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How connectionist simulations fail to account for developmental disorders in children*

Abstract

Using connectionist modelling, Thomas and Karmiloff-Smith claim that developmental disorders in children are characterised by atypical trajectories and an ultimate functional architecture that is fundamentally different from normal. We argue that there is no empirical evidence for these claims in any developmental disorder and that the available evidence provides support for residual normality in both developmental and acquired disorders. We also refute the claim that modular accounts cannot encompass developmental trajectories in children with developmental disorders.

A fundamental debate concerning developmental disorders is the relationship between established skills and normal skills. An assumption of cognitive neuropsychology is that impaired performance reflects normal function minus impaired function, such that exhibited skills are part of the normal functional architecture (Ellis & Young 1988; Temple 1997). This has been termed subtractivity (Saffran 1982), transparency (Caramazza 1984) or residual normality (Thomas & Karmiloff-Smith). Evidence against subtractivity would come from cases in which the functional architecture of the system had altered with development of cognitive modules that do not exist in the normal brain. However, there is no such empirical evidence in relation to either adults or children.

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A contrasting view to subtractivity is that performance in developmental disorders does not relate to normality but reflects abnormal development of the entire system. Thomas and Karmiloff-Smith argue for a qualitatively different end-state to normal with “a different functional structure in children”. On this basis developmental disorders provide no insight into normal development and child neuropsychology is erroneous in its method and conclusions. Thomas and Karmiloff-Smith argue from simulations but there remains no empirical evidence in any developmental disorder that the ultimate functional architecture has fundamentally different organization from normal, rather than merely lacking or having reduced development of components of normal functional architecture. Consequently, their simulations are not a valid reflection of developmental disorders in children.

In contrast to absence of empirical evidence supporting a different functional structure in abnormal development, there is extensive empirical evidence of residual normality in both developmental and acquired disorders in children. For example, in procedural dyscalculia, despite pervasive impairment in arithmetical procedures, knowledge of numerical facts can have 95% accuracy. In number fact dyscalculia, severely impaired knowledge of numerical facts co-exists with excellent mastery of procedural algorithms (Temple 1991; 1994; Sokol et al. 1994). Similar arguments apply to developmental perceptual disorders that selectively affect perception of either movement or location (Ahmed and Dutton 1996; McCloskey et al. 1995). Another example is selective impairment in components of complex visual recognition with severely impaired face recognition but excellent visual word recognition in reading for Dr S (Temple 1992; 1997) and severely impaired visual recognition for irregular words yet intact visual memory for other complex material in surface dyslexia (Castles & Coltheart 1996). In each case, impaired skill co-exists with excellent development of contrasting skill within the same domain.

Such cognitive neuropsychological analyses highlight the focal modular impairments seen within developmental disorders.

Thomas and Karmiloff-Smith suggest that evidence for a distinct functional structure in a developmental disorder is found in face recognition in Williams syndrome [WS]. Here it is argued that development is completely different from normal and cannot be explained as a normal brain with "parts intact and parts impaired" (Karmiloff-Smith 1997). Early studies argued for good face processing in WS but based only on face matching. Feature-based componential analysis of faces, dependent on local cues, is intact in WS but global processing of the configuration of faces is impaired (Deruelle et al. 1999; Karmiloff-Smith 1997). However, normal face recognition begins with a featural componential strategy (Carey & Diamond, 1977). This ability remains existent in the normal repertoire of skill. Normal children and adults can perform both local and configural analyses of faces successfully. Children with WS can perform only the former. In terms of the normal brain, componential processing is intact and configural processing impaired, a straightforward modular dissociation within face processing systems. There is no evidence for a new and different end-state.

A further example of modular impairment is the intact ability to form regular inflected words but selective impairment in irregular inflection in WS (Clahsen & Almazan 1998). Thomas and Karmiloff-Smith dismiss this study, referring to their own study reporting absence of such selective effects when scores are averaged across diverse ages and mental ages rather than in age bands (Thomas et al. 2001). Clahsen and Temple (2003) offered a reanalysis of this data that remains compatible with a modular account and emphasises the importance of appropriate control matching within developmental levels. Furthermore, recent data from further cases of WS confirms the original finding of a selective (lexical) impairment with irregular inflection

(Clahsen, Ring & Temple 2003). Thomas and Karmiloff-Smith also ignore the comparable selective impairment in comparative-adjective formation (Clahsen & Temple 2003) and irregular plural formation (Clahsen & Almazan 2001) yet intact complex syntax in WS (Clahsen & Almazan 1998). They appear unable to account for subtle selective deficits of this kind.

