Developmental disorders: what can be learned from cognitive neuropsychology?

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The discipline of cognitive neuropsychology has been important for informing theories of cognition and describing the nature of acquired cognitive disorders, but its applicability in a developmental context has been questioned. Here, we revisit this issue, asking whether the cognitive neuropsychological approach can be helpful for exploring the nature and causes of developmental disorders and, if so, how. We outline the key features of the cognitive neuropsychological approach, and then consider how some of the major challenges to this approach from a developmental perspective might be met. In doing so, we distinguish between challenges to the methods of cognitive neuropsychology and those facing its deeper conceptual underpinnings. We conclude that the detailed investigation of patterns of both associations and dissociations, and across both developmental and acquired cases, can assist in describing the cognitive deficits within developmental disorders and in delineating possible causal pathways to their acquisition.

1. Introduction

The discipline of cognitive neuropsychology developed in the late 1970s, at a point when cognitive psychologists and neuropsychologists began to see how valuable the detailed investigation of cases of acquired brain impairment could be for informing models of cognition [1]. This approach has since produced a wealth of fascinating case studies that have contributed to our understanding of cognitive functioning across a wide range of domains. In addition, the approach has led to improved understanding of the nature of underlying impairments in cognitive disorders, and consequently informed the diagnosis and treatment of these disorders. However, cognitive neuropsychology has not been without its controversies, particularly in relation to its usefulness for understanding developmental disorders. Bishop [2] and Karmiloff-Smith [3], in particular, have raised important concerns about whether the cognitive neuropsychological approach can inform our understanding of how particular cognitive processes develop or the factors that may impede their normal course of acquisition.

In this paper, we revisit this issue. We begin by outlining the basic tenets of the classic cognitive neuropsychological approach as it was initially formulated within the acquired domain. We then move to considering our key question of whether and how the cognitive neuropsychological approach can be meaningfully applied to the study of developmental disorders. In doing so, we make a distinction between the methods of cognitive neuropsychology and its conceptual underpinnings, and we consider challenges presented to each of these in turn from a developmental perspective. Throughout, we draw on examples from the domain of reading. We conclude that a cognitive neuropsychological approach, which draws on both associations and dissociations, and from both developmental and acquired cases, can be of value in identifying the cognitive deficits in developmental disorders and their underlying causes.

2. The cognitive neuropsychological approach

The dissociation of function, in which a patient is found to be impaired on a task assessing one cognitive process (say, written word recognition) but unimpaired...
on a task measuring another cognitive process (say, face recognition), has traditionally formed the backbone of the cognitive neuropsychological approach. As Ellis & Young [4, p. 5] put it in their classic text, ‘On such evidence alone, many cognitive neuropsychologists would feel justified in saying that the normal cognitive system must be organized with face recognition and written recognition handled by different sets of cognitive processes, thereby allowing one set to be impaired while the other continues to function normally’. Even stronger evidence for functional independence is argued to come from a double dissociation, where a second patient is found with the reverse pattern of strengths and weaknesses, thus reducing the likelihood that any single dissociation observed could be attributed to other factors such as differences in task difficulty [5] (although see [6,7]).

In classic cognitive neuropsychology, dissociations and double dissociations form the empirical basis for a proposed functional architecture. This architecture or cognitive model represents the sequence of discrete information processing components involved in performing some particular cognitive operation, such as reading aloud or identifying a face. The processing components may be considered ‘modules’ of the form proposed by Fodor [8] although, as discussed in more detail by Caramazza & Coltheart [1], a strong modularity assumption is not necessarily required. The important feature is that the components are relatively autonomous and can be selectively impaired by brain damage. According to this logic, cases of brain impairment should exist in which each of these components dissociates from the others (even though this may occur infrequently), and the performance of every case should be explicable in terms of the level of functioning of the combined set of components.

