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Network neuropsychology: The map and the territory

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Abstract

In “network neuropsychology”, network modelling and graph theory is applied to the neuropsychological test scores of patients with neurological disorders to investigate cognitive functioning. This review identifies the emerging literature on several disorders before focusing on the assumptions about cognition underlying the studies; specifically, that cognition can be thought of as a network of interrelated variables and that changes in these interrelationships, or cognitive rearrangement, can occur in neurological disorders. Next the review appraises how well network models can provide a “map” of this cognitive “territory”. In particular, the review considers the lack of correspondence between the variables and properties of network models and cognitive functioning. The challenges of explicitly accounting for latent cognitive constructs and making inferences about cognition based on associative, as opposed to dissociative, methods are also discussed. It is concluded that the validity of network neuropsychological models is yet to be established and that cognitive theory and experiments, as well as network models, are needed to develop and interpret better maps.

1. Introduction

Some neuropsychological approaches focus on the subtractive effect of cerebral damage on cognition, such as cognitive neuropsychology (Ellis and Young, 1996; McCarthy and Warrington, 1990). These reductionist approaches help to identify the major cognitive effects of neurological disorders. However, they may struggle to model the complex interrelationships amongst cognitive functions which typify normal cognition (Kan et al., 2019; van der Maas et al., 2017) and may change in response to brain pathology.

In the last six years, several studies have used network modelling and graph theory to investigate cognitive functioning in various neurological disorders. Network modelling is a holistic way of representing relationships amongst many variables and graph theory provides a set of metrics to describe patterns of associations within these network models (Christensen, 2019). This modelling strategy may broaden understanding of cognitive functioning in neurological disorders. For example, in a sample of stroke patients, Massa et al. (2015) estimated domain-specific network models based on tests of each of the five Birmingham Cognitive Screen domains (i.e., attention, memory, language, praxis, and number) and an across-domain model. In the reductionistic within-domain models, tests of specific cognitive subcomponents tended to be linked with each other and unrelated to tests of other subcomponents. By contrast, in the holistic between-domain model, in which tests were conditioned on tests of all other domains, multiple tests were associated with others outside of their purported domains. These findings suggest that patterns of test associations, and, perhaps, interrelationships amongst cognitive functions, differ when holistic investigations are performed.

As network models represent interrelationships, they may benefit clinical practices that invoke these. For example, case formulations often consider the influence, whether compensatory or detrimental, of one function on another (Lamberty and Nelson, 2015, pg. 55). If an assessment reveals impaired free recall but preserved recognition memory, attention, visuospatial functioning, and so on, a formulation might be that impaired retrieval underlies difficulties with episodic memory. As this formulation references functioning in one area *relative* to others, it makes sense to use an approach that explicitly models interrelationships. Graph theory metrics might also highlight potential avenues for treatment. For example, if a function is the most central, it can be thought to strongly influence other variables. Supporting this function might be expected to also benefit others (Rodebaugh et al., 2018).

The disorders studied with these methods include adult and paediatric epilepsies (Garcia-Ramos et al., 2021, 2015; Kellermann et al., 2016, 2015), mild cognitive impairment (Ferguson for the Alzheimer's Disease Neuroimaging Initiative, 2021), Alzheimer's disease (Ferguson for the Alzheimer's Disease Neuroimaging Initiative, 2021; Nevado et al., 2021a; Tosi et al., 2020; Wright et al., 2021), vascular encephalopathy (Tosi et al., 2020), stroke (Massa et al., 2015), and frontal and non-frontal lesions arising from various aetiologies (Jonker et al., 2019). Comparable studies have created network models of more specific aspects of cognition, including intelligence (Kan et al., 2019; Schmank et al., 2019; van der Maas et al., 2017), reading (Angelelli et al., 2021; Goring et al., 2021; Zoccolotti et al., 2021), and semantic fluency (Bertola et al., 2014; De Marco et al., 2021; Goni et al., 2011~ ; Lerner et al., 2009; Nevado et al., 2021b), or developmental processes like ageing (Garcia-Cabello et al., 2021; Konigs et al., 2021~), in clinical and non-clinical groups. Other related studies have combined neuroimaging and cognitive, behavioural, and/or self-report data in clinical groups (Bathelt et al., 2020; Hilland et al., 2020; Simpson-Kent et al., 2021), or investigated relationships amongst neuropsychological variables and the symptoms of autism spectrum disorder (Ibrahim et al., 2016) and attention deficit hyperactivity disorder (Eadeh et al., 2021).

The studies summarised in Table 1 are united by four characteristics, outlined a posteriori, which are not ubiquitous across the other related studies:

- 1 Groups of patients with neurological disorders are studied.
- 2 There is predominant, or exclusive, use of neuropsychological test scores in the network models.
- 3 There is a focus on cognitive functioning, and, occasionally, on neuropsychological measurement.
- 4 Finally, neuropsychological profiles and scores across tests are focused on rather than singular functions and individual neuropsychological tests.

