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Computational modelling of interventions for developmental disorders

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Abstract

Computational modelling has been used to advance understanding of the mechanistic causes of developmental disorders, particularly in the domains of language and literacy. In principle, such models provide the foundation to explore the effects of interventions to remediate developmental deficits. However, thus far, this avenue has been little explored. The current article lays the foundation for future work, considering the issues surrounding, and important factors influencing, interventions. Its initial perspective is on behavioural disorders that arise from computational limitations to a single developmental mechanism, focusing mainly on models drawn from the domain of language and literacy development. Based on this perspective, we argue that behavioural interventions will not be effective in producing full remediation unless: (1) the computational limitation of the mechanism is one of plasticity not capacity; (2) the previous informational input to the mechanism has not been representative of the target cognitive domain; (3) when additionally stimulated, the mechanism changes its computational properties; (4) there are compensatory pathways accessible beyond the single mechanism (i.e., other mechanisms that can do the same/similar task); (5) the problem that the learning system is trying to solve can be changed or simplified by altering its input/output representations; or (6) the system is not trained on the target problem but a subset of the problem that should be sufficient for most behaviours in the individual's environment.

(224 words)

If you know why a system is broken, one assumes this would help you to fix it. In the discipline of psychology, however, theories about causes of deficits have not always informed interventions. Indeed, Michie and Prestwich commented that 'despite the apparent advantages of applying theory to interventions, a substantial proportion of studies, identified in reviews, fail to make explicit reference to theory, let alone explicitly apply or test it' (Michie & Prestwich, 2010, p.1). To take one example, disorders of language, it has been argued that despite extensive theories about the causes of behavioural deficits, such theories have played a relatively small role in the intervention practices of speech and language therapists; and indeed, theories of treatment have often developed relatively independently of theories of deficit (Law et al., 2008).

The gap between a mechanistic understanding of the causes of deficits and everyday therapeutic practice exists for a number of reasons. These include the complexity of the therapeutic situation involving treatment of the whole child via a social interaction with the therapist (where the techniques employed are often dependent on the characteristics of the individual child and the therapist's experience and intuitions); the diverse real-world constraints on interventions, including resources like time and cost; the primary focus of intervention on behavioural outcomes, which do not in themselves necessitate an understanding of cause; frequent lack of an evidence-based consensus on the most effective treatment for a given deficit; the fact that children often do not have a single 'deficit' either behaviourally or in terms of underlying mechanisms; and, even when a theory of deficit exists, the difficulty of moving straightforwardly from that theory to a prediction of best treatment. As Byng (1994) argued, while theories of deficit are a necessary precursor to developing

therapies, 'simply having a detailed analysis of the deficit does not by itself suggest the formulation of specific therapeutic procedures to effect change' (p.270). In that sense, what is needed is a theory of therapy. Moreover, debates in education have even questioned whether the deficit itself is the best focus to consider remediation. Using terminology from rehabilitation, Bowers recently argued: '[it] is not clear that interventions should target an underlying disorder. Instead, interventions might be best when they are designed to enhance alternative skills (compensatory approach) as opposed to ameliorating the deficits themselves (restitutive approach)' (Bowers, 2016; see Howard-Jones et al. 2016).

Nevertheless, it remains an important ambition to narrow the gap between theories of deficit and practices of intervention. In a seminal paper, Plaut (1996, see also Plaut, 1999) demonstrated how connectionist computational models of cognitive processes could contribute to this endeavour, in that case informing rehabilitation following acquired brain damage in adults. Computational models of cognition focus on causal mechanisms and use implementation both to test theoretical proposals and derive novel predictions. Using explicit computational simulations, Plaut sought to provide a theoretical framework for understanding how and when experience-driven therapy would be most effective at remediating cognitive impairment in the domain of reading.

Connectionist models are computational systems loosely based on the principles of neural information processing. They are positioned at a level of description above biological neural networks and aim to explain behaviour on the basis of the same style of computation as that employed by the brain. Connectionist models construe cognitive processes in terms of activation

patterns over large numbers of simple processing units organised into networks. The units represent idealised versions of neurons, and influence each other's activation states via weighted connections. The strengths of connections store long-term knowledge in the network and are gradually learned through exposure to a structured learning environment (see, e.g., Thomas & McClelland, 2008).

Plaut's (1996) investigation focused on rehabilitation in a model of reading. In the model, information about a word's written form (orthography) was mapped to a representation of the word's meaning (semantics). Following damage to the network (removal of some subset of connections), the model exhibited *deep dyslexia*, whereby words were misread as their semantic neighbours. Plaut then explored opportunities to remediate reading deficits following damage by giving the system additional training experiences. There were two important findings. First, the degree of relearning, and the extent to which treatment extended to untreated words, depended on the location of the damage within the network. Outcomes were better for damage closer to semantic output than orthographic input. This provided a possible explanation of variation between patients in their recovery from brain damage. Remediation itself caused changes in weights not just in the region of damage but triggered reorganisation throughout the network. Second, generalisation of retraining to untreated words was more effective when the network was retrained with words that were *atypical* exemplars of their semantic category rather than typical members, an unanticipated and counter-intuitive finding. This provided a novel prediction about how to improve therapeutic intervention. Plaut concluded that the model had extended the relevance of connectionism in

cognitive neuropsychology by providing insight into the nature of recovery from brain damage and methods for remediation (see also Hinton & Sejnowski, 1986; Wilson & Patterson, 1990).

While the number of subsequent models of remediation of acquired deficits has been more trickle than deluge, there have been clear demonstrations of the utility of the approach. For example, Abel and colleagues showed how a connectionist model of word naming in adults (Dell, Schwartz, Martin, Saffran & Gagnon, 1997; Foygel & Dell, 2000) could be used to generate predictions about the locus of damage in individual cases of acquired aphasia, and these in turn could be used both to guide intervention and to explore the neural basis of intervention effects (Abel & Huber, 2009; Abel, Weiller, Huber & Willmes, 2014; Abel, Willmes & Huber, 2007).

Our focus in this article is to assess the utility of connectionist models that simulate interventions for cases of *developmental* disorders. It has been argued that, despite occasional similarities at a behavioural level, developmental disorders require a different type of causal explanation to acquired deficits, an explanation that incorporates the developmental process itself (Bishop, 1997; Karmiloff-Smith, 1998; Thomas & Karmiloff-Smith, 2002). Over 20 years, a wide range of connectionist models of developmental disorders has been published, targeting deficits in disorders such as dyslexia, specific language impairment, autism, and attention deficit hyperactivity disorder (see Thomas, Baughman, Karaminis & Addyman, 2012; Thomas & Mareschal, 2007; Thomas & Karmiloff-Smith, 2003a, for reviews). Much of the research has been in the field of language and literacy acquisition. Connectionist models of developmental disorders have served to progress theories of deficit, sufficient that some authors have foreseen

an influential role for connectionism in intervention research. In his 2002 volume *Connectionist Approaches to Clinical Problems in Speech and Language: Therapeutic and Scientific Applications*, Daniloff argued that connectionism 'will, in conjunction with other theories, explain the swiftness and accuracy of language processing during acquisition and maturation and thus form the backbone of much of language therapy in the near future' (Daniloff, 2002, *p.viii*).

Almost a decade later, Poll (2011) argued that a theoretical foundation for language interventions is an important step in evidence-based practice. He advanced *emergentism* as a leading theory of language development. Emergentism is the view that language skills are the product of interactions between the language environment and the learning abilities of the child (Elman et al., 1996). Similarly to Daniloff, Poll identified connectionist modelling as a key approach underpinning emergentism and argued that 'insights from connectionist research on the acquisition of early morphology and syntax can provide theoretical guidance for language intervention' (Poll, 2011, p. 583).

Despite the enthusiasm shown by Daniloff and Poll, this promise has yet to be realised. To date, only two connectionist models of developmental deficits have paid serious attention to the simulation and evaluation of interventions (Best et al., 2015; Harm, McCandliss & Seidenberg, 2003).

This article considers in more detail the potential application of connectionist models of developmental disorders to intervention research, in the service of closing the gap between theories of deficit and theories of treatment. We take a similar approach to that of Plaut (1996), focusing on single network models of specific abilities. We will advance the following argument: if the reason why an experience-dependent learning mechanism cannot acquire a

target behaviour is a limitation in the mechanism's neurocomputational properties, then behavioural intervention will be unable to fully remediate the deficit. *Totally successful intervention will not be possible*. Nevertheless, we argue several exceptions exist to this general rule, which in themselves provide an insight into the challenge of linking theory of deficit to most effective treatment. Before we articulate this argument more fully, we give a brief introduction to intervention practices, using developmental language disorders as our main example, and briefly review existing connectionist models of intervention.

Behavioural interventions for developmental disorders of language

Interventions encompass a wide range of activities. One definition, in the context of improving the language skills of children with speech, language, and communication needs, describes an intervention as 'an action or technique or activity or procedure (or indeed combinations of these) that reflects a shared aim to bring about an improvement, or prevent a negative outcome … this can also include the modification of factors that are barriers or facilitators to change and the modification of an environment to facilitate communication development' (Roulstone, Wren, Bakopoulou & Lindsay, 2010, p. 327). Roulstone et al. identify several terms that are used interchangeably, including *treatment*, *therapy*, *intervention*, and *remediation*.

One principal determining factor influencing choice of intervention method is the child's age. *Implicit* techniques are employed with younger children, while *explicit* techniques are employed with older children (Stokes, 2014; Laws et al., 2008). For younger children (less than 6 years), the main aim is skill acquisition. Techniques are informal and naturalistic, with implicit goals

and methods embedded in child-directed learning contexts. For older children (more than 6 years), intervention also targets meta-cognitive abilities and the development of compensatory strategies. There is greater use of formal methods, employing explicit goals and instruction in a therapist-directed learning context. While there is a general view that targeting causal processes early in disordered development may be more effective than waiting until outcomes are established (Wass, 2015), systematic evaluations of timing-of-intervention effects are less common. Important dimensions of the intervention method include the precise nature of the intervention itself; who delivers the respective components of the therapy (e.g., a speech and language therapist [SLT], an SLT assistant, a teaching assistant, teacher, parent, or a computer); if the therapy is delivered one to one, or in a group; and the dosage of the intervention, including intensity and duration (Ebbels, 2014).

Later we will consider interventions applied to a computational model of children's acquisition of the English past tense. To give a concrete example of an intervention relevant to this domain, Seeff-Gabriel, Chiat and Pring (2012) evaluated an intervention to improve performance in producing regular English past tenses for a 5-year-old child with speech and language difficulties. The intervention was delivered one-to-one by an SLT, with carryover from the mother and the school. Facilitation methods were used, including modelling and elicitation, to help the child produce the correct past tenses, combined with visual symbols to provide meta-linguistic support. The intervention dose was 30 minutes a week for 10 weeks with the SLT for a total of 5 hours, plus the additional input from the mother and school. This pattern is representative of a single block of intervention: in a survey of over 500 SLTs in the UK, Lindsay et al.

(2010) reported the most common frequency of delivery of a language therapy was once a week for 6 weeks or more, with 42% asking teachers and parents to deliver the intervention more frequently between visits to increase the dosage. Blocks may be repeated. This typical dose and duration can be contrasted with much larger dosages for other developmental disorders, for instance to address the wider socio-communicative deficits in autism. In one form of the early intensive behavioural intervention (EIBI), intervention begins by 2 years of age, with a range of 20 to 40 hours per week across one to four years of the child's life, for a range of intervention dose of between 1000 and 8000 hours (Eikeseth, 2009; Smith, 2010).

Children can vary widely in their response to interventions. Apart from the age of the child, other characteristics are relevant to intervention outcome, including the severity of the developmental deficit and the presence of other co-morbid deficits (Ebbels, 2014). The relationship between dosage and the effect size of the behavioural improvement produced by the intervention also varies, and depends on the target ability. For example, Lindsay et al. (2010) summarised meta-analysis data to indicate that for interventions targeting phonology, intensive interventions were more effective than those of long duration; for syntax, interventions of long duration were more effective than short intensive ones; for vocabulary, long duration was important but not intensity – children did better with short bursts over an extended time. In a well controlled study of a grammar treatment for 5-year-olds with SLI, Smith-Lock et al. (2013) found that the same dose of 8 hours was more effective delivered weekly over 8 consecutive weeks than daily over 8 consecutive days. These differences presumably relate

to the functional plasticity of the underlying mechanisms, including time for consolidation and opportunities for practice.

Practice varies as to whether the primary aim of intervention is to remediate the deficit or to encourage development of potential compensatory strengths. We will later consider a computational model of vocabulary development addressed to simulating children with word-finding difficulties (WFD). In WFD, children struggle to produce words that they can nevertheless comprehend. WFD is viewed as a heterogeneous disorder, with possible causes either in phonological access or impoverished semantic representations (Best, 2005; Faust, Dimitrovsky & Davidi, 1997). In a survey, Best (2003) reported that SLTs listed phonological awareness difficulties as co-occurring with WFD 46% of the time, while semantic problems co-occurred only 13% of the time. Thus, phonological deficits appeared to be the more frequent cause. Nevertheless, intervention approaches that targeted semantics were used more frequently than those that targeted phonology (79% of the time compared to 54%). SLTs, therefore, often sought to buttress areas of strength within the child to improve naming skills.

The order of targeting skills within a domain may also be important. For example, in the usage-based approach to remediating developmental problems in syntax, structures are targeted in an order that takes into account the gradual ordered development of grammatical structures in typically developing children (e.g., Riches, 2013). The order of acquisition reflects the interaction between the challenges of the particular domain and the constraints of developmental mechanisms.

Which intervention should a therapist choose? The decision is influenced by multiple factors. A key factor, of course, should be the intervention's effectiveness. However, Roulstone et al. (2010) noted that evidence for effectiveness incorporate clinical experience or local evaluations, in addition to research evidence. Roulstone et al. identified several other factors influencing intervention choice, including reference to underlying theoretical positions, and pragmatic reasons related to efficiency, accessibility, popularity and cost. Other researchers have taken a wider perspective on the factors influencing the design and success of interventions aiming to change behaviour. For example, Michie and colleagues (e.g., Michie, van Stralen & West, 2011) constructed a framework that incorporates not just the internal cognitive mechanisms able to deliver behavioural change (which they termed 'capability'), but also motivation and opportunity to change. The framework identifies environmental influences and structures, such as resources and policy, which operate as constraints on or incentives for success.

There are two important dimensions in the evaluation of interventions. The first is the extent to which the intervention *generalises* to other items or skills beyond those targeted in the intervention itself. The second is the *persistence* of the benefits of intervention after the intervention has ceased. Using our example study of Seeff-Gabriel et al. (2012) targeting English past tense, the 5-hour intervention was found to generalise to untrained regular verbs but not to irregular verbs, while progress was maintained at follow-up 8 weeks later. Generally, achieving generalisation and persistence of interventions has proved challenging. For example, in her review of interventions for grammar difficulties in school-aged children, Ebbels (2014) concluded that follow-up

generally shows that the progress produced by the intervention is maintained, but does not continue after the intervention has ceased. The gains are retained but no further gains are stimulated. Bailey, Duncan, Odgers and Yu (2017) identify the diminishing effect of an intervention after its cessation, so-called 'fade-out', as a characteristic of many interventions targeting cognitive and socioemotional skills and behaviours.

Other important factors include: (i) child preferences (e.g., a child's willingness to work on target A but not B); (ii) parental involvement (what are appropriate activities for home practice to maximise dose); (iii) context (e.g., selecting vocabulary items to mirror those currently being taught in the school curriculum); and (iv) outcome of intervention (such that the therapist may modify targets, methods, and feedback according to the response to previous intervention).

Lastly, even if an intervention has been shown to be effective, unless its key 'active ingredient' has been understood, it is not guaranteed that the effect will be similar when applied to new children, when delivered by less expert practitioners, or when adapted to new contexts. Identification of the active ingredient in turn requires comparison to a control group whose treatment differs only in the active ingredient. And this in turn requires a theory about how the intervention remediates the deficit or supports a compensatory strategy.

In sum, interventions involve activities to improve developmental outcomes in children; their outcomes are variable depending on the characteristics of the child and therapist; both the design and the dosage of the intervention are important for outcome; and outcomes need to be evaluated against key criteria of generalisation to other items or skills beyond those

targeted in the intervention itself, and maintenance of gains once the intervention has ceased.

Connectionist models of interventions for developmental language disorders

In connectionist simulations of development, changes in behaviour are the result of experience-dependent alterations to the structure of an artificial neural network, caused by its interaction with a learning environment with particular informational content. A connectionist network model has intrinsic constraints that affect its learning ability and rate of development. These constraints include properties such as the number of simple processing units, the pattern of connections between units, the rate at which connection strengths change in response to experience, and the way external or environmental inputs are encoded for processing. Models of developmental deficits propose that these constraints are atypical in some children, deflecting developmental trajectories outside the normal range of variation (Thomas & Karmiloff-Smith, 2003a, b).

Early models took what might be called a *monogenic* approach to explaining developmental disorders. Connectionist models usually have several free parameters. For example, most models specify the values of parameters such as the number of internal or 'hidden' units, the learning rate, and the momentum. Values for these parameters are determined so that the model captures the trajectory of typical development. The model might be run several times with different initial randomised connection weights, to show that its behaviour is not highly sensitive to starting conditions, with the reported behaviour an average of these runs. Sometimes, the multiple runs are equated

with simulating different children in a group (Thomas, 2003a). In the disordered case, just one parameter is then set to a different value, such as reducing the number of hidden units in a model of reading to simulate dyslexia (e.g., Harm & Seidenberg, 1999; Plaut et al., 1996), or increasing the number of hidden units in a model of categorisation to simulate autism (Cohen, 1994). The disorder, then, has a single cause, against a background of very small or zero variation in all other computational parameters across individuals.

More recently, models have adopted a *polygenic* approach (e.g., Thomas, Forrester, & Ronald, 2015; Thomas & Knowland, 2014; Thomas, 2016a). Individual variation in the development of cognitive abilities is viewed as arising from small variations in many neurocomputational parameters, including those involved in the construction, activation dynamics, adaptation, and maintenance of network architectures. The approach involves simulating development in large populations of individuals. The cumulative effect of many small contributions produces a normal distribution of the development of behaviour in the population, against which a 'normal range' of variation can be defined, and cases of developmental delay identified (Thomas, 2016b). Disorders are thus viewed as the lower tail of a continuous distribution of developmental variation in a population.

