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ARTICLE

Neuroplasticity and the logic of cognitive neuropsychology

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ABSTRACT

More than thirty years ago, Alfonso Caramazza laid out assumptions for drawing inferences about the undamaged cognitive system from individuals with brain damage. Since then, these assumptions have been challenged including the transparency or subtractivity assumption, that the cognitive system does not reorganize following brain damage. It has been repeatedly demonstrated that brains are highly plastic. However, there is no clear connection between brain plasticity and cognitive reorganization. Brain plasticity research does not require a rethinking of the core logic of cognitive neuropsychology. Differences in task-based activation between damaged and undamaged brains provide little insight into the cognitive architectures of brain-damaged patients. Theory and methods are needed to understand cognitive neuroplasticity, or how neural reorganization that follows brain damage relates to reorganization of functions. We discuss alternative types of cognitive neuroplasticity that may occur in damaged brains and consider how they impact the basic logic of cognitive neuropsychology.

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Empirical observations can only inform psychological science given a rich set of background assumptions that link data to theory. These background assumptions are necessary for all types of empirical observations, though their exact nature is rarely made explicit. A historic strength of cognitive neuropsychology is that these assumptions—those things that need to be true of the human mind and brain in order for researchers to draw inferences about the undamaged cognitive system by studying individuals with brain damage—have been explicitly outlined and discussed great detail (Caramazza, 1984, 1986, 1992; Caramazza & McCloskey, 1988; Coltheart, 2001; McCloskey & Caramazza, 1988; Shallice, 1988; see also Coltheart, 2017). We only briefly review these assumptions here: *functional modularity*, that the mind can be described as functional architecture, or a configuration of cognitive modules; *anatomical modularity*, that local brain damage can selectively impair one cognitive module while leaving other cognitive modules intact; *universality*, that all individuals have roughly the same functional architecture; and finally *transparency* (or what Coltheart, 2001, called *subtractivity*), that brain damage does not result in the creation of new cognitive functions that differ substantially from those used in the unimpaired system (Coltheart, 2001).

Caramazza (1986) further argues that with this set of assumptions the only valid inferences about normal cognition from brain-damaged participants come from single-case methods. Furthermore, he advocates focusing this single-case cognitive neuropsychological research on questions relevant to normal *cognitive* models, rather than questions about the neural correlates of cognitive functions. This approach to cognitive neuropsychological research, with its focus on single-case studies and lack of concern about localization of function, has been dubbed alternatively "orthodox" (Shallice, 1988), "radical" (Robertson, Knight, Rafal, & Shimamura, 1993), or "ultra" (Coltheart, 2004; Harley, 2004) cognitive neuropsychology. Unsurprisingly, given these modifiers, not all cognitive neuropsychological research falls into this category. In particular, in recent years there has been an increase in case series cognitive neuropsychology (Schwartz & Dell, 2010), which looks for associations across a large sample of participants, and in particular in case series studies that look for associations between performance on specific tasks and lesion location as a method for localizing cognitive functions (e.g., Bates et al., 2003; Rorden & Karnath, 2004). Therefore, as we reflect on the logical assumptions of cognitive

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neuropsychology, it is critical to consider both ultra cognitive neuropsychology and case series/functional localization cognitive neuropsychology.

Practitioners typically treat the logical assumption of cognitive neuropsychology pragmatically. Rather than these being testable hypotheses about how the mind works, the claim has been that the ability to draw reasonable conclusions about the normal cognitive system from cognitive neuropsychological cases depends on this set of assumptions to be true. The best support for these assumptions, therefore, is the success of the cognitive neuropsychology programme. As many people have argued, including McCloskey and Chaisilprungraung (2017), theoretical advances made with the methods of cognitive neuropsychology have been supported with converging evidence from other sources. These successes indicate that the assumptions are more than likely true.

Still, critics of the cognitive neuropsychological approach frequently question whether these assumptions are correct characterizations of how the mind and brain work (e.g., Farah, 1994; Kosslyn & Intriligator, 1992; Kosslyn & Van Kleeck, 1993; Patterson & Plaut, 2009; Shallice, 2015). For example, Farah (1994) challenged whether either the brain or the mind is as modular as is typically assumed by cognitive neuropsychology, while Shallice (2015) argues that the universality assumption ignores important premorbid individual differences in functional architectures. Many of these critiques are not specific to cognitive neuropsychology, as the same assumptions are required for making inferences from other types of neuroimaging and behavioural data. Standard approaches to neuroimaging that draw inferences about the function of specific brain regions from task-related differences in blood-oxygen-level-dependent (BOLD) response across a group of subjects rely on the universality, functional modularity, and anatomical modularity assumptions.