The common approach in the studies in Table 1 can be provisionally termed “network neuropsychology”. A summary of the main findings of network neuropsychology studies is presented in Table 1 (the search strategy is reported in the supplementary materials); although, a systematic review would be premature. Network neuropsychology is in its infancy and it is not yet possible to draw conclusions about cognitive interrelationships in neurological disorders or to direct clinical practice.

Significant theoretical issues are present and it is important to investigate these sooner rather than later. Namely, the fundamental issue of how well network models and graph theory can represent cognition is yet to be appraised. Korzybski famously wrote that “A map is not the territory it represents, but, if correct, it has a similar structure to the territory, which accounts for its usefulness” (Korzybski, 1995). This review aims to uncover the cognitive “territory” that network neuropsychology is interested in and evaluate the usefulness of network modelling and graph theory in providing a “map” of this.

Table 1

Summary of network neuropsychology studies

Study	Groups	Domains tested	Associations	Main findings
Ferguson for the Alzheimer's Disease Neuroimaging Initiative (2021)	• Mild cognitive impairment (n = 395) • Early Alzheimer's disease (n = 191) • Cognitively normal controls (n = 229)	• Attention/processing speed • Demographic variables • Episodic memory • Fluency • Language • Premorbid ability • Visuospatial • Working memory	Partial correlations	• Network structures differed across models for CN and eAD and aMCI and eAD groups. • Two putative communities (not statistically determined) in models for clinical groups in contrast to fractionated domains in HC model: 1 Attention-working memory-processing speed. 2 Memory-language-semantic.
Garcia-Ramos et al. (2021)	• Temporal lobe epilepsy phenotype groups (n = 104): ○ No cognitive impairment (n = 53) ○ Focal cognitive impairment (n = 31) ○ Generalised cognitive impairment (n = 20) • Healthy controls (n = 30)	• Executive • Executive/processing speed • Intelligence • Language • Language/ executive • Memory • Motor speed • Processing speed • Visuospatial	Partial correlations	• Reduction of positive associations amongst tests across models for control and TLE phenotype groups. • Increased clustering and modularity from control to phenotype models. • Decreased global efficiency across models for groups. • Different communities in clinical models compared to HC model. • Lower strength centrality of nodes in models for focal- and generalised-cognitive impairment epilepsy groups.
Garcia-Ramos et al. (2015)	• Paediatric epilepsy (various syndromes) (n = 104) • Healthy controls (n = 74)	• Academic achievement • Executive • Intelligence • Language • Memory	Correlations	• Higher harmonic mean in the model for the epilepsy group compared to that for the healthy control group. • Lower clustering in epilepsy group model relative to healthy control model. • Three communities in control group model: 1 Intelligence-achievement. 2 Memory-semantic-language. 3 Speed-executive. • Five communities in epilepsy group model:

				1 Speed-executive. 2 Working memory-fluency. 3 Intelligence-achievement. 4 Memory-language. 5 Visuospatial-speed. • Category switching and verbal intelligence tests showed high betweenness centrality in models for both groups.
Jonker et al. (2019)	• Frontal lesions (n = 62) • Non-frontal lesion patients (n = 66) • Controls (n = 67)	• Working memory • Memory • Executive function • Frontal symptoms	Partial correlations	• Three communities in HC model: 1 Memory. 2 Letter fluency-working memory. 3 Divided attention-interference control-frontal symptoms. • Two communities in frontal model: 1 Memory-letter fluency-working memory. 2 Divided attention-interference control-frontal symptoms. • Four communities in non-frontal model: 1 Memory. 2 Letter fluency-working memory. 3 Frontal symptoms. 4 Divided attention-interference control. • Immediate memory most degree central in all models.

				• Working memory and divided attention less central in frontal model than control and non-frontal models.
Kellermann et al. (2015)	• Paediatric epilepsy (various syndromes) (n = 127) • Healthy controls (n = 80)	• Intelligence • Academic performance • Language • Memory • Executive function • Psychomotor speed	Correlations	• Epilepsy group displayed poorly separated modules in comparison with the control group. • Higher clustering coefficient, greater degree connectivity, and shorter path length in epilepsy group, indicative of poorer segregation.
Kellermann et al. (2016)	• Temporal lobe epilepsy (n = 100) • Healthy controls (n = 82)	• Academic achievement • Attention • Executive function • Fluency • Language • Memory • Motor • Processing speed • Visuospatial skills • Working memory	Correlations	• Four communities in HC model: 1 Verbal memory. 2 Language-perception-intelligence. 3 Speed-fluency. 4 Visual memory-executive. • Three communities in TLE model: 1 Executive-verbal/visual memory. 2 Speed-fluency. 3 Speed-executive-perception-language-intelligence-nonverbal memory.

