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studies has now established cases that show a very selective reading deficit parallel in nature to acquired dyslexias (Stuart & Howard 1995; Temple 1984; 1997; Temple & Marshall 1983).

Peripheral dyslexias, which have been less widely explored with respect to developmental origin, also show modular impairments in developmental forms. Letter position dyslexia (LPD), a selective deficit in letter position encoding with unimpaired letter identification, was initially reported in acquired cases (Friedmann & Gvion 2001). This dyslexia has now been identified in 12 Hebrew-speaking children and adolescents whose reading patterns show striking similarities to acquired LPD (Friedmann & Gvion 2002; Friedmann & Rahamim 2002; Precel & Friedmann 2002).

Both acquired and developmental LPD manifest modular impairment, as only one submodule of the visual analysis system, letter position encoding, is impaired, whereas another submodule, letter identification, is unimpaired. Both in acquired and in developmental LPD, individuals show the dissociation between making predominantly letter migration errors within words (reading "bread" for "beard") and very few or no letter substitutions in a wide variety of tasks. In reading aloud, individuals with acquired LPD had 21% letter-order errors and only 1% letter substitutions; individuals with developmental LPD had 15% letter-order errors and 1% letter substitutions. In a same-different task, individuals in both groups could detect differences in letter identity between words but failed to detect letter-order differences: The individuals with acquired LPD made 48% errors in letter order but only 7% errors in letter identification; the 12 individuals with developmental LPD had 37% versus 4% errors.

Moreover, exactly the same pattern of migration errors occurs in acquired and developmental LPD. In both cases the migration errors occur almost exclusively in medial-letter positions. In word reading and definition tasks, individuals with acquired LPD made 16% medial-letter position errors, compared to 0.01% exterior-letter position errors. Similarly, the individuals with developmental LPD had 15.1% medial errors and 0.8% exterior errors.

However, many of the individuals with developmental dyslexias show errors in addition to the errors that characterize their reading deficits. We argue that these stem from their incomplete orthographic-lexical knowledge because of incomplete and flawed input to the lexicon and lack of sufficient exposure to written words and texts that is only a side effect of their reading impairment. For example, the individuals with developmental LPD also showed, in addition to letter migrations, 5.7% errors in reading aloud that resulted from insufficient lexical-orthographic knowledge: They made errors on heterographic homophones (or heterophones with homophonic letters), which can be read correctly only after being lexicalized, as well as regularization errors and errors of the vocalic pattern of unvoweled words (in Hebrew, vowels are underrepresented, and so lexical knowledge is required for reading). The same was true for the Hebrew-speaking neglect-dyslexic child in Friedmann and Nachman-Katz (in press), who made neglect errors in 50% of the words. He too had homophone and vocalic-pattern errors that are not characteristic of the reading of individuals with acquired neglect dyslexia. These error types were not found in the reading of the individuals with acquired LPD in Friedmann and Gvion (2001) or in acquired neglect dyslexia, probably because the onset of the dyslexia in the acquired cases followed reading acquisition and the loading of the orthographic input lexicon, whereas the developmental or early-onset cases did not have the chance to fill up their orthographic lexicon.

To conclude, selective impairments that indicate the modular nature of developmental disorders are reported both in SLI and in peripheral dyslexias. Additional errors can result from the lack of lexical knowledge rather than lack of Residual Normality.

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Nativism, neuroconstructivism, and developmental disorder

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Abstract: Either genetically specified modular cognitive architecture for syntactic processing does not exist (neuroconstructivism), or there is a module but its development is so abnormal in Williams syndrome (WS) that no conclusion can be drawn about its normal architecture (moderate nativism). Radical nativism, which holds that WS is a case of intact syntax, is untenable. Specific Language Impairment and WS create a dilemma that radical nativism cannot accommodate.

