



Figure 2 (Briscoe).

T&K-S are critical of assumptions of “Residual Normality,” that is, that “in the face of a selective developmental deficit . . . the rest of the system can develop normally” (sect. 3.1) and that Residual Normality is an assumption regarding “how development takes place.” A closer look at children with SLI shows that they are not selected directly from an underlying genotype. Instead, selectivity of language impairment occurs despite appropriate marking of developmental milestones, that is, “in the context of normal development” (Bishop 1997b, p. 21). Residual Normality is, under these circumstances, a rational application of a species-specific developmental pattern, not a misguided assumption from cognitive models. The problem of tying a cognitive framework to developmental disorders is really the problem of providing explanatory power at multiple levels of analysis, not the use of cognitive models per se. Ultimately, the truth behind the process of development lies with exceptional children whose abilities defy explanation by simplistic models and challenge cognitive psychology to provide answers.

Raising the bar for connectionist modeling of cognitive developmental disorders

Morten H. Christiansen,^a Christopher M. Conway,^a and Michelle R. Ellefson^b

^aDepartment of Psychology, Cornell University, Ithaca, NY 14853;

^bDepartment of Psychology, University of Warwick, Coventry CV4 7AL, United Kingdom. mhc27@cornell.edu cmc82@cornell.edu

M.Ellefson@warwick.ac.uk

<http://cni.psych.cornell.edu/people/chris.html>

<http://www.psych.cornell.edu/people/Faculty/mhc27.html>

<http://www.warwick.ac.uk/fac/sci/psychology/staff/research.html#ME>

Abstract: Cognitive developmental disorders cannot be properly understood without due attention to the developmental process, and we commend the authors’ simulations in this regard. We note the contribution of these simulations to the nascent field of connectionist modeling of developmental disorders and outline a set of criteria for assessing individual models in the hope of furthering future modeling efforts.

Thomas & Karmiloff-Smith (T&K-S) make an important theoretical contribution to our understanding of cognitive developmen-

tal disorders. We find their arguments regarding the problems associated with the assumption of Residual Normality very compelling. In particular, we agree that the developmental process must be taken into account when considering the possible causes of cognitive developmental disorders.

Connectionist modeling plays a crucial role in the arguments put forward by T&K-S. As highlighted in the target article, connectionists have recently begun to model various developmental disorders, including dyslexia (Brown 1997; Brown & Loosemore 1995; Harm & Seidenberg 1999), autism (Cohen 1998; O’Laughlin & Thagard 2000), selective language impairment (Hoeffner & McClelland 1993), Williams syndrome (WS) (Thomas & Karmiloff-Smith, in press), mental retardation (Bray et al. 1997), and schizophrenia (Hoffman & McGlashan 1997). However, in a recent review of connectionist modeling of cognitive developmental disorders, we found that most models suffer from a variety of shortcomings (Conway et al., in preparation). In this regard, we believe that the simulation approach taken by T&K-S provides a positive step forward. Using their simulations as a starting point, we discuss how connectionist modeling of cognitive developmental disorders may be improved further.

When modeling cognitive developmental disorders, one of the critical questions concerns how a particular deficit should be implemented. That is, how should the disordered startstate differ from the “normal” case? T&K-S explored three different ways of manipulating the startstate of their networks: removal of weights (lesioning), addition of noise, and changes in unit discriminability. They found that different types of startstate damage can result in very similar patterns of endstate performance. This is an important result from the viewpoint of connectionist modeling of cognitive developmental disorders. Many connectionists typically report only a single type of startstate damage (though they may have investigated others). But if a particular startstate manipulation is to have theoretical significance in terms of explaining a developmental deficit, it is crucial to establish that *this* manipulation, and no other, is the right causal factor. We therefore think that it is imperative to follow the T&K-S example and explore several types of startstate damage.

Of course, there is more to the modeling of developmental disorders than manipulating the startstate. It is also important that the model captures relevant aspects of behavior given reasonable input and a plausible learning task. Although existence-proof

models are crucial to establish the feasibility of a particular psychological modeling approach, the long-term success of any such approach requires close and substantial coverage of empirical data (Christiansen & Chater 2001). To further the modeling of cognitive developmental disorders, we have proposed four criteria to assess the strengths and weaknesses of individual models: deficit implementation, data contact, task veridicality, and input representativeness (Conway et al., in preparation). We see the T&K-S simulations as being on a par with existence-proof models and will instead discuss our criteria in the context of the related model of past-tense formation in WS patients (Thomas & Karmiloff-Smith, in press) described in T&K-S.¹

The first criterion, *deficit implementation*, refers to how well the manipulations used to simulate a particular deficit are motivated by theoretical and/or empirical research. Thomas and Karmiloff-Smith (in press) implemented several different types of deficits corresponding to different theoretical views of the underlying cause. They found that both manipulations to phonology and the integration of phonology and lexical-semantics allowed for the simulation of the appropriate pattern of WS past-tense formation, suggesting a good, theoretically informed deficit implementation.