Massa et al. (2015)

- Stroke (various aetiologies and locales) (n = 287)
- No control group.
- Affect
- Attention/executive
- Language
- Memory
- Motor performance
- Number skills
- Praxis

Partial correlations

- Within-domain analyses suggested separation between tests tapping different elements of each domain.
- Between-domain analyses showed that cognitive tests were linked within their purported domains; however, many tests were also linked beyond domains.
- Between-domain analyses suggested cognitive functioning was not associated with anxiety, mood, and motor functioning.

Nevado et al. (2021a)

- Dementia, mixed aetiologies (n = 2040)
- Mild cognitive impairment (n = 5981)
- Healthy controls (n = 7623)
- Attention
- Executive function
- Memory
- Language

Partial correlations

- Largely invariant network structure across models for all groups.
- Four communities in models for all groups:
 - 1 Attention.
 - 2 Executive.
 - 3 Memory.
 - 4 Language.
- Executive (composite) was most the central (composite) across groups.
- More interrelationships were seen amongst memory and language variables in the dementia model.

Tosi et al. (2020)	• Alzheimer's disease (n = 191) • Vascular encephalopathy (n = 129) • Healthy controls (n = 165)	• Attention • Demographics • Episodic memory • Fluency • Intelligence • Language • Screening • Visual memory • Visuospatial • Working memory	Partial correlations	• Segregated profile for HC group model. • Reduced impact of age and education in clinical models • Two putative communities (not statistically determined) in the AD model: ¹ Memory. ² Executive. • Preserved integration and segregation but weaker associations amongst tests in VE network model. • Category fluency was the most central node in the AD and VE models.
Wright et al. (2021)	• Healthy controls ◦ Young (n = 75) ◦ Middle aged (n = 75) ◦ Older (n = 70) • Mild cognitive impairment ◦ naMCI (n = 60) ◦ aMCI (n = 75) • Alzheimer's disease (n = 60)	• Attention/processing speed • Fluency • Inhibition • Intelligence • Language • Visuospatial • Working memory	Correlation (conditioned on age and education only before network estimation.)	• Control groups showed increased average global and local efficiency, clustering coefficients and connection density across an increasing age range. • Clinical groups displayed higher network segregation, clustering coefficients, and local efficiency than healthy control groups. • Clinical groups displayed lower betweenness centrality for semantic processing than control groups.

NB. cognitive domain labels were taken from the original articles for [Ferguson for the Alzheimer's Disease Neuroimaging Initiative \(2021\)](#), [Garcia-Ramos et al. \(2021\)](#) and [\(2015\)](#), [Kellermann et al. \(2015\)](#) and [\(2016\)](#), [Massa et al. \(2015\)](#), and [Nevado et al. \(2021a\)](#); domains for [Tosi et al. \(2020\)](#) and [Wright et al. \(2021\)](#) were assigned post-hoc. Community titles for [Ferguson for the Alzheimer's Disease Neuroimaging Initiative \(2021\)](#), [Kellermann et al. \(2015\)](#) and [Kellermann et al. \(2016\)](#), and [Tosi et al. \(2020\)](#) were taken from the original papers

2. The territory

2.1. A network system of cognition

Most network neuropsychology studies assume that cognitive functions influence each other and can therefore be modelled as a web-like structure or a network (Kan et al., 2019; van der Maas et al., 2006). A helpful metaphor for this view of cognition is found in network neuropsychology studies which sought to characterise the cognitive network “topology” or “landscape” of “cognitive landmarks” (Jonker et al., 2019; Garcia-Ramos et al., 2015, 2021; Kellermann et al., 2015, 2016). “Topology” means the way in which constituent parts are interrelated or arranged and “landmark” means a recognisable feature of a landscape and/or an event marking an important stage or turning point in something (Oxford English Dictionary). “Cognitive landmarks” then can refer to cognitive functions and/or changes in them over time, such as maturation or decline. Studies in Table 1 sought to investigate both the arrangement of interrelationships amongst variables (e.g., Ferguson for the Alzheimer’s Disease Neuroimaging Initiative, 2021; Jonker et al., 2019; Garcia-Ramos et al., 2021; Kellermann et al., 2015, 2016; Nevado et al., 2021a; Massa et al., 2015; Tosi et al., 2020) and developmental changes in these (e.g., Garcia-Ramos et al., 2015; Wright et al., 2021).

Auxiliary assumptions about cognition are sometimes made. For example, Massa et al. (2015) referred to domain-general and domain-specific factors following stroke. They stated that domain-general factors, such as working memory and sustained attention, can influence the functioning of domain-specific factors, such as language, memory, and attention. That some aspects of cognition affect others is consistent with the network intuitions previously described. However, Massa et al.’s assumptions about cognition are also hierarchical, as some domains (e.g., working memory) control others, and directional, as these higher domains control others in top-down, non-reciprocal manner. These additional assumptions do not prohibit viewing cognition as a network, they just specify the nature of the relationships between variables.