The monogenic and polygenic approaches are not mutually exclusive. For example, Thomas and colleagues demonstrated how autism might combine two groups, monogenic cases with a genetic mutation causing a given neurocomputational parameter to take up extreme values, and polygenic cases with the same parameter falling in the upper normal range but having its effect on behaviour amplified by a combination of risk factors that vary across the

whole population (Thomas, Davis et al., 2015; Thomas, Knowland & Karmiloff-Smith, 2011). Furthermore, interaction of a monogenic cause and population-wide polygenic individual differences can give rise to apparent sub-groups within the developmental disorder despite it having a single pathological cause (Thomas, Davis et al., 2015; Thomas, 2016b).

The simulation of intervention builds on the foundation of an implemented model of a developmental deficit. From the point of view of a single processing mechanism, *behavioural* interventions can be construed as the addition of new information to the structured learning environment (or in simulation terms, new / replacement patterns to the training set). *Biological* interventions can be construed as direct alterations to the existing computational properties of the system, for example through pharmacological treatments, transcranial magnetic stimulation, or neurofeedback. In some types of models, changes in computational properties might subsequently serve to alter the system's sampling of its learning environment.

The statistical learning perspective of which connectionism is a part has generated a growing understanding of environmental factors that produce stronger or weaker learning in typical development (Borovsky & Elman, 2006; Gomez, 2005; Onnis et al., 2005). This includes the importance of factors such as the frequency of training items, their variability, and the provision of novelty in familiar contexts. Here's an example of a heuristic that arises from this approach: to improve acquisition of *compositional* domains, in which concepts are made up of different combinations of the same primitives, show the system the component primitives either in isolation or in many different combinations. This will also encourage subsequent generalisation to novel instances.

Potentially, these kinds of lessons can provide guidance on how to design intervention sets to achieve the best behavioural outcome for a model with atypical computational constraints. However, this would be to assume that an understanding of the experiences that improve or hinder learning in *typically* developing systems is informative about how to influence developmental outcomes in cognitive systems with *atypical* constraints. The usage-based approach for grammar deficits in the previous section (Richie, 2013) is an example of extrapolating from typical development, in that case in the order of acquisition of syntactic structures, to inform intervention in cases of disorder. Poll (2011) explicitly argued that (connectionist-influenced) emergentist principles from typical development should be applied to language interventions for atypical development. He identified three such principles: that the structure and quantity of the input is important in driving development; that language development does not occur through passive exposure but via experiences related to the child's own expectations; and that language development concerns learning the relationship between language form and language meaning such that contextual cues which narrow the hypotheses will aid learning.

Before we consider the main implemented simulations of intervention, it is important to draw a distinction between two aspects of behaviour in cognitive computational models. The first is *performance on the training set*, that is, the range of experiences the system encounters in its structured learning environment. The second is *performance on a generalisation set*, that is, items which are novel to the system but which bear similarity to those with which it has experience. Systems with a so-called inductive bias (Mitchell, 1997) can take advantage of their existing knowledge to produce responses to novel inputs. If –

externally, as modellers – we stipulate that the structured learning environment in fact contains some underlying regularity or function, we can assess the generalisation performance of a system depending on whether it has extracted this underlying function from its training examples, and is then able to apply it appropriately to novel items. This distinction is important for several reasons. As we shall see, developmental deficits may operate differentially across performance on the training set and the generalisation set. Interventions are often assessed specifically on their ability to produce generalisation beyond items treated in the therapy. And interventions may themselves be targeted differentially towards training set or generalisation performance.

The Seidenberg and McClelland 'triangle' model of reading (Seidenberg & McClelland, 1989; Plaut et al., 1996; Harm & Seidenberg, 1999) provides perhaps the best example of extending a model of a developmental deficit to investigate interventions. Harm, McCandliss and Seidenberg (2003) were concerned with understanding the apparent paradox that, while a phonological deficit is often viewed as the principal cause of developmental dyslexia, interventions that target spoken language (phonology) alone are relatively ineffective at remediating reading deficits once a child has learnt to read. Instead, interventions need to combine work on phonology and on decoding, that is, learning the mapping between print and sound (Bus & Ijzendoorn, 1999).

Harm et al.'s (2003) model of reading involved a phonological component, which first learned a lexicon of English words. An orthographic component then provided representations of the written forms of words, which had to be associated with the existing phonological representations. Dyslexic versions of the model were produced by applying atypical constraints to the phonological

component, which impacted on its initial phase of acquisition. Specifically, prior to training, 50% of the connection weights were set to and held at zero, and weight decay was applied to the remaining weights, thereby limiting the maximum magnitude that they could reach during training. Before reading acquisition commenced, phonology was atypical. The outcome of reading acquisition was a system with a particular deficit in its nonword reading, that is, its generalisation of reading to novel forms. Such a deficit has been termed 'phonological' developmental dyslexia (Castles & Coltheart, 1993).

Harm et al. (2003) then compared two interventions, each applied at two different points in training. One intervention simply alleviated the phonological deficit – unfroze the 50% of weights and removed weight decay. One could view this as a sort of biological intervention that directly targeted neurocomputational properties. The second intervention added new items to the training set, to simulate a particular behavioural intervention (the Word Building Intervention; McCandliss et al., 2003). This took the form of extra 'lessons', triggered with low probability during each training cycle, commencing after a given point in training. The lessons represented training on an ordered sequence of words each of which differed by changing or moving only one grapheme (e.g., sat, sap, tap); where the model made an error, extra training was given on the individual component grapheme-phoneme mappings of a word (for 'sat', s=>/s/ in first position, a=>/a/ in second position, etc.).

Both interventions produced benefits to nonword reading, albeit without fully remediating the deficit to the levels observed in the typically developing model. The timing of intervention was also important. Alleviating the phonological deficit alone only showed benefits when applied early in training,

while the simulated behavioural intervention that targeted decoding showed benefits across training. The explanation for this *age-related* effect, paralleling the observed empirical data, was that once the network began to learn mappings between orthography and impoverished representations of phonology, these were hard to undo even if phonology was remediated later on. An apparent sensitive period for remediation by training phonology alone, therefore, was explained by *entrenchment*: the difficulty of resetting inappropriately configured connection weights (Thomas & Johnson, 2006).

To note from the Harm et al. (2003) model, then, the initial developmental deficit was mainly in generalisation rather than performance on the training set. The deficit was remediated by showing the network the component parts of holistic representations (in line with the heuristic identified in statistical learning approaches) through the particular sequence of presentation of items in the 'lesson', and the addition of new information to the training set in the form of individual grapheme-phoneme correspondences. Lastly, there was a contrast between what one might view as a biological intervention (directly changing computational properties) and a behavioural intervention (adding something new to the training set and/or changing the frequency distribution within the training set).

The second model by Best et al. (2015) is more recent and explored interventions for children with WFD. Developmental deficits in productive vocabulary may be caused in at least two ways: by impairments in the semantic representations driving naming or by impairments in accessing phonological output forms. The naming model had two components: a semantic component and a phonological component, each of which underwent its own developmental

process to establish its internal representations. There were then pathways to learn the mappings between these representations as they developed, from semantics to phonology to simulate naming, and from phonology to semantics to simulate comprehension. Constraints applied to either of these components, or to the pathways between them, could then produce developmental naming deficits. The model was used to predict the outcome of interventions on two individual 6-year-old children diagnosed with WFD. Two atypical models were calibrated to resemble the developmental profiles of the individual children, according to measures of the children's phonological knowledge, semantic knowledge, naming, and comprehension abilities. The model manipulations involved removing connections, reducing the number of hidden units, or altering the activation dynamics of the simple processing units, either in the components or the pathways, but always prior to training.

The individual models were then given either a 'semantic' or a 'phonological' intervention. The semantic intervention involved additional training for the semantic component to improve its internal representations, while the phonological intervention involved additional training for the phonological component. The interventions were interleaved with the normal training regime for vocabulary development. The result was a prediction for which type of intervention would work best for each child. The model predictions were then tested in reality by giving each child both a semantic and a phonological intervention in turn (1 session of 30 minutes per week for 6 weeks, for a total of 3 hours for each intervention type, and a 6-week wash-out period between interventions). It was then determined which improved naming skills more. For one child, the model's prediction was correct (only the phonological

intervention benefited naming performance); for the other child it was not (the model predicted both interventions would work; the child only benefited from the semantic intervention).

To note from the Best et al. (2015) model, once more a behavioural intervention was simulated by modifying the training set, in this case altering the relative amount of training on different components of the system, but without the addition of new information. Intervention success was measured against performance on the training set, although the intervention occurred only on a subset of the full training set. The model focused on differential effects of therapy type and did not report whether deficits were fully remediated in either case.

We now move to consider what we believe is the central lesson from viewing interventions in terms of manipulating a single network model of a developmental deficit.

Computational modelling

The central argument

Assume there is a single processing mechanism, where typical development corresponds to the mechanism acquiring a given set of input-output mappings. We refer to the input-output mappings as a training set, which captures the structured learning environment to which the system is exposed via the simulated child's experiences, either through exposure to natural experiences and/or the specific experiences of a given educational environment (such as learning to read).

In the case of a developmental disorder, the mechanism fails to acquire the expected input-output mappings through its history of experience-dependent learning, represented by a divergence of its developmental trajectory outside the range of variation found in typical development. Assume that the explanation for this deficit is that the processing mechanism has been created with atypical computational constraints. Constraints might include insufficient numbers of processing units, insufficient connectivity, sub-optimal activation dynamics, insufficient adaptivity, or poor input and output representations. Assume a behavioural intervention is simulated in terms of either the addition of some further information to the training set, or modulation of the frequency distribution of the training set to make some experiences more frequent / salient than others, or a combination of both of these methods. Lastly, assume that the evaluation of the intervention assesses the extent to which it improves performance on the training set.

With this set of assumptions in place, behavioural interventions *should not be successful in fully remediating developmental deficits*. If the existing

computational properties of the processing mechanism are insufficient to allow it to acquire the training set, why should the addition of further information help acquisition of the training set itself? The new information would just further burden a system that already has insufficient resources to learn its training set. If intervention changes the frequency distribution within the training set, it can only enhance performance on the now-more-frequent items at the expense of the now-less-frequent.

This conclusion paints a bleak picture of the prospects for behavioural intervention. However, there are at least six exceptions to this general rule; where one or more assumptions are violated and better outcomes would be predicted. The exceptions are: (1) if the computational problem is one of plasticity not capacity; (2) if the previous informational input to the mechanism has not been representative; (3) if when additionally stimulated, the mechanism changes its computational properties; (4) if there are compensatory pathways beyond the single mechanism (i.e., other mechanisms that can do the same/similar task); (5) if the problem that the learning system is trying to solve can be changed or simplified by altering its input/output representations; and (6) if the system is not trained on the target problem but a subset of the problem that should be sufficient for most behaviours. In the following sections, we address each of these, with illustrating connectionist simulations. We begin by considering what long-term compensatory outcomes might be available within a single mechanism without intervention.

Compensatory outcomes without intervention

An implemented model of a developmental deficit provides the foundation to investigate different possible interventions applied in childhood. But the modeller can also refrain from intervening, and use the model to predict the ultimate developmental outcome.

For some computational limitations, sufficient exposure to the training set eventually permits performance to reach the normal range on this set. However, close inspection of these networks indicates that the underlying processing itself has not normalised. This can be demonstrated by observing a persisting deficit on generalisation. Such an effect was observed in a connectionist model of English past tense formation simulating children with specific language impairment (SLI).

The model of Thomas (2005) explored the theoretical proposal of Ullman and Pierpont (2005) that children with SLI might have a specific deficit in morphosyntax because of a more general deficit in their procedural memory systems. The so-called 'procedural deficit hypothesis' addressed the observation that children with SLI often exhibit greater impairment in grammar development than vocabulary development. According to the hypothesis, the disparity stems from a differential reliance of the normal language system on two separate, more domain-general memory systems: grammar development on the procedural memory system, whose characteristics are implicit learning, slow acquisition, fast automatic execution and sequence processing; and vocabulary development on the declarative memory system, whose characteristics are parallel processing, slow recall, and explicit/conscious learning. Notably, the hypothesis proposed a central role for compensation in explaining observed behavioural impairments

in SLI: the profile of language skills is a consequence of the procedural system's sub-optimal attempts to acquire the structural aspects of language combined with the attempts of the declarative memory system to compensate for this shortcoming through lexical strategies.

Thomas (2005) explored this idea with a model of English past tense acquisition in which the production of phonologically encoded past tense forms at the output was driven by a combination of lexical-semantic and phonological information about the verb at the input (Joanisse & Seidenberg, 1999). SLI was simulated as a *monogenic* disorder, by decreasing the discriminability of the internal processing units prior to development. Unit discriminability was reduced such that units were less able to make large changes in their output for small changes in their input, achieved by reducing the 'temperature' parameter in the sigmoid activation function from 1 to 0.25. This impaired the network's ability to form sharp categorical boundaries in its internal representations. Figure 1 demonstrates the match of model data to empirical data in a past tense elicitation task for children around 10 years of age. As well as capturing the profile of reduced accuracy, the model captured a key 'compensatory' feature identified by Ullman and Pierpont in the inflection of regular verbs: increased frequency effects (2005; see van der Lely & Ullman, 2001). These were taken to be a hallmark of the operation of declarative memory and reflect its unusual involvement in morphosyntax in SLI. In the connectionist model, this compensatory effect was instantiated as a greater role for lexical information in driving past tense formation, rather than learning the phonological regularities relating base and inflected verb forms that capture the past tense rule in the emergentist account of acquisition.

Figure 1 now shows what happened when the atypical model was allowed to run to its 'adult' state. Performance on the training set, on both regular and irregular verbs, reached ceiling. Notably, however, there was a residual deficit on generalisation, the extension of the regular past tense rule to novel forms. The model, with its atypical processing properties, had not managed to extract the general function within the training set; but with enough exposure to the training set, had eventually managed to produce normal-looking behaviour on that set. Even in the adult state, the atypical network relied more on lexical information at input to drive its inflections.[1]

Reducing the discriminability of processing units particularly impacted on generalisation because it affected the formation of sharp category boundaries. Categorical functioning allows novel forms to be treated in the same way as existing category members. In unpublished work, the simulations reported in Thomas (2005) were run with other *monogenic* causes of the initial deficit. For two other deficits, excessive processing noise and a purely lexical strategy for producing inflections, a similar pattern of resolving delay on the training set and a residual generalisation deficit was found; for restricted numbers of internal processing units, there was a residual generalisation deficit but the early deficit on the training set did not resolve, either; for a very slow learning rate, there was no generalisation deficit but a residual deficit in irregular verb performance within the training set. It is evident, then, that long-term compensatory avenues

[1] See Thomas and Redington (2004) for similar developmental findings in a connectionist model of syntax processing. Simple recurrent networks with atypical sequence processing properties could eventually find compensatory solutions, but only for grammatical constructions that could be comprehended via locally available lexical cues, not those relying solely on sequencing information for decoding.

within this single mechanism model were sensitive to the type of initial processing deficit.

In sum, a system that exhibits early delays through atypical processing properties may be 'forced' through massive exposure to show normal-looking behaviour on the training set – the items that are intensely practised. However, this does not normalise processing properties. Residual deficits may remain, observable in generalisation. This pattern of eventual good accuracy on practised items along with subtle residual deficits is observed in some developmental disorders. For example, large dosages of reading experience can sometimes remediate reading accuracy deficits in dyslexia, but residual deficits can be found in reading speed and in spelling, both of which suggest the internal representations have not been normalised (Hulme & Snowling, 2009).

<Insert Figure 1 about here>

Six exceptions to the General Rule, where intervention can succeed

1. Behavioural intervention will not work except if the computational problem is a plasticity limitation rather than capacity limitation

Behavioural interventions will not be fully successful if the mechanism does not have the capacity to acquire the domain. However, capacity limitations are not the sole cause of delayed development. Thomas and Knowland (2014) used the same connectionist model of past tense acquisition as Thomas (2005) to investigate why early-identified delay sometimes resolves. The resolution of deficits in some individuals has been reported in several developmental disorders, including language (e.g., Dale, Price, Bishop, & Plomin, 2003), autism

(e.g., Charman, 2014a; Fein et al., 2013), and attention deficit hyperactivity disorder (ADHD; e.g., Biederman et al., 2010) and has generated theoretical debate in each case. What does resolution of delay imply about underlying cause?

Thomas and Knowland (2014) argued that limitations in the plasticity of developmental mechanisms can initially produce similar behavioural patterns as limitations in computational capacity. Systems with limited plasticity require more exposure to learning events to produce an equivalent improvement in performance. Mechanisms exhibiting early delays through limited plasticity should therefore respond to interventions that simply enrich the level of naturalistic experience. Such systems should remediate to the normal range just through greater 'practice', without requiring specially designed interventions.

Unlike the Thomas (2005) model of past tense formation, Thomas and Knowland (2014) took a *polygenic* approach to language delay. Variation in rates of development was modelled in a large population of simulated children (N=1000). Variation was caused by differences in 14 computational parameters, as well as in the richness of the language environment in which the child was raised. The computational parameters influenced properties of the learning mechanism such as network construction (e.g., number of internal units), network activation (e.g., unit discriminability, processing noise), network adaptation (e.g., the learning algorithm, the learning rate), and network maintenance (e.g., the level of pruning to eliminate unused connectivity, weight decay). Across the 14 parameters, Thomas and Knowland identified four broad types of *processing role* that parameters might serve. These roles were *capacity*, *plasticity*, *signal*, and *regressive events*. Some parameters contribute mainly to

one role, such as the number of processing units and denseness of connectivity contributing to capacity. Some parameters contribute to more than one role. For example, the nature of the learning algorithm determines both what can be learned and also how quickly; the unit discriminability influences the quality of the signal propagating through the network but also modulates the rate of connection changes and therefore plasticity.

Of the 1000 networks in the simulated population, 287 were diagnosed with language delay at an early point in development, based on falling 1 standard deviation below the population mean. The subsequent developmental trajectories of these delayed networks were followed, and 169 networks later resolved back into the normal range. Persisting deficits were observed in the remaining 118. Figure 2 shows the mean trajectories of the typically developing and delayed groups. The proportions are similar to those reported in the literature, where early-diagnosed delay (e.g., aged 3-4) resolves in more than half of cases (e.g., by age 6) (Bishop, 2005; Dale, Price, Bishop, & Plomin, 2003; see also Ukoumunne et al., 2012, for resolution at younger ages).

