impaired for all patients, while recognition memory was not (Braun, Guimond, Payette, & Daigneault, 2013). Thus, we see two abilities (EF and recognition memory) that covary, that must covary for some causal reason, yet manipulation to one ability leads to no change in the other. This suggests theories entailing direct or indirect causal connections between cognitive abilities cannot explain such dissociation.

Nevertheless, the only way to truly disentangle causal developmental models will be longitudinal follow up of sufficiently sized samples with proper psychometric assessments in developmentally appropriate timescales (e.g. monthly/yearly during periods of rapid growth), with consideration of focal lesions (to exploit exogeneity) and a full range of psychometric data. As it stands, the state of the literature does not contain enough such data to warrant any strong conclusions, but evidence is mounting. As is plain from our review, however, the fullest evidence testing these interaction models must be the place of future work (and we explain why). This is a further direction for neuropsychological work to take, as it is clearly a needed avenue of testing and reporting childhood focal lesions, a large battery of disparate tasks, and long-term follow-up (over the course of years).

6. Conclusion

In conclusion, focal chronic cortical lesions cause local instead of global deficits and we have discussed here how this pattern may have remarkable consequences for different psychometric models trying to account for the cause of the positive manifold. The discussed evidence leads to the conclusion that not all these models are consistent with the pattern of lesion effects and their necessarily causal explanations of why all cognitive performances covary (Table 1).

We explored three classes of models: common cause models, interconnected models, and sampling models, with a number of alternatives

within each class (Table 2). The fact that disparate models can accommodate –from a psychometric perspective—the positive manifold is simply an extension of a well-known scientific feature of underdetermination of theory by data. As several models are able to accommodate the positive manifold *equally well*, statistical fit indices on the same data cannot demarcate which models are more likely. Strict causal tests and indirect evidence must be accumulated for finding a way out.

One proposed solution is to adopt an experimental psychometrics approach, where effects on a potential ability/driver node are imposed and the dissipation of effects is observed to strictly test the measurement-implied causal paths (Protzko, 2017). Here we have explored the opposite side of the causal coin. Instead of attempting to increase cognitive abilities through training, stimulants, or other means, we explored the effects of focal chronic cortical lesions *decreasing* specific cognitive abilities.

This is an important distinction as instances of increasing local abilities through avenues such as cognitive training has been fraught with concerns over teaching to the test, 'hollow' effects not to the construct under question, and lack of reproducibility (e.g. Colom et al., 2013; Green et al., 2019). Thus, the applicability of cognitive training studies on transfer and causal connections can sometimes be tenuous. Lesion effects, however, rarely suffer from such concerns, and provide an additional test of causal implications through manipulability.

The pattern of lesion effects observed in neuropsychology research is that lesions to one part of the cortex have local instead of global effects. In one of the case examples considered here, the rIFC causes decrements in inhibitory control but not in covarying abilities such as vocabulary, reading ability, or processing speed. Using this example, we saw hierarchical models like the classic *g*-model could accommodate local deficits, but not phenomena like cognitive reserve. Bifactor models are also able to accommodate the local pattern of deficits caused by focal cortical lesions. These two types of models are therefore indirectly supported by the available lesion evidence.

Some sampling models are able to accommodate lesion effects if they engage in semicircular reasoning, which is suboptimal from a theoretical perspective. A sampling model like POT was unable to accommodate local lesion effects without corresponding effects on *general* ability. Therefore, lesion research provides indirect evidence against such sampling models.

Correlated factor models must, as a necessity, decompose into a different type of model such as a network model or other interconnected model. We saw network models are unable to accommodate local lesion effects unless they decompose themselves into a hierarchical model or give up on explaining the correlation between causally unconnected variables. Causal interactionist models, such as Cattell's investment theory and dynamic mutualism, were likely unable to accommodate lesion effects, as the necessary causal connections between either some (investment) or all (mutualism) cognitive abilities is not held up by the absence of spreading effects from (for example) inhibition to vocabulary or processing speed.