2.2. Segregation and integration

It may also be assumed that cognitive networks display patterns of segregation and integration (Garcia-Ramos et al., 2015, 2016, 2021; Kellermann et al., 2016; Tosi et al., 2020; Wright et al., 2021). For example, Garcia-Ramos et al. (2015) observed that patients with paediatric epilepsy displayed both lower integration and segregation across neuropsychological tests.

Patterns of segregation and integration have been investigated in graph theoretical rather than cognitive, or information processing, terms (e.g., Ellis and Young, 1996; Fodor, 1983). In graph theory, segregation is a property of network models that allows substructures to exist within the wider model whereas integration involves connections between elements of the wider model (Garcia-Ramos et al., 2016). These properties *might* reflect specialised and integrative processes occurring within the cognitive network. However, this cannot be readily determined based on network models alone. This issue will be explored in the second part of the review, which considers how well network models provide a map of the cognitive territory outlined here.

2.3. Latent cognitive functions

Network theories are sometimes presented as alternative explanations to latent variable theories in intelligence, psychopathology, and personality research (Cramer et al., 2016; Guyon et al., 2017; Kan et al., 2019; van der Maas et al., 2006). For example, the mutualism hypothesis disavows the existence of latent

general intelligence. Instead, it proposes that the development of a given cognitive function (e.g., abstract reasoning) occurs because of the influence of the development of other cognitive abilities (e.g., working memory) as well as autonomous development in that function. This gives rise to a network of mutually reinforcing abilities which can explain the positive associations commonly seen amongst cognitive tasks without recourse to latent general intelligence (Kan et al., 2019; van der Maas et al., 2006).

None of the studies in Table 1 posit that latent cognitive functions, such as working memory or divided attention, do not exist. Across the discipline of neuropsychology, it is widely accepted that latent cognitive functions exist, even though the exact nature and the existence of some functions is contested (e.g., see Morey, 2018 regarding visuospatial short-term memory). Consistent with the assumption of influences amongst cognitive functions in network neuropsychology, latent cognitive constructs are intercorrelated in some latent variable models of cognition (e.g., Agelink van Rentergem et al., 2020).

2.4. The “normal” network

A fairly typical pattern of interrelationships is present across the adult control group models in Table 1, providing an impression of normative functioning against which clinical models can be compared. Generally, relatively distinct communities of tests probing the same cognitive domains and weaker associations between tests probing different domains are observed (Ferguson for the Alzheimer’s Disease Neuroimaging Initiative, 2021; Jonker et al., 2019; Garcia-Ramos et al., 2021; Kellermann et al., 2016; Nevado et al., 2021a, Tosi et al., 2020). These findings are broadly consistent with factor models of cognitive functioning in healthy groups which allow latent cognitive functions to correlate (e.g., Agelink van Rentergem et al., 2020). Also, this pattern is consistent with cognitive theories proposing patterns of specialisation and integration or domain specific and domain general processing (Massa et al., 2015). However, this pattern should not be over-interpreted. It is likely to change across the lifespan (Kan et al., 2019); reflecting, for example, the progressive specialisation of cognition in childhood (D’Souza and Filippi, 2017) and neural dedifferentiation in older age (Goh, 2011; Koen and Rugg, 2019; Wright et al., 2021).

2.5. Cognitive rearrangement

Network neuropsychology is also concerned with the changing relationships amongst cognitive functions in neurological disorders. Historically, the possibility of cognitive rearrangement has been implied by several neuropsychologists (Kertesz, 1983, pg. 8; Riddoch and Humphries, 1994, pg. 6; Shallice, 1988, pg. 241). Modelling such changes is important for informing the assessment and treatment of patients with neurological disorders as well as understanding cognition. Returning to the metaphor of the “cognitive network topology” of “cognitive landmarks”, cognitive rearrangement can be understood as the rearrangement of this map following illness or injury. Patient-control group network neuropsychology studies generally report differences in the overall network structure of neuropsychological test scores of neurological groups relative to control groups (Ferguson for the Alzheimer’s Disease Neuroimaging Initiative, 2021; Garcia-Ramos et al., 2015, 2021; Jonker et al., 2019; Kellermann et al., 2015, 2016; Tosi et al., 2020; Wright and Woods, 2020; Wright et al., 2021), with one notable exception (Nevado et al., 2021a), which may suggest that cognitive rearrangement occurs. Note that rearrangement refers to changes in the interrelationships amongst cognitive functions and not the invention of new cognitive functions, which is implausible in the mature brain (Ellis and Young, 1996, pg. 19; Saffran, 1982). It is important to hold this in mind when interpreting communities and associations amongst neuropsychological test scores in network models.

