ably, there were no differences in the number of items and in overall frequency effects between the WS and the control group. However, WS subjects produced more repetitions ($p < 0.001$), more noncategory member items ($p < 0.05$), and more members ($p < 0.01$) that are not listed in the normative adult data (Kónya & Pintér 1986). A more fine-grained analysis of the latter difference reveals that items listed by the WS subjects that are not listed in the normative database are often not unusual or rare but are pet names for food or animals, examples of which are not found in the control sample, in which children are past the age at which typically developing children use such names.

So WS individuals do not differ from their controls matched on receptive vocabulary scores in the frequency effects governing retrieval and production of lexical items belonging to a semantic category. Repetitions and the answers not obeying category restrictions are in agreement with the conclusions of earlier studies (Jarrold et al. 2000; Johnson & Carey 1998; Temple et al. 2002) – that instead of the lack of frequency effects, it is atypical organization or underspecification of conceptual knowledge that can explain peculiarities of language use in Williams syndrome.

The issue of rules versus items – and the implied double dissociation – is certainly crucial to the overall debate between dual-system (Clahsen 1999; Pinker 1999) and single-system approaches to the development and mental representation of language. As Thomas and Karmiloff-Smith (2002) point out, the much-discussed observations that WS subjects show strong morphological overgeneralizations (Clahsen & Almazan 1998) can be reinterpreted in the frame of single-route systems.

> While individuals with WS do show poorer performance on exception verbs than regular verbs, this appears to be in step with their delayed performance, because younger children also find exception verbs harder than regular verbs. There is no *specific* deficit for irregular verbs. (Thomas & Karmiloff-Smith 2002, p. 17)

The Hungarian nominal allomorphy system is especially relevant to test the rule issue because of the fact that there are subclasses of different generality, and saturation and regularity and frequency are much less tied there than in the English past tense (see Lukács & Pléh 1999). Earlier we reported data on a group of Hungarian WS subjects showing a rather strong tendency to overgeneralize, thus supporting a crude version of the dual-route system (Lukács et al. 2001). Recently, more elaborate analyses were performed on the same data set from the impaired group with a set of more properly matched controls. Table 2 shows correct percentages in an elicited morphological suffixation task in which subjects had to provide accusatives and plurals to nouns varying in both regularity and frequency.

The superiority of performance on regulars over irregulars was observed in WS ($p < 0.05$), whereas frequency effects were not significant. VCs seemed to follow a similar pattern in that they also had more errors on irregulars, but in their case overregularization of irregulars was clearly related to frequency. This may be interpreted as evidence for a slower language development in WS, as suggested by Thomas and colleagues (Thomas et al. 2001). However, there are still some signs to the effect that in WS subjects even frequent irregular items lead to overgeneralizations, and they seem to be delayed on regulars as well, arguing against a clear case of a selective impairment with irregulars.

Table 2 (Pléh et al.). *Correct performance of different groups with noun suffixation*

Noun type	Williams syndrome	Age control	Vocabulary control
Regular, frequent	89	100	99
Regular, rare	85	100	99
Irregular, frequent	74	100	92
Irregular, rare	66	99	64

Therefore, taken together our data on Hungarian WS subjects suggest that frequency does operate as a regulatory factor weighting connections in the memory system of this impaired population. In general, they seem to have relative difficulty stabilizing items in their memory, which shows up in different lexical tasks. But frequency does not disappear as a factor, it merely has a harder time. At the same time these subjects seem to have a looser semantic organization and a peculiar tendency to overgeneralize with irregulars, but they also err on regulars. These facts imply some peculiarities in their language representation, though not necessarily a two-process theory.

ACKNOWLEDGMENTS

Financial support for most of the research discussed in the commentary was provided by an OTKA grant (Hungarian National Science Foundation) T 029514 and an NKFP grant (5/0079/2002) to Csaba Pléh and by an NSF grant No. BCS01226151 to Ilona Kovács with Csaba Pléh as co-principal investigator. The empirical worked referred to was made possible by the Hungarian Williams Syndrome Association.