The one unique assumption for cognitive neuropsychology is the assumption of *transparency* or *subtractivity*. Basically, this assumption states that the brain-damaged system is the same as the unimpaired system except with some pieces missing. Caramazza (1986, p. 52) defined the *transparency* assumption in the following way:

> [The transparency] assumption essentially says that the cognitive system of a brain-damaged patient is fundamentally the same as that of a normal subject except for a "local" modification of the system.

Coltheart (2001, p. 10) defined the *subtractivity* assumption in a similar manner.

> Cognitive neuropsychology treats the functional architecture of an impaired cognitive system as the functional architecture of the intact system with one or more of its components damaged or deleted. The assumption here is that brain damage can impair or delete existing boxes or arrows in the system, but it cannot introduce new ones.

For the purpose of the current article, we follow Coltheart (2017) and treat these two assumptions as synonymous. Both assume that the cognitive systems of brain damage subjects are not fundamentally different from those of unimpaired participants, except for local disruptions caused by the damage. Because other methods in cognitive science do not depend on drawing inferences from damaged brains, critiquing the *subtractivity* assumption poses a specific challenge to inferences drawn from cognitive neuropsychology, without challenging other aspects of cognitive science. Caramazza (1984, p. 11) already imagined what such a critique would look like.

> The position could be taken that there is a *de novo* organization of the remaining components such that the processes involved in the original complex function now work differently. In this case, the pathological performance would not have a transparent relation to the working of the normal system and would make the analysis of pathological cases irrelevant for the understanding of normal cognition.

As discussed above, pragmatically, this does not appear to be the case, as cognitive neuropsychological evidence has made clear and lasting contributions to theory building. Still, researchers have challenged the validity of the *subtractivity* assumption, largely on the grounds that there is evidence that the brain reorganizes following brain damage (e.g., Johnson, Halit, Grice, & Karmiloff-Smith, 2002; Kosslyn & Intriligator, 1992; Kosslyn & Van Kleeck, 1993; Thomas & Karmiloff-Smith, 2002; Welbourne & Lambon Ralph, 2005; Welbourne, Woollams, Crisp, & Lambon Ralph, 2011). This critique is somewhat odd, as neither the *subtractivity* assumption nor the *transparency* assumption makes any reference to neural mechanisms, instead referring only to the cognitive mechanisms that brain-damaged patients use to

perform the behavioural task of interest. These researchers implicitly assume that this brain reorganization must have some important consequence at the cognitive level of description that would result in a violation of the *subtractivity* assumption.

However, we argue that there is no existing evidence that brain reorganization has these consequences for the cognitive systems of brain-damaged individuals. In order to make the case from neural data that the *subtractivity* assumption is incorrect, it is necessary to demonstrate not only that the brain has reorganized but also that the resulting reorganized brain instantiates cognitive functions that are substantially different from those used by unimpaired individuals. But this literature has largely ignored links between brain plasticity and cognitive reorganization. Indeed, many of the ways that we can conceive of the cognitive consequences of brain plasticity are consistent with the *subtractivity* assumption, and therefore do not detract from the ultra cognitive neuropsychology approach. This is not to say that new insights in brain plasticity have no impact on the variety of ways that we draw conclusions about unimpaired minds/brains from brain-damaged participants. As we discuss below, reorganization following brain damage might have a major impact on the validity of conclusions drawn from cognitive neuropsychological approaches that try to draw inferences about the location of specific functions in the brain, even if the resulting brain reorganization does not result in a de novo cognitive system.

Imaging brain reorganization during recovery from stroke

Research with humans on how the brain reorganizes following brain damage has largely involved functional neuroimaging with individuals who have naturally occurring brain damage, frequently from a stroke. While widespread changes in task-based activity maps following brain damage have been reported for a number of different tasks, what these changes mean in terms of cognition has not been well articulated.