<Insert Figure 2 about here>

If the nature of intervention should be differentiated by whether delay resolves or persists, it is important to be able to predict outcomes for children with early-diagnosed delay as soon as possible. However, researchers have found this challenging. For example, in a large empirical study, Dale et al. (2003) explored whether it was possible to predict if children would fall in the persisting delay (n = 372) or resolving delay (n = 250) group on the basis of their

'time 1' profiles at 2 years of age, compared against 'time 2' outcome at 4 years. Children whose delays would persist scored reliably lower across a number of parental rating measures, including vocabulary, grammar, displaced reference (use of language to refer to past and future events), and nonverbal skills, as well as scoring reliably lower maternal education and showing a greater incidence of ear infection. Nevertheless, the effect sizes were small (.01–.06), and logistic regression analyses found that children's profiles at age 2 offered only modest classification of outcome at age 4. The statistical regression model including vocabulary, displaced reference, and nonverbal scores at time 1 correctly predicted only 45% of cases of persisting delay (chance = 50%), but 81% of cases of resolving delay. Addition of gender and maternal education level brought up the prediction of persisting delay to 52%.

A similar analysis was possible in the Thomas and Knowland (2014) model. Here, time 1 behavioural measures were broadly similar across persisting and resolving delay groups. There were subtle differences in past tense accuracy, with the persisting delay group performing reliably worse on regular verbs and generalisation of the past tense rule to novel verbs (that is, in extracting the underlying regularities of the domain) compared to the resolving group. But while these effects were highly reliable, as with the empirical data, they were of small effect size. A logistic regression model entering just time 1 behavioural profiles was 80% accurate in predicting persisting delay but only 54% accuracy in predicting resolving delay. Accuracy was not increased by adding in the richness of the language environment to which each network was exposed (an implementation of one proposed pathway by which differences in maternal education influence language development; see Thomas, Forrester & Ronald,

2013). As per Dale et al. (2003), measures of the environment didn't help to predict developmental outcome, even though in the model, experience of the language environment was the primary driver of development itself.

Computational implementations, however, provide the opportunity to investigate the mechanistic reasons why a model captures a given behavioural profile. In the current case, we can identify which of the computational parameters in fact predicted whether delay would resolve or not. Table 1 indicates which parameters had predictive power on developmental outcome. Limits on capacity tended to predict persisting delay, while limits in plasticity predicted resolving delay. When the full set of computational parameters was added into the logistic regression, a combination of time-1 behaviour and information about processing properties was able to predict persisting delay at 72% accuracy and resolving delay at 84%.[2] It is notable that these levels did not reach 100%. That not all the variance in outcome could be predicted in a relatively simple and well-controlled model points to the complex dynamics involved in development of non-linear learning systems.[3] More importantly, the model suggested that to predict behavioural outcomes in cases of atypicality, *measures of behaviour need to be complemented with measures of processing*, as argued by Fernald and colleagues (e.g., Fernald & Marchman, 2012).

[2] In clinical practice, 80% sensitivity and specificity is sometimes viewed as the requirement of a good screening test for developmental disabilities; it is less than 100% since clinical science is accepted as often imprecise (Charman et al., 2015; Glascoe, 1999).

[3] Predictive power is lost due to the interactions between the computational parameters in such mechanisms, where many of the effects are non-linear (Thomas, Forrester & Ronald, 2016).

In sum, the single-network perspective suggests that early developmental deficits may be fully remediated by interventions *if the cause is a limitation in plasticity*, rather than in capacity; and that these interventions need only increase the dosage of naturalistic experience rather than employ a specially designed intervention. However, identifying early on whether an emerging delay is due to a plasticity rather than a capacity limitation requires attention to processing properties rather than just behavioural profiles and environmental measures.

<Insert Table 1 about here>

2. Behavioural intervention will not work except if the behavioural experiences thus far haven't been representative

Behavioural intervention can fully remediate a developmental deficit if the cause of the deficit is not a limitation in the learning mechanism itself, but the information with which it has been provided. Although clinicians usually attempt to rule out environmental causes in diagnosing developmental disorders, language disorders are often observed with increased frequency in children from low SES backgrounds (All Party Parliamentary Group on Speech and Language Difficulties, 2013; Locke et al., 2002; Nelson et al., 2011). One factor associated with low SES that impacts language development is the richness of the language environment in which children are raised (Hart & Risely, 1995). A number of longitudinal studies have shown that differences in the richness of linguistic input result in an increasing gap in children's language development

(Huttenlocher et al., 2010; Reilly et al., 2010; Rowe, Raudenbush, & Goldin-Meadow, 2012; Hoff, 2013).

From the point of view of a single mechanism embedded within a wider cognitive system, the deficit in input need not be a property of the external environment, but could stem from deficits in other parts of the system. For instance, one theory of why components of the social cognitive system (such as those underlying face recognition) do not develop typically in autism is that the infant as a whole does not pay attention to the relevant social cues that are nevertheless present in his or her environment (e.g., Elsabbagh et al., 2011; though see Elsabbagh & Johnson, 2016). Thus a face recognition system might not develop because it is not exposed to sufficient information about faces.

Behavioural intervention should therefore involve enriching the learning environment from the perspective of the relevant mechanism, to ensure sufficient information is present to acquire the target ability. In the domain of language, there are initiatives to encourage parents from lower SES backgrounds to talk more to their children (e.g., Leffel & Suskind, 2013; Suskind & Suskind, 2015). Within autism, interventions are being developed that specifically train infants at familial risk of autism to pay attention to social cues (Wass & Porayska-Pomsta, 2014).

Restoration of an enriched input should bring atypically developing systems back towards the typical range of development. There is one caveat concerning timing. Certain domains, particularly those involving low-level perceptual skills, may exhibit sensitive periods in development, such that later acquisition does not reach the same ultimate levels of proficiency (Huttenlocher, 2001). Restoration of enriched input that occurs after the plasticity of the system

has begun to reduce may not be as successful; in effect, the early disadvantage will be imprinted on the structure of the system. One example of such an account is the proposal that SLI is caused by an early auditory deficit even though not all children with SLI show auditory deficits. The idea is that an early auditory deficit may resolve in some children, but due to sensitive periods in the development of the language system, the now-enriched auditory input cannot bring the development of the language system (and specifically, its phonology) back onto the typical trajectory (Bishop, 1997).

Figure 3 shows data from a polygenic model of individual differences (Thomas, 2016a), again employing the example domain of English past tense. Here, development is simulated in 1000 children. Individual variability is due to two sources, variation in multiple computational parameters and variation in the richness of the information present in the learning environment. The population depicted in Figure 3 experienced wide variation in the richness of individuals' learning environments and relatively less in their computational learning parameters (see Thomas, 2016a, for simulation details; GNEW population). Variation in the environment was implemented by a one-time filter on the training set applied to each family, analogous to the effects of SES on language input (Thomas, Forrester & Ronald, 2013). Solid lines show how the distribution of performance changes for this population across development (in this case, a lifespan of 1000 epochs of training, where 1 epoch was a single exposure to the individual's family training set). Data are shown separately for regular verbs (Figure 3a) and irregular verbs (3b).

At epoch 50, relatively early in development, every simulated child's environment was fully enriched to provide the maximum possible training set.

Dashed lines show the effect on the population distribution. Regular verbs immediately showed an acceleration in response to this whole-population intervention, with variation reducing and the lowest performers eventually performing above the 50th-centile of the original population. Irregular verbs took more time to exhibit the acceleration, indeed initially showing a decline, but eventually exhibited large gains. Acquisition of irregular verbs in these associative models tends to be more sensitive to the computational properties of the network. For irregular verbs, variation in computation properties continued to produce individual differences in performance despite the enriched environment. Thus Figure 3b shows that the population standard deviation did not change in the developmental phases following enrichment (i.e., distributions after 50 epochs). In other words, *the gap between simulated children did not close following enrichment*. Instead, the whole population increased its performance level. In contrast, gaps *did* close for the easier regular verbs, where computational properties did not constrain performance so strongly; poorer performing children caught up once the hindrance of a disadvantaged environment was lifted. In short, the effects of universal enrichment on narrowing gaps between children depended on the extent to which internal computational properties constrained development.

Functional plasticity can reduce in associative networks with 'age' via a number of mechanisms (Thomas & Johnson, 2006). In connectionist models, age may be indexed by the amount of training the system has experienced or a maturational schedule acting on computational properties. Among the mechanisms that can reduce plasticity are the loss of resources, reductions in the malleability of connections in response to training signals, entrenchment of

connectivity (i.e., established connections take longer to reset), and assimilation (where top down processes reduce the detection of differences in an altered learning environment, thereby mitigating drivers of change). The population under consideration here experienced aged-related reductions in plasticity through pruning of connectivity, which reduced available resources. Pruning had its onset at around 100 epochs. Figure 3(c) shows the effect of population-wide enrichment on irregular verb performance at 250 epochs compared to, respectively, normal (untreated) development and early intervention. Intervention had reduced effectiveness when it commenced after the onset of pruning. For regular verbs, by the end of training, the mean improvement in population accuracy following early enrichment was 22%, while that following later enrichment was 16%. For irregular verbs, the improvement following early enrichment was 31% and after later enrichment 13.5% (t-test, both $p<.001$).

If early impoverished environments cause deficits, the size of the treatment effect available through enrichment should be inversely proportional to the quality of that early environment. In other words, children who are held back more by an impoverished early environment should have greater scope for improvement following enrichment. In the simulation of early enrichment, this correlation was observed both for regular and irregular verbs, with correlations between environmental quality and treatment effect of -.86 and -.77, respectively (Figure 4a).

However, sensitive periods in development eventually translate the consequence of being raised in a poor environment into a deficit in the structure of the network, which later enrichment is less able to undo. In this scenario, the greater the early impoverishment, the greater the impact on the development of

processing structures, and the poorer the predicted treatment effect. One might thus expect the inverse correlation of early environmental quality and treatment effect to weaken or even reverse. In line with this expectation, the equivalent correlations following late enrichment were -.76 and -.25 for regular and irregular verbs, respectively (Figure 4b). The reduction in scope for treatment across development for networks raised in poorer environments was larger for irregular verbs than regular verbs, since they are more sensitive to the processing capacity of the network (in a fully factorial ANCOVA of treatment effects with factors of verb type and timing, and environmental quality as the covariate, all main effects and interactions were highly significant).

The pattern of more sustained early deprivation leading to less easily remediated deficits can be seen in data from a recent follow-up study of Romanian orphans exposed to severe early deprivation but then adopted into enriched environments. Sonuga-Barke et al. (2017) found that, when followed up into young adulthood, Romanian adoptees who experienced less than 6 months in an institution had similarly low levels of symptoms as typically developing controls. By contrast, compared to controls, Romanian adoptees exposed to more than 6 months in an institution had persistently higher rates of symptoms of autism spectrum disorder, disinhibited social engagement, and inattention and over-activity through to young adulthood.

<Insert Figure 3 about here>

Thus, enrichment interventions to alleviate deficits caused purely by a lack of appropriate experience need to pay attention to possible timing effects

impacting plasticity. If plasticity reduces, enrichment alone will be insufficient as an intervention. How should interventions alter if plasticity has reduced? The best behavioural intervention method in the case of late intervention will depend on the particular mechanism causing the plasticity loss for the domain and mechanism in question (see, e.g., McClelland et al., 1999; Thomas & Johnson, 2006). It may involve more intense practice, more feedback, or perceptually exaggerated stimuli.

<Insert Figure 4 about here>

Lastly, behavioural deficits produced by impoverished learning environments will not necessarily act independently of differences in intrinsic learning properties. Figure 5 shows the difference between impoverished and enriched learning environments for the simulated population, stratified by their unit discriminability. The effect of learning environment interacted with this internal computational constraint, such that the less optimal computational constraint tended to exaggerate the impact of the impoverished environment, albeit this was a marginal effect against the variation of other computational parameters in the population (main effect of environment: $F(1,996)=89.61$, $p<.001$, $\eta_p^2=.083$, main effect of temperature: $F(1,996)=10.73$, $p=.001$, $\eta_p^2=.011$, environment x temperature: $F(1,996)=3.51$, $p=.061$, $\eta_p^2=.004$). This interaction occurred because both influences act on the strengthening of network connections, which in turn drives behaviour. An increase in the incidence of developmental disorders in low SES families may, therefore, represent an

interaction between risk factors, rather than a misdiagnosis of pure environmental effects.

<Insert Figure 5 about here>

In sum, interventions to remediate deficits stemming from insufficient stimulation of a developing cognitive system may either target the external environment, or the internal environment of the system by seeking to alter those aspects of the external environment to which the child attends. Interventions involving enrichment to remediate environmentally caused deficits may be sensitive to timing, inasmuch as timing impacts plasticity. If environmental factors (such as SES) inversely predict response to treatment in younger but not older children, this is the hallmark of the operation of sensitive periods. Enrichment interventions will eliminate gaps between children unless the target behaviours are sensitive to other (independently occurring) individual differences in computational properties of learning mechanisms. In the latter case, enrichment can improve the whole population level of performance without narrowing gaps between children. Lastly, environmental effects may interact with and exacerbate underlying computational risk factors.

2a. Atypical computational properties may mean the system needs to see more of the problem to learn the general function

So far we have seen how continued exposure to the training set can eventually bring performance into the normal range for some past tense networks, and how enrichment of a previously impoverished training set can improve performance

across a whole population, so long as it is applied sufficiently early. In both cases, no specially designed intervention was added to the training set. It was just a case of 'more of the good stuff'. Previously, we introduced the important distinction between performance on the training set and generalisation of a function that is implicit in the training set. If the target is to improve generalisation performance rather than just performance on the training set, some atypical computational properties may mean that greater exposure to the training set will never be sufficient. In this case, additional information may need to be added to the training set to support generalisation.

Fedor et al. (2013) explored how the addition of specially designed input-output mappings could support generalisation in networks with atypical processing properties. These authors also employed a feedforward connectionist model drawn from the field of language development, in this case acquisition of the Arabic plural (Forrester & Plunkett, 1994). The aim was to visualise the formation and mediation of atypical representations of categories. The model was trained to learn categorisations defined over a 2-dimensional input space using high-dimensional internal representations. Fedor et al. considered different categorisation problems, in each case only giving the network a limited sample of the categorisation problem, and testing its ability to acquire (generalise to) the full function.

Developmental disorders were then simulated by start-state changes to parameters such as the denseness of connectivity, numbers of internal processing units, the learning rate, the unit discriminability, and processing noise. Next, cases of developmental deficits were re-run and interventions applied early in development. Interventions comprised additional input-output

mappings (no more than 10% of the size of the training set), which offered different information about the categories. For example, interventions might mark out prototypical members of categories, or demarcate the edges of category boundaries in the input space. The results of these exploratory simulations indicated that the best interventions either sampled the whole problem space or provided a representative 'slice' across all categories. There was some evidence, however, that interventions were differentially effective depending on the problem domain (mapping problem) and depending on the type of deficit.

Figure 6 illustrates one example of a training problem used by Fedor et al. (2013). It shows the architecture, the full categorisation problem, the training set (which represents a subset of the full problem), and then an example intervention set. Here, the network had to learn a category that spanned a zone around a diagonal of the two-dimensional input space, with different categories either side. The training set only provided examples at either end of the diagonal, and the network had to learn to *interpolate* the general function linking the two ends. Figure 7 demonstrates an example of a network learning this general function successfully. Although the internal representations of the network had high dimensionality, their structure could be visualised by determining the network's categorisation of all 10,000 possible locations in the input space. Figure 7 shows that in the typical case, there was quick formation of the diagonal category but with fuzzy boundaries, which were then progressively sharpened through further training. The figure also shows the formation of atypical representations in a case of a developmental deficit, in this case, a network with only 30% of the normal level of connectivity. Interpolation was unsuccessful, and

eventual performance retained accuracy only in the region of the training set. Finally, the figure demonstrates the consequence of adding an effective intervention (a slice across all categories) early in training. These additional input-output mappings improved performance on the training set, but crucially were also able to support acquisition of the general function despite the atypical processing properties.

In sum, as a corollary to eventual compensation in untreated atypical systems, atypical processing properties may require the design of special intervention sets to support generalisation, even in cases where high accuracy on the training set can eventually be reached through extended exposure.

<Insert Figures 6 & 7 about here>

3. Behavioural intervention will not work except if stimulation of the system changes its computational properties

We have suggested that if a disorder is the result of a restriction in the computational properties of a learning mechanism, behavioural intervention alone cannot succeed. However, not all theoretical approaches view the computational properties of learning mechanisms in the cognitive system as fixed throughout development. Should the behavioural intervention serve to alter the learning mechanism's computational properties, then fully successful remediation might be possible.

In its development, the brain undergoes a phase of elaboration of connectivity followed by regressive events that prune away connectivity; in addition, some existing connectivity is enhanced by myelination (Huttenlocher,

2001). It is as yet unclear what direct bearing such brain-level changes have on cognitive development. Researchers have sometimes included both increases in connectivity and decreases in connectivity in their developmental cognitive models. For example, constructivist approaches employ networks that can increase the number of processing units and connections in an experience-dependent manner (see, e.g., Quartz & Sejnowski, 1997; Mareschal & Shultz, 1999; Westermann & Ruh, 2012). Other models have included pruning of connectivity, where the connections removed are those that have not been strengthened by experience (e.g., Thomas, 2016a). Yet other models have included the assumption that some computational properties alter according to a maturational schedule. For example, Munkata (1999) captured age-related differences in a connectionist model of the infant A-not-B task partly through a maturational increase in the system's ability to maintain active representations, implemented by a gradual increase in the strength of recurrent connections.

In principle, then, one could conceive of a behavioural intervention modulating a mechanism's computational properties through altering the way certain parameters change across development. For example, this might equate to stimulation causing greater elaboration of connectivity in the target mechanism, or greater resistance to loss of connectivity during pruning of connectivity.

To illustrate how this might work, consider a model of autism proposed by Thomas, Knowland and Karmiloff-Smith (2011). This account initially focused on the regressive sub-type. It proposed that autism is caused by an exaggeration of the normal phase of pruning of connectivity occurring from infancy onwards; over-pruning occurs and particularly impacts long-range connectivity. Thomas,

Davis et al. (2015) later showed how differences in the timing of onset of over-pruning could link early onset, late onset, and regressive sub-types of autism (Landa et al., 2013). Davis (2017) then considered whether the behavioural deficits shown by the atypical connectionist models could be remediated by interventions of different types and applied at different times. As we shall later see, behavioural improvements were on the whole relatively small, and individual networks show variation in their response to intervention. However, some networks did show a marked behavioural benefit from a short, discrete intervention applied early in development.