Overall, it cannot be the case that performance on two different cognitive subtests are correlated without being causally connected in some way. The pattern of evidence from cortical lesions provides an interesting test of proposed causal connections from numerous competing models of the positive manifold. Only models that can accommodate a deficit in a local ability *without* effects on other *covarying* abilities are able to account for lesion evidence. Bifactor models are best able to accommodate such evidence. Hierarchical models, like the classic *g*-model, can also accommodate lesion effects, but not cognitive reserve. Some sampling models are able to accommodate the effects as well. For developmental models, the evidence appears consistent with non-spreading deficits from early focal lesions, but more evidence is required to draw firm conclusions.

The core argument of this article is not a takedown of any type of model nor an impassioned defense of another. All models are incomplete and the cause of the positive manifold will not be found in one of the models examined here. This may be an underwhelming conclusion, as the work here does not definitively declare a winner, but that is the nature of scientific exploration. The approach we take here is one of falsifiability of theories, instead of searching for confirming evidence. Such an approach cannot declare uncontested winners. Furthermore, as the sciences here involve individual differences, strict falsification may not be possible from one study alone. That is why, throughout, we have eschewed using the term 'falsifies' and instead used terms more like 'provides evidence against' a theory. Bringing together the two worlds of psychometrics and neuropsychology shows they may (and should) inform one another, give wider context to the implication of their work, and help move each other forward. What is clear, however, is that future theories that try to explain the positive manifold must consider the focality of lesion effects data in their explanations and (necessarily) causal implications of their theories. This is perhaps our main take home message.

Crucially, our argument here goes from neuroscience informing psychometrics. The same relation does not necessarily hold going from psychometrics to neuroscience. Future research and theorizing may draw such connections.

Available measurement models are substantially more than ways of picturing an idea. They are causal models, scientific theories, with necessarily causal connections and, importantly, testable predictions (Borsboom et al., 2003). These qualitative testable predictions must be borne out if a theory is to accurately explain the positive manifold. While many explanations so far can provide closely similar statistical fit, theories must do more than simply account for data. It is in these testable predictions that lesion studies can weigh in. Lesion effects being local, even years after the damage, has implications for the predictions all past present and future models make. Future theories and explanations of the positive manifold must also consider the fact that the covariance in the positive manifold must necessarily be causal *in some capacity*, yet manipulation of one local ability does not correspond to cross-sectional nor longitudinal effects on other abilities.

Conflicts of interest

The Authors Declare no Conflicts of Interest.

Acknowledgments

References

Abad, F. J., Colom, R., Juan-Espinosa, M., & García, L. F. (2003). Intelligence differentiation in adult samples. *Intelligence, 31*, 157–166.

Ackerman, P. T., Weir, N. L., Holloway, C. A., & Dykman, R. A. (1995). Adolescents earlier diagnosed as dyslexic show major IQ declines on the WISC-III. *Reading and Writing, 7*(2), 163–170.

Anderson, M. (1992). *Intelligence and development: A cognitive theory*. Blackwell Publishing.

Anderson, M. (2005). *Marrying intelligence and cognition. Cognition and intelligence* (pp. 268–287). New York: Cambridge University Press.

Anderson, M. (2017). Binet's error: Developmental change and individual differences in intelligence are related to different mechanisms. *Journal of Intelligence, 5*(2), 24. https://doi.org/10.3390/jintelligence5020024.

Anderson, V., Jacobs, R., & Harvey, A. S. (2005). Prefrontal lesions and attentional skills in childhood. *Journal of the International Neuropsychological Society, 11*(7), 817–831.

Anderson, V. A., Catroppa, C., Rosenfeld, J., Haritou, F., & Morse, S. A. (2000). Recovery of memory function following traumatic brain injury in pre-school children. *Brain Injury, 14*(8), 679–692.

Aram, D. M., & Eisele, J. A. (1994). Intellectual stability in children with unilateral brain lesions. *Neuropsychologia, 32*(1), 85–95.

Aram, D. M., & Ekelman, B. L. (1986). Cognitive profiles of children with early onset of unilateral lesions. *Developmental Neuropsychology, 2*(3), 155–172.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences, 8*(4), 170–177.

Assem, M., Glasser, M. F., Van Essen, D. C., & Duncan, J. (2020). A domain-general cognitive core defined in multimodally parcellated human cortex. *Cerebral Cortex, 30* (8), 4361–4380. https://doi.org/10.1093/cercor/bhaa023.

