sider "local phase synchrony" in the neural activity of particular regions of the brain, there is also evidence of "large-scale synchronization" among neural assemblies in different regions of the brain (the analogy used is of several computers linked through a program like Napster over the World Wide Web). This program, I think, adds ammunition to the claims made by T&K-S against a simple modular view of brain development, but also challenges them to find ways to integrate first-person experiences and aims into their account of atypical development.

There may also be critical "turning points" that orient how biological development will unfold in cases of genetic abnormalities, just as there are turning points in personal autobiography (Bruner 2001). This is especially plausible when one considers that autobiography in general is initially coconstructed with parents (Nelson 1993) and so is not divorced from the cultural context in which such constructions take place. This coconstruction is just as true in cases of abnormal development, as we have shown in a detailed biographical study of an autistic child (Vuletic & Ferrari, in press). Our study shows that how a particular autistic child develops is deeply influenced by how his parents conceive of his life and his possible development and not by neurobiological considerations alone. Of course, parents must adapt to the reality of how their child is acting and experiencing the world; but that adaptation can range from making every attempt to assure a self-sufficient and happy life for their child, as these parents and grandparents do, to despair at making any positive impact on their developing child, as many doctors schooled in the modular view of fixed potential sadly recommend.

This leads to the second point of this commentary. Psychological development itself cannot be understood as a uniquely individual thing involving only an individual's brain and how that brain interacts with the world. Development depends crucially on the sociocultural context in which (normal and abnormal) children develop. In cases of infant attachment, for example, an infant's inner working model of his or her relationship to an adult caregiver is constructed from the history of his or her interactions with that caregiver, as both are integrated into a sociohistorical/cultural context (Bretherton 1996). According to Hacking (1998) and Morris (1998), cultural context is one of the factors that helps give a specific form to an illness or disability as it interacts with biological abnormality through development. In other words, while it is true that neurobiology is an important aspect of human development, it is equally true that how particular children are treated, and the general sociocultural conditions under which they develop (which extend to basic nutrition and medical care as well as to parental expectations about children's physical and social obligations as part of their community), will all have a huge impact on how children with genetic abnormalities develop (Keating & Miller 1999). Hacking (1998) shows how an entire type of mental illness disappeared when social conditions changed – even if trace elements of that illness remain and have been reclassified.

Both of these points are meant only to add to what is a very strong and important critique of the widespread notion of Residual Normality. I think that it is time to broaden our understanding of psychology beyond the limited neurobiological framework that many psychologists have already found too constricting for a natural science of psychology over a century ago.

Modularity in developmental disorders: Evidence from Specific Language Impairment and peripheral dyslexias

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Abstract: Evidence from various subtypes of Specific Language Impairment and developmental peripheral dyslexias is presented to support the idea that even developmental disorders can be modular. However, in developmental letter position dyslexia and neglect dyslexia we show that additional errors can occur because of insufficient orthographic-lexical knowledge.

One of the intriguing questions regarding disorders of language and reading is whether these disorders are modular. Thomas & Karmiloff-Smith (T&K-S) suggest that although acquired disorders might be modular, developmental disorders are not. Of course, there might be multifaceted cases of developmental aphasias and dyslexias, and many types of impairment can coexist in the same individual. Cases like these also exist in acquired aphasia and dyslexia. However, we believe that the main question is: Can modular impairments be found in cases prior to training? We believe there can. The structure of our argument will be as follows: first, we will demonstrate some evidence for modular impairments in developmental aphasia and dyslexia, focusing on their modular nature and the similarities between acquired and developmental forms of the same disorder, and then we will show, taking the reading domain as an example, that such a modular and selective impairment might cause a deficit in other reading abilities, stemming from the lack of exposure to written text and possibly from lack of well-formed input to the orthographic lexicon during reading acquisition.

Starting with developmental aphasia or Specific Language Impairment (SLI), it is possible to distinguish various types of impairments, differing, just as in acquired aphasia, in the linguistic module which was impaired. Friedmann and Novogrodsky (2003) reported 10 children with syntactic SLI (G-SLI) whose syntactic deficit manifests in chance-level performance on the comprehension of object relative sentences and referential object questions, and the inability to produce relative clauses and sentences that include verb movement; these children are unimpaired in naming and their phonological abilities are normal for their age, as tested by pseudoword repetition, nonword judgment and phonological awareness tasks.

A group of children with a severe naming disorder (stemming either from lexical-semantic or lexical-phonological deficit) was also identified, who show intact syntactic abilities, as manifest by their normal comprehension of relative clauses, normal production of relative clauses (except for paraphasias, which preserve the syntactic structure of the sentence), and normal production of sentences that include verb movement (Novogrodsky & Friedmann 2002). This dissociation indicates that it is possible to see linguistic modules such as syntactic movement, phonology, and lexical access selectively impaired even in developmental aphasias.

The same selective and modular nature is also manifest in dyslexia. Acquired dyslexias can appear in very selective forms, stemming from a circumscribed deficit in one of the processes or lexicons that participate in the reading process or in the connection between them (Ellis 1993; Ellis & Young 1996; Marshall & Newcombe 1973; Patterson 1981). This is the way modularity reflects in reading disorders. What about developmental dyslexias? Indeed, children and adults with dyslexia of early onset – before reading acquisition – can show a myriad of reading deficits and sometimes additional disorders such as attentional and phonological deficits. But does this mean that there is no modularity in developmental reading deficits? We believe it does not. A group of

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-