Since functional neuroimaging became a widespread technique in cognitive neuroscience, researchers have been investigating how activation maps in brain-damaged patients relate to those in controls (Kiran, 2012; Meinzer, Lindenberg, Antonenko, Flaisch, & Flöel, 2013). It is common for researchers to report that different cortical regions are activated in response to the task in the patients versus controls. Specifically, when controls show activity for a task in regions that are damaged in the patients, it is common for the patients to show task-related activity in both contralesional and perilesional regions. Another common approach has been to compare functional magnetic resonance imaging (fMRI) between pre- and post-treatment sessions, showing that contralesional and perilesional regions show increased activation with successful treatment (Rapp, Caplan, Edwards, Visch-Brink, & Thompson, 2013). The fact that brain-damaged individuals show different patterns of activation from controls, and that brain-damaged individuals show different patterns of activation following treatment, has been argued to demonstrate the presence of neural plasticity, a dynamic reorganization of the neural system following damage (e.g., Fridriksson, 2010; Fridriksson, Richardson, Fillmore, & Cai, 2012; Leger et al., 2002; Meinzer et al., 2006; Saur et al., 2006; Turkeltaub, Messing, Norise, & Hamilton, 2011; Vitali et al., 2007).

What exactly this neural plasticity means in cognitive terms is less well understood. The debate has largely focused on whether brain changes indicate a shift in the location of an otherwise normally operating cognitive function, reflect the use of some type of compensatory mechanism for carrying out this task, or even reflect some type of maladaptive response that inhibits performance on the task. For example, in unimpaired readers, it is typical for a region of the left ventral occipitotemporal, frequently referred to as the visual word form area (VWFA), to be more activated during word reading than other visual processing tasks (Dehaene & Cohen, 2011). When undergoing task-related fMRI, patients with damage to the VWFA typically show greater activation for words compared to baseline in the right-hemisphere homologue of this region (e.g., Cohen et al., 2004; Ino et al., 2008; Pyun, Sohn, Jung, & Nam, 2008; Tsapkini, Vindiola, & Rapp, 2011) and/or in regions just adjacent to the lesion (e.g., Ino et al., 2008; Tsapkini et al., 2011). The fact that patients show increased activation for words in regions not typically observed in the unimpaired population has been used to argue that the orthographic function of the damaged region is reorganized into other regions that do not typically carry out that function (e.g., Cohen et al., 2004; Tsapkini et al., 2011). Note,

however, that this type of brain reorganization does not result in a "*de novo* organization of the remaining [cognitive] components such that the processes involved in the original complex function now work differently" (Caramazza, 1984, p. 11). The argument is that the same type of orthographic function as that typically carried out by left VWFA has shifted either to the right VWFA or to left-hemisphere perilesional regions. Therefore, this type of neural plasticity poses no challenge for the *subtractivity* assumption.

Still, it remains an open debate about whether these differences observed in fMRI even suggest a shift in the location of the cognitive function. Residual reading ability following brain damage could be due to the refinement of an alternative neural pathway for word reading that exists even in the undamaged brain. For example, the right hemisphere might have some rudimentary capability for visual word processing. When the left hemisphere is intact, readers do not make use of this right-hemisphere pathway. But when the left hemisphere is damaged, brain-damaged readers have to make use of this right-hemisphere pathway, with recovery over time resulting from participants learning to more efficiently use these alternative pathways, rather than from a dynamic change in the neural organization of the reading system (e.g., Behrmann, Black, & Bub, 1990; Behrmann & Plaut, 2014; Miozzo & Caramazza, 1998; Seghier et al., 2012). This account is entirely consistent with the *subtractivity* assumption; components of the normal cognitive system are impaired in the brain-damaged participants, and new novel components are created.

Both of these accounts assume that these changes in brain activation support the ability of brain-damaged patients to do the task. Even this assumption is under debate. Naeser et al. (2005) showed that disruption from transcranial magnetic stimulation in contralesional areas activated during language tasks in aphasic patients actually improves/improved language production in those patients. This finding has been interpreted to indicate that at least contralesional activation may reflect the engagement of a dysfunctional process that inhibits the ability to do the task (see also Postman-Caucheteux et al., 2010). However, when a second stroke damages contralesional regions, whatever function has recovered is severely impacted (e.g., Barlow, 1877; Bartolomeo, Bachoud-Lévi, Degos, & Boller, 1998; Basso, Gardelli, Grassi, & Mariotti, 1989; Turkeltaub et al., 2012), suggesting that the contralesional region had been supporting the residual capacity following the stroke.

All of this is to illustrate how difficult it is to interpret what changes in task-related activation patterns following stroke mean in terms of changes in cognitive function. These activated regions may reflect reorganization of the location of a function, an existing alternative pathway, or even a dysfunctional process. It is clear that the brain changes following stroke. However, we know little about what these brain changes mean with respect to changes in the underlying cognitive architectures. Evidence against the *subtractivity* assumption requires evidence that brain damage leaves patients with fundamentally different cognitive architectures from those of neurotypical individuals. Current research using fMRI to investigate neuroplasticity following brain damage cannot address critical issues of what brain reorganization means in terms of changes to the cognitive system.