A further debate concerns the nature of development itself. Thomas and Karmiloff-Smith discuss cognitive neuropsychology and its presupposition of modularity and residual normality as if it necessarily ignores development. However, a modular account does not preclude the study of development or a developmental dimension to the functional architecture of normal systems. Indeed the multiple route cognitive neuropsychological models, for example of literacy (e.g. Coltheart et al. 2001), have enabled more flexible interpretation of development than traditional post-Piagetian stage-models with one-dimensional rigidity and invariant sequence (e.g. Frith 1985). Another example comes from Specific Language Impairment (SLI). Longitudinal studies of English (Rice 1999) and German SLI children (Rothweiler & Clahsen 1994) demonstrate selective delayed onset of verb-finiteness marking, the same developmental trajectory as normal children, and persistent selective delay in verb-finiteness marking into late childhood. Thus, impairments in onset do not necessarily lead to atypical developmental trajectories.

Thomas and Karmiloff-Smith's simulation demonstrates development that proceeds differently in a damaged single-mechanism system. However, disorder can only be interpreted as delay or distortion in a single-mechanism system since it does not have any other mechanism able to continue to operate normally despite initial damage. The introduction of multiple-route connectionist models has been an attempt to respond to the limitations of unitary systems in explaining neuropsychological phenomena (e.g. Plaut et al. 1996). Thomas and Karmiloff-Smith claim that even in a dual-mechanism system residual normality is absent. Their second simulation tries to demonstrate that if one route is damaged the other will not develop normally. However, this does not imply that the impaired route works improperly for its domain. For example, damage to the indirect-route system in their dual-route simulation does not produce impairments for regular past-tense forms and the end-state performance on regulars is intact in this damaged system.

An alternative are multiple-component cognitive neuropsychological models with potential to account for both individual variation in developmental sequence of skill acquisition and varied developmental disorders. Their success in modelling dynamic development can be seen in modelling of changes in language associated with progressive dementia (e.g. Hodges et al. 1992). The system changes with development although the underlying functional architecture remains the same. Just as components of the functional architecture may degrade with dementia, so in development components of the architecture may unfold over time and become more complex in relation to the representations upon which they can act. A genetic blueprint may unfold with different modules coming into play or maturing of capabilities within modules over time. In developmental disorders, components may fail to unfold as in face processing in WS or the representations upon which they act may be reduced or distorted as in irregularly inflected word forms in WS. The success of cognitive neuropsychology in assessing dynamic change is also seen in studies charting progress of theory-driven remediation upon development (e.g. Brunsdon et al. 2002; Cardell & Chenery 1999). This approach provides a constructive starting point and framework for the study of development and in the delineation of patterns of performance and disorder seen across development the extent of modularity itself within development can be properly assessed.

In any developmental analysis, a systematic approach is needed to define the states that change. Combined with a longitudinal perspective, cognitive neuropsychology provides a method and framework for investigation and description of developmental change. Thomas and Karmiloff-Smith argue for the importance of development but use empirical data averaged across the life span (e.g. Thomas et al. 2001) and provide no protocol with which to describe and measure empirical change in performance. There is no theoretical account of the mental representations that are changing within their postulated dynamic processes and how these changes are to be quantified. Hence, from an empirical perspective their model remains untestable.

References

Ahmed, A. & Dutton, G.N. (1996) Cognitive visual dysfunction in a child with cerebral damage. *Developmental Medicine & Child Neurology 38*: 736-743.

Brunsdon et al. 2002; Brunsdon R.K., Hannan T.J., Nickels L & Coltheart M. (2002) Successful treatment of sublexial reading deficits in a child with dyslexia of the mixed type. *Neuropsychological Rehabilitation 12*: 199-229.