Figure 8 shows the mean performance of a group of such networks that exhibited a strong response to early intervention. Networks were trained for 1000 epochs, with the onset of pruning between 25 and 50 epochs; atypical networks were exposed to an intervention at epoch 30, lasting 40 epochs; the intervention was designed to enhance generalisation by including novel examples of items following the implicit regularity present in the training set, with the intervention set approximately 10% the size of the training set. Figure 8(a) shows the behavioural deficit of the impaired networks, compared to a control condition of the same networks trained without the atypical setting of the pruning parameter. The short intervention showed a marked benefit on accuracy, which sustained until the end of training. The size of the intervention effect was highest in mid-training, and did not increase at the later measurement point. A similar patterns has been reported for interventions with autism: in a systematic review of the effects of early intense behavioural interventions, Howlin, Magiati and Charman (2009) found that IQ changes were smaller between first and second follow-up points than the changes produced by the

intervention from baseline to first follow-up point. Figure 8(b) shows the total number of connections in the atypical networks in the untreated and treated conditions. Notably, during the intervention, connection loss accelerated as the internal representations underwent reorganisation. Thereafter, the treated condition retained a greater proportion of connections (t-test: 250 epochs $t(8)=3.91$, $p=.004$, Cohen's $d=.43$; 1000 epochs, $t(8)=3.85$, $p=.005$, $d=.37$). Connection number is associated with improved computational power.[4] The behavioural intervention for these atypical networks, then, served to improve their computational properties during subsequent development compared to the untreated condition.

<Insert Figure 8 about here>

Under a strictly maturational view, computational properties may alter with development, but the schedule is not influenced by behavioural interventions, or more broadly, by experience. In such a scenario, behavioural interventions could still be rendered successful simply by waiting until the computational properties have improved. Maturational accounts have been proposed in disorders such as SLI (Bishop & McArthur, 2004) and ADHD (Batty et al., 2010; Shaw et al., 2007). Evidence from neuroscience has been used to argue that interventions for anxiety disorders may be more effective after adolescence due to the developmental state of the underlying mechanisms

[4] In an equivalent population of 1000 networks without atypical pruning, the number of connections in a network correlated .134 with behaviour at epoch 250 and .163 with behaviour at epoch 1000 (both $p<.001$).

(Hartley & Casey, 2013). Within the field of education, the broader notion of 'school readiness' is predicated on the assumption that development of skills such as executive function needs to have reached a certain level before the classroom-based behavioural methods can be properly effective (Noble, Tottenham & Casey, 2005).

In sum, behavioural interventions may be successful if they trigger experience-dependent changes in the computational properties of a processing mechanism, or if an intervention is delayed until the computational properties exhibit maturational changes that permit behavioural interventions to be effective.

4. Behavioural intervention will not work except if there are compensatory pathways (other mechanisms that can do the same/similar task)

From within our single-mechanism perspective, behavioural intervention could be successful if it caused the recruitment of other mechanisms or pathways able to deliver or support the target behaviour. Of the models we considered in the Introduction, several make reference to pathways outside of the single implemented mechanism to explain behavioural patterns. In the case of Abel, Huber and Dell's (2009) naming model, the authors referred to respectively, visual input, the conceptual-semantic system, an editor component, and a phonetic component as possible sources of naming errors that their implemented system could not accommodate. Plaut (1996)'s model could not accommodate a certain pattern of reading errors during relearning after damage, which led Plaut to argue that the pattern originates from the operation of an unimplemented phonological route. In their reading model, Harm, McCandliss

and Seidenberg (2003) speculated that interventions acting on an unimplemented semantic route could improve word reading rather than just the nonword reading improvements shown by the implemented architecture.

Evidence from functional brain imaging of developmental disorders has encouraged the view that in some cases of good developmental outcomes, usually following intensive interventions, compensatory mechanisms are engaged beyond normal circuitry. For example, arguments have been made in the case of dyslexia (compensatory activation in right inferior frontal gyrus; Hoeft et al., 2011) and autism (compensatory activations in several left- and right-lateralised regions identified in a language comprehension task; Eigsti et al., 2015). Researchers then hope that identification of alternative brain pathways will lead to new interventions that will encourage adoption of compensatory strategies.[5] Similar claims for compensatory outcomes have been made on behavioural evidence alone. For example, De Haan (2001) pointed out that in children with autism, despite evidence that individuals processed faces atypically (such as the unusual absence of categorical perception of facial expressions), some nevertheless performed in the normal range on expression-recognition tasks. These individuals tended to have higher IQs. De Haan argued that there must be "a degree of plasticity in the developing system that allows for

[5] For example: https://www.nih.gov/news-events/news-releases/brain-activity-pattern-signals-ability-compensate-dyslexia, retrieved 17 August 2016: "Understanding the brain activity associated with compensation may lead to ways to help individuals with this capacity draw upon their strengths. Similarly, learning why other individuals have difficulty compensating may lead to new treatments to help them overcome reading disability" (Alan E. Guttmacher, M.D., director of the NIH's Eunice Kennedy Shriver National Institute of Child Health and Human Development, commenting on Hoeft et al., 2011)

development of alternative strategies/mechanisms in face processing" (2001, p. 393).

The proposal that alternative combinations of mechanisms can deliver similar behaviours requires a certain kind of developmental theory to be true. Price and Friston (2002) have argued for *degeneracy* in the brain's realisation of cognition. This is a biological concept, whereby elements that are structurally different can perform the same function or yield the same output. For example, objects can be recognised either on the basis of their global shape or by the presence of distinguishing features. The different cognitive functions of either global form or local feature processing can therefore deliver the same output: accurate object recognition. How well a processing component performs a task then depends on the fit of its structure (i.e., its neurocomputational properties) to the intended function; and how much training the component has had in performing the task. Even within the normal range, individuals may follow developmental trajectories that harness different combinations of components to perform the same task. Degeneracy may therefore explain both individual variation in functional brain activations, and variation in impairments following the same localised brain damage (Price & Friston, 2002).

Relatively few computational accounts have explicitly considered how development integrates multiple mechanisms to perform complex tasks, let alone variation between individuals. In the mixture-of-experts approach (Jacobs, 1997, 1999; Jacobs et al., 1991), the initial architecture is comprised of components that have different computational properties. A specific mechanism gates the contribution of these components to the output. When the overall architecture is presented with a task, the gating mechanism mediates a

competition between the set of components, allowing the most successful component for each training pattern both to drive output performance and to update its weights to become better at that pattern. Across training, certain mechanisms come to specialise on sets of patterns, by virtue of having an initial (perhaps small) advantage in processing those patterns. Why might such a process of emergent specialisation differ between individuals? Presumably, variation in outcomes could arise from differences in the set of 'experts', differences in the experts' respective computational properties, the operation of the gating mechanism, and the composition of the training set (see Thomas & Richardson, 2005).

As yet, no computational accounts have considered how an intervention might alter the organisation of a set of mechanisms to improve accuracy on a given behaviour. We do know that in practice, clinicians tend to shift from implicit to explicit methods with older children, in order to encourage compensatory strategies. However, there is a missing link in the argument. While there is evidence of individual variability in the use of mechanisms, and evidence of compensatory engagement of new mechanisms in some disorders where individuals show good outcomes, this does not guarantee that we can generate interventions to encourage the use of alternative sets of mechanisms. Evidence of different outcomes across individuals is not the same as evidence that all outcomes are equally accessible to a single individual. One view is that individual variability in the use of different mechanisms for a task indexes the scope for compensatory reorganisation (e.g., in the domain of reading: Kherif, Josse, Seghier & Price, 2009; Richardson et al., 2011; Seghier et al., 2008). But evidence from the functional imaging of compensated brains minimally requires

translation to the cognitive level to understand what the compensations represent, before a facilitatory intervention can be developed.

How might an intervention prompt use of compensatory mechanisms? Perhaps a behavioural method could emphasise different task-relevant information, or different modalities; or encourage differential reliance on motor versus sensory demands of the task; or engagement of different representational formats, such as gesture to support language, or language to support spatial cognition. This remains to be clarified.

In sum, intrinsic computational limitations in a target mechanism might be overcome by recruiting other mechanisms able to support task performance, but a computational analysis of this strategy is not far advanced, nor an understanding of how to encourage such recruitment via a specific behavioural intervention.

5. Behavioural intervention will not work except if the problem that the learning mechanism is trying to solve can be changed or simplified

The structured environment presents a problem domain to the learning mechanism, and in typical development, this problem can be solved. The relevant behavioural repertoire is acquired. From a computational perspective, the problem is specified by the way the domain is encoded, with respect to the input representations and the output representations. Where a learning mechanism has insufficient computational resources to solve the problem, development occurs more slowly, may asymptote at a lower level, show acquisition of some parts of the domain but not others, or show generalisation deficits. We have so far considered behavioural intervention as adding some further information to

the structured environment or altering its frequency distribution. However, a behavioural intervention could serve to alter the nature of the input or output representations. Changing the representations might simplify the problem that the learning mechanism has to solve, and bring it within what can be achieved with the existing computational constraints. That is, a less powerful mechanism may be able to learn a simpler problem.

Behavioural interventions for dyslexia and WFD both appeal to this idea. For reading, interventions target the structure of the phonological representations, the output of the decoding system. For WFD, interventions additionally target improvements in semantic representations, the drivers of naming. Computational models of intervention have also appealed to this method. Seidenberg and McClelland's original connectionist model of reading (1989) was later deemed to be closer to the performance of a dyslexic, because it had representations that didn't show sufficient similarity between written letters or between speech sounds to allow the learning mechanism to generalise the reading problem to novel words. The presentation of the problem domain made it too hard for the learning mechanism to solve. A later implementation utilised more componential input and output representations and was taken to be a better model of typical development (Plaut et al., 1996). One of the interventions considered to alleviate dyslexia in the Harm, McCandliss and Seidenberg (2003) model was to improve the output representations developed by the phonological component. Best et al.'s (2015) model considered interventions to improve naming – captured as the mapping between semantic and phonological representations – by treatments that improved the representations of semantics or phonology in isolation, rather than simply more

practice in using the compromised pathway linking these representations. Lastly, Harm et al. demonstrated that improvements stemming from changes in input or output representations may be subject to timing effects; previous learning may cause entrenched connections that mean the mechanism responds less readily when representations are changed later in development.

The Best et al. (2015) model used fairly idealised depictions of semantics and phonology. Figure 9 shows results from a model with more realistic representations (Alireza, Fedor & Thomas, 2017). Using the same architecture as the Best et al. model, this implementation employed a training set of 400 English words taken from the Masterson, Stuart, Dixon and Lovejoy (2010) corpus of words found in children's books. Phonology was encoded in a slot-based scheme using articulatory features, while semantics used a feature-based scheme of over 1000 features drawn from Vinson and Vigliocco's (2008) adult ratings of word meanings. Figure 9a depicts the typical model in its development of semantic knowledge, phonological knowledge, single word comprehension, and single word naming; and an atypical network, which had a computational restriction to the naming pathway that linked emerging semantic and phonological representations. For the atypical network, Figures 9b-c depict the effect on naming of a relatively short intervention early in training (between 100 and 200 epochs, in a lifespan of 1000 epochs, depicted by the shaded area). Intervention was triggered at a point when the typical model had acquired a productive vocabulary size of 67 words, while the atypical models had a vocabulary size of 36 words. Five different interventions were contrasted, of three types: (1) remediating the weakness – the model was provided with additional training on the naming pathway; (2) improve the strength – the model was provided with

additional training to improve the (otherwise typically developing) semantic representations, the phonological representations, or both at once; (3) both types 1 and 2 were combined into an intervention that sought to simultaneously improve strength and remediate weakness.

<Insert Figure 9 about here>

The intervention to target the naming weakness, extra practice for the semantics-to-phonology pathway, improved performance initially, but served only to propel the system further along its atypical trajectory. The final level of performance was no higher; eventually, the untreated condition caught up with the treated condition. Interventions to target strengths, the semantic and phonological representations, produced more gradual improvements, but these were long-term and raised the final level of performance. Extra training on the input and output representations for naming served to make them more distinguishable, and therefore make the task of learning the arbitrary mappings between meaning and sound easier for the restricted pathway. The largest benefit occurred when both semantic input and phonological output representations were improved (Fig. 9c). When the input/output intervention was combined with extra training on the semantics-to-phonology pathway, both short-term and long-term benefits were observed (Fig. 9b).

Alireza et al. (2017) also considered the effects of timing, contrasting interventions at 100, 250, and 750 epochs. In all models, unused network connections were pruned away with a small probability from 100 epochs onwards, reducing the plasticity of older networks. Later in training, improving

strengths became less effective and remediating weaknesses became more effective. Echoing the findings of Harm et al. (2003), the benefit of improving input and output representations was more marked early in development, and reduced once pathways had committed to utilising the (potentially poor) initial representations. At that point, maximising the performance of the pathway through intense practice became the best recourse.

In sum, behavioural interventions that improve either the input or output representations involved in acquiring a cognitive domain may improve the ultimate level of performance that is attainable by the system with atypical computational constraints, but such improvements may be subject to timing effects. Remediating weakness did produce improvements, but these only propelled the system more quickly along the same atypical trajectory. In this model, long-term benefits of an early intervention arose from improving strengths, not from focusing on weaknesses. However, the opposite was true of a late intervention.

6. Behavioural intervention will not work except if the system is trained not on the target problem but a subset of the problem that should be sufficient for most behaviours in the domain

If the model is unable to learn the training set to a given performance level through limitations in processing capacity, adding further input-output mappings to the training set is unlikely to enhance accuracy on the patterns in the original training set. What one might call normalisation through behavioural intervention is therefore difficult if one conceives of developmental deficits as arising from limitations in individual systems. We define *normalisation* here as

the acquisition of the abilities and knowledge that any typically developing system acquires through exposure to the normal training set.

However, one might take the view that, for adequate functioning of a child in his or her day-to-day environment, learning the full repertoire of behaviours in the target domain is not necessary. Perhaps it is sufficient to learn *just some items* in the training set, the most frequently required, the most prototypical? This more modest objective might suggest interventions that focus only on a subset of the training set. For example, in the past tense domain, one might select the most frequently used verbs, be they regular or irregular. Alternatively, one might take the view that what the atypical system needs to learn is not the training set per se (even though this is what typical systems acquire), but *a general function* implicit in the items in the training set. Acquisition of this general function can be assessed by performance on generalisation sets rather than the training set. There may then be input-output mappings that can be added to the training set which could improve the network's ability to extract the general function, even if performance on the original training set did not improve (or even worsened). In contrast to normalisation, we could term this approach *compensation*, since the aim is to optimise a subset of behaviours present in the original training set. In the past tense domain, such an approach might seek to improve acquisition of the regular past tense rule by showing its use across a variety of verb forms.

The distinction between these two intervention aims – improving performance on the full training set versus on a sub-set or a function implicit in the training set – allows us to draw a formal distinction between normalisation and compensation, with respect to our single-mechanism perspective. It poses

the challenge of how one might derive interventions that achieve these goals. So far, we have conceived of a behavioural intervention as the addition of training patterns to the network's training set for some duration. Which additional patterns would support normalisation, under our definition? Which additional patterns would support compensation?

Yang and Thomas (2015) explored one method to derive intervention sets within a machine-learning framework. The method assumes the availability of an artificial neural network that is able to successfully acquire the target domain through exposure to the training set. A genetic algorithm technique is then used to identify which input units were most important for generating good learning on, respectively, the training set or the generalisation set. Intervention items can be produced which embody the features that support either training set acquisition or generalisation. An intervention set then comprises a selection of these items, for example which span the internal representational space of typically developing models. The internal representational space can be characterised by principal component analyses of hidden unit activations produced by the training set. Davis (2017) used this method to derive intervention sets to encourage either normalisation or compensation, and applied them to a model of autism. Intervention sets contained around 10% the number of patterns as the training set. The results in that case indicated that compensation was more effective than normalisation for networks with compromised connectivity, since in artificial neural networks, regularity is less demanding on representational resources.

The Yang and Thomas method for deriving intervention sets is model dependent. It requires the availability of a fully specified training set, and

commitment to the representational format in which the problem is specified. Moreover, compensation requires specification of the implicit function in order to identify the key input dimensions that embody the function – in other words, a theory of the information that is most important in a domain.

In sum, behavioural interventions may be successful in mechanisms with atypical computational constraints if the goal of intervention is revised from normalisation (fully behavioural competency) to a subset of skills, which we termed compensation. Machine-learning methods suggest possible ways of identifying items that will support normalisation and compensation.

Biological interventions

A connectionist perspective on intervention lends itself to the consideration of experience-dependent factors to change the developmental trajectory of cognitive mechanisms. Thus far, we have focused on how experience-based interventions may yield limited returns where the cause of a developmental disorder is an atypical computational constraint within a processing mechanism. An alternative form of intervention would be to directly manipulate the computational properties of the processing mechanism. We refer to these as *biological* interventions, since they need not involve behavioural methods directly relevant to the target skill.

Biological interventions most obviously would include pharmacological treatments that alter the levels of neurotransmitters (e.g., dopamine for ADHD, Volkow et al., 2002; serotonin for repetitive behaviours in pervasive developmental disorders, McDougle, Kresch & Posey, 2000; oxytocin in autism, Preckel et al., 2016). More speculatively, biological methods might target neural

activity via electrical methods (e.g., direct cortical stimulation for dyslcalculia; Iuculano & Cohen Kadosh, 2014) or brain plasticity via drug treatments (e.g., valproate acid for auditory learning; Gervain et al., 2013). Biological methods might also employ behavioural practices that do not directly target cognition but influence brain function, such as exercise and diet (e.g., for treating ADHD: alterations of diet, Konikowska, Regulska-Ilow & Rózańska, 2012; use of exercise, Silva et al., 2015). Or they might employ methods that indirectly target cognition, for example through the effect of sleep on memory consolidation, or mindfulness training on attention, or action video game playing on visual attention (e.g., role of sleep in developmental disabilities: Ashworth, Hill, Karmiloff-Smith & Dimitriou, 2015; Dodge & Wilson, 2001; mindfulness treatments for autism, dyslexia, ADHD: Sequeira & Ahmed, 2012; Tarrasch, Berman & Friedmann, 2016; video game playing for dyslexia: Franceschini et al., 2013).

It should be possible to construe all such biological effects in terms of manipulations to parameters within computational models of development. For example, impulsivity in ADHD has been modelled in terms of a computational constraint on reward-based or reinforcement learning. Williams and Dayan (2004, 2005; Richardson & Thomas, 2006) used one form of reinforcement learning, Temporal Difference learning, to simulate a developmental profile of impulsivity in ADHD, based on a model of the role of dopamine in operant conditioning. In this model, the agent (child) had to learn to delay an immediate action that gained a small reward in favour of a later action that gained a larger reward. Williams and Dayan simulated ADHD by altering the 'discounting rate' parameter, which determined the relative weighting of immediate versus long-term rewards in guiding action. The atypical setting of the parameter

corresponded to the lower levels of dopamine found in the brains of children with ADHD. A system that discounted long-term rewards developed impulsive behavioural patterns, by allowing small immediate rewards to guide action. Although this model was not extended to consider intervention, the common pharmacological treatment for ADHD, methylphenidate hydrochloride, is a stimulant that operates by increasing levels of dopamine in children's brains (Gottlieb, 2001). In the model, the effects of the biological intervention could be simulated by altering the discounting rate parameter, thereby removing the atypical constraint on subsequent development of impulse control in reward-based action decision-making.