Evidence for a domain-specific deficit in developmental dyslexia

Franck Ramus

Laboratoire de Sciences Cognitives et Psycholinguistique (EHESS/ENS/CNRS), 75006 Paris, France. **ramus@lscp.ehess.fr**
http://www.lscp.net/persons/ramus/

Abstract: Thomas & Karmiloff-Smith (T&K-S) claim that "Residual Normality" is a priori unlikely, that is, that specific cognitive deficits should not exist in developmental disorders. Here I review evidence that a specific cognitive deficit is at the core of developmental dyslexia and I provide a possible neurological account thereof.

Thomas & Karmiloff-Smith's (T&K-S's) argument against Residual Normality (RN) is based on (1) ideological preconceptions concerning the development of cognitive modules; and (2) neural network simulations that suggest that RN is unlikely to occur. However, as they note, whether RN exists or not is an empirical issue, not one that will be solved by theoretical considerations or computer simulations. I propose to examine empirical data from developmental dyslexia. As this suggests that RN does sometimes occur, it follows that T&K-S's ideological preconceptions are mistaken and that their neural network simulations do not adequately model brain and cognitive development.

Developmental dyslexia is traditionally viewed as a specific developmental disorder: One that initially affects exclusively phonological representations and processes, with consequences for the acquisition of grapheme/phoneme correspondences and reading processes (Snowling 2000; Vellutino 1979). Because all other cognitive components seem entirely unaffected, dyslexia is usually seen as a case of RN.[1] However, a number of researchers point out that dyslexics' difficulties are not restricted to phonology and reading but include auditory (Tallal 1980), visual (Lovegrove et al. 1980), tactile (Stoodley et al. 2000), motor (Fawcett et al. 1996), learning (Nicolson & Fawcett 2000), and attention deficits (Hari & Renvall 2001). It is even suggested that dyslexia is a general sensorimotor syndrome (Stein & Walsh 1997). Obviously, all these findings undermine the claim for RN. The difficulty here is in seeing the forest for the trees.

Evidence for sensorimotor dysfunction in dyslexia comes from many studies reporting significant group differences between dyslexics and controls in sensory or motor tasks. However, when individual data is examined, it invariably shows that there is a large overlap in performance between the two populations and that the group effect is carried by a subgroup of dyslexics performing ab-

normally (for a review, see Ramus 2003). This could simply reflect measurement error and task reliability. But many of the results reported in recent studies rely on several measures of the same psychophysical threshold in each individual, obtained using adaptive procedures. There are therefore good reasons to believe in individual data points. Hence, on any given sensory or motor task, a good proportion of dyslexics seems to have intact performance. This contrasts with the quasi-universality of the phonological deficit.

It could be that any single low-level deficit fails to account for all dyslexics, but that, taken together, they do: Perhaps one third of dyslexics have an auditory-based phonological deficit, one third have a visual deficit, and the rest have a cerebellar learning disorder; all of these would lead to reading difficulties. My collaborators and I have addressed this possibility by administering a comprehensive 10-hour test battery of psychometric, phonological, auditory, visual, and motor skills to 16 adult dyslexics and 16 controls. We found that all 16 dyslexics had a phonological deficit, 10 had an auditory deficit, 4 had a motor dysfunction, and 2 had visual problems (with partial overlap between the sensorimotor deficits). Most interestingly, 5 dyslexics seemed to have a pure phonological deficit, as their performance on the whole battery of auditory, visual, and motor/cerebellar tests was entirely normal (Ramus et al. 2003) – a clear demonstration of RN. These results are not an artifact of data collection cut short prematurely; on the contrary, we have adopted all the main state-of-the-art tasks used to reveal sensorimotor deficits. Yet, some dyslexics seem entirely untouched by these low-level deficits. Similar results have been obtained with dyslexic children (Kronbichler et al. 2002; White et al., submitted). Moreover, it is becoming increasingly clear that the auditory deficits observed in certain dyslexics have little causal relationship to the phonological deficit (Bishop et al. 1999a; see review by Ramus 2003).