2.6. Adaptation and maladaptation

If present, cognitive rearrangement may also be adaptive or maladaptive. There is evidence for these phenomena independent of network neuropsychology. Compensatory rehabilitation strategies provide evidence for a temporary form of adaptive cognitive rearrangement which may cease if the strategies are discarded (e.g., [Pamir et al., 2021](#); [Signoret and Lhermitte, 1976](#); [Shallice, 1988](#), pg. 374). For example, semantic scaffolding and mental imagery appear to aid learning on verbal paired associates in patients with anterior communicating artery lesions ([Signoret and Lhermitte, 1976](#); [Shallice, 1988](#), pg. 374). The upregulation of attention has also been linked with language recovery in stroke ([Geranmayeh et al., 2014](#)). Regarding the deleterious impact of aspects of some aspects of cognition on others, unilateral neglect, a predominantly spatial attention issue, can be exacerbated by secondary deficits in executive functioning and working memory ([Malhotra et al., 2005](#); [Massa et al., 2015](#)).

In network neuropsychology, [Kellermann et al. \(2016\)](#), for example, observed less segregation between purported domains in a group of people with temporal lobe epilepsy compared to controls. They speculated that this interdependency across domains reflected compensatory attempts to maintain cognitive functioning. It seems likely that the nature of cognitive rearrangement is shaped by the nature of the pathology, premorbid cognitive functioning, and developmental stage.

3. The map

3.1. The basics of network models

A network model consists of a set of variables and the associations amongst them. In network neuropsychology, these variables are generally neuropsychological test scores. The associations amongst test scores can be correlations or partial correlations after conditioning on all the test scores in the model. Network models can represent the presence/ absence and strength of associations amongst test scores. If the assumption of multivariate normality is met, connected scores in partial correlation networks can be assumed to be conditionally dependent given the other scores in the model ([Christensen, 2019](#); [Epskamp et al., 2018](#); [Epskamp and Fried, 2018](#)). The network can be visualised in a plot and graph theory metrics can also be calculated (see the [supplementary materials](#) for an overview of these).

3.2. Why network models and graph theory?

In network neuropsychology, the application of network modelling and graph theory generally stems from the assumptions about cognition, especially regarding interrelationships and cognitive rearrangement. Historically, the clinical utility of modelling interrelationships amongst cognitive functions in neurological diseases has been hinted at ([Riddoch and Humphries, 1994](#), pg. 6):

In the clinic the rehabilitationist will typically be faced with patients with multiple deficits, where the damage has not respected the strict boundaries between functionally independent processing modules. It may be clinically relevant to understand how much one deficit impinges on another, or even more particularly, whether an associated deficit constrains rehabilitation of a given ability.

Network modelling enables the estimation of a structure in which connected variables can be thought to influence each other and graph theory provides measures of the interrelationships amongst individual elements as well as their participation in the wider system, making it a natural choice for analysis in network

neuropsychology ([Garcia-Ramos et al., 2016](#)). However, the ability of network modelling and graph theory to provide a useful map of the cognitive terrain of interest is yet to be established.

The use of network modelling and graph theory can also be pragmatic, as there are scenarios where the properties of network models are desirable but the assumption of a network system of cognition is not relevant ([Epskamp et al., 2018](#)). For example, in modelling the relationships within screening tests ([Massa et al., 2015](#)) or amongst screening tests and tests designed to measure specific constructs ([Tosi et al., 2020](#)), potentially to ascertain convergent and discriminant validity. Network modelling and graph theory could also be motivated by detecting disease (as proposed by [Wright et al., 2021](#)). In these situations, network modelling and graph theory can be used to model statistical relationships between tests, while conveniently conditioning on multiple relevant variables, without making any assumptions about the relationships amongst the cognitive functions underlying test performance. Neither do these applications necessitate a detailed representation of the cognitive terrain.

3.3. Problems with parallelism

There are two immediate ways in which network models may struggle to map cognition. First, neuropsychological test scores are not the same as the cognitive functions they are sensitive to. This is for at least four reasons, including a lack of specificity of a test to a cognitive function ([Koekkoek et al., 2014](#); [Lezak et al., 2012](#), pg. 127), the challenge of defining functions ([Bilder et al., 2009](#); [Kent, 2018](#); [Poldrack et al., 2011](#)), the ubiquity of measurement error ([Crawford, 2004](#)), and the lack of process homogeneity amongst people who score differently on the same test ([Borsboom, 2005](#), pg. 84). By extension, it is unclear what exactly covariance amongst test scores, the basis for estimating network models, means *cognitively*. Certainly, it cannot be equated with information processing in vivo (e.g., the recall of information from long term to working memory to solve a problem).

Second, the correspondence between the parameters and properties of network models and cognitive functioning is opaque ([Bringmann et al., 2019](#); [Fried and Cramer, 2017](#)). The former are statistical descriptions which can reflect multiple causal relationships ([Dablander and Hinne, 2019](#)). Betweenness centrality, for example, is taken as a measure of how well a variable facilitates communication between other variables ([Garcia-Ramos et al., 2015, 2016](#); [Tosi et al., 2020](#); [Nevado et al., 2021a](#); [Wright et al., 2021](#)). Mathematically, it quantifies how often a score is in the shortest route from one score to another in the network model ([Bringmann et al., 2019](#)). One might predict that a working memory score would display high betweenness centrality, given the role of working memory role in co-ordinating thinking and behaviour ([Baddeley, 2007](#)). However, the supervisory properties of working memory could not be inferred from network models alone. Both cognitive theory and experimental research are needed to establish such claims based on network models ([Fried, 2020](#)).