Harm, McCandliss and Seidenberg's (2003) reading model in effect included a biological intervention. In one of its conditions, an initial computational limitation in the phonological component (lower connectivity and restrictions on weight size) was simply eliminated by an intervention. Lost connections were restored and weights were allowed to take on larger sizes. It is worth noting that in this model, this biological intervention was subject to timing effects. Later interventions were less effective because they could not reverse entrenched weight values produced by earlier learning in the network connecting orthographic inputs to atypical phonological outputs. On the face of it, biological interventions might seem more powerful, but they too may be subject to limitations.

One question that arises for biological interventions is how long they should continue. Must they continue into adulthood, to maintain the remediating effects on the targeted computational properties? Sustained biological intervention both incurs practical costs and risks potential side effects. The non-

linearity of neurocomputational systems suggests that in some cases, the answer may be no: a biological intervention may only need to continue until robust internal representations have been acquired. For example, Figure 10 shows the effects of different levels of neural processing noise applied to a past tense network, with the noise either present throughout development or applied only at the end of development (Thomas & Karmiloff-Smith, 2002). Small amounts of noise, less than 10% of the dynamic range of the artificial neurons, was enough to entirely disrupt development. However, if development proceeded without noise, once robust internal representations were established, the system's performance was unaffected by the same level of noise applied at the end of training: the system only began to show small decrements when the noise level reached 20% of dynamic range. Therefore, if biological treatments ameliorate atypical computational constraints long enough for robust representations to be acquired, cessation of treatment and re-instantiation of the identical atypical constraints may not then impact behaviour.

In sum, biological interventions that directly target computational properties of cognitive mechanisms are an alternative to experienced-based, behavioural methods. Behavioural methods may also target brain function or indirectly target cognition. These interventions can also be explored through computational modelling, which may provide insights into, for example, how long the intervention needs to continue for a robust, long-lasting effect.

<Insert Figure 10 about here>

Principles and dimensions for modelling intervention

When implementing a computational model of intervention, the researcher has to make several design decisions. Recall, we have construed an intervention as the provision of additional training patterns to an atypical network beyond what might be called its 'natural' experience or training set.

The first decision is the composition of the intervention set, with respect to its aim (to improve performance on the natural training set; to encourage compensation to a subset or a general function). The second is the number of items in the intervention set, compared to the size of the natural training set. In our simulations, we have restricted the intervention set to 10% of the size of the natural training set, under the assumption that intervention experiences are dwarfed by contemporaneous natural experience. However, as we saw in the Introduction, in reality interventions may vary from 1 hour per week for language disorders (perhaps 1% of waking experience, assuming 14 hour waking days) to 40 hours per week for pervasive disorders like autism (perhaps 40% of waking experience).

The third decision is whether in the simulation the intervention set replaces the natural training set for the period of the intervention, or whether it is added to and interleaved with natural experience. Given that natural experience resumes once the child leaves the intervention environment, interleaving might seem more appropriate. In artificial neural networks using distributed representations, temporary replacement of the natural training set by the intervention set risks disruption of established learning in the network, particularly where the new knowledge is not consistent with established knowledge, an effect called catastrophic interference (McCloskey & Cohen, 1989;

Ratcliff, 1990). When Davis (2017) simulated interventions for a population of 1000 past tense networks at risk of an over-pruning disorder, the response of networks even with interleaving was often an initial worsening of performance (as representations were reorganised), with improvements often emerging after intervention had ceased (as reorganised representations were integrated with natural experience). Figure 11(a) shows the mean treatment effect for the whole population, either on regular verbs in the training set or application of the past tense rule to novel verbs in a generalisation set. Davis (2017) used either a normalisation set or a compensation set, following Yang and Thomas (2015). The intervention period is shown in shaded green. Both intervention sets produced initial worsening of performance, followed by small but reliable long-term gains. The compensation set, designed to support the regular past tense rule, was more consistent with the training set, and so produced less interference. Figure 11b shows two example cases of atypical networks, allowed to develop either under treated or untreated conditions, where the benefit of the intervention in the treated condition was not observed until after the intervention had ceased.

<Insert Figure 11 about here>

The fourth implementation decision involves the dosage of the intervention. This may include the duration – how long the intervention set is added to the natural training set during intervention – and the intensity, determined by the number of intervention items and their token frequency with respect to items in the natural training set. For example, Davis (2017) used an intervention set of 50 items against 500 in the natural training set, with a token

frequency (number of repetitions of each item) four times higher than the natural training set, to mark the intensity of the intervention situation. The greater the intensity of the simulated intervention, the greater the risk of disruption of previously established knowledge. Real-life interventions are usually limited in duration, leading to considerations of whether any benefits sustain after the intervention ends (Bailey et al., 2017).

In a simulation, a straightforward implementation is addition of the intervention set for a limited phase of the network's training. However, a more nuanced view of the operation of the cognitive system is that intervention experiences are also stored in the child's episodic memory; under some views, such as the complementary learning systems theory (McClelland, McNaughton & O'Reilly, 1995; O'Reilly et al., 2014), the episodic memory system continues to transfer its knowledge to conceptual systems over an extended period of time. Moreover, one approach argued to support persistence of intervention effects beyond cessation of the treatment is the creation of a sustaining environment, perhaps through interventions that partly target the parent-child interaction, home or school environment (Bailey et al., 2017). Either way, one could make a case for an implementation that continued to expose the system to the intervention set for a more extended time during training.

Davis (2017) contrasted the effect of a short-lived or discrete intervention versus the permanent addition of intervention items to the training set. Figure 12 shows the results for the at-risk population, for the compensatory intervention applied early in training (at 30 epochs of a lifespan of 1000), either for a discrete duration of 40 epochs or as a permanent addition. Both interventions produced small benefits to generalisation observable at the end of

training, with the effect of permanent addition only marginally larger. While in this case the discrete intervention did produce long-term benefits, this may depend on an implementation decision about changes in developmental plasticity in the base architecture. Davis (2017) used a model that had reducing plasticity across development (through pruning; see Thomas & Johnson, 2006), tending to give early experiences long-term impact. When Yang and Thomas (2015) considered interventions to past tense networks with consistent lifespan plasticity, the effects of early discrete interventions tended to dissipate over subsequent training.

<Insert Figure 12 about here>

This leads onto the fifth implementation decision, the age at which interventions are applied. This can be made with reference to the typical developmental trajectory and the point at which the atypical trajectory of a network diverges from that trajectory. Several simulations we have considered – universal enrichment, the treatment of input and output representations in the Best et al. (2015) model, the reading interventions carried out by Harm, McCandliss and Seidenberg (2003) – demonstrated that the point in training at which the intervention is applied can have a material effect on its success, depending on the level of functional plasticity in the network.

None of the simulations so far have considered the potential role of factors such as the child's attention, motivation, and engagement. This is despite these factors being important dimensions of the child's interaction with the therapist. In principle, such factors might alter the computational processing

within the target mechanism (e.g., in the way processes of selective attention have been implemented as serving to boost processing pathways, such as in a connectionist model of the Stroop task; Cohen, Dunbar & McClelland, 1990; see Filippi, Karaminis & Thomas, 2014, for a similar approach in a developmental model of bilingual reading).

We presume, however, that it is not a lack of attention / motivation / engagement that is the primary cause of atypical development, rather some computational constraint more intrinsic to the target mechanism (though any such lack in the child may of course have secondary cascading consequences on other systems and on behaviour). We assume instead that the child's attention / motivation / engagement in the therapeutic situation is necessary for the intervention to gain access to and alter the functioning of the target mechanism. The simulation of intervention therefore already accepts and is predicated upon the engagement of the child in the intervention situation.

A final important decision is the way in which individual differences will be implemented. A key dimension of intervention is the source of differential response across children observed in many therapeutic situations. Monogenic models of disorders give some basis to consider differential responses to intervention. For example, in their model of word finding difficulties, Best et al. (2015) were able to use three different atypical constraints (operating on hidden units, connectivity, and unit activation function) to simulate the language profiles of individual children. Figure 13 shows the response to two different interventions (semantic therapy, phonological therapy) for the three different 'versions' of each child with WFD. Notably, the different ways of producing the

same atypical behavioural profile were associated with different responses to intervention.

<Insert Figure 13 about here>

Polygenic models of disorders offer a more ready framework to capture differential response. Using population-level models, atypical computational constraints can be simulated against a background of small population-wide variations in many computational constraints, such as those involved in specifying the network architecture, processing dynamics, and plasticity, as well as differences in environmental stimulation. One might think of this as the general intelligence of a network. Figure 14 shows distributions of treatment effects from the simulations of Davis (2017) discussed above, considered separately for training set performance or generalisation performance, and in response to normalisation or compensation interventions. The treatment effects were generally small, of the order of a few percentage points of accuracy against deficits of 20-40%; however, they varied widely across individual networks, including cases of large gains and large losses in response to intervention. The parameter sets of individual networks can then be used to predict the size of the treatment effect, to construct a mechanistic account of the origin of variable response to intervention.

Table 2 shows a set of standardised coefficients from linear regressions for each intervention type, assessed on training set and generalisation. The shaded rows represent parameters related to the pathological process (over-pruning), the rest to general intelligence. Several points are notable. First, the

main effects of these parameters explained the minority of the variance in response to intervention. While there was a stochastic element to the response, replication indicated that the test-retest correlation was around 0.5, indicating that a fair proportion of the response to intervention depended on the network's developmental conditions (its parameters and its environment). Mostly likely those development conditions arose from higher order interactions between computational parameters, enabling some networks to gain from intervention, others not to gain, and some to lose. Second, some predictors of individual response depended on intervention type (normalisation versus compensation). Third, predictors could be differentially important for intervention responses on the training set versus generalisation, that is, dependent on the target behaviour. And last, while some predictors were involved in modulating the impact of the atypical connectivity pruning process, others represented parameters *unrelated to the pathology*, consistent with the idea that general individual differences factors influence the effectiveness of behavioural therapy.

In sum, the use of computational models to simulate interventions for developmental disorders necessitates explicit design decisions around several dimensions, including the composition and size of the intervention set, the dosage (intensity, duration) of the intervention, how interleaved it is with natural experience, the age at which it is applied, plasticity conditions across development, and specification of possible sources of individual differences in response to intervention.

<Insert Figure 14 about here>

<Insert Table 2 about here>

Discussion

We have highlighted the gap between theories of deficit and theories of treatment in developmental disorders, and considered the potential contribution of connectionist modelling in closing the gap. Although connectionist models of developmental deficits are many, extensions to simulating interventions are few.

What have we learned?

We adopted the perspective of the processing abilities of a single cognitive mechanism, and the assumption that atypical development usually arises from computational limitations that constrain its learning properties. From that perspective, the outlook was pessimistic. Behavioural interventions are unlikely to be successful in fully remediating developmental deficits, because they will not remove the computational limitations. Perhaps this chimes with the difficulty of treating developmental disorders, particularly those with pervasive effects such as autism (Charman, 2014b). But there was also a more optimistic note. A number of situations provided exceptions to the general conclusion. First, if the deficit is a case of delay through limits in the plasticity of the mechanism rather than its capacity, the deficit should eventually resolve (Thomas & Knowland, 2014); and its resolution might be accelerated by a greater dosage of otherwise naturalistic experience (i.e., practice). Second, if the deficit in fact arises through insufficient stimulation of the mechanism, whether externally in richness of the environment to which the child is exposed or internally in the information provided to the single mechanism (for instance, by attentional orienting systems), then the deficit can be treated by alleviating this shortfall. This might amount to enriching the environment (for example, in the domain of language,

with more child-directed speech; e.g., Suskind & Suskind, 2015); or to training attentional mechanisms (for example, in the case of young children with autism, training attention to social cues, e.g., Powell, Wass, Erichsen & Leekam, 2016; Wass & Porayska-Pomsta, 2014).

If neither of these exceptions holds, then several alternative cases were identified where greater success might be achieved through intervention. A more positive outcome might be expected if stimulation of the target mechanism causes a change in its computational properties. In addition, should the target mechanism experience changes in its computational properties through maturation, waiting to apply the intervention may produce a better outcome. If there are compensatory pathways beyond the target mechanism that can produce the same or similar behaviour, and an intervention can serve to encourage the engagement of these alternative mechanisms, a better outcome is also possible. If the domain that the target mechanism is struggling to acquire can be simplified by altering the quality of the mechanism's input and/or output representations, a better outcome is possible. If the intervention chooses to train the target mechanism not on the full cognitive domain but a subset of the problem adequate for everyday functioning, a better outcome is possible. Finally, if the computational properties of the target mechanism can be altered more directly by biological means (e.g., pharmacological treatments, diet), or indirectly through other behavioural techniques (e.g., exercise, sleep, mindfulness training, action video game playing), more positive outcomes can be achieved. Simulations demonstrated that such treatments may not need to be permanent, just sufficient to allow skills to be established, after which representations can become robust to atypical processing properties.

These findings prompt two questions. First, there are existing connectionist models of behavioural interventions that have reported more positive results. Given our pessimistic conclusion here, how did these simulated interventions nevertheless succeed? Second and perhaps more importantly, for the clinician, if an intervention isn't working with a child, how can it be altered to engage one of the exceptional cases?

Let us briefly revisit the previous connectionist models of intervention. Harm, McCandliss and Seidenberg (2003) implemented two interventions that were effective in a developmental model of reading. The first intervention directly altered the computational properties of the system (the limitations in the phonological component), one of the exceptions. The second added new information to the training set, in line with the heuristic that learning of compositional domains can be improved by showing the system the 'joints' of stimuli, that is, the individual components. This condition showed an improvement of generalisation (nonword reading) in the atypical system, but no evidence that performance on the training set (word reading) was improved. The findings are consistent with our demonstration that atypical computational properties may mean the system needs to see more of the problem domain to learn the general function. Best et al. (2015) showed how a model of atypical productive vocabulary development could benefit from interventions improving either semantic or phonological representations. The model focused on immediate benefits of the interventions, in line with the behavioural data of the children it was simulating, but did not establish that the interventions had raised the final performance levels achievable by the system. If these levels were no higher, then the intervention may have served only to accelerate progress along

the same atypical trajectory. If the system eventually achieved higher levels, this is in line with the exception case that improving the input (semantic) or output (phonological) representations can enhance what is learnable by a mechanism required to associate the two.

We now turn to ways to engage the exception cases to improve the effectiveness of an intervention. There are several possibilities. If there is domain evidence supporting maturation in the target mechanism, waiting to apply the intervention may yield benefits, since computational limitations may reduce with time. Note, this is at odds with the general rubric of intervening earlier at a time of purportedly highly plasticity. Hence there is a requirement of a specific evidence base of the importance of maturation for a given process (see, e.g., Karmiloff-Smith et al., 2014, for discussion of the efficacy of CBT to treat anxiety disorders at different ages, depending on the maturation of fear extinction mechanisms).

For older children, explicit interventions may increase the opportunity to engage alternative mechanisms to drive the impaired behaviour. An analysis of the cognitive domain may indicate subsets of behaviour that could provide adaptive functioning in everyday life, and so form a compensatory intervention. As we saw above, generalisation might be enhanced by an intervention that highlights key cues, or in compositional domains, component parts of stimuli, which would normally be extracted by a typically developing system but need to be included in the training of a system with atypical properties. If a behaviour requires learning associations between representations in different domains, improving these representations may aid an intervention targeting the associations themselves. Lastly, alternative interventions might be considered to

alter the computational properties of the target mechanism by biological or indirect behavioural means, as indicated above.

The advantages of modelling

Computational modelling brings several advantages through implementation. It forces theoretical clarity, tests the viability of theoretical proposals, and generates new predictions. The greater clarity at stake concerns a mechanistic understanding of children's learning difficulties and their response to intervention. Connectionist modelling draws on principles of brain function, and therefore targets the complex interaction between learning, brain development, genetic differences, and prior experience that is likely to underlie children's differential response to interventions (Jolles & Crone, 2012). Modelling provides a test bed to consider how biological processes such as neural plasticity and pruning provide opportunities for resilience and positive adaptation (Beauchaine et al., 2008). Across the simulations, we encountered several dimensions of implementation or model behaviour that correspond to the practice of interventions identified in our review of SLT. Among them were the design of intervention items and treatment regimes, the role of age, the persistence and generalisation of intervention effects, the possibility of compensation, and individual differences in response to intervention.

In terms of the design of intervention items, we drew a distinction between additional practice on items in the child's natural experience of the domain and the introduction of new items that highlight key information for the child, such as indicating compositional structure. We additionally distinguished information intended to support generalisation of implicit regularities of the

cognitive domain to new situations. We distinguished tasks that directly target a behaviour compared to those that enhance representations that drive the behaviour. We emphasised principles derived from statistical learning theory as candidates to improve learning: the richness of learning experiences, their variability, the provision of novelty in familiar contexts, and the construction of more complex representations from simpler ones. These principles were caveated by the possibility that what works in a system with typical computational learning constraints may not have the same effect in systems with atypical constraints. Lastly, implementation encouraged a focus on the dosage, duration, and regime of training. In distributed connectionist models, modification of the training set can cause interference with prior established knowledge (so-called catastrophic interference). Interference can be reduced by lowering the dosage of new information, extending its duration, and interleaving it with training on the old information. Indeed, we saw simulation results where the performance worsened during the intervention, and benefits only emerged once it had ceased (Figure 11).

Age is an important dimension in clinical situations influencing the selection of implicit versus explicit intervention methods. Age was represented in two ways in the models we considered. First, it could be indexed by an accumulation of previous experience. The Harm, McCandless and Seidenberg (2003) reading model demonstrated a negative effect of prior learning on the potential for intervention, to explain why oral language interventions would have limited success in alleviating difficulties once the child had started to read. Even if the oral language intervention alleviates a core problem in phonology, it cannot undo prior learning linking orthography to atypical phonology. These

sub-optimal mappings must be over-written by complementary intervention targeting decoding. Second, age could index maturational changes in the computational properties of the learning mechanism. In the simulated intervention to treat delays caused by impoverished input, we saw how early treatment had greatest effects on those networks with the poorest environments; but late interventions could not benefit these networks to the same extent, as maturational pruning of connectivity had consolidated an environmental disadvantage into a structural deficit. Researchers have speculated about the cognitive domains in which maturational constraints may have most impact on training effects (Jolles & Crone, 2012). Sensitive periods suggest early intervention is better, but these reducing profiles of plasticity tend to be limited to lower level sensory and motor domains, rather than high-level cognitive abilities (Huttenlocher, 2002). In some domains, such as attention, training may indeed be more effective in later childhood – at younger ages, the target systems may be computationally immature (e.g., at 4 years instead of 6 years for attention training; Rueda et al., 2005). A lifespan perspective suggests that while behaviour is changeable at all ages, behavioural changes rely on the brain systems that are most plastic at the age when training takes place (Bengtsson et al., 2005).