What might cognitive neuroplasticity mean?

It is critical for research in neural plasticity to move beyond descriptions of task-based changes in brain activity towards determining what these brain changes mean regarding the cognitive systems of brain-damaged patients, or what we call *cognitive* neuroplasticity (see also Grafman, 2000). This research will deepen our understanding of how reorganization relates to recovery of function. Standard methods for mapping task-based activation patterns might not be the best approach to addressing these issues, though newer methods that can decode cognitive function from patterns of activation might provide some insights (e.g., Fischer-Baum, Jang, & Kajander, 2017).

Below we lay out different types of cognitive neuroplasticity that could occur following brain damage. In discussing these different types of cognitive neuroplasticity, we discuss how each impacts the logic of cognitive neuropsychology, both in terms of ultra cognitive neuropsychology and in case series/functional localization cognitive neuropsychology. As we show below, most types of cognitive neuroplasticity do not undercut the *subtractivity* assumption, and therefore do not limit the ability to draw inferences in the ultra cognitive neuropsychology framework, though cognitive neuroplasticity might place limits on the

ability to draw inferences about functional localization from brain-damaged individuals.

When a piece of cortex is permanently lesioned follow a stroke, what happens to the function normally subserved by that region? Figure 1 illustrates a series of possibilities. In Figure 1a, three brain regions are shown (X, Y, Z). In Figures 1b–1e, a stroke has damaged Brain Region X, and each figure shows an alternative for how the brain has reorganized the function normally subserved by Region X.

Figure 1b shows what we call *simple subtraction*. For this type of cognitive neuroplasticity, once the brain region is damaged, the ability to implement that function is also lost. That is, Brain Regions Y and Z continue to compute the same function as the function that they compute in the undamaged brain. Even in this case, it is possible to observe that the patient will show a task-based increase in activity in either Region Y or Z compared to controls, under the mechanism of compensatory masquerade (Grafman, 2000). Consider again the case of reading. It is possible that all people have available to them multiple pathways for reading: an efficient left-hemisphere pathway and a less efficient right-hemisphere pathway (Behrmann & Plaut, 2014; Coltheart, 2000; Miozzo & Caramazza, 1998). When the left hemisphere is intact, control participants rely less on their right-hemisphere pathway. When the left-hemisphere pathway is missing due to stroke, patients depend more on their right-hemisphere pathway and therefore show increased activation in this region. This type of cognitive neuroplasticity would be consistent with the assumptions of cognitive neuropsychology laid out over 30 years ago while simultaneously explaining many of the neuroimaging results during recovery from stroke. This type of cognitive neuroplasticity also means that there are no issues in drawing conclusions about the localization of function from brain-damaged patients, though if there are multiple neural regions that support specific cognitive functions even in the unimpaired population, multivariate approaches to lesion–symptom mapping might be more appropriate than traditional univariate approaches (e.g., Mah, Husain, Rees, & Nachev, 2014; Zhang, Kimberg, Coslett, Schwartz, & Wang, 2014).

An alternative type of cognitive neuroplasticity is *no-cost functional takeover*, as shown in Figure 1c. Here, the brain has reorganized, such that the function normally subserved from the damaged brain region (X) is now subserved by a different region typically assumed to be either the region just adjacent to the lesion (Y) or in the homologous region in the opposite, undamaged hemisphere (Z). Additionally, in this view the function has not fundamentally changed what type of information is being computed. One possibility is that the new region is able to fully take over the function normally computed by Region X, and therefore the patient should fully recover functionally. However, it is also possible that when the function shifts to a new region it is unable to compute the function as efficiently as is typical when it is computed by Brain Region X, and therefore some degree of cognitive impairment might be observed. Furthermore, this view assumes that introducing this new function to a brain region has no impact on the function(s) normally computed by that region—that is, the functional takeover happens at no cost to other functions. In the case of reading, what this might mean is that a function typically computed by a left-hemisphere region —for example, the orthographic function normally associated with the left visual word form area (Dehaene & Cohen, 2011)—is being computed by the right-hemisphere homologue (Region Z) of the visual word form area (e.g., Cohen et al., 2004; Fischer-Baum et al., 2017), despite the fact that Region Z typically does not compute that function.