3.4. Error is ever present

Even though neuropsychological test scores are not perfect representations of cognitive functions ([Crawford, 2004](#)), many network models do not assume measurement error ([Epskamp and Fried, 2018](#)). Clearly, this contradicts the conventional wisdom in neuropsychology that measurement error is ubiquitous. In addition to the error that is part and parcel of neuropsychological assessment, error is likely to be introduced into network models by including multiple tests of the same cognitive function ([Epskamp et al., 2017](#)). Multiple tests of the same function are present in all the studies in [Table 1](#), but this source of measurement error is also ignored. Latent variable models of cognition can mitigate this error by reducing

a larger number of test scores to a smaller number of latent variables based on shared variance, although these factors are not necessarily cognitively meaningful (Delis et al., 2003; Kent, 2013).

3.5. Network versus latent variable models of cognition

Network neuropsychology does not disavow latent cognitive functions. However, the studies in Table 1 model associations amongst test scores, even though cognitive functions are not synonymous with the tests used to measure them (Borsboom, 2005; Poldrack et al., 2011). By modelling shared variance across scores, latent variable models can suggest latent cognitive functions (e.g., Agelink van Rentergem et al., 2020; Jewsbury et al., 2017). Although, this approach is not without limitation (Delis et al., 2003); long-term memory would not exist across healthy subjects if factor analytic models were taken as facsimiles of cognition (Kent, 2013)! The question of whether network or latent variable models provide the best map of the cognitive terrain emerges.

Mathematically, a hard distinction is redundant. Network and latent variable models are often statistically equivalent (van Bork et al., 2021) and strength centrality is nearly perfectly correlated ($r > 0.97$) with factor loading when latent variables exist (Hallquist et al., 2019). Indeed, while network models do not assume latent variables, dense communities of associations amongst variables imply that they are present (Epskamp et al., 2017, 2018; Epskamp and Fried, 2018). For example, in network models of Weschler Adult Intelligence Scale normative data, Schmank et al. (2019) found three communities of tests in the working memory, processing speed, and crystallised intelligence domains and an enmeshed community of subtests across fluid reasoning and visuospatial ability domains. These findings can suggest latent variables, although the authors did not.

While network models and latent variable models are often statistically equivalent, the emphasis in interpretation is shifted from shared variance to the unique variance between tests in network models (Costantini et al., 2015; Epskamp and Fried, 2018). It is unclear whether unique variance is clinically meaningful in neuropsychological assessment (Crawford, 2004, pg. 133), although current network neuropsychological models depend on it being so.

A potential step forwards is in using latent network modelling (Epskamp et al., 2017) to model a network of associations amongst theoretically specified latent variables. This method is consistent with the assumption of interrelationships amongst cognitive functions while explicitly handling latent constructs and error. However, it is not a panacea for mapping the cognitive terrain in network neuropsychology. Latent factors or communities of test scores, like the scores themselves, are not necessarily the same as cognitive functions (Delis et al., 2003; Kent, 2013). Also, network models of latent cognitive factors may provide a more invariant representation of cognition and therefore reduce sensitivity to any cognitive rearrangement (Nevado et al., 2021a).

3.6. Modelling associations versus dissociations

Communities and graph theory metrics reflect associations, being based, in one way or another, on covariance or the number of connections amongst test scores (Garcia-Ramos et al., 2016). It has been argued that associations amongst test scores are less powerful than dissociations, especially double dissociations, for making inferences about cognitive functioning (Ellis and Young, 1996, pg. 19; McCarthy and Warrington, 1990, pg. 19–20). Unlike dissociations, associations amongst test scores may occur for anatomical rather than cognitive reasons as proximal but distinct neural substrates are damaged (Ellis and

Young, 1996, pg. 6). It follows that graph theory may be of limited use for understanding cognition per se, although changes in graph theory metrics may reflect the nature of cerebral damage.

For example, in network models for groups with dementia, stronger interrelationships are observed amongst tests of episodic memory, semantic knowledge, and language (Ferguson for the Alzheimer's Disease Neuroimaging Initiative, 2021; Nevado et al., 2021a; Tosi et al., 2020; Wright et al., 2021). These associations may reflect mesial temporal lobe pathology rather than rearrangement amongst cognitive functions per se (e.g., semantic memory offering some compensation for episodic memory impairment). Of course, cognitive functioning ultimately depends on the brain and the level of neurocognition which network models reflect may not be an issue in all cases (e.g., for ruling pathology in or out). However, indications for cognitive rehabilitation (e.g., using semantic strategies to attenuate the impact of episodic memory deficits) may be groundless if a network model is presumed to reveal interrelationships amongst cognitive functions but actually reflects neural damage without rearrangement at the cognitive level.