Interventions for developmental deficits usually take place for a discrete time, and an important concern is whether intervention effects persist or dissipate in the longer term after it ceases. In a review of persistence and fadeout in the impacts of child and adolescent interventions, Bailey et al. (2017) argued that impacts are likely to persist for interventions that build skills influencing future development (especially that allow the individual to 'stay on track' in

home, school, or community) and in the case of environments that sustain the gains. Skills most likely to yield long-term impact are those that are fundamental for success, malleable through intervention, and would not develop eventually in the absence of the intervention. The simulations we considered either implemented intervention as an alteration to the training set for a discrete period, or as a permanent alteration. The latter could be viewed as the provision of a sustaining environment for the intervention (such as training parents to permanently altering their interactions with the child, perhaps in their level of language input). Simulation results pointed to persisting benefits of the intervention if the change to the training set was permanent. Discrete interventions could have persisting benefits, but only when plasticity reduced during training (Davis, 2017), not when it was constant across training. In the latter case, Yang and Thomas (2015) found that early interventions showed dissipating effects across development once the intervention was discontinued, with the exact type of intervention becoming less relevant. In these models, therefore, early discrete interventions had long-term benefits if the consequent gains were consolidated in the structure of the target mechanism. This reveals the double-edged sword of plasticity: if plasticity is consistent across age, interventions can be applied at any age, but the effects of early discrete interventions will be lost; if plasticity reduces with age, interventions must be early, but their effects will persist.

The single mechanism perspective did not permit us to consider the wider issue of transfer / generalisation of training effects to different skills. Nevertheless, when simulating interventions, at no time did we consider improvements in performance on the intervention items themselves – in a sense,

this would be trivial, since in error-correction networks, performance on the intervention items will almost always improve. We instead considered transfer from the intervention set to items either in the network's usual experience (the training set) or to previously unencountered items (the generalisation set). This might explain the relatively small size of the intervention effects in a number of cases (e.g., see Figure 14). Results also pointed to the importance of the composition of the intervention set in supporting performance on the training set versus generalisation. In networks with atypical computational properties, generalisation (transfer to novel items) needed additional support from intervention items selected to highlight implicit regularities in the domain, regularities that typical networks could extract from normal experience. Atypical networks often best generalised through interpolation rather than extrapolation, since their properties could not support processing of items very different from those previously encountered.[6]

Implementation suggested that the concept of compensation might have multiple related senses. First, we saw one principled way to define compensation, by contrasting it with normalisation (Yang & Thomas, 2015). In *normalisation*, the aim of intervention is to provide the full range of abilities and knowledge that any typically developing system acquires through exposure to the normal training set. In (this sense of) *compensation*, the aim of the intervention is to optimise a subset of behaviours present in the original training set. However,

[6] Plaut (1996) found that simulated recovery of reading following acquired damage was better supported by retraining on atypical semantic category members than prototypical category members. This can be seen as an example of encouraging training transfer by interpolation. In Plaut's implementation, atypical category members surrounded prototypical category members in semantic space. Training on the surrounding members transferred to those lying in between.

other models provided alternative senses of a 'compensated' system. These include (2) forcing a system to find a partial solution to the cognitive domain through over-training, but leaving residual deficits; and (3) recruiting other mechanisms to deliver the same or similar behaviour. These three senses would then translate to three distinct approaches to intervening upon an atypical system: (1) selecting an intervention that targets a subset of the target cognitive domain; (2) providing greater practice to force greater accuracy from an atypical system, or simply leaving the system to improve through more experience; (3) employing explicit strategies to encourage the use of alternative mechanisms.

Lastly, the simulations addressed possible mechanistic explanations of individual differences in response to interventions. More recent polygenic models of developmental disorders were useful here, since they simulated the atypical mechanism against a background of typical variation in a range of developmental factors, or indeed captured the developmental deficit as lying on a continuum of population-wide variation (Thomas et al., 2016). A model investigating the causes of language delay (Thomas & Knowland, 2014) pointed to the limited power of early behavioural markers in predicting whether delays would resolve, since early profiles are largely conditioned by the structure of the task domain. The model suggested that predictive power could be increased by measures of underlying cognitive processes (see Fernald & Marchman, 2012). A model investigating individual differences in response to intervention (Davis, 2017) demonstrated that responses could be highly variable, and that both differences in the severity of atypical computational constraints and in other population-wide individual differences factors predicted the response. However, there were stochastic factors, and the predictive factors themselves showed

strong interactions such that much variance in outcome remained unexplained, despite replicable individual differences in response to intervention. Finally, a lower level of stimulation from the environment could also play a role, exaggerating the effect of atypical computational constraints (Figure 5), or itself causing deficits in combination with maturational changes in network connectivity (Figure 3). Overall, this avenue of modelling is important to support the search for stratification biomarkers in research on developmental disorders, work which seeks to isolate measures (e.g., age, gender, intellectual ability, comorbidity of deficits) that predict developmental outcomes and response to intervention.

Limitations to modelling

Models require simplification, and one might argue that in applying them to intervention, the level of simplification is too great. We should be clear, then, the ways in which the computational work we have reviewed falls short with respect to the practice of therapeutic intervention for developmental disorders.

On a broader scale, a focus on cognitive mechanism does not capture the complexity of the intervention situation, which can depend on dynamics of the interaction between the child and the therapist, and where intervention is sometimes a process of discovery of what works for individual children in the context of their family and school environment. To some extent, even fairly mechanism-focused interventions involve substantial behavioural and interactional interchange between the children and the therapist (and parent, if also coached), which may yield collateral benefits. Simulations do not address some of the complexities, such as distinguishing the effects of explicit instruction

from implicit, the role of the expertise of the therapist, the effects of adaptive vs. non-adaptive instruction, the distinction between 1-to-1 versus group instruction, the difference between therapist-delivered and parent-delivered interventions. Moreover, as Beauchaine et al. argue: 'opponents of biological approaches to prevention and intervention also argue that by emphasising genetic and neurobiological processes, we divert attention and resources away from important psychosocial causes of maladjustment, such as stress, parenting, and family interactions' (2008, p.748). Work in the implementation sciences has also pointed to wider limiting, enabling and incentivising factors for changing behaviour beyond cognitive mechanisms, such as resources and policy (e.g., Michie, van Stralen & West, 2011).

On a narrower scale, our focus was on a limited set of computational architecture: associative networks. It is possible that other architectures, such as self-organising maps or recurrent networks, might provide different plasticity conditions or effects of intervention on generalisation. These remain to be explored. The observation that interventions for different language skills required different levels of intensity, duration, and interleaving (Lindsay et al, 2010) is consistent with the view that different types of mechanism are in play. Speculatively, it may be that intensity is more important than duration to change sensory representations (self-organising systems); that repeated short bursts over an extended time are necessary to alter access to representations (associative systems); and that an extended duration of practice is necessary to extract regularities in complex sensori-motor sequences (recurrent networks). In addition to different architectures, it is necessary to consider control systems, mechanisms of executive function and reward-based learning, in order to

address the origin and malleability of deficits in behavioural regulation, such as the restricted repertoire of interests in autism, or attentional deficits in Fragile X syndrome, or impulsivity in ADHD. Lastly, the model framework captures development in terms of a plastic mechanism exposed to a structured learning environment. However, this does not readily lend itself to considering the possibility that the disorder may change the structure of learning environment via indirect pathways. For example, poor reading levels may reduce the child's motivation to spend time reading, or parents may respond differently to children with learning disabilities than they would typically developing children.

As with Plaut (1996), we took a simplifying step of first adopting a single mechanism perspective. However, behaviour is generated by the interaction of multiple mechanisms. A multiple-mechanism framework is necessary to consider, variously, interventions to encourage alternative strategies, the use of executive function skills to compensate for weaknesses in domain-specific systems (Johnson, 2012), and interventions that might address deficits in functional connectivity between mechanisms (e.g., as sometimes proposed as a deficit in autism; see Thomas et al., 2016, for discussion). The Best et al. (2015) model holds some promise in this regard, since it captures separate behaviours stemming from the operation of components (nonword repetition, semantic categorisation) and from the interaction between components (naming, comprehension), where each behaviour exhibits its own developmental trajectory. Within such a multiple-mechanism framework, it is apparent that a single mechanism can nevertheless serve as a limiting factor on performance, even if it is not the sole generator of behaviour.

Conclusion: The importance of narrowing the gap

Advances in mechanistic, computational models of developmental disorders (and more widely, individual variability) set the foundation for an investigation of intervention. Implementation can provide a driver for advances in theory, although questions remain about whether the simplification necessary for modelling omits key dimensions of the therapeutic situation, notably its usual basis in social interaction. It is important to narrow the gap between theories of deficit and theories of intervention, in order to place intervention on an evidence-driven, mechanistic basis. Practice-based approaches naturally emphasise behavioural consequences of therapy and are less focused on understanding mechanisms: for these approaches, what is important is what works behaviourally and what can enable success. This emphasis on proximate goal is one of the reasons for the gap. It is a view recently echoed in education, where behaviour change through instruction is the aim. Scepticism has been expressed regarding the relevance of any lower level understanding of mechanism than psychological constructs in the field of education, because there too, it is argued that only behavioural outcomes are important (Bowers, 2016; see Howard-Jones et al., 2016, for discussion).

However, understanding the active agent underpinning a successful intervention is key to understanding what will work in which contexts for what disorders, as well as the flexibility of the application of a given technique (Law et al., 2008). As Nathan and Alibali (2010) argue, to narrow the gap, we need a combination of scaling-up from the elemental, mechanistic models of cognitive science and scaling-down from the complexity of real-life therapeutic situations. That in turn requires clinicians to be interested in mechanism, despite it being an

understandably lower priority than behavioural outcomes for the children they treat.

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Tables

Table 1.

Computational parameter	Processing role	Effect size of PD vs. RD comparison
Number of internal units	Capacity	.031**
Pruning threshold	Capacity / Regressive events	.021*
Learning algorithm	Capacity / Plasticity	.104**
Lexical-semantic learning rate	Plasticity	.024**
Unit discriminability	Plasticity / Signal	.025**
Processing noise	Signal	.026**

PD = persisting delay; RD = resolving delay

Scores show η_p^2 effect sizes from ANOVA comparing PD and RD groups (see Thomas & Knowland, 2014, Table 2, for parallel analyses using logistic regression methods)

*Effect reliable at $p < .05$. **Effect reliable at $p < .01$

Table 2. Standardised beta values for linear regressions predicting individual differences in treatment effect sizes following two different types of intervention, normalisation and compensation, in simulated networks with a connectivity over-pruning disorder (Davis, 2017). N=790 networks (only those from the population showing a behaviourally assessed performance deficit). Separate regressions were carried out for performance on the training set and generalisation set. The shaded area shows parameters related to the pathological process, elevated values of the pruning threshold, permitting larger connections to be removed following the onset of pruning.

	Intervention type			
	Normalisation		Compensation	
Parameter	Training set performance	Generalisation performance	Training set performance	Generalisation performance
Number of hidden units	-0.016	0.012	0.011	0.023
Sigmoid temperature	-0.040	-0.001	**-0.098**	**-0.127**
Processing noise	0.028	0.032	0.007	-0.012
Learning rate	**-0.065**	**-0.086**	-0.053	-0.016
Momentum	-0.014	-0.011	-0.013	-0.011
Initial weight variance	-0.015	-0.002	-0.031	-0.023
Architecture	**-0.110**	**-0.101**	**-0.112**	**-0.092**
Learning algorithm	-0.006	-0.059	-0.011	0.010
Response threshold	-0.055	-0.063	0.000	0.036
Pruning onset	0.022	-0.007	0.057	0.045

Pruning rate	-0.006	0.006	-0.062	**-0.075**
*Pruning threshold	0.014	**0.082**	0.039	-0.047
Weight decay rate	0.021	0.007	0.036	0.025
Sparseness of connectivity	0.027	**0.065**	0.049	0.052
Richness of environment	-0.030	-0.036	-0.028	-0.028

Bold shows significant at p<.05

* This parameter was set to atypical values to produce the developmental disorder

Figure captions

Figure 1. Simulation of typical and atypical past tense acquisition. Empirical data (per cent accuracy) for typically developing children from Thomas et al. (2001) for a group of typically developing children on a past tense elicitation task for regular verbs, irregular verbs, novel verbs, and over-generalisation errors; and for a group of children with SLI from van der Lely and Ullman (2001), using the same elicitation task. The simulation data are from Thomas (2005) for a connectionist past tense model, either in a typical condition or an atypical condition where the discrimination of the simple processing units was reduced by lowering the 'temperature' of the sigmoid activation function (1=>0.25). The typical and atypical models are shown at two points in development, at a point which approximately matched the performance of real children (250 epochs of training) and at the end of training when performance had reached asymptote (5000 epochs). Accuracy on the training set eventually achieved ceiling performance in the atypical network but a deficit in generalisation to novel forms remained. Error bars show standard error over 10 replications with different initial random seeds.

Figure 2. Group averaged developmental trajectories for 1000 simulated children in a model of English past tense formation, assuming a polygenic model for language delay (Thomas & Knowland, 2014). Delay was defined at Time 1 as networks whose performance fell more than 1 standard deviation below the population mean. Networks were defined as having Resolving delay if their performance fell within this normal range by Time 5; and as having Persisting delay if their performance remained below the normal range by Time 5 (See

Thomas & Knowland, 2014, for further details). Error bars show standard deviations.

Figure 3. Performance frequency distribution for a population (N=1000) of networks learning English past tense. Individual differences exist in the richness of the learning environment (Normal – solid lines). In an intervention condition, these differences are universally alleviated (Intervention – dashed lines). Networks also had intrinsic individual differences in their learning ability. Each network was trained for 1000 epochs. Training environments for all individuals were fully enriched either following 50 epochs (early) or 250 epochs of training (late). X-axis shows performance level (proportion correct). (a) Change in distribution of performance across development for regular verbs, following early universal enrichment. (b) Change in distributions for irregular verbs following early universal enrichment. (c) Change in distribution for irregular verbs following late universal enrichment, comparing Normal (solid), Early intervention (EI, wide dashed) and Late intervention (LI, narrow dashed). For clarity, data are idealized normal distributions based on means and standard deviation of the simulated populations.

Figure 4: The relationship between treatment effects (change in proportion correct assessed at end of training) and the quality of the early environment for each simulated child (varying between 0 and 1) following universal enrichment of the population, for (a) Regular and (b) Irregular verbs. Poorer family language environment predicted a larger treatment effect, but this reduced when enrichment occurred later in development, and more so for irregular verbs.

(Early enrichment = 50 epochs, Late = 250 epochs, treatment effects assessed at 1000 epochs. Linear fits are shown for all conditions. Early enrichment for regular verbs was better fit by a log function (R^2=.87), while linear functions explained more variance for the other three conditions.)

Figure 5. Population performance on regular verbs early in development (50 epochs), split by individuals in impoverished or enriched environments, and stratified by individuals with different unit discriminability (temperature values 0.5-1.5). Interaction effect was at trend level (p=.06). Error bars show standard deviations. (Impoverish env = Impoverished environment; Enriched env = Enriched environment).

Figure 6. A simple associative network was trained to learn category boundaries under typical and atypical conditions (e.g., reduced connectivity): (a) network architecture; (b) example categorisation problem, with 10,000 data points; (c) the training set given to the network, sufficient to learn the problem under typical conditions; (d) an example intervention set added to the training set to aid development under atypical conditions. Networks had 50 internal units (backpropagation network; learning rate=.1, momentum=.3, temperature=1)

Figure 7. Developmental trajectories and internal representations in a typical case (TD), an atypical case with low connectivity (30%, C=0.3) and the same atypical case with an intervention. Top panel: Developmental trajectories; intervention commenced at 100 epochs. The intervention set was added to the training set for the duration of training. Vertical lines show epochs at which

snapshots were taken. Lower panels: snapshots of the activation pattern of the unit for output category 2 in the three cases, which should respond only to the central band (see Figure 6). Hot colours represent more activity. (Fedor et al., 2013).

Figure 8. (a) Performance of a group of 9 networks with a disorder caused by greater-than-usual loss of connectivity (red), compared to control networks (blue). Also shown are the disorder networks following an early behavioural intervention (green). Effects of the intervention sustain to the end of development. (b) The number of network connections for the disorder group in untreated and intervention conditions. The intervention caused initial acceleration of loss but final preservation of a greater proportion of connections, associated with improved computational power. Mid-training = 250 epochs; End of training = 1000 epochs.

Figure 9. (a) Developmental trajectories for naming and comprehension in a model acquiring the meanings (semantics) and word names (phonology) of 400 vocabulary items (averaged over 3 replications). The typical model shows the usual comprehension-production asymmetry. In the Word-Finding Difficulty (WFD) model, there was a restriction in the capacity of the pathway linking semantics to phonology (from 175 to 70 hidden units), which impacted on the development of naming, while comprehension trajectories did not reliably differ. (b) Early intervention targeting the naming pathway (weakness). (c) Early intervention targeting the development of the phonological representations, the semantic representations, or both (strengths). A combined intervention is also

shown in (b). Intervention comprised training at 5 times the frequency on acquisition of these representations compared to naming and comprehension, beginning at 100 epochs and lasting for 100 epochs, shown by the shaded area. (Alireza, Fedor & Thomas, 2017).

Figure 10. Effects of increasing levels of neural noise on performance in a past tense network, when the noise is present throughout development (early deficit), or applied when representations are established (late deficit). [Fully connected feedforward network learning past tense problem: 90 input units, 50 hidden units, 100 output units, training set 500 patterns, learning rate 0.1, momentum 0, performance evaluated after 500 epochs of training. Noise was added to the activation levels of the units in the hidden layer, with a Gaussian distribution mean zero and a standard deviation that determined the severity of the damage. Standard deviations of 0, .025, .05, .0625, .075, .0875, .1, .2, .3, .4, .5, .6, and .7 were used. Units had a maximum activation level of 1 and a minimum of 0, and noise could not take the activation state of a unit outside of these limits. Error bars represent standard errors over 10 replications with different initial random seeds.] Data from Thomas & Karmiloff-Smith (2002).