This leads us to a second key feature of the cognitive neuropsychological approach: its preference for individual cases as the unit of empirical investigation [9–11]. As each case of brain injury will produce its own unique pattern of associations and dissociations, cognitive neuropsychologists have traditionally argued that to average results across cases in a group study may disguise the very phenomena that are most of interest. Two cases showing a double dissociation between written word recognition and face recognition, for example, would have scores on these tasks which, averaged out, would suggest a pattern of a mild impairment in both skills. Cognitive neuropsychologists have therefore traditionally favoured individual cases as the primary unit of analysis, with each case in a sense serving as a separate experiment, or replication of an experiment.

The cognitive neuropsychological approach was applied at first almost exclusively to patients with acquired brain injuries. That is, the subjects of investigation were individuals who had previously acquired a fully functioning cognitive system but had then lost some aspect of that system owing to brain injury or disease. However, as the field became more established, some researchers began to explore whether the same approach could be used when the subjects of investigation were individuals (often children) who had never acquired a fully functioning cognitive system in the first place [12,13–15], a branch of the discipline referred to as developmental cognitive neuropsychology [16].

This extension of the approach has been controversial, with Bishop [2] and Karmiloff-Smith [3], in particular, articulating strong arguments against its validity. In the following sections, we respond to some of the key challenges that have been raised. We address first some issues surrounding the methods of cognitive neuropsychology, before moving to examining more fundamental conceptual problems.

3. Challenges for cognitive neuropsychology in the study of developmental disorders

(a) Methodological issues

(i) Dissociations and their reliability

A concern raised by Bishop [2] and others more recently [17] relates to the reliability of dissociations in the context of developmental disorders. All tests are, of course, subject to measurement error, and no test is ever a pure measure of the underlying process it purports to index, performance being subject to attentional, motivational and other extrinsic factors. This being the case, how do we know when an observed dissociation is ‘real’ and therefore theoretically informative? This concern applies both to the study of acquired and developmental disorders (see [18,19] for discussions in the acquired domain), but arguably the problem is greater in the case of developmental cognitive neuropsychology [2]: a deficit in an adult with an acquired disorder is in many instances quite clear-cut, given that the impairment is often of sudden onset and can be observed on tasks on which typical adults perform at or near ceiling. But, in the case of developmental disorders, drawing the line between ‘deficit’ and ‘no-deficit’ may be challenging, involving setting arbitrary cut-offs in continuous distributions.

Clearly, as in any type of research, it is essential to establish that our measures are valid and that our findings are robust and replicable (this requirement is of course not restricted to case studies: the potential for spurious findings arising from multiple comparisons could be seen as a corollary issue in large multivariate group studies). As Nickels et al. [20, p. 477] put it, ‘blind acceptance of dissociating cases is no better than blind rejection’. There are means within the cognitive neuropsychology toolkit, however, of meeting this requirement, and we would argue that the best examples of this research do so as well as or better than many group-based studies. First, in such studies, a dissociation will be confirmed through repeated testing, and across a range of convergent measures. Second, although the dissociation may occur in just one case, its veracity will be established across often large numbers of experimental items, and through careful manipulation of theoretically relevant item characteristics. Finally, any deficit that has been proposed based on quantitative scores will be further tested with a qualitative analysis of error types, providing a particularly direct and transparent test of the proposed functional deficit (for some good examples in the developmental domain, see [21,22]). In these ways, we believe meaningful dissociations can generally be distinguished from statistical or methodological artefacts in both developmental and acquired disorders.