Our second criterion, *data contact*, assesses how well a model provides a fit with the relevant psychological data. We further distinguish between primary data contact, which refers to contact with data from specific experiments, and secondary contact, referring to contact with general trends of a population. An example of the former is a model that directly simulates dependent measures from psychological experiments, such as reaction time; an example of the latter is a model that recreates the general trend that children with mental retardation perform worse on a memory task than normal children. With startstate damage to either phonology or the integration of phonology and lexical-semantics, Thomas and Karmiloff-Smith (in press) found close contact with primary human data.

The third criterion, *task veridicality*, is aimed at the extent to which the learning task given to the model is realistic relative to what human participants may face. We further differentiate training-task veridicality – which is the degree to which the network training task maps onto what the target population faces – from test-task veridicality – the degree to which the network test task maps onto what participants do in the actual human experiment(s). In the case of the Thomas and Karmiloff-Smith (in press) study, test-task veridicality is good because the networks are faced with a close approximation of the human test task. Although the stem-to-past-tense training task is typical in connectionist modeling of past-tense morphology, it is arguably not the primary way in which children acquire their inflectional skills,² leading to a relatively poor training-task veridicality.

Our final criterion, *input representativeness*, refers to the degree to which the information provided to a model matches the input available to a child. Although we realize that it is often necessary for practical reasons to simplify the input, models should be made as realistic as possible. The phonological input to the Thomas and Karmiloff-Smith (in press) models is based on an artificial language of 500 triphonemic verb stems taken from Plunkett and Marchman (1991). The lexical-semantic input came in different types of nonreferential formats (both localist and distributed). Overall, input representativeness is low, but both the phonological and lexical-semantic representations are not far from the state of the art in current connectionist past-tense modeling.

In conclusion, we see the T&K-S simulations as providing a valuable contribution to the field of connectionist modeling of developmental cognitive disorders. On a theoretical level, their simulations illuminate the problematic aspects of the Residual Normality assumption. On a connectionist modeling level, the simulations have pointed to the importance of exploring several deficit implementations. Although this emerging field of connectionist modeling is very much in its infancy, the simulations presented by T&K-S here and in Thomas and Karmiloff-Smith (in press) highlight the promise that it holds. We hope that the crite-

ria we have outlined here may additionally help raise the bar for connectionist modeling of developmental cognitive disorders.

NOTES

1. Briefly, in this model the phonological pattern of a verb stem was mapped onto a phonological form of its past tense through a set of hidden units – in some versions of the model, lexical-semantic information was additionally provided as input.

2. The version of the model in which both phonological and lexical-semantic information is mapped onto a past-tense form may be less prone to this criticism.

The residual normality assumption and models of cognition in schizophrenia

Ruth Condray and Stuart R. Steinhauer

Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213.

condrayr@msx.upmc.edu sthauer@pitt.edu

Abstract: Thomas & Karmiloff-Smith's (T&K-S's) argument that the Residual Normality assumption is not valid for developmental disorders has implications for models of cognition in schizophrenia, a disorder that may involve a neurodevelopmental pathogenesis. A limiting factor for such theories is the lack of understanding about the nature of the cognitive system (modular components versus global processes). Moreover, it is unclear how the proposal that modularization emerges from developmental processes would change that fundamental question.

In their target article, Thomas & Karmiloff-Smith (T&K-S) make several important arguments. The primary contribution is their critical evaluation of the assumption that atypical development can produce selective cognitive deficits while the rest of the system develops normally (the Residual Normality assumption). Additionally valuable is their illustration that behavioral outcomes may be influenced jointly by the types of task and central nervous system (CNS) damage, which in turn may be further qualified by the developmental period at the time of CNS damage (before vs. after training).

We will focus our comments on the relevance of T&K-S's challenge for current theories of cognition in schizophrenia. There are two important points of contact between these two areas of research. The first is the modular versus global distinction made regarding cognition in both sets of discussions; the second concerns the assumption of a developmental etiology. Schizophrenia is a severe psychiatric disorder that affects approximately one percent of the general population, includes cognitive dysfunction as an important clinical characteristic, and may involve a neurodevelopmental pathogenesis. Adult schizophrenia patients exhibit performance decrements on a wide range of cognitive tasks. However, their reduced performance differs significantly from that of non-clinical controls on only some, not all, tasks.

A current trend is to view this overall pattern in a polarized fashion, which commonly takes the following form: Do patients exhibit reduced performance on X, Y, Z tasks because of compromises to specific cognitive components, or is their suboptimal performance merely due to an overall reduction in cognitive ability that is manifested differently as a function of task difficulty? Most writers agree that an accurate partitioning of the relative contributions of global and specialized dysfunction will advance our understanding about cognition in schizophrenia. Writers differ, however, about what constitutes acceptable theoretical and methodological approaches, with much of the disagreement concerning how task difficulty is viewed; as a process-oriented variable or as a nuisance variable requiring psychometric solutions (see Chapman & Chapman 2001; Knight & Silverstein 2001; Strauss 2001). We suggest that T&K-S's proposed framework, in which task is an independent factor that may interact with etiology and developmental phase, represents a more interesting formulation, from a cognitive