Figure 11. (a) Treatment effect size for an entire population of 1000 networks, experiencing either a normalisation or compensation intervention. Green shaded area shows the period of the intervention. (b) Two case studies of atypical networks, developing in either treated or untreated conditions, showing worsening of performance on the training set during the intervention, but a subsequent longer term benefit. (See Davis, 2017, for further details).

Figure 12. Treatment effects across a population of 1000 past tense networks. The networks were given a compensatory intervention to enhance generalisation (the past tense rule). Under the Discrete condition, the intervention was applied at 30 epochs for a duration of 40 epochs. Under the Continuous condition, the intervention set was permanently added to each network's training set. Treatment effects are shown at the end of intervention (70 epochs), a short period after (100 epochs) and the end of training (1000 epochs). Error bars show 95% confidence intervals for the population. (Davis, 2017)

Figure 13. Treatment effects of phonological versus semantic interventions for the Best et al. (2015) model of word-finding difficulties. The atypical language profiles of two individual children were simulated, and then interventions applied (here measured in how much naming development was advanced). The profile of each child could be simulated by atypical settings to three different computational parameters: reduced connectivity (Deficit C), reduced hidden units (Deficit H), or a shallower sigmoid activation function in the artificial neurons (Deficit T). Intervention responses were subtly different depending on how the deficit was implemented. Error bars show standard errors of 10 replications of each intervention (See Best et al., 2015, for further details).

Figure 14. Distribution of treatment effect sizes for atypically developing networks with an over-pruning disorder (Davis, 2017). X-axis shows treatment effect in terms of change in proportion correct. (a) Distribution for performance

on the training set following either normalisation or compensation treatment; (b) distribution for performance on the generalisation set following either normalisation or compensation treatment. [Population of 1000 networks, intervention for duration of 40 epochs applied early in development, epoch 30 out of a lifespan of 1000, performance tested at 100 epochs]

Figures

Figure 1

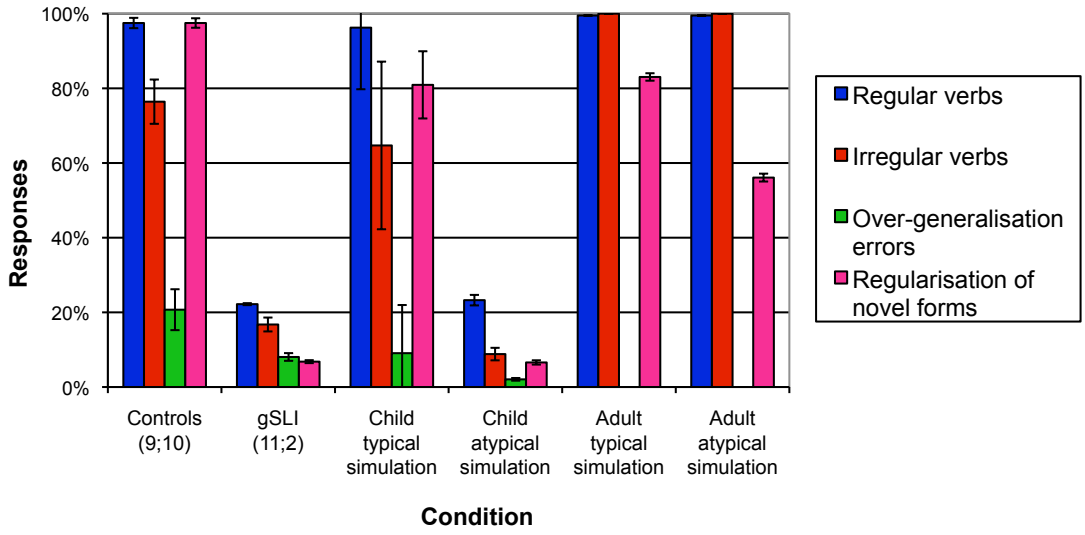


Figure 2

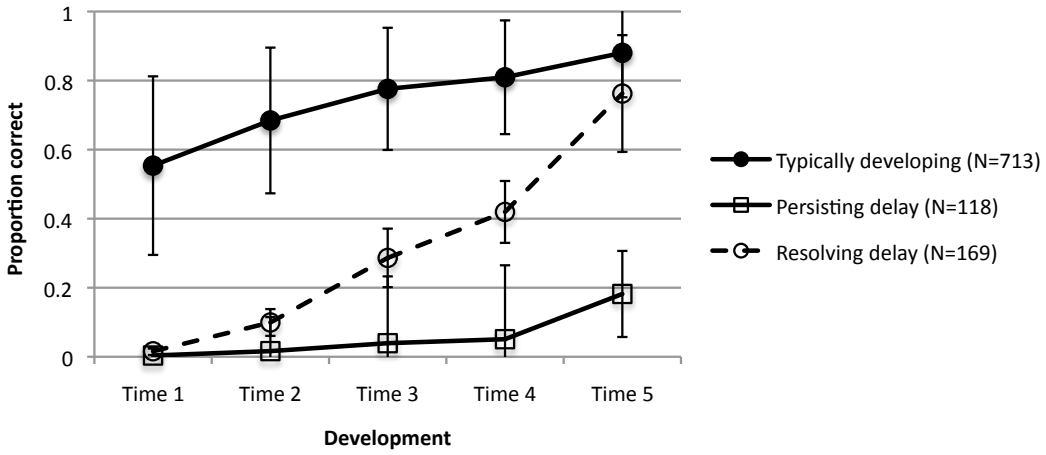
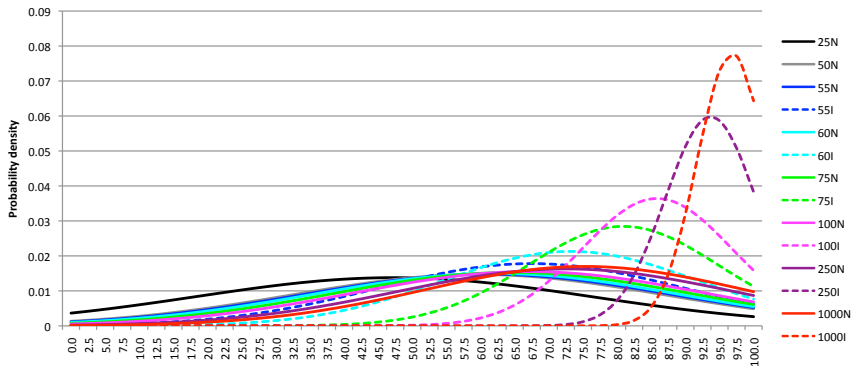
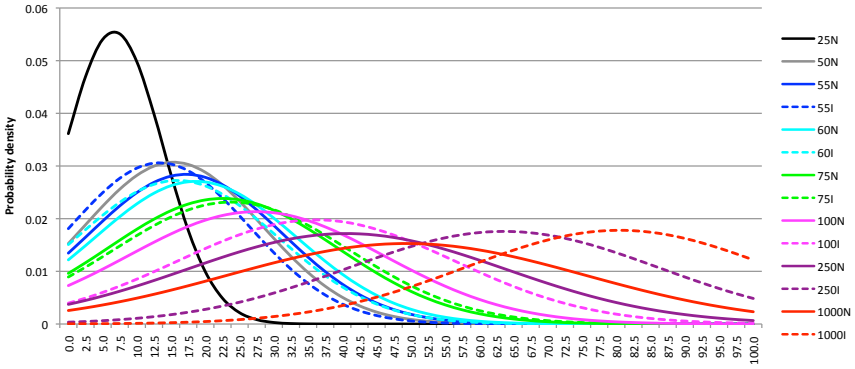

1
0.8
0.6
0.4
0.2
0
Proportion correct
Time 1
Time 2
Time 3
Time 4
Time 5
Development
Typically developing (N=713)
Persisting delay (N=118)
Resolving delay (N=169)

Figure 3

(a)


Probability density
25N
50N
55N
55I
60N
60I
75N
75I
100N
100I
250N
250I
1000N
1000I

(b)


Probability density
25N
50N
55N
55I
60N
60I
75N
75I
100N
100I
250N
250I
1000N
1000I

(c)

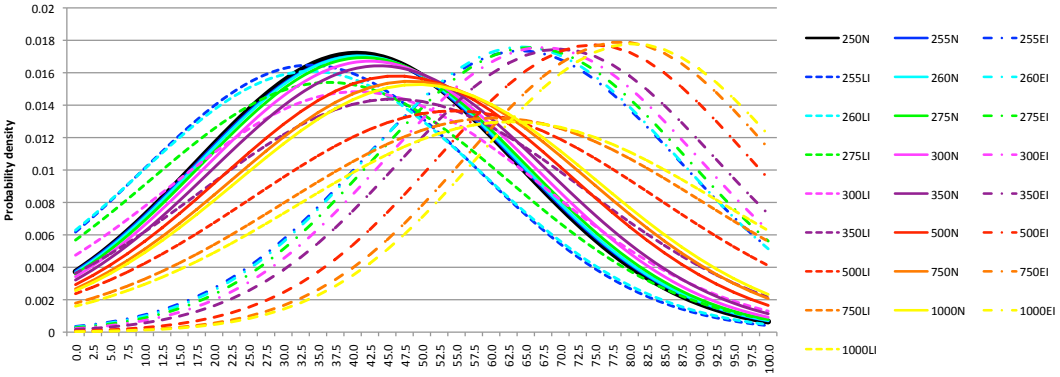
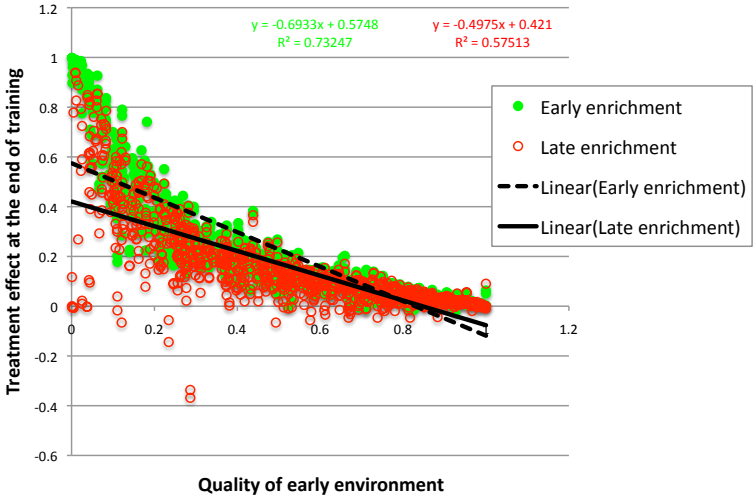

Probability density
250N
255N
255EI
255LI
260N
260EI
260LI
275N
275EI
275LI
300N
300EI
300LI
350N
350EI
350LI
500N
500EI
500LI
750N
750EI
750LI
1000N
1000EI
1000LI

Figure 4

(a) Regular verbs



(b) Irregular verbs

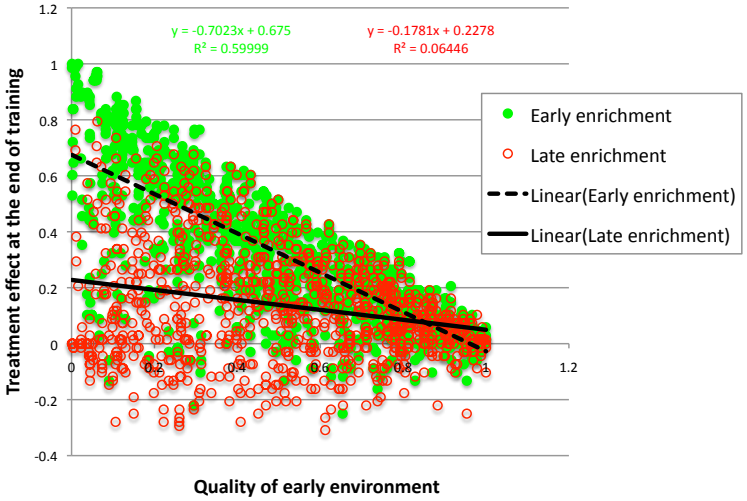


Figure 5

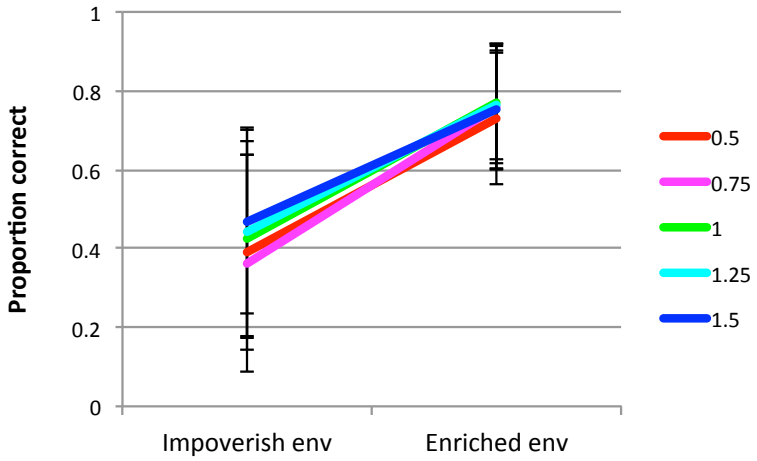
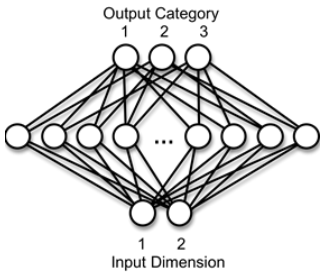
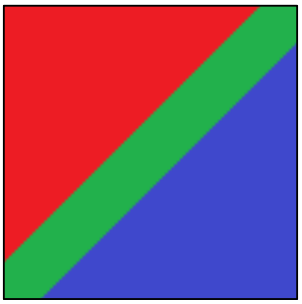

1
0.8
0.6
0.4
0.2
0
Proportion correct
Impoverish env
Enriched env
0.5
0.75
1
1.25
1.5

Figure 6

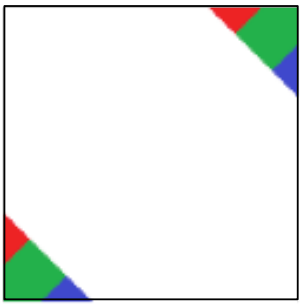
(a) Network architecture



(b) Full problem



(c) Training set



(d) Intervention set

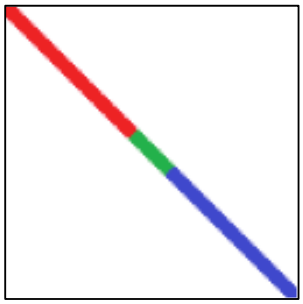


Figure 7

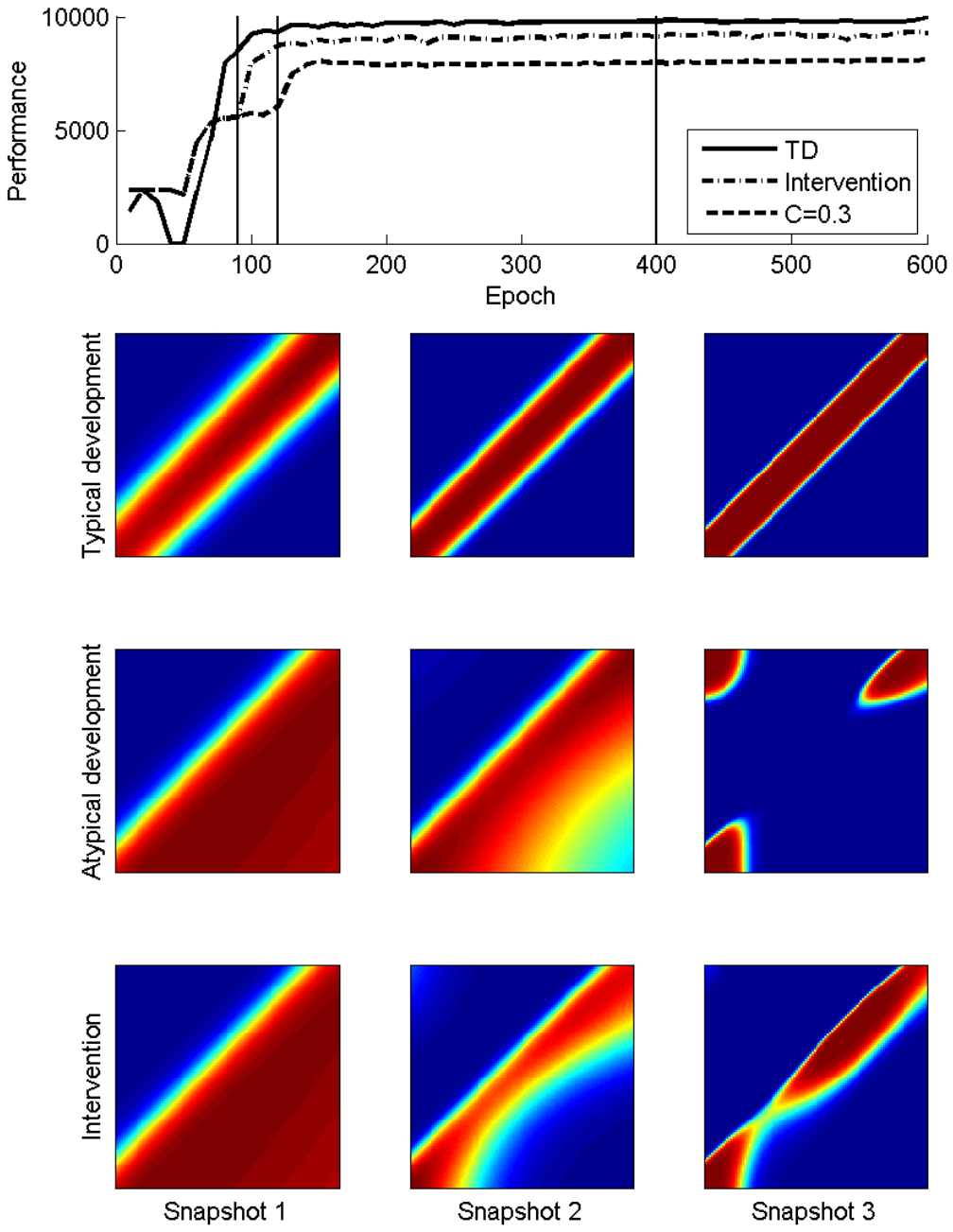
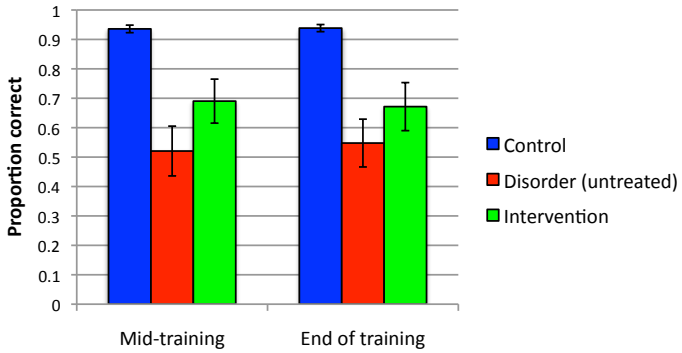