Barabási, A. L. (2016). *Network science*. Cambridge University Press.

Barbey, A. K. (2017). Network neuroscience theory of human intelligence. *Trends in Cognitive Sciences, 22*(1), 8–20.

Barbey, A. K., Colom, R., Paul, E. J., & Grafman, J. (2014). Architecture of fluid intelligence and working memory revealed by lesion mapping. *Brain Structure & Function, 219*(2), 485–494.

Barbey, A. K., Colom, R., Solomon, J., Krueger, F., Forbes, C., & Grafman, J. (2012). An integrative architecture for general intelligence and executive function revealed by lesion mapping. *Brain, 135*(4), 1154–1164.

Bartholomew, D. J., Deary, I. J., & Lawn, M. (2009). A new lease of life for Thomson's bonds model of intelligence. *Psychological Review, 116*(3), 567–579.

Basten, U., Hilger, K., & Fieback, C. (2015). Where smart brains are different: A quantitative meta-analysis of functional and structural brain imaging studies on intelligence. *Intelligence, 51*, 10–27.

Bailey, D., Duncan, G. J., Odgers, C. L., & Yu, W. (2017). Persistence and fadeout in the impacts of child and adolescent interventions. *Journal of research on educational effectiveness, 10*(1), 7–39.

Berkson, J. (1946). Limitations of the application of fourfold table analysis to hospital data. *Biometrics Bulletin, 2*(3), 47–53.

Blair, J. R., & Spreen, O. (1989). Predicting premorbid IQ: A revision of the National Adult Reading Test. *The Clinical Neuropsychologist, 3*(2), 129–136.

Borsboom, D. (2005). *Measuring the mind*. Cambridge University Press.

Borsboom, D., Cramer, A. O., Kievit, R. A., Scholten, A. Z., & Franić, S. (2009). The end of construct validity. In *The concept of validity: Revisions, new directions and applications, Oct, 2008*. IAP Information Age Publishing.

Borsboom, D., Mellenbergh, G. J., & Van Heerden, J. (2003). The theoretical status of latent variables. *Psychological Review, 110*(2), 203–219.

Braun, C. M., Guimond, A., Payette, J. F., & Daigneault, S. (2013). Specific early vulnerability of high-order executive function to focal brain lesions and long-term impact on educational persistence: Sparing of incidental episodic memory. *Developmental Neurorehabilitation, 16*(2), 89–101.

Braun, C. M. J., Montour-Proulx, I., Daigneault, S., Rouleau, I., Kuehn, S., Piskopos, M., … Rainville, C. (2002). Prevalence, and intellectual outcome of unilateral focal cortical brain damage as a function of age, sex and aetiology. *Behavioural Neurology, 13*(3–4), 105–116.

Carroll, J. B. (1991). No demonstration that g is not unitary, but there's more to the story: Comment on Kranzler and Jensen. *Intelligence, 15*(4), 423–436.

Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor-analytic studies*. Cambridge University Press.

Catroppa, C., Anderson, V. A., Morse, S. A., Haritou, F., & Rosenfeld, J. V. (2007). Children's attentional skills 5 years post-TBI. *Journal of Pediatric Psychology, 32*(3), 354–369.

Cattell, R. B. (1963). The theory of fluid and crystallized intelligence: A critical experiment. *Journal of Educational Psychology, 54*, 1–22.

Cattell, R. R. (1943). The measurement of adult intelligence. *Psychological Bulletin, 40*, 153–193.

Colom, R. (2014). From the earth to the brain. *PAID, 61–62*, 3–6.

Colom, R., Karama, S., Jung, R. E., & Haier, R. J. (2010). Human intelligence and brain networks. *Dialogues in Clinical Neuroscience, 12*, 489–501.

Colom, R., Román, F. J., Abad, F. J., Shih, P. C., Privado, J., Froufe, M., & Jaeggi, S. M. (2013). Adaptive n-back training does not improve fluid intelligence at the construct level: Gains on individual tests suggest that training may enhance visuospatial processing. *Intelligence, 41*(5), 712–727.