Nativists about syntax argue that syntax (1) is innate; (2) depends on rule-based processing; (3) is modularised; *and* (4) Williams syndrome (WS) and Specific Language Impairment (SLI) are a double dissociation (DD) which identifies a syntax module damaged in SLI and "spared" in WS. Thus, Pinker explains the presence of inflectional morphology in WS as follows:

> Their grammar is running smoothly but their word-fetcher doesn't have the usual bias to fetch frequent and appropriate words quickly. Irregular verbs survive on that basis, so occasionally an irregular form doesn't survive quickly enough and the rule is ready and waiting to step in. (Pinker 1999, p. 262)

The word-fetcher is a memory system whose functioning is statistical, not rule-based (hence the salience of irregulars), whereas the grammar module, presumed intact in WS, is a rule-based system. Nativists model the interaction of these two systems in a dual-route neural network which stores and retrieves stems and regular and irregular suffixes. If the word-fetcher cannot find an irregular suffix, the default regularisation mechanism automatically produces the regular construction.

Karmiloff-Smith and neuroconstructivist collaborators have undermined point 4 by producing a model that captures the linguistic performance of SLI and WS subjects in a single-route network whose functioning is statistical. The network approximates the linguistic performance of SLI if the noise/signal ratio in phonological input is increased and WS if the pattern is reversed and the signal/noise ratio is increased.

These findings have different consequences for different forms of nativism. They strongly undermine a reading of point 4 which depends on Residual Normality (RN). Heather van der Lely, for example, has argued that in SLI subjects have "*normal cognitive and auditory abilities* alongside impaired grammatical abilities" (van der Lely 1999, p. 286).

Thomas & Karmiloff-Smith (T&K-S) point out, however, that performing *within the normal range on standard tests* is not sufficient to justify the RN hypothesis of spared or intact function. In the case of phonological processing, fine-grained testing can disclose subtle deficits with major developmental consequences. For example, in the extraction of a signal from a variable acoustic stream, temporal interval as well as amplitude can affect performance. Hence, a standard hearing test may not detect subtle deficits in the ability to overcome the masking effects of the surrounding stream. One explanation of dyslexia is that it is primarily a result of such basic processing deficits rather than a problem with higher-level linguistic processing (Nagarajan et al. 1999; Wright et al. 1997a; 1997b). Normal can thus mean "test within the normal range," or (the RN hypothesis) "processed by the same mechanism(s) in the same way as normal subjects." It is this sense of normality as RN which is required by van der Lely.

A nativist cannot accept T&K-S's data and abandon RN without abandoning the hypothesis that WS and SLI are a DD which identifies a syntax module. A nativist might initially argue that SLI is a consequence of impairment to the syntax module, but then they face a dilemma regarding WS. Is the module intact in WS? If it is, why do language and syntax have an unusual developmental tra-

jectory in WS? (Donnai & Karmiloff-Smith 2000; Laing et al. 2002). Possibly, increased sensitivity to phonology overrides the default mechanism for regularisation. So a WS subject's syntax module develops abnormally because the syntax module is crowded out. However, if this is the case, RN must be abandoned. Points 1 to 3 can be maintained, but point 4 cannot.

The nativist who abandons point 4 might then argue that abnormal syntax in WS is the result of absence of the syntax module. But in that case, WS should resemble SLI, when in fact they present initially as a DD. So the best nativist hypothesis is to abandon point 4 and retain the idea that what we see in WS is the anomalous development rather than absence of a syntax module. But distinguishing between these two hypotheses on the grounds of performance alone seems very difficult.

On either story – and the former is the most plausible – it seems that a nativist must eventually agree that apparent linguistic fluency in WS is essentially due to nonsyntactic factors, and hence that whatever the fate of nativism about syntax, the hypothesis of RN for SLI or WS is unsustainable.

The radical neuroconstructivist conclusion is, of course, that nativist modular hypotheses about high-level cognitive processing such as syntax or theory of mind are mistaken. The appearance of such modularity is an artefact of interactive development of general cognition and low-level processing modules (in this case, phonology; in the theory-of-mind case, perhaps functions such as recognition of emotional and facial expression and intentional movement) (Gerrans 2002; 2003; Gerrans & McGeer, 2003).

The nice thing about T&K-S's article is that it shows that the resolution of the issue requires a theory that models the neural implementation of computational properties considered essential to processing the domain in question. A simple inference from performance to cognitive architecture of high-level, abstract cognition is not licensed by the data.

