
Title: The Residual Normality Assumption and Models of Cognition in Schizophrenia
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Abstract: Thomas and Karmiloff-Smith’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 and Karmiloff-Smith 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 CNS damage, which, in turn, may be further qualified by developmental period at the time of CNS damage (prior to versus after training).

We will focus our comments on the relevance of Thomas and Karmiloff-Smith’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 1% 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 sub-optimal 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 Thomas and Karmiloff-Smith’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 perspective, and may supply a more informative
approach for examining the nature of cognitive dysfunction in schizophrenia.

Thomas and Karmiloff-Smith correctly emphasize that a major difficulty for the Residual
Normality assumption is the current lack of understanding about which aspects of the cognitive
system are global and which are modular (for a recent discussion of this general issue, see Fodor,
2000). Data from our studies (Condray, Steinhauer, van Kammen, & Kasparek, in press)
illustrate the importance of such uncertainty for models of cognition in endstate schizophrenia.
Accuracy on a grammatical parsing task (Who did What to Whom?) was reduced for
schizophrenia patients, compared to non-clinical controls. More important, grammatical parsing
accuracy was correlated with measures of general intelligence and semantic knowledge for
healthy controls, but these factors were not correlated for schizophrenia patients. Why the
coupling of grammatical parsing accuracy with general intelligence and semantic knowledge in
the healthy non-patient participants? Why the disconnection in the patient group? It is possible
that the disconnection in patients’ responding merely underscores the relative independence
(modularity) of at least some portion of the grammar parsing system (e.g., Chomskian
movement). And, while it could be argued that our non-patient control data (association of
intelligence, semantics, and grammar) are problematic for such an independence account of
parsing, it is also possible that controls’ performance merely reflects the smooth
interconnectivity of systems that work optimally. Minimally, grammatical parsing appeared to
be connected with controls’ global cognitive processes; patients’ grammatical parsing appeared
more local (compartmentalized). Thus, patients were able to perform the task, albeit less
accurately, but they may have accomplished that performance in a different way. The source of
this grammatical parsing problem is unknown; patients’ compromised function could be due to
problems with that grammar (sub-)system, other cognitive systems, the connective linkages
(interfaces) between cognitive systems, or the combined effects of some or all of the above.
Finally, fundamental to the position of Thomas and Karmiloff-Smith is the possibility that
damage to cognitive systems during childhood may produce different developmental trajectories,
which could affect how such systems are organized and function during adulthood. It is unclear,
however, whether Karmiloff-Smith’s theory of emergent modularization (specialized
components that emerge through developmental processes) would necessarily change the central
question concerning which cognitive functions are modular and which are global. Thus, while it
is possible that atypical development could transform a module into something else (or vice
versa), such a possibility would not alter interest in compartmentalized versus global functions
and their inter-connectivity. If our data are representative for schizophrenia, then an important question concerns how modular and global functions may be interconnected (e.g., some type of binding mechanism) in this patient population.

The pathogenesis of adulthood schizophrenia may originate in utero or during early childhood, and may affect brain development (Gooding and Iacono, 1995; Marenco and Weinberger, 2000). Some version of the scenario described by Thomas and Karmiloff-Smith may therefore apply to this disorder. The precise etiology is yet to be determined, however, with strong candidates including abnormalities of selected neural circuitry and neurochemistry function (Cohen & Serven-Schreiber, 1992; Weinberger, 1996); cytoarchitecture (Barbeau, Liang, Robitaille, Quirion, & Srivastava, 1995; Weinberger, 1996); and cell membrane structure and function (Horrobin, 1998). Thus, models of cognitive dysfunction in schizophrenia will require reconciliation with the mechanism(s) of pathogenesis eventually revealed. An etiology that involves a general mechanism (e.g., abnormality of cell membrane structure) might influence the cognitive system in a pervasive manner so that disruption occurs at multiple levels (e.g., specific, global, and binding functions). In contrast, an etiology involving more restricted circuitry (e.g., prefrontal cortex) might disrupt more specialized functions.

We believe the idea that types of cognitive task and CNS compromise should be considered within the context of developmental phase is important. We wish to emphasize, however, that questions about modular function and interconnectivity remain of interest despite the hypothesized alterations in developmental trajectory.

References