Colom, R., Shih, P. C., Flores-Mendoza, C., & Quiroga, M. A. (2006). The real relationship between short-term memory and working memory. *Memory, 14*(7), 804–813.

Colom, R., & Thompson, P. M. (2011). Understanding human intelligence by imaging the brain. In T. Chamorro, S. von Stumm, & A. Furnham (Eds.), *Handbook of individual differences*. Wiley-Blackwell.

Corkin, S., Rosen, T. J., Sullivan, E. V., & Clegg, R. A. (1989). Penetrating head injury in young adulthood exacerbates cognitive decline in later years. *Journal of Neuroscience, 9*(11), 3876–3883.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin, 52*(4), 281–302. https://doi.org/10.1037/h0040957.

Damasio, H., Grabowski, T., Frank, R., Galaburda, A. M., & Damasio, A. R. (1994). The return of Phineas Gage: Clues about the brain from the skull of a famous patient. *Science, 264*(5162), 1102–1105.

Detterman, D. K. (1987). Theoretical notions of intelligence and mental retardation. *American Journal of Mental Deficiency, 92*(1), 2–11.

Detterman, D. K., & Sternberg, R. J. (1993). *Transfer on trial: Intelligence, cognition, and instruction*. Ablex Publishing.

Dickens, W. T., & Flynn, J. R. (2001). Heritability estimates versus large environmental effects: The IQ paradox resolved. *Psychological Review, 108*(2), 346.

Dittrich, L. (2017). *Patient HM: A story of memory, madness, and family secrets*. Random House Trade Paperbacks.

Dubois, J., & Adolphs, R. (2016). Building a science of individual differences from fMRI. *Trends in Cognitive Sciences, 20*(6), 425–443.

Duncan, J., Assem, M., & Shashidhara, S. (2020). Integrated intelligence from distributed brain activity. *Trends in Cognitive Sciences, 24*(10), 838–852. https://doi.org/10.1016/j.tics.2020.06.012.

Duncan, J., Burgess, P., & Emslie, H. (1995). Fluid intelligence after frontal lobe lesions. *Neuropsychologia, 33*(3), 261–268.

Duncan, J., & Owen, A. M. (2000). Common regions of the human frontal lobe recruited by diverse cognitive demands. *Trends in Neurosciences, 23*(10), 475–483. https://doi.org/10.1016/S0166-2236(00)01633-7.

Duval, J., Braun, C. M., Montour-Proulx, I., Daigneault, S., Rouleau, I., & Bégin, J. (2008). Brain lesions and IQ: Recovery versus decline depends on age of onset. *Journal of Child Neurology, 23*(6), 663–668.

Epskamp, S., Rhemtulla, M. T., & Borsboom, D. (2016). Generalized network psychometrics: Combining network and latent variable models. *Psychometrika, 82*(4), 904–927.

Felton, R. H., Naylor, C. E., & Wood, F. B. (1990). Neuropsychological profile of adult dyslexics. *Brain and Language, 39*(4), 485–497.

Filley, C. M., Young, D. A., Reardon, M. S., & Wilkening, G. N. (1999). Frontal lobe lesions and executive dysfunction in children. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 12*(3), 156–160.

Fodor, J. A. (1983). *The modularity of mind*. MIT Press.

Fodor, J. A. (1985). Precis of the modularity of mind. *Behavioral and Brain Sciences, 8*(1), 1–42.

Garlick, D. (2002). Understanding the nature of the general factor of intelligence: The role of individual differences in neural plasticity as an explanatory mechanism. *Psychological Review, 109*(1), 116.

Friedman, N. P., Miyake, A., Corley, R. P., Young, S. E., DeFries, J. C., & Hewitt, J. K. (2006). Not all executive functions are related to intelligence. *Psychological science, 17*(2), 172–179.

Geary, D. C. (2018). Efficiency of mitochondrial functioning as the fundamental biological mechanism of general intelligence (g). *Psychological Review, 125*(6), 1028–1051.

Gignac, G. E. (2016). The higher-order model imposes a proportionality constraint: That is why the bifactor model tends to fit better. *Intelligence, 55*, 57–68.