However, even if it is possible to establish that cases of developmental disorders do exist in which two skills are reliably dissociated, a further issue is how we might interpret that ‘dissociation’. Specifically, if two skills are correlated imperfectly in the general population, it is inevitable that there will be some degree of dissociation, so do we need to set some further criterion for likelihood or extremeness of a
dissociation in order for it to be meaningful? We would argue that the answer to this question is no. On the contrary, in making the case that a particular developmental case shows a dissociation between X and Y, we are precisely arguing that there are two (at least partially) independent cognitive mechanisms in operation in typical development. We therefore expect any tests of X and Y to be partially decorrelated (i.e. correlated less than would be expected based on the reliability of each measure) in the general population. In almost every domain of developmental disorders, our cases of dissociation will not be examples drawn from unexpected ‘humps’ in a normal distribution (see [23] for developmental dyslexia); they will simply be cases selected from the population as (reliably) falling at the lower end of the normal distribution on one skill but not another. Thus, the frequency of their occurrence should be perfectly predictable from the true population correlation between those skills, were we to know it (see also [24]).

Nevertheless, the above discussion indicates the need for a more sophisticated approach to analysing dissociations within developmental disorders and we have begun applying recent methodologies in measurement and statistics to this problem [25]. In addition, a promising ‘hybrid’ approach is to conduct group-level analyses that allow for and to some extent quantify variation between individuals. For example, we have been conducting mixed random effects analyses in which both subjects and items are treated as random factors [26]. Here, rather than averaging across trials to obtain a single score for each participant, the objective is to model the data at the level of individual trials, either as a continuous variable or, in the case of accuracy data, as a binary logistic function (see [27]). One advantage of this approach is that it can provide information on the reliability of the data at the level of individual participants. We can see, for example, if the model fit is significantly improved if the effect of condition (e.g. words versus nonwords) is allowed to vary across individuals. We can also identify participants who appear to be reliable outliers in this regard.

(ii) The value of associations

As mentioned earlier, dissociations have traditionally been the ‘bread and butter’ of cognitive neuropsychology, with associations being assigned a lower status because of their lack of specificity and interpretability [4]. This has been viewed as a key point of difference in approach by researchers from other traditions, particularly those involved in studying developmental disorders: developmental psychologists tend not to trust dissociations, because they may focus attention on the exception rather than on the rule, and because they may reflect processing which is the outcome of an aberrant developmental pathway [2]. These researchers promote the status of associations as being the best means by which reliable patterns of co-occurrence among complex variables can be identified.

In our view, this point of difference has been somewhat exaggerated and may have led to an unwarranted perception of discord across different research traditions. Dissociations are certainly valued within cognitive neuropsychology, but it is not the case that associations have no place or are not used. Let us take an example from a recent paper examining three cases of apparent developmental letter-position dyslexia in English [28]. All three cases show a specific dissociation of interest: they are poor in reading aloud anagram words (which are susceptible to letter-position processing difficulties; e.g. *slime, smile*), but not other kinds of words. The problem is also shown to dissociate from difficulties in phonological processes. Thus far, therefore, the focus is on dissociations. However, the authors then hypothesize that the children’s difficulties arise at a pre-lexical stage of reading and proceed to test this hypothesis by examining predicted associations within the cases: they demonstrate that the reading aloud problem is also associated with difficulties on other tasks believed to involve pre-lexical processing, including nonword reading, visual lexical decision and same-different matching. Thus, a profile of the reading deficit in these cases, and its interpretation in the context of a theoretical model, is built up through a detailed investigation of the pattern of both associations and dissociations within each case. It should also be noted that the case series design in cognitive neuropsychology explicitly involves examining associations across cases. We discuss this method further in the section on case studies below.

Thus, associations are drawn upon in many cognitive neuropsychological studies. Conversely, it is also the case that dissociations are drawn upon in many traditional group-based studies (often in the guise of ‘uneven cognitive profiles’). Studies of developmental disorders almost invariably use at least some exclusion criteria. Many require normal range intelligence quotient (IQ); others exclude on the basis of attentional or low-level perceptual deficits. Although the degree of specificity may vary, we would argue that the reasons for adopting these exclusion criteria are essentially the same as those for identifying cases with dissociations: individuals are selected who are of interest because they show an impairment within one somewhat circumscribed domain but relative sparing in other domains, allowing research investigations to focus in a more targeted way on the cognitive impairments of interest. It is also notable in this context that the diagnostic process for many developmental disorders, such as autism, dyslexia, specific language impairment and Asperger syndrome also entails a dissociation logic. Thus, it would appear that both associations and dissociations are drawn upon widely across a range of research traditions, with each serving different purposes.