Distinguishing proximal from distal causes is useful and compatible with accounts of compensatory processing in developmental disorders of cognition

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Abstract: Models of the architecture of mature cognitive systems can inform the study of normal and disordered cognitive development, if one distinguishes between proximal and distal causes of performance. The assumption of residual normality need not be made in order to apply adult models to performance early in development, because these models can be modified to reflect the results of compensatory processing.

Thomas & Karmiloff-Smith (T&K-S) agreed with us (Jackson & Coltheart 2001; hereafter J&C) that one can distinguish proximal causes of abnormal performance (explanations in terms of the current architecture of a cognitive system) from distal causes (a category in which we include everything else, including developmental history). J&C argued that this proximal-distal distinction permits models of the architecture of mature cognitive systems to inform the study of normal and disordered cognitive development. We did not assert that adult models should be *assumed* adequate to describe children's behavior or used to restrict the search for differences between developmental and acquired impairments. However, we remain convinced that models of mature performance are useful starting points for describing both typical and atypical performance during the development of skills such as reading. For example, if T&K-S had not been familiar with dual-route models of skilled performance, would they have attempted to falsify a hypothesis of similarity between early- and late-damaged systems?

We have *not* assumed that those components in an abnormally developing system that are not directly implicated in abnormal performance will show Residual Normality (RN). On the contrary, we explicitly disavowed the assumption of RN in developing systems. Indeed, rather than arguing for RN, we proposed that: "[i]n a developing system, single deficits might be more likely to have broad implications than is the case when a previously intact system has been damaged" (J&C, p. 152), and went on to speculate about ways in which impaired development of either a lexical or nonlexical route might cause problems throughout a child's developing reading system:

> If each kind of deficit impedes development, we should not be surprised to find that children who are atypical at exception-word reading often are atypical at pseudoword reading, and vice versa, even though these two tasks depend on different parts of the reading system. If what we have proposed about how learning based on each route's operations contributes to the development of the other route is true, we would expect deficits in both types of reading to be the most common, but not the invariable pattern. Indeed this is what has been found. (J&C, p. 193)

What, then, of T&K-S's claim that inferences from observed behavior to underlying cognitive structure must be conditional on "developmental constraints under which processing structures have emerged" (sect. 10, para. 4)? Given the likelihood of compensatory processing in early-damaged systems, how can models of skilled cognition be useful for understanding developmental disorders?

T&K-S assumed that static models cannot deal with phenomena such as compensatory processing. They argued that if RN does not hold, then behaviors that appear similarly intact in acquired and developmental disorders could reflect operation of an intact processing module in the acquired case but *qualitatively different* compensatory processing in the developmental case. But what constitutes a qualitative difference? Is compensatory processing at a particular point in development really beyond the scope of nondevelopmental models such as the Dual Route Cascaded Model of Word Recognition and Reading Aloud, or DRC (Coltheart et al. 2001)? We counter that it is not and that *quantitative* changes in how the parameters of such a model are set can alter its function in a way that might be called strategic and compensatory.

For example, Rastle and Coltheart (1999; see also J&C) showed that the DRC model can simulate strategic effects that might occur if a reader expected to read only nonwords. This is not a developmental scenario; but such tuning of system parameters is analogous to what might happen if a child's prior experience in using either the lexical or the nonlexical route of a modular system like DRC had been repeatedly unsuccessful. J&C also sketched how the DRC might be modified to simulate the effects of the restricted orthographic lexicon and incomplete knowledge of grapheme-phoneme correspondence rules that characterize normal beginning readers (J&C, Fig. 5.1). The extent to which a modified version of a static model such as the DRC might account for observed reading performance at different points in development is an empirical question that has already been answered in the affirmative in some instances (Coltheart et al. 2001, Fig. 15).

Static models such as DRC say nothing about processes by which reading systems change over time as children develop reading ability. Nonetheless, such models can offer successive snapshots of what the system might look like, for normal and impaired readers, as reading develops. However, given that static models do not seek to explain processes of development, why do we still prefer such models to apparently more comprehensive and undoubtedly more dynamic connectionist models? To answer this question, we return to the distinction between proximal and distal cause.

Distal causes that have been proposed for developmental reading failure are diverse, ranging from genetic anomalies to restric-