3.7. Independencies, dissociations, and deficits

Network models can represent conditional independence relationships (Epskamp and Fried, 2018; Epskamp et al., 2018), which are akin to single dissociations, insofar as they suggest that one cognitive function is not affected by another. A conditional independence relationship is present in a partial correlation network model when two variables are not connected after conditioning on all variables in the model. For example, in their network model of language test scores in stroke patients, Massa et al. (2015) observed that language comprehension was conditionally independent given sentence construction, sentence reading, picture naming, and nonword reading. This finding is consistent with cognitive neuropsychological models of object recognition and language (e.g., Ellis and Young, 1996, pg. 31, 222), which posit distinct cognitive functions underlying these tests. While conditional independence relationships can suggest that processing in one area is not dependent on processing in another, they do not encode deficits relative to spared functioning, just the lack of statistical association. The sparing and impairing of functions needs to be considered simultaneously.

3.8. Variable selection

The variability in tests used across network neuropsychological models makes it harder to draw inferences about cognition. As shown in Table 2, there is generally weak overlap across test/scores (e.g., digit span backwards) used in studies of the same disorder, although overlap at the levels of subdomains (e.g., auditory working memory) and domains (e.g., working memory) is considerably better. Two network neuropsychology studies (Tosi et al., 2020; Wright et al., 2021) also suffered from a lack of a standardised battery across healthy and clinical groups, limiting comparison of network model structures.

Progress in network neuropsychology is likely to be limited unless tests are used consistently across studies. Without consistency, pragmatic studies aiming to identify disorders would be undermined as the parameters and properties of network models heavily depend on the specific task scores included (Burger et al., 2020). Investigations of cognition would also be problematised because test scores are not equivalent to cognitive functions. In effect, two tests of a particular cognitive function in two otherwise identical network models based on two identical samples may engender two different impressions of the role of the same function in the disorder.

A similar issue is the need to probe the same underlying cognitive functions across studies. Table 2 shows that broad cognitive domains (e.g., attention) are well represented across studies. However, subdomains (e.g., divided attention, focussed attention, sustained attention, attention switching), which are closer to specific cognitive functions, are less consistently probed across studies. Like variability in the tests used, discrepancies in the underlying functions probed by tests problematise meaningful understanding of cognitive rearrangement in neurological disorders. Firm conclusions cannot be drawn from a series of studies aiming to understand interrelationships amongst cognitive functioning in a given disorder if the sampled functions differ across studies.

A potential remedy is to use standardised batteries, which exist for several neurological disorders, including mild cognitive impairment (Boccardi et al., 2021), Alzheimer's disease (Morris et al., 1989; Weintraub et al., 2018), and frontotemporal lobar degeneration (Haanpa"ä"ä et al., 2015). Generally, these batteries identify reliable tests sensitive to differences in key cognitive constructs. However, standardised batteries are not present for all disorders and established batteries do not cover all relevant cognitive subdomains.

Table 2

Overlap of tests/measures used and domains and subdomains probed by topic.

Disorder	Studies	Content overlap (Jaccard Index)		
		Measure	Subdomain	Domain
Alzheimer's disease	Ferguson for the Alzheimer's Disease Neuroimaging Initiative (2021) Tosi et al. (2020) Wright et al. (2021)	0.21* (Weak)	0.38* (Weak)	0.58* (Moderate)
Paediatric epilepsy	Garcia-Ramos et al. (2015) Kellermann et al. (2015)	0.17 (Very weak)	0.5 (Moderate)	0.8 (Very strong)
Temporal lobe epilepsy	Kellermann et al. (2016) Garcia-Ramos et al. (2021)	0.83 (Very strong)	0.88 (Very strong)	0.93 (Very strong)

NB. Overlap is described with the Jaccard Index with reference to interpretive guidelines provided by Evans (1996), as in Fried (2017). The assignment of the tests/measures to domains and subdomains, which formed the basis of the Jaccard Index calculations, was based on that of the source studies, where reported. Otherwise, assignment was performed by the review author (see data and code at <https://osf.io/f9g2e/>).

*An average of the Jaccard Index for each possible pairwise comparison of studies (Evans, 1996; Fried, 2017).

3.9. Maps for individuals and groups

Neuropsychological network models of groups do not necessarily provide accurate characterisations of individual patients. There is evidence of intra- and inter-individual variation in cognitive functioning over time in both health and disease (Karr et al., 2013; Vaughan et al., 2013; Gomes et al., 2014; Villard and Kiran, 2015; Felice and Holland, 2018; Jones et al., 2019), which is not well accounted for in network neuropsychology. Most of the studies in Table 1 were based on cross-sectional, group level data. Group-based network models can offer an impression of individual patients' cognitive networks (Garcia-Ramos et

al., 2016). However, they are helpful approximations in the same way that an average can be a useful summary statistic. Like an average, the parameters and properties of a group network model are not necessarily displayed by any of the individuals in the group. The extent to which a model for an individual can be derived from a group model is a continuum rather than an all or nothing property (Adolf and Fried, 2019), but stronger resemblances enable better inferences, which are important in clinical practice.