Gignac, G. E., & Kretzschmar, A. (2017). Evaluating dimensional distinctness with correlated- factor models: Limitations and suggestions. *Intelligence, 62*, 138–147.

Gingras, B., & Braun, C. M. (2018). Intellectual outcome after a cortical lesion with versus without epilepsy: A life span neurodevelopmental view. *Epilepsy & Behavior, 85*, 129–140.

Gläscher, J., Rudrauf, D., Colom, R., Paul, L. K., Tranel, D., Damasio, H., & Adolphs, R. (2010). Distributed neural system for general intelligence revealed by lesion mapping. *Proceedings of the National Academy of Sciences, 107*(10), 4705–4709.

Gläscher, J., et al. (2009). Lesion mapping of cognitive abilities linked to intelligence. *Neuron, 61*, 681–691.

Green, C. S., et al. (2019). Improving methodological standards in behavioral interventions for cognitive enhancement. *Journal of Cognitive Enhancement, 3*(1), 2–29.

Guilford, J. P. (1967). *The nature of human intelligence*. New York: McGraw-Hill.

Haier, R. J. (2017). *The neuroscience of intelligence*. Cambridge University Press.

Hajek, C. A., Yeates, K. O., Anderson, V., Mackay, M., Greenham, M., Gomes, A., & Lo, W. (2014). Cognitive outcomes following arterial ischemic stroke in infants and children. *Journal of Child Neurology, 29*(7), 887–894.

Hausman, D., & Woodward, J. (1999). Independence, invariance, and the causal Markov condition. *British Journal for the Philosophy of Science, 50*, 1–63.

Heckman, J. J. (2006). Skill formation and the economics of investing in disadvantaged children. *Science, 312*, 1900–1902.

Horn, J. L. (1976). Human abilities: A review of research and theory in the early 1970's. *Annual Review of Psychology, 27*, 437–485.

Jacobs, R., Harvey, A. S., & Anderson, V. (2007). Executive function following focal frontal lobe lesions: Impact of timing of lesion on outcome. *Cortex, 43*(6), 792–805.

Jacobs, R., & Anderson, V. (2002). Planning and problem solving skills following focal frontal brain lesions in childhood: Analysis using the Tower of London. *Child Neuropsychology, 8*(2), 93–106.

Jensen, A. R. (1998). *The g factor: The science of mental ability*. Westport, CT: Praeger.

Johnson, W., & Bouchard, T. J., Jr. (2005). The structure of human intelligence: It is verbal, perceptual, and image rotation (VPR), not fluid and crystallized. *Intelligence, 33*(4), 393–416.

Jung, R. E., & Haier, R. J. (2007). The Parieto-Frontal Integration Theory (P-FIT) of intelligence: Converging neuroimaging evidence. *Behavioral and Brain Sciences, 30*(2), 135.

Kan, K., van der Maas, H., & Levine, S. (2019). Extending psychometric network analysis: Empirical evidence against g in favor of mutualism? *Intelligence, 73*, 52–62.

Katzman, R., Terry, R., DeTeresa, R., Brown, T., Davies, P., Fuld, P., … Peck, A. (1988). Clinical, pathological, and neurochemical changes in dementia: A subgroup with preserved mental status and numerous neocortical plaques. *Annals of Neurology, 23*, 138–144.

Kievit, R. A., Lindenberger, U., Goodyer, I. M., Jones, P. B., Fonagy, P., Bullmore, E. T., … Dolan, R. J. (2017). Mutualistic coupling between vocabulary and reasoning supports cognitive development during late adolescence and early adulthood. *Psychological Science, 28*(10), 1419–1431.

Kievit, R. A., Brandmaier, A. M., Ziegler, G., van Harmelen, A. L., de Mooij, S. M., & Moutoussis, M. (2018). NSPN Consortium. Developmental cognitive neuroscience using latent change score models: A tutorial and applications. *Developmental cognitive neuroscience, 33*, 99–117.

Kovacs, K., & Conway, A. R. (2016b). Has g gone to POT? *Psychological Inquiry, 27*(3), 241–253.

Kovacs, K., & Conway, A. R. (2016a). Process overlap theory: A unified account of the general factor of intelligence. *Psychological Inquiry, 27*(3), 151–177.