(iii) The validity of case studies

Related to the concerns about the lack of emphasis on associations in cognitive neuropsychology have been concerns about the overemphasis on single case studies [2,18,19]. As Bishop [2, p. 904] argues, ‘single-case methodology is not helpful for studying associations, because we cannot establish which correlated impairments are just chance associations and which correspond to reliable patterns of co-occurrence’. In part, this again touches on the methodological issues already discussed: if associated deficits can be identified within individual cases that are reliable, persistent across converging measures, and linked at an item level in systematic ways, confidence in some functional relationship between them is greatly increased. For example, there are several case studies of semantic dementia in which an association between irregular word reading deficits and semantic deficits has been demonstrated at an item level: the patients have difficulty reading the same words that they cannot understand [29,30]. Of course, no association in either a case study or a group study can establish a causal relationship,
but we would argue that associations of this kind, even in single cases, require explanation.

It should also be reiterated in this context that group studies present their own set of challenges—indeed, it was concerns about the group design as applied to clinical populations that motivated the move to case-based investigations in the first place. A reliance on group averages is really only justified if the condition being studied is homogeneous and we know that, particularly in the case of developmental disorders, this is very rarely the case (see [31] in relation to autism). In the presence of heterogeneity, group means can be highly misleading: the scores of a small number of participants can drive a significant effect that does not characterize the majority, and indeed may not even characterize any individual participant [10]. In addition, differences between samples can result in highly variable results across different studies, and problems with replicability [32].

Many group-based studies deal well with these concerns by going beyond overall means to look at correlates of particular profiles [33]. In addition, valuable middle ground may be found in cognitive neuropsychological work that extends beyond single cases into extensive case series analyses [34]. Here, patient variation on one variable is examined in relation to variation in other theoretically relevant variables across a large number of cases, as exemplified by work in acquired disorders such as semantic dementia [35] and anomia [36]. As Schwartz & Dell [34] point out, these kinds of investigations should not be seen as comparable to traditional group-based designs: many group studies treat within-group variability as noise, and seek to remove it by averaging across it, whereas case series designs treat the variability as the focus of interest and actively make use of individual data by characterizing the distributions of scores and the factors that covary with them. We hope to see this approach becoming more widespread within developmental cognitive neuropsychology, with work in developmental dyslexia [37–39] and in autism [40] demonstrating its promise.

In summary, developmental cognitive neuropsychology, such as every research tradition, is faced with methodological challenges. However, we feel that these challenges can be met and that they might not be so different from those faced by researchers within different traditions. Schwartz & Dell [34, p. 480] list the following as basic criteria by which cognitive neuropsychological research should be judged: ‘are the measures appropriate to the question being asked and the patients being studied? Have they generated data that are valid and reliable? Have important confounds been addressed? Do the results advance knowledge about the nature of human cognition? These seem like standards to which we would all hold our research, regardless of the specific approach.

(b) Conceptual issues
(i) Can cognitive neuropsychology shed light on the causes of developmental disorders?

Researchers examining developmental disorders typically ask somewhat different questions than those exploring acquired disorders. Perhaps most prominently, they are often not just interested in the impairment displayed by the case and how it can be accounted for within a cognitive model, but what caused the impairment to arise in the first place. Unlike acquired cases, where the cause of the impairment is generally a known neurological event, the cause of the deficit in developmental cases is typically unknown and may be a chain of events that has affected the acquisition of key skills over an extended period of time. Researchers are naturally keen to identify these causal pathways, as they may allow early identification and even prevention of the developmental disorder in future cases. The question becomes: can the cognitive neuropsychological approach be usefully applied to addressing these kinds of problems?