This type of neuroplasticity introduces real complications in drawing inferences about the localization of function from damaged brains. The logic of mapping lesions to symptoms would say that, if Region X is the neural locus of a specific function, then patients with damage to Region X should be unable to compute that function, and patients without damage to Region X should be able to compute this function. Given this type of cognitive neuroplasticity and this logic, researchers would erroneously conclude that Region X is not the only region involved in processing this function, since individuals without this region still have this function elsewhere in the brain.

However, this type of neuroplasticity has little impact on our ability to draw inferences about cognitive architectures in unimpaired participants from brain-damaged subjects. The function that has been recovered here is not carrying out some fundamentally new representation and process not observed in unimpaired participants, and therefore the *subtractivity* assumption has not been violated. If patients still have some measurable cognitive impairment even

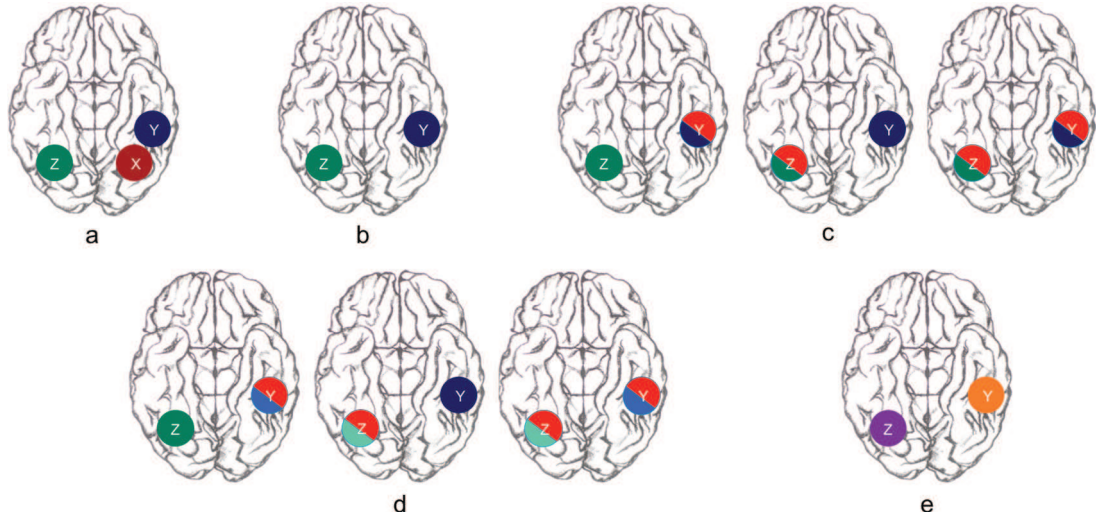

Figure 1. Types of cognitive neuroplasticity that could follow stroke. Intensity of colour represents intactness of a region's corresponding function. **(a)** Healthy brain with undamaged Regions X, Y, and Z, computing functions represented by colours red, blue, and green, respectively. **(b)***Simple subtraction* neuroplasticity: Following a lesion to X, Regions Y and Z continue to compute the same function as the function that they compute in the undamaged brain at the same capacity. **(c)***No-cost functional takeover:* the function normally subserved by Region X is now subserved by perilesional Region Y or homologous Region Z or both, without affecting the efficiency of the functions normally subserved by those regions. **(d)***Zero-sum functional takeover:* The function normally subserved by Region X is now subserved by Regions Y or Z or both, at the cost of efficiently computing the functions normally subserved by Regions Y and/or Z. **(e)***Creation of a new kind of function*, with the manner of processing in Regions Y and Z now distinctly different from their manner of processing in the undamaged brain and that of Region X in the undamaged brain, thus represented by two new colours: orange and purple, respectively. [To view this figure in colour, please see the online version of this Journal.]

after this type of cognitive neuroplasticity, then the patient can still be used to draw inferences about the unimpaired cognitive system within the ultra cognitive neuropsychology approach.

Related to *no-cost functional takeover* is the idea of *zero-sum functional takeover*, as shown in Figures 1d. For this type of neuroplasticity, the function normally subserved by the damaged brain region (X) is now subserved by the adjacent region (Y) or the homologous region (Z), though some degree of cognitive impairment might be observed. However, we additionally assume that the fact that this region (Y or Z) is now responsible for computing the damaged function means that the function that the region normally computes is less efficient. By calling this type of functional takeover "zero-sum", we highlight the fact that there should be consequences of retuning a region to subserve the damaged function in addition to the original function. Dehaene et al. (2010) argue for a similar type of reorganization during the acquisition of literacy. Specifically, they showed how, in illiterate participants, the left occipitotemporal cortex is specialized for face processing. When participants are learning to read, this same region starts to selectively respond to written words. This transformation comes at the cost of losing left-hemisphere face processing. Similarly, we might imagine that if a literate person loses the left occipitotemporal lobe to a stroke, the reading function might shift to the right occipitotemporal lobe, impacting the face-processing capacity of that region.