Kovacs, K., & Conway, A. R. (2019). What is IQ? Life beyond "general intelligence". *Current Directions in Psychological Science, 28*(2), 189–194.

Krakauer, J. W., Ghazanfar, A. A., Gomez-Marin, A., MacIver, M. A., & Poeppel, D. (2017). Neuroscience needs behavior: Correcting a reductionist bias. *Neuron, 93*(3), 480–490.

Kranzler, J. H., & Jensen, A. R. (1991). The nature of psychometric g: Unitary process or a number of independent processes? *Intelligence, 15*(4), 397–422.

Lee, S., & Hershberger, S. (1990). A simple rule for generating equivalent models in covariance structure modeling. *Multivariate Behavioral Research, 25*(3), 313–334.

Levine, S. C., Huttenlocher, P., Banich, M. T., & Duda, E. (1987). Factors affecting cognitive functioning of hemiplegic children. *Developmental Medicine and Child Neurology, 29*(1), 27–35.

Lichtenberger, E. O., & Kaufman, A. S. (2009). *Essentials of WAIS-IV assessment. 50*. John Wiley & Sons.

Long, B., Spencer-Smith, M. M., Jacobs, R., Mackay, M., Leventer, R., Barnes, C., & Anderson, V. (2011). Executive function following child stroke: The impact of lesion location. *Journal of Child Neurology, 26*(3), 279–287.

Lumsden, J. (1976). Test theory. *Annual Review of Psychology, 27*, 251–280.

Luria, A. R. (2012). Higher cortical functions in man. *Springer Science & Business Media*.

van der Maas, H. L. J., Dolan, C. V., Grasman, R. P. P. P., Wicherts, J. M., Huizenga, H. M., & Raijmakers, M. E. J. (2006). A dynamical model of general intelligence: The positive manifold of intelligence by mutualism. *Psychological Review, 113*, 842–861.

Maul, A., Irribarra, D. T., & Wilson, M. (2016). On the philosophical foundations of psychological measurement. *Measurement, 79*, 311–320.

McAuley, T., & White, D. A. (2011). A latent variables examination of processing speed, response inhibition, and working memory during typical development. *Journal of Experimental Child Psychology, 108*(3), 453–468.

McFarland, D. J. (2017). How neuroscience can inform the study of individual differences in cognitive abilities. *Reviews in the Neurosciences, 28*(4), 343–362.

McFarland, D. J. (2019). A brief history of theory and testing of mental abilities. In D. McFarland (Ed.), *General and specific mental abilities* (pp. 1–24). Cambridge Scholars.

McFie, J. (1960). Psychological testing in clinical neurology. *Journal of Nervous and Mental Disease, 131*, 383–393.

McFie, J. (1961). Intellectual impairment in children with localized post-infantile cerebral lesions. *Journal of Neurology, Neurosurgery, and Psychiatry, 24*(4), 361–365.

McGrew, K. S. (2009). CHC theory and the human cognitive abilities project: Standing on the shoulders of the giants of psychometric intelligence research. *Intelligence, 37*, 1–10.

Mendez, M. F., Adams, N. L., & Lewandowski, K. S. (1989). Neurobehavioral changes associated with caudate lesions. *Neurology, 39*(3), 349–354.

Merkle, E. C., You, D., & Preacher, K. J. (2016). Testing nonnested structural equation models. *Psychological Methods, 21*(2), 151–163.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*(1), 49–100.

Molenaar, D., Dolan, C. V., Wicherts, J. M., & van der Maas, H. L. (2010). Modeling differentiation of cognitive abilities within the higher-order factor model using moderated factor analysis. *Intelligence, 38*(6), 611–624.

Montour-Proulx, I., Braun, C. M., Daigneault, S., Rouleau, I., Kuehn, S., & Bégin, J. (2004). Predictors of intellectual function after a unilateral cortical lesion: Study of 635 patients from infancy to adulthood. *Journal of Child Neurology, 19*(12), 935–943.

Muter, V., Taylor, S., & Vargha-Khadem, F. (1997). A longitudinal study of early intellectual development in hemiplegic children. *Neuropsychologia, 35*(3), 289–298.