Answering this question first requires some consideration of what is meant by ‘causes’ in a developmental context. Imagine that we have a developmental disorder, characterized by impairments on a particular cognitive task, and we identify a putative causal factor which we hypothesize may play a direct role in producing the impairments observed. There are two possibilities as to what the nature of this direct role might be: one is that the presence of the factor impedes a child from being able to perform the cognitive task, and another is that the presence of the factor impedes the child from being able to acquire the skills needed to perform the cognitive task. That is, in the former case the factor impairs processing; in the latter case, the factor impairs acquisition, and is a ‘developmental’ cause in the sense that researchers typically mean that term (see also [41,42]). Jackson & Coltheart [43] make this same distinction in referring to proximal and distal causes of reading impairments: a proximal cause of a reading impairment is, by definition, part of the cognitive model of the reading system, whereas a distal cause is not part of the reading system but affects the acquisition of some component of it.

In a thought-provoking recent article, Apperly et al. [41] suggest that, somewhat paradoxically, data from acquired cases may provide insights about these different types of causes of developmental disorders that data from developmental cases themselves cannot. Apperly et al. focus on acquired and developmental impairments of theory of mind. However, in keeping with the rest of this paper, we consider these issues in relation to reading, specifically the difficulty in reading nonwords that is widespread in children with developmental dyslexia [44].

One cognitive deficit that has been linked with impaired nonword reading is poor phonological short-term memory—the ability to hold phonological information in a temporary store for future use—as measured by tasks such as digit span and nonword repetition [45]. If, for the purposes of illustration, we assume that poor phonological short-term memory is directly implicated in poor nonword reading in some way (of course it may not be), one possibility is that this is because phonological memory is involved in performing the task of nonword reading online. It might be, for example, that the products of letter-sound translation are transferred to a short-term memory store (or buffer) prior to being produced as a single utterance. This idea is represented diagrammatically in figure 1a. Here, a hypothetical cognitive model of the processes that might be required for nonword reading is presented, with phonological short-term memory represented as one of the processing components. An impairment of phonological short-term memory (in grey) leads to an impairment in nonword reading. Importantly, in this case, poor phonological short-term memory has not actually affected the acquisition of the processes required to read nonwords in any way. It is not a developmental cause of poor nonword reading.
Figure 1. Three hypothetical ways in which impaired phonological short-term memory (STM) might cause impaired nonword reading. Impaired components are in grey. (a) Impaired phonological short-term memory (buffer) directly affects online processing; (b) impaired phonological short-term memory affects the acquisition of letter-sound links; and (c) impaired phonological short-term memory affects the acquisition of letter-sound links and also directly affects online processing.

Alternatively, it may be that a sufficient level of phonological short-term memory is actually needed to acquire one or more aspects of the processing system for reading nonwords. For example, it may be that sounds, or sequences of sounds, need to be held in memory for long enough such that their association with printed letters can be learned, and that impaired phonological short-term memory compromises this letter-sound learning process. However, once that learning has taken place, phonological short-term memory is no longer used in any significant way to perform the task of nonword reading. This scenario is represented in figure 1b. Here, to represent a genuine developmental cause, we have adapted the ‘horseshoe’ notation of Morton & Frith [46,47]. A horseshoe link from a particular process (in this case, phonological short-term memory) means that how well that process is functioning affects how well some aspect of nonword reading ability is acquired. In this case, the problem with phonological memory is a developmental cause of impaired nonword reading as it has directly affected the learning process.

Of course, it is also possible that both of these scenarios are the case: poor phonological short-term memory disrupts the acquisition of one or more of the skills needed for successful nonword reading as well as directly disrupting online processing. This would be represented graphically in a form something like figure 1c, where impaired phonological short-term memory is depicted twice, once for its effect over time on the acquisition of letter-sound rules and once for its direct effect on reading nonwords.