Caramazza, A. (1984) The logic of neuropsychological research and the problem of patient classification in aphasia. *Brain & Language* 21: 9-20.

Cardell E.A. & Chenery H.J. (1999) A cognitive neuropsychological approach to the assessment and remediation of acquired dysgraphia. *Language Testing 16*: 353-388

Carey, S., & Diamond, R. (1977) From piecemeal to configurational representation of faces. *Science 195*: 312-314.

Castles, A., & Coltheart, M. (1996) Cognitive correlates of developmental surface dyslexia: A single case study. *Cognitive Neuropsychology 13*: 25-50.

Clahsen, H. & Almazan, M. (1998) Syntax and morphology in children with Williams syndrome. *Cognition 68*: 167-198.

Clahsen, H. & Almazan, M. (2001) Compounding and inflection in language impairment: Evidence from Williams Syndrome (and SLI). *Lingua 111*: 729-757.

Clahsen, H. & Temple, C. (2003) Words and rules in children with Williams Syndrome. In *Language competence across populations*, ed. Y. Levy & J. Schaeffer. Erlbaum.

Clahsen, H., Ring, M. & Temple, C. (2003) Lexical and morphological skills in English children with Williams Syndrome. In *Language competence and development in Williams Syndrome,* ed. S. Bartke & J. Siegmueller. Benjamins.

Coltheart, M., Rastle, K., Perry, C., Langdon, R. & Ziegler, J. (2001) DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychological Review 108*: 204-256.

Deruelle, C., Mancini, J., Livet, M., Casse-Perot, C., & de Schonen, S. (1999) Configural and local processing of faces in children with Williams Syndrome. *Brain & Cognition 41*: 276-298.

Ellis, A.W. & Young, A. (1988) *Human cognitive neuropsychology*. Erlbaum.

Frith, U. (1985) Beneath the surface of developmental dyslexia. In *Surface dyslexia*, ed. K.E. Patterson, J.C. Marshall & M. Coltheart. Erlbaum.

Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992) Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain 115*: 1783-1806.

Karmiloff-Smith, A. (1997) Crucial differences between developmental cognitive neuroscience and adult neuropsychology. *Developmental Neuropsychology 13*: 513-524.

McCloskey, M., Rapp, B, Yantis, S. et al. (1995) A developmental deficit in localising objects from vision. *Psychological Science 6*:112-117.

Plaut, D. C., McClelland, J. L., Seidenberg, M. S., & Patterson, K. E. (1996) Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review 103*:56-115.

Rice, M. (1999) Specific grammatical limitations in children with Specific Language Impairment. In *Neurodevelopmental disorders*, ed. H. Tager-Flusberg. MIT Press.

Rothweiler, M. & Clahsen, H. (1994). Dissociations in SLI children's inflectional systems: A study of participle inflection and subject-verb agreement. *Journal of Logopedics & Phoniatrics 18*: 169-179.

Saffran, E. M. (1982) Neuropsychological approaches to the study of language. *British Journal of Psychology 73*:317-337

Sokol, S. M., Macaruso, P. & Gollan, T. H. (1994) Developmental dyscalculia and cognitive neuropsychology. *Developmental Neuropsychology 10*: 413-441.

Temple, C. M. (1991) Procedural dyscalculia and number fact dyscalculia: Double dissociation in developmental dyscalculia. *Cognitive Neuropsychology 8*: 155-176.

Temple, C. M. (1992) Developmental memory impairment: faces and patterns. In *Mental lives: cased studies in cognition*, ed. R. Campbell. Blackwell.

Temple, C. M. (1994) The cognitive neuropsychology of the developmental dyscalculias. *Cahiers de Psychologie Cognitive/ Current Psychology of Cognition 133*: 351-370.

Temple, C. M. (1997) *Developmental cognitive neuropsychology*. Psychology Press.

Thomas, M.S.C., Grant, J., Barham, Z., Gsödl, M., Laing, E., Lakusta, L. Tyler, L.K., Grice, S., Paterson, S. & Karmiloff-Smith, A. (2001) Past tense formation in Williams syndrome. *Language & Cognitive Processes 16*: 143-176.