Nass, R., Peterson, H. D., & Koch, D. (1989). Differential effects of congenital left and right brain injury on intelligence. *Brain and Cognition, 9*(2), 258–266.

Nelson, H. E., & Willison, J. (1991). *National adult reading test (NART)*. Windsor: Nfer-Nelson.

Neubert, J. C., Kretzschmar, A., Wüstenberg, S., & Greiff, S. (2015). Extending the assessment of complex problem solving to finite state automata: Embracing heterogeneity. *European Journal of Psychological Assessment, 31*(3), 181–194.

Owen, A. M., Downes, J. J., Sahakian, B. J., Polkey, C. E., & Robbins, T. W. (1990). Planning and spatial working memory following frontal lobe lesions in man. *Neuropsychologia, 28*(10), 1021–1034.

Pavlovic, J., Kaufmann, F., Boltshauser, E., Mori, A. C., Mercati, D. G., Haenggeli, C. A., … Perez, E. R. (2006). Neuropsychological problems after paediatric stroke: Two year follow- up of Swiss children. *Neuropediatrics, 37*(01), 13–19.

Pearl, J. (2009). *Causality*. New York, NY: Cambridge University Press.

Pósfai, M., Liu, Y. Y., Slotine, J. J., & Barabási, A. L. (2013). Effect of correlations on network controllability. *Scientific Reports, 3*, 1067.

Price, C. (2018). The evolution of cognitive models: From neuropsychology to neuroimaging and back. *Cortex, 107*, 37–49.

Protzko, J. (2015). The environment in raising early intelligence: A meta-analysis of the fadeout effect. *Intelligence, 53*, 202–210.

Protzko, J. (2016). Does the raising IQ-raising g distinction explain the fadeout effect? *Intelligence, 56*, 65–71.

Protzko, J. (2017). Effects of cognitive training on the structure of intelligence. *Psychonomic Bulletin & Review, 24*(4), 1022–1031.

Raykov, T., Marcoulides, G. A., Menold, N., & Harrison, M. (2018). Revisiting the bi-factor model: Can mixture modeling help assess its applicability?. Structural equation modeling. *A Multidisciplinary Journal*, 1–9.

Raz, S., Foster, M. S., Briggs, S. D., Shah, F., Baertschi, J. C., Lauterbach, M. D., … Sander, C. J. (1994). Lateralization of perinatal cerebral insult and cognitive asymmetry: Evidence from neuroimaging. *Neuropsychology, 8*(2), 160.

Riva, D., & Cazzaniga, L. (1986). Late effects of unilateral brain lesions sustained before and after age one. *Neuropsychologia, 24*(3), 423–428.

Rohrer, J. M. (2018). Thinking clearly about correlations and causation: Graphical causal models for observational data. *Advances in Methods and Practices in Psychological Science, 1*(1), 27–42.

Roid, G. H., & Barram, R. A. (2004). *Essentials of Stanford-Binet intelligence scales (SB5) assessment. 39*. John Wiley & Sons.

Ruíz Sánchez de León, J. M., Quiroga, M. A., & Colom, R. (2019). Intelligence and executive function: Can we reunite these disparate worlds? In D. McFarland (Ed.), *General and specific mental abilities* (pp. 311–339). Cambridge Scholars Publishing.

Santarnecchi, E., Rossi, S., & Rossi, A. (2015). The smarter, the stronger: Intelligence level correlates with brain resilience to systematic insults. *Cortex, 64*, 293–309.

Santonja, J., Martínez, K., Román, F. J., Escorial, S., Quiroga, M. A., Álvarez-Linera, J., … Colom, R. (2021). Brain resilience across the general cognitive ability distribution: Evidence from structural connectivity. *Brain Structure & Function, 226*(3), 845–859.

Schmiedek, F., Lövdén, M., von Oertzen, T., & Lindenberger, U. U. (2019). Within-person structures of daily cognitive performance cannot be inferred from between-person structures of cognitive abilities (No. e27576v1). *PeerJ Preprints*.