The question we ask here is: can dissociations be identified within cases of developmental disorders that will assist us in distinguishing between factors that are genuine developmental causes, affecting the acquisition of knowledge within the cognitive system in question, from those which are processing causes, affecting the online functioning of the cognitive system but not necessarily its acquisition? The answer would appear to be no. As is apparent from figure 1a–c, regardless of which role phonological short-term memory plays, there will be no dissociation between it and nonword reading performance: both will be impaired, leading to an association between the two deficits.

Studying dissociations within cases of acquired cognitive deficits, however, presents a different picture. Taking the same example, let us imagine an acquired case whose phonological short-term memory is suddenly severely compromised after brain injury. If a sufficient level of phonological short-term memory is required to successfully perform the task of nonword reading, then that case will immediately demonstrate nonword reading deficits, exactly as represented in figure 1a. However, if the role of phonological short-term memory in nonword reading is a purely developmental one, then that case should not show any nonword reading impairments. As letter-sound rules will already have been successfully learned by the individual, the loss of a skill associated with their acquisition will have no negative impact, leading to a dissociation between phonological short-term memory (impaired) and nonword reading (intact).

As it happens, dissociations between phonological short-term memory and nonword reading have been reported in cases of acquired brain injury. Howard & Nickels [48] present the case of HB, a 60-year-old businessman who had acquired severe phonological impairments as a result of a left temporoparietal stroke. He displayed significant phonological awareness deficits and had severely impaired phonological short-term memory. In a digit span task, he could not reliably report more than two digits forwards. He was also severely impaired on nonword repetition, with less than perfect accuracy on even the shortest nonwords. Yet HB’s nonword reading was completely normal: his performance in reading aloud a set of 100 nonwords fell within the range of controls for both accuracy and response times. In an earlier paper, Howard & Franklin [49] reported the case of MK, who displayed a similar pattern.

According to Apperly et al.’s [41] reasoning, the dissociations displayed by these two acquired cases indicate that, as in figure 1b, phonological short-term memory is in some way involved in the acquisition of one or more of the processes required for reading nonwords, but is not involved in online cognitive processing. Of course, this assumes that the association is direct and leaves open the question of the precise nature of that developmental causal mechanism. Importantly, however, we would not have progressed even this far by studying developmental cases of impaired phonological short-term memory, where effects on processing and effects on development can be very difficult to separate. Thus, cognitive neuropsychological data—in this case from acquired cases—may provide valuable insights in relation to causes of developmental disorders.

There is, however, a critical assumption being made here—that the cognitive architecture (in this case of nonword reading) is the same in children as it is in adults. We must, therefore, consider another possibility—that phonological short-term memory is essential for the online process of
nonword reading in children but is not necessary for that online process in adults. Indeed, the belief that such qualitative changes do take place across development has formed the basis of much of the opposition to the developmental cognitive neuropsychological approach [2,3]. We now turn to this issue.

(ii) Can adult models be helpful in understanding development?
A major concern raised in relation to the cognitive neuropsychology of developmental disorders has been that the models used typically represent the ‘static’ adult system rather than the developing system [2]. It is certainly true that the models used have often been formulated based on data from the adult cognitive psychological research literature, and that the value of these models is therefore questionable if processing changes qualitatively during reading acquisition. How can having a model of adult processing assist us in understanding developmental disorders if children at different stages of acquisition perform those operations quite differently?

At least in the case of reading, however, it is our view that the evidence for qualitative shifts in processing is not persuasive, and that, as such, considering development with reference to skilled processing models is less problematic than has been suggested. Of course we cannot presume that this is the case for all domains, but the domain of reading does serve as a particularly good example here, because it is widely agreed to be a skill that must be learned as it has been acquired too recently in evolutionary terms to be innate [50].