Consistent with the currently known data, my view of dyslexia is that of a specific phonological deficit sometimes accompanied by a sensorimotor syndrome with variable manifestations and little additional impact on phonology and reading. According to T&K-S, such a disorder makes no neurological and developmental sense. Indeed, as the authors note, "Karmiloff-Smith and colleagues have argued that a priori, the effects of genetic abnormalities are likely to be widespread throughout the brain and unlikely to be isolated to single high-level cognitive modules" (target article, sect. 3.3). Yet I propose that there is a plausible neurodevelopmental model for this view of dyslexia.

Two types of brain anomalies that make cognitive sense have been observed in dyslexics' brains: (1) cortical layer ectopias and microgyri (focal anomalies of neural migration in the outer layer of the cortex) are located mainly in left perisylvian areas and can be seen as underlying the phonological deficit (Galaburda et al. 1985; Humphreys et al. 1990); (2) the magnocells of the lateral and medial geniculate nuclei of the thalamus are disordered, which is thought to underlie the visual and auditory deficits, respectively (Galaburda et al. 1994; Livingstone et al. 1991). Interestingly, the thalamic anomalies seem secondary to the cortical ones, rather than the other way around. Moreover, they arise only under certain conditions. Indeed, based on animal models, it seems that the thalamic anomalies arise from the conjunction of cortical anomalies and certain hormonal conditions, notably excessive testosterone concentrations during brain development (Fitch et al. 1997; Herman et al. 1997; Rosen et al. 1999).

These neurological data naturally suggest a model of developmental dyslexia in which cortical anomalies occurring in early brain development and located in cortical areas important for the development of phonology and reading are the main underlying cause of the phonological and reading impairments. In certain individuals only (under certain hormonal conditions), the disruption spreads to the thalamus and engenders sensory deficits.[2] This is consistent with behavioral-genetic studies showing that dyslexics' phonological deficit is highly heritable, whereas their auditory and visual deficits are not (Bishop et al. 1999a; Davis et al. 2001; Olson & Datta 2002).

Interestingly, the early focal cortical anomalies documented above are quite similar to the focal lesions seen in adult neurology. Of course, there is no doubt that focal cortical damage may have very different effects in a developing brain and in an adult brain (as already pointed out by Frith 1986). But this is a clear example of how the effects of genetic abnormalities can be isolated to certain *anatomical* modules.[3] Furthermore, the dependence of the spread of the disruption on hormonal factors demonstrates that "malformation in the cortex is not always associated with plasticity changes in connectionally related areas" (Galaburda 2001, pp. 456–57). Nevertheless, in order to explain the dyslexia data, our model requires that certain *anatomical* modules are unique in their ability to support certain *cognitive* modules; if they are disrupted, no amount of "plasticity" or "compensation" will allow the brain to relocate the function to another area. This seems to be a necessary conclusion from the observation that dyslexics never fully recover from their specific congenital phonological deficit. Note that the functional uniqueness of certain brain areas does not necessarily imply the existence of a detailed genetic blueprint; it may simply reside in the combination of their connections to certain inputs and outputs together with their representational and computational properties.

In summary, developmental dyslexia, when not accompanied by the sensorimotor syndrome, seems to be a prime example of residual normality, and there is a plausible neurological model supporting this view. I submit that such empirical work and neuroscience-based modeling should weigh more than ideological preconceptions and computer simulations. In fact, the incapacity of T&K-S's neural network models to generate or accommodate the sort of neurodevelopmental scenario proposed only highlights the fact that these models are too simplistic even to begin to capture the complexity and sophistication of neural and cognitive development. It therefore seems unwise to rely on them to draw very general conclusions about the brain, development