Zero-sum functional takeover cognitive neuroplasticity would have a clear impact on how we draw inferences about functional localization from brain-damaged participants. For questions of localization, we might incorrectly infer that the function normally subserved by Region Y or Z is actually subserved by Region X. A lesion to Region X will correspond to a decline in the function subserved by Region Y or Z, because, as the function normally carried out by Region X moves to these other regions, the functions typically computed in these regions will be impacted. With the example above, damage to the left occipitotemporal lobe impacts the face-processing ability because of reorganization in the right occipitotemporal lobe. This pattern might lead us to erroneously conclude that the left occipitotemporal is a face-processing region.

This issue with interpretation is not simply hypothetical. Behrmann and Plaut (2014) present a series of

individuals with left occipitotemporal damage who have severe reading problems and slight face-processing impairments, and a series of individuals with right occipitotemporal damage who have severe face-processing impairments and slight reading impairments. Their interpretation of this pattern is that neither the faces nor the words are subserved by independent neural mechanisms. Rather, they argue, both hemispheres are involved in both face and word processing, albeit with a slightly different set of computations in each hemisphere. This result has been used as evidence against both the functional and anatomical modularity assumptions of cognitive neuropsychology and cognitive neuroscience more broadly (e.g., Behrmann & Plaut, 2013).

An alternative interpretation of this pattern is that in the undamaged brain there is functional and anatomical modularity, with the left occipitotemporal lobe subserving a reading function and the right occipitotemporal lobe subserving a face recognition function. However, if either region is damaged, there is *zero-sum functional takeover*, meaning that with a left occipitotemporal lobe lesion, the reading function is somewhat preserved at the slight expense of face processing, and the opposite pattern is observed with a right occipitotemporal lobe lesion. Perhaps Behrmann and Plaut's (2014) inferences about the function of the left and right occipitotemporal lobe from patient data are correct. However, depending on the type of cognitive neuroplasticity that occurs after damage to those regions, there are alternative interpretations of their data.

At the same time, this type of cognitive neuroplasticity still does not violate the *subtractivity* assumption. The modification still results in local disruptions to the cognitive system, though multiple cognitive functions that might be somewhat distinct in the location of their neural instantiations might be impacted. For neither of the impaired functions does brain damage result in de novo cognitive functions. We can still draw the type of inferences about unimpaired cognitive systems from brain-damaged patients using the ultra cognitive neuropsychological approach.

Finally, the cognitive consequences of neural plasticity could be the *creation of a new kind of function*, as shown in Figure 1e. That is, after damage, the brain relearns how to read using a type of cognitive architecture not observed in the broader population. The function normally subserved by Region X is lost. Regions Y and/or Z start processing information in a manner distinct from both their manner of processing in the undamaged brain and Region X's manner of processing in the undamaged brain. Only this type of cognitive neuroplasticity clearly violates the *subtractivity* assumption; if this is the type of cognitive neuroplasticity that occurs following brain damage, we cannot draw inferences about the unimpaired cognitive architecture from brain-damaged individuals. We also cannot draw inferences about the location of unimpaired cognitive functions in the brain, since the brain-damaged individuals rely on cognitive functions not observed in the unimpaired population. However, if this is the type of cognitive neuroplasticity that typically occurs after brain damage, it is unclear why there has been so much converging evidence from other methods for conclusions drawn from cognitive neuropsychological studies.

Conclusions

Critics of cognitive neuropsychology have argued that the fact that the brain reorganizes after damage poses a problem for drawing inferences about the unimpaired cognitive system from individuals with damaged brains. Given the state of current research on neural plasticity, that simply does not appear to be the case. While there is clear evidence of brain changes following stroke, we know little about what these changes mean in terms of changes in cognitive function. Given the major contributions that cognitive neuropsychological research has made to our understanding of the unimpaired cognitive system over the past three decades, it seems very unlikely that we will discover that reorganization after brain damage results in the creation of entirely new cognitive systems. However, a more careful consideration of the cognitive consequences of brain reorganization may have some impact on the kinds of conclusions that we draw from damaged brains, namely in our ability to localize functions using lesion–symptom mapping methods.

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