Network neuropsychology might establish greater similarity between group models and the cognitive functioning of individual patients through refining group status, comparing similarities amongst multiple pairs of scores, or by explicitly modelling within-subjects changes. Regarding the former approach, one study used machine learning methods to group temporal lobe epilepsy patients into cognitive phenotypic groups prior to network modelling (Garcia-Ramos et al., 2021). This ensured more homogeneous groups; however, group status was based on exploratory techniques which do not apply to individual patients. In clinical practice, cognitive phenotypes are often what is to be ascertained rather than known a priori, which limits the utility of phenotype-based attempts to draw conclusions about individual patients from group models.

Outside of Table 1, in a study of healthy young adults, Konigs et al. (2021) estimated networks for individuals based on the difference in Z scores between multiple pairs of tests before referencing to a group model and only maintaining connections in individual models that were also present in the group model. Unsurprisingly, they found that models for healthy individuals and groups were consistent. This method could highlight quantitative differences in the strength of associations amongst tests between an individual and a group, but not qualitative differences; that is, different patterns of associations, as predicted by inter-subject variability and the assumption of cognitive rearrangement.

Alternatively, idiographic network models can be used to explicitly model both inter-subject and intra-subject interrelationships amongst test scores over time. However, this method must contend with practice effects, the availability of parallel test versions, test-retest reliability, and the burden of high-frequency testing on patients. Idiographic models also assume that the structure of the relationships and variances must remain the same over the entire period of data capture (Wright and Woods, 2020), which may not be the case for individual patients with neurological disorders.

4. Conclusions

Network neuropsychology makes two core assumptions about cognition. First, that cognitive functions are interrelated. Second, that cognitive rearrangement can occur in neurological disorders. Additionally, network neuropsychology does not disavow the presence of latent cognitive functions or patterns of segregation and integration at a cognitive level, although these are not well defined in the literature. These assumptions signify the cognitive terrain that network neuropsychology attempts to map with network modelling and graph theory. However, the correspondence between the maps and the terrain is not straightforward. Neither the variables within nor the metrics used to summarise network models readily correspond to cognitive processes. Tension also arises as latent cognitive functions are accepted but modelling strategies that do not explicitly account for these and for measurement error are used. At present, the validity of network models and graph theory for overall cognitive functioning is unestablished. Cognitive theory and experimental studies, as well as statistical models, are needed to build and interpret better network maps of cognition.

5. General recommendations

- Neuropsychological test scores should not be presented as synonymous with underlying cognitive functions. For example, digit span backwards should not be equated with working memory manipulation. Researchers should specify which tests are known to be sensitive to differences in which functions.
- Network parameters and properties should not be conflated with cognitive processing. For example, associations amongst test scores sensitive to attention, working memory, long term memory, and executive functioning should not be taken to denote the transfer of attended to information from working memory to long term memory with influence from executive encoding strategies.
- Latent network modelling (LNM) should be used to explicitly account for latent cognitive constructs and measurement error. For example, if several groups of tests sensitive to episodic memory differences, attentional capacity, and visuospatial processing are included in the same network model, LNM should be used to model associations amongst these latent constructs.
- If researchers are interested in deficits as well as interrelationships amongst functions, then patterns of spared/impaired performance and associations amongst scores should be considered simultaneously while interpreting network models.
- Before making inferences about interrelationships amongst functions based on patterns of associations amongst test scores, researchers should consider the possibility that neural changes account for these patterns as opposed to cognitive rearrangement per se.
- Standardised batteries should be used across studies of the same disorder to ensure consistent test use, which will facilitate replicability, and sampling of the same cognitive functions, which will avoid further complication with making inferences about interrelationships amongst cognitive functions based on associations amongst test scores.
- The feasibility of using idiographic network modelling methods should be explored, particularly regarding the test-retest reliability and availability of parallel forms of neuropsychological tests and the acceptability of repeated assessment to patients.
- Cognitive theory and experiments, as well as network models, should be utilised. For an intuitive example, the central executive is theorised to direct and regulate other functions. In network terms, one might anticipate high centrality for a central executive related variable in a network model as several other variables, including one related to long term memory acquisition (for example), would presumably be associated with it. An independent experiment might then test whether manipulating central executive demands during encoding of new memoranda impacts on long-term storage to (dis) confirm the influence of the central executive on memory acquisition. Cognitive theories might also arise based on network models. These should also be investigated with independent experiments.

6. Author contributions

Cameron E. Ferguson - conceptualization, writing, and editing.

Declaration of Competing Interest

The author has cited their own work in this review.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at <https://doi.org/10.1016/j.neubiorev.2021.11.024>.

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