Performance
10000
5000
0
0
100
200
300
400
500
600
Epoch
TD
Intervention
C=0.3
Typical development
Atypical development
Intervention
Snapshot 1
Snapshot 2
Snapshot 3

Figure 8

(a)


Proportion correct
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
Mid-training
End of training
Control
Disorder (untreated)
Intervention

(b)

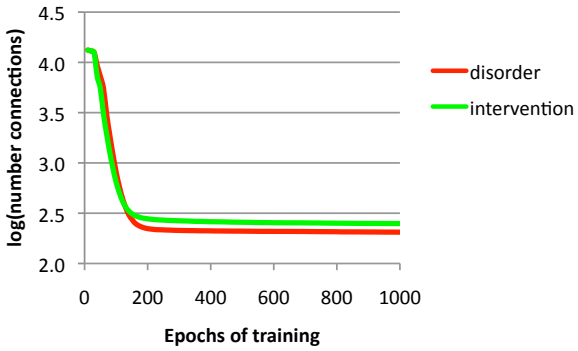
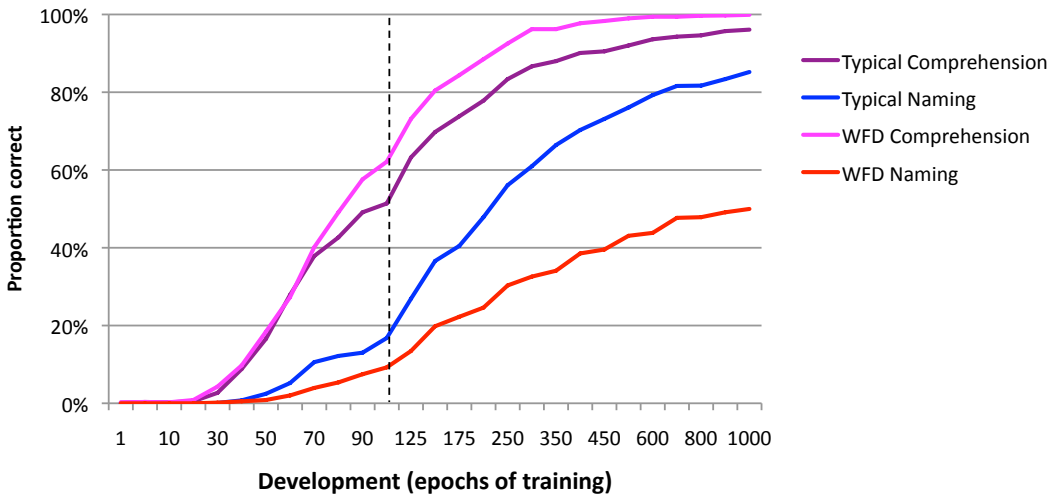
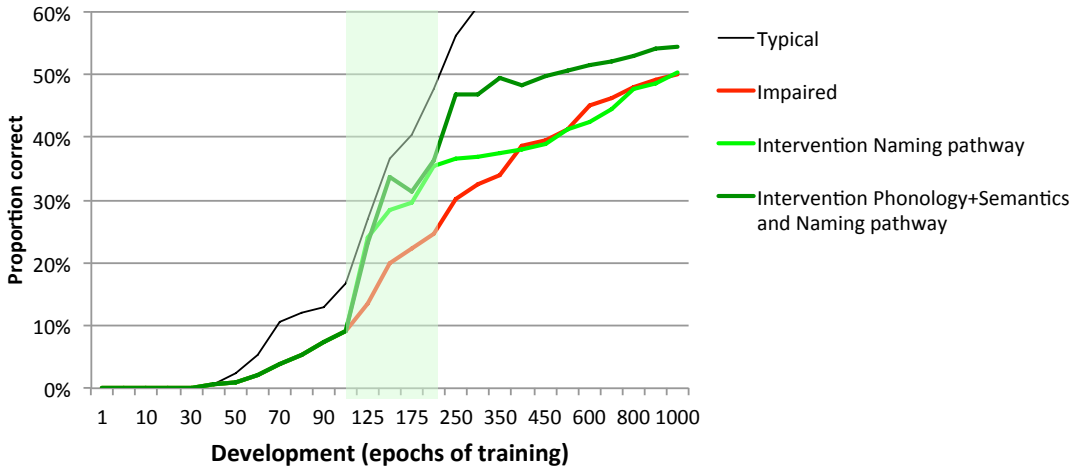

log(number connections)
4.5
4.0
3.5
3.0
2.5
2.0
0
200
400
600
800
1000
Epochs of training
disorder
intervention

Figure 9

(a)


100%
80%
60%
40%
20%
0%
Proportion correct
1 10 30 50 70 90 125 175 250 350 450 600 800 1000
Development (epochs of training)
Typical Comprehension
Typical Naming
WFD Comprehension
WFD Naming

(b)


60%
50%
40%
30%
20%
10%
0%
Proportion correct
1 10 30 50 70 90 125 175 250 350 450 600 800 1000
Development (epochs of training)
Typical
Impaired
Intervention Naming pathway
Intervention Phonology+Semantics and Naming pathway

(c)

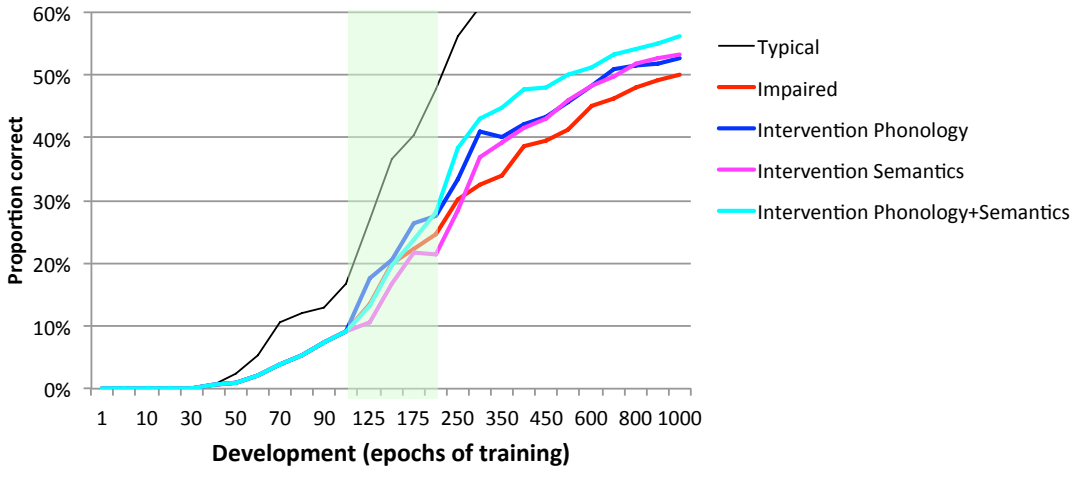

60%
50%
40%
30%
20%
10%
0%
Proportion correct
1 10 30 50 70 90 125 175 250 350 450 600 800 1000
Development (epochs of training)
Typical
Impaired
Intervention Phonology
Intervention Semantics
Intervention Phonology+Semantics

Figure 10

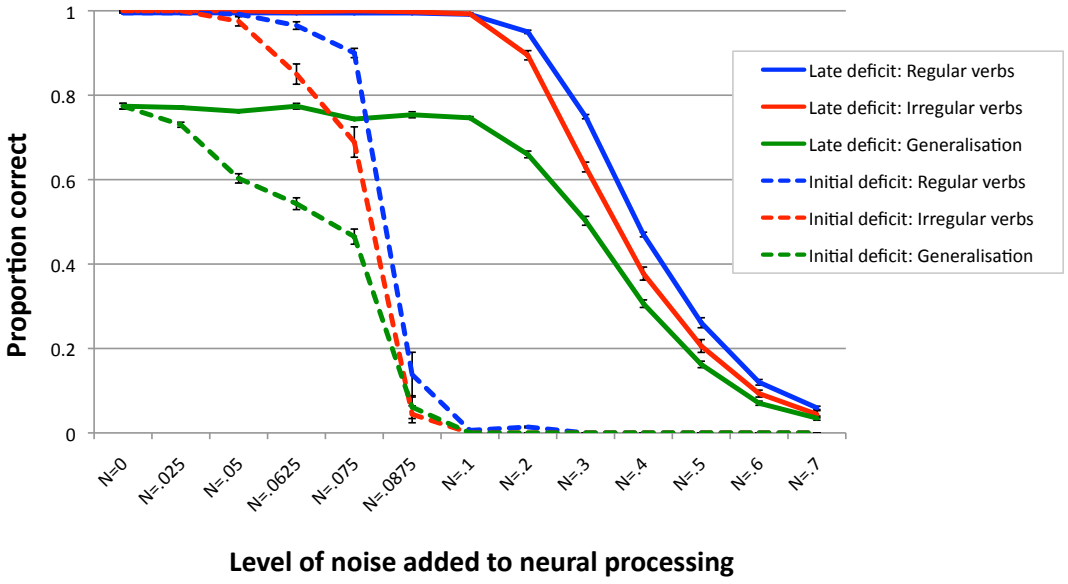
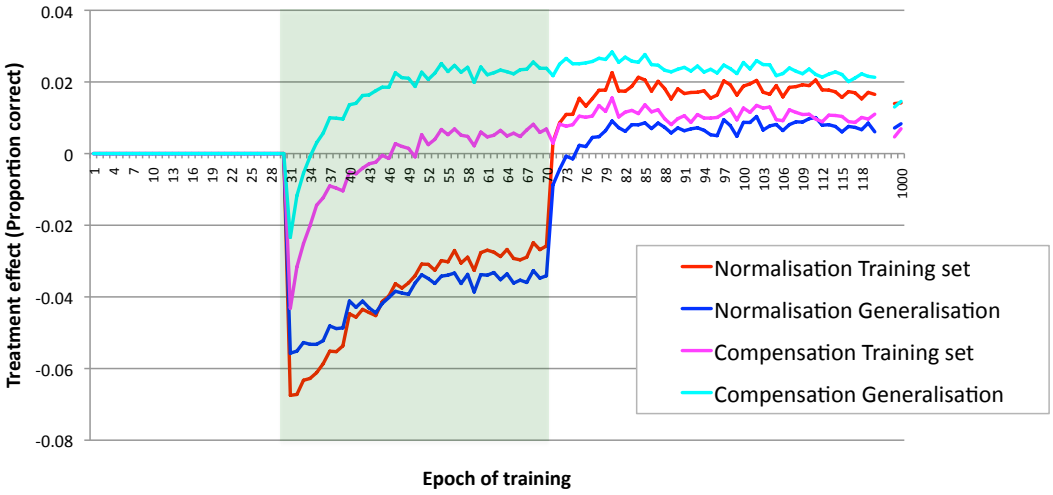

Proportion correct
1
0.8
0.6
0.4
0.2
0
N=0
N=.025
N=.05
N=.0625
N=.075
N=.0875
N=.1
N=.2
N=.3
N=.4
N=.5
N=.6
N=.7
Level of noise added to neural processing
Late deficit: Regular verbs
Late deficit: Irregular verbs
Late deficit: Generalisation
Initial deficit: Regular verbs
Initial deficit: Irregular verbs
Initial deficit: Generalisation

Figure 11

(a)


0.04
0.02
0
-0.02
-0.04
-0.06
-0.08
Treatment effect (Proportion correct)
Epoch of training
Normalisation Training set
Normalisation Generalisation
Compensation Training set
Compensation Generalisation

(b)

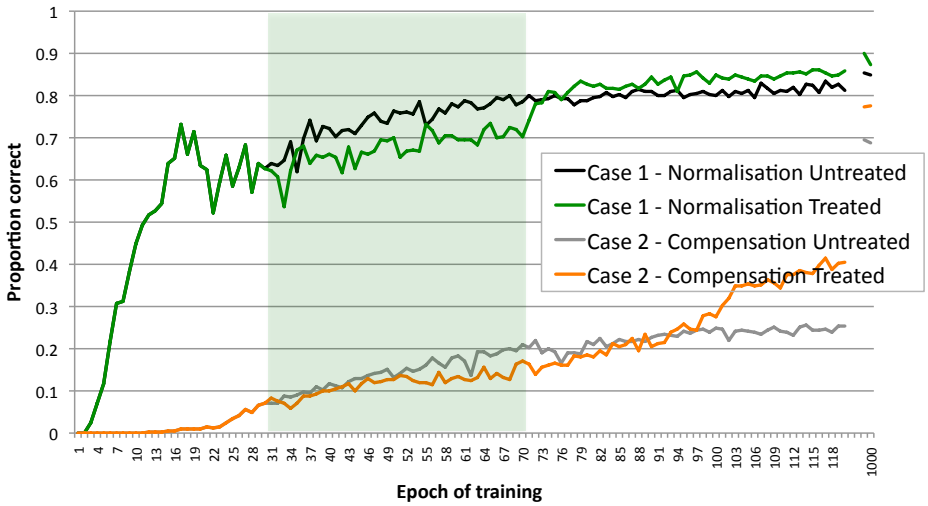

1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
Proportion correct
Epoch of training
Case 1 - Normalisation Untreated
Case 1 - Normalisation Treated
Case 2 - Compensation Untreated
Case 2 - Compensation Treated

Figure 12

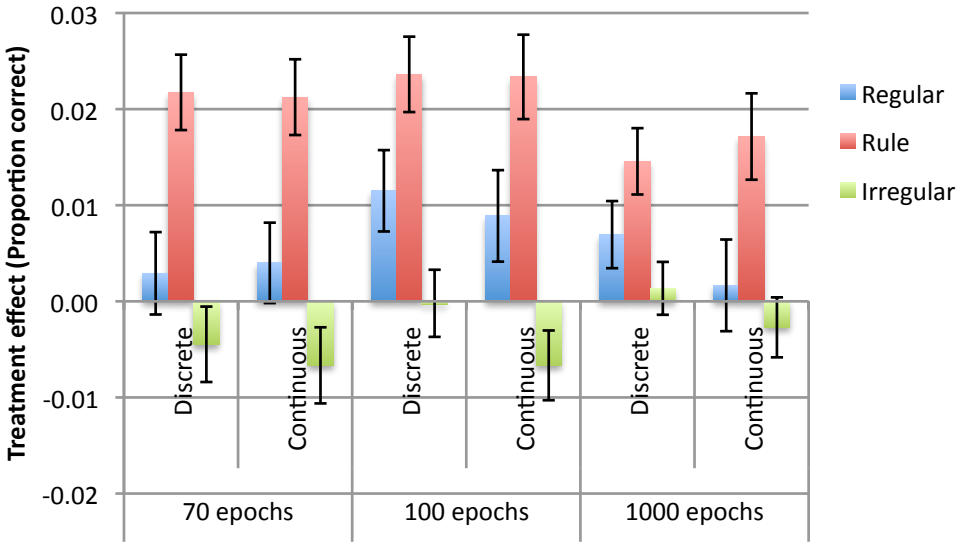

0.03
0.02
0.01
0.00
-0.01
-0.02
Treatment effect (Proportion correct)
Discrete
Continuous
Discrete
Continuous
Discrete
Continuous
70 epochs
100 epochs
1000 epochs
Regular
Rule
Irregular

Figure 13

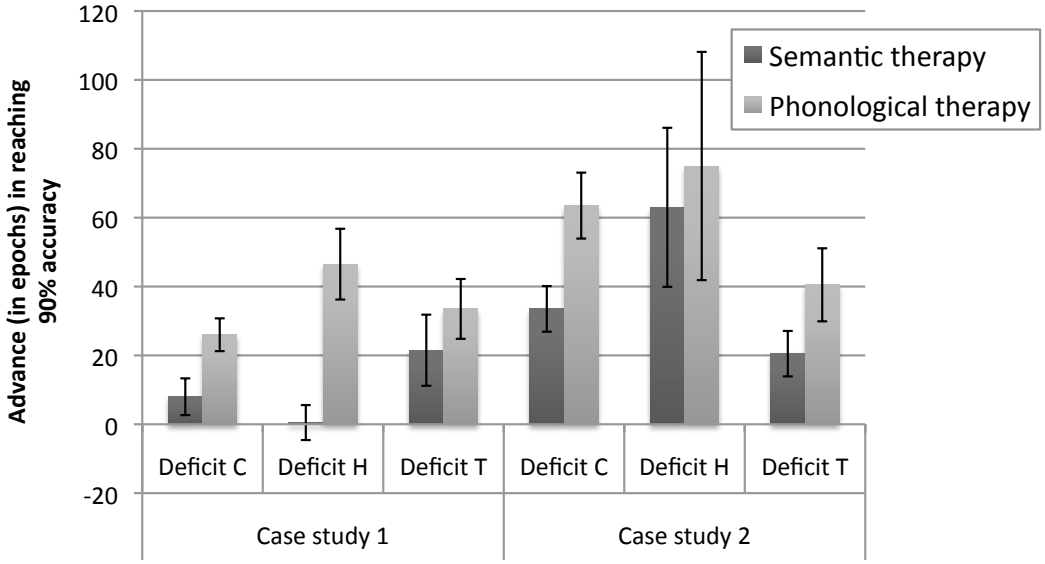
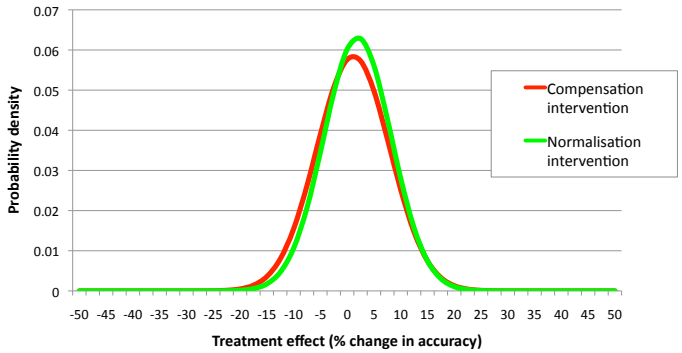

Advance (in epochs) in reaching 90% accuracy
120
100
80
60
40
20
0
-20
Semantic therapy
Phonological therapy
Deficit C
Deficit H
Deficit T
Deficit C
Deficit H
Deficit T
Case study 1
Case study 2

Figure 14

(a) Training set performance



(b) Generalisation set performance