There is a strong research tradition of modelling the skilled reading process, with several prominent theories having been successfully implemented computationally [51–53]. Although these models differ in some respects, all involve a system of connections between orthographic, phonological and semantic representations. In addition, in all these models, different sets of connections are required for successful nonword reading, typically referred to as alphabetic or phonological processes, than are required for successful exception word reading, typically referred to as orthographic or lexical processes. It is also important to note in the present context that all are models of the skilled reading system: although some, such as the parallel distributed processing (PDP) model of Pault et al. [53], are distinguished by having a learning phase, it is the ‘processing’ model achieved after the learning cycles are complete that forms the basis of most research using these frameworks.

The question we need to ask then is: can children’s reading throughout acquisition be represented accurately within these models? The 1980s saw the development of several ‘stage-based’ theories of learning to read, which have continued to be elaborated on in a range of ways [54–56]. These theories propose that there is a certain phase in acquisition when children primarily process written material in one way (e.g. the alphabetic phase) but that, as they became more skilled, they move away from this and begin processing it primarily in a different way (e.g. the orthographic phase). The theories therefore propose a qualitative transformation across learning that, if correct, would indeed render the skilled processing models irrelevant—a model where skilled reading was orthographic would be a completely inaccurate description of the reading of a child in the alphabetic phase.

However, more recently, stage-based theories have been challenged by more gradual, ‘item-based’ theories of reading acquisition, such as Shan’s [57] self-teaching hypothesis. These theories arose in response to the observation that the kind of step functions that might be expected to be associated with stage-based changes do not appear to be evident in reading acquisition—changes that occur are typically gradual and seem to depend more on the level and nature of the input than on the child’s stage (see also [58] in relation to spelling). For example, children (and adults) will read highly familiar words orthographically but less familiar words alphabetically, and the balance at any point in acquisition will be a function of their experience and the frequency of the words. There is no evidence that we are aware of that any type of reading processing, once acquired, actually disappears at a later point in learning. Skilled readers are much less likely to read alphabetically than children, but they can still do so when the input demands it—which is why the models of skilled reading described above represent both processes.

So what does change with reading acquisition? Obviously, knowledge within each of the processing components must be acquired, and the question of exactly what factors influence this learning process is an empirical one. However, we would argue that again there is evidence for considerable continuity across development. For example, members of our group have recently modelled the process by which children acquire alphabetic rules, basing that modelling on the hypothesis that order of acquisition is a function of (i) the simplicity of the mapping (one-to-one correspondences first, followed by more complex rules), and (ii) the frequency of that mapping in the language of exposure [59]. The effect of this is that, at different points in learning, children’s alphabetic knowledge will look quite different (more experienced readers will know and use mappings like ‘the final e rule’ that less experienced readers do not), but it does not mean that there has been any qualitative shift in processing at any point. Recent work by Taylor et al. [60] demonstrates that similar factors influence alphabetic learning of artificial orthographies in skilled adult readers. Importantly, if the factors influencing learning are similar across acquisition, then the ‘skilled’ processing model will represent the outcome of learning in a systematic and predictable way, and so represents a reasonable endpoint on which to anchor studies of development.

Another example from reading comes from the investigation of learning within another processing component: orthographic lexical representations. Members of our group have presented evidence that orthographic representations become gradually ‘tuned’ as children learn more and more words, so as to maximize ease of discrimination of highly similar looking words [61,62]. In some ways, this could be considered a qualitative shift with development. However, once again, the changes in processing are governed by the same factors all across development—the frequency and neighbourhood size (N) of the words that the child is exposed to—and these changes take place in a continuous way at an item level right through into adulthood. Some words become tuned very early, because they are so frequent and so difficult to discriminate, whereas others never become tuned even in adulthood. Indeed, reflecting this, the same factors of frequency and N are well established as modulating skilled lexical processing, and it was in fact exactly these observations that generated the developmental investigations [63,64].

Reading acquisition will also be reflected in changes outside of the processing components themselves: in the strengths of the links between those components. Indeed, it is changes of this kind, rather than any qualitative shifts in processing, that characterize the learning cycles in the PDP models of reading
acquisition [53]. It is also possible that developmental change will be characterized by the introduction of a degree of redundancy or ‘degeneracy’ into the processing system—the same functional outcome might be achieved by more than one alternative means (cf. Price & Friston [65]). But these changes are again not qualitative and can be accurately represented with reference to the skilled processing model. No particular processing mechanism is acquired and then subsequently lost.

In summary, in our view, there are two important features of adult cognitive models that make them meaningful and valuable in a developmental context. First, if accurate, they represent the endpoint of the acquisition process: ultimately, any theory of development must conclude with them. Second, they are models of processing; they represent the best description we have of the key operations involved in actually performing a particular cognitive task at any point in acquisition. To understand developmental disorders, what seems important is to map these models onto developmental theories that describe the way in which the processes are acquired. The cognitive models specify what children need to learn, and the developmental theories specify how they learn it. These are two separate enterprises, but each can and should complement the other.

4. Conclusion and future directions

In conclusion, solutions to complex problems usually require converging approaches. The limitations of one approach can be offset with the benefits of another. In our view, the methods of cognitive neuropsychology complement those of more traditional group-based developmental approaches, and drawing on insights from both will maximize progress in understanding the nature and causes of developmental disorders. Importantly, those insights will involve looking at patterns of both association and dissociation within and across individuals. They will also involve drawing on data from both developmental and acquired cases. Developmental cases will not necessarily only inform ‘developmental’ questions, and acquired cases will not necessarily only inform questions about the adult cognitive system. On the contrary, researchers studying acquired cases should be encouraged to consider developmental questions when carrying out their investigations, because dissociations they find may stimulate hypotheses about developmental causal pathways.

So can the cognitive neuropsychological approach applied to developmental disorders contribute to our understanding of the causal pathways that may have led to those conditions? We would argue yes, although this moves beyond cognitive neuropsychology’s more traditional aims. It can contribute, because the search for causes will be most informative if cases of developmental disorders are examined in the context of an explicit cognitive model of the relevant cognitive processes, and if patterns of sparing and impairment within that model are identified and drawn upon when looking for underlying developmental causes. To draw on genetic terminology, the better we characterize the ‘phenotype’, the more successful will be our search for developmental causes.

This can be conceptualized in the following way: the level of functioning of each process within a cognitive model can be seen as representing the endpoint of a set of causal pathways. For each process, this will consist of both general and specific causal pathways. There will undoubtedly be some general developmental pathways that will be broad in their impact, and that will affect the acquisition of more than one process within the model. However, if our model is accurate, then there must be at least one developmental pathway that will selectively affect the acquisition of a single aspect of processing. We know that this must be the case because, if it were not, that process within the model would not be dissociable from the other processes. Stated differently, it is impossible for one cognitive process to function independently of another cognitive process if there is complete overlap in the causal pathways to their acquisition.

Thus, developmental cases should be able to be identified who show dissociations that are meaningful and that directly reflect the compromise of a specific causal pathway. Investigating such cases provides an opportunity to explore hypotheses about the nature of that compromise. Ultimately, the only way to test developmental causal hypotheses is experimentally—through intervention studies. There are many examples of group-based treatment studies that test causal hypotheses [66,67], but we would argue that treatment designs within individual cases and case series also have the potential to be powerful [20,68]. Here, the cognitive profile of cases of developmental disorder is characterized in detail at baseline, and a proposed developmental cause is introduced as a targeted intervention. The effect of that intervention is then explored at a fine-grained level, examining its general and specific effects on the subsequent acquisition of different components of the system, and potentially providing direct support for a proposed causal mechanism. Such designs allow the best features of the cognitive neuropsychological approach to be drawn on, and demonstrate its potential for contributing to our understanding of the nature and causes of developmental disorders.

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