Schneider, W. J. K. S. (2018). McGrewThe Cattell-Horn-Carroll theory of cognitive abilities. In D. P. Flanagan, & E. M. McDonough (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (4th, pp. 73–162). New York, NY: Guilford.

Schweizer, K., & Koch, W. (2002). A revision of Cattell's investment theory: Cognitive properties influencing learning. *Learning and Individual Differences, 13*(1), 57–82.

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry, 20*(1), 11–21.

Spearman, C. (1904). "General intelligence," objectively determined and measured. *The American Journal of Psychology, 15*, 201–293.

Sporns, O., & Betzel, R. F. (2016). Modular brain networks. *Annual Review of Psychology, 67*, 613–640.

Süß, H. M., & Beauducel, A. (2015). Modeling the construct validity of the Berlin intelligence structure model. *Estudos de Psicologia (Campinas), 32*(1), 13–25.

Swick, D., Ashley, V., & Turken, U. (2008). Left inferior frontal gyrus is critical for response inhibition. *BMC Neuroscience, 9*(1), 102.

Theiling, J., Petermann, F., & Daseking, M. (2013). WAIS-IV profiles in first-ever unilateral ischemic stroke patients. *Zeitschrift für Neuropsychologie, 24*(4), 239–252.

Thomson, G. H. (1916). A hierarchy without a general factor. *British Journal of Psychology, 8*, 271–281.

Thomson, G. H. (1951). *The factorial analysis of human ability* (5th ed.). London: University of London Press.

Thurstone, L. L. (1935). *The vectors of the mind*. Chicago: Chicago University Press.

Tucker-Drob, E. M., Braindmaier, A. M., & Lindenberger, U. (2019). Coupled cognitive changes in adulthood: A meta-analysis. *Psychological Bulletin, 145*(3), 273–301.

Unsworth, N., & Engle, R. W. (2007). On the division of short-term and working memory: An examination of simple and complex span and their relation to higher order abilities. *Psychological Bulletin, 133*(6), 1038.

Vaessen, M. J., Saj, A., Lovblad, K. O., Gschwind, M., & Vuilleumier, P. (2016). Structural white-matter connections mediating distinct behavioral components of spatial neglect in right brain-damaged patients. *Cortex, 77*, 54–68.

Vaidya, A. R., Pujara, M. S., Petrides, M., Murray, E. A., & Fellows, L. K. (2019). Lesion studies in contemporary neuroscience. *Trends in Cognitive Sciences, 23*(8), 655–673.

Van Horn, J. D., Irimia, A., Torgerson, C. M., Chambers, M. C., Kikinis, R., & Toga, A. W. (2012). Mapping connectivity damage in the case of Phineas gage. *PLoS One, 7*(5), Article e37454.

Vernon, P. E. (1964). *The structure of human abilities*. London: Methuen.

Warne, R. T., & Burningham, C. (2019). Spearman's g found in 31 non-Western nations: Strong evidence that g is a universal phenomenon. *Psychological Bulletin, 145*(3), 237–272.

Wechsler, D., de la Guía, E., & Vallar, F. (2012). *WAIS-IV: escala de inteligencia de Wechsler para adultos-IV*. Madrid: Pearson.

Westmacott, R., Askalan, R., Macgregor, D., Anderson, P., & Deveber, G. (2010). Cognitive outcome following unilateral arterial ischaemic stroke in childhood: Effects of age at stroke and lesion location. *Developmental Medicine and Child Neurology, 52*(4), 386–393.

Westmacott, R., MacGregor, D., Askalan, R., & deVeber, G. (2009). Late emergence of cognitive deficits after unilateral neonatal stroke. *Stroke, 40*(6), 2012–2019.

Williams, D., & Mateer, C. A. (1992). Developmental impact of frontal lobe injury in middle childhood. *Brain and Cognition, 20*(1), 196–204.

Woods, B. T. (1980). The restricted effects of right-hemisphere lesions after age one; Wechsler test data. *Neuropsychologia, 18*(1), 65–70.

Woods, B. T., & Teuber, H. L. (1978). Changing patterns of childhood aphasia. *Annals of Neurology, 3*(3), 273–280.

Woodward, J. (2003). *Making things happen: A theory of causal explanation*. Oxford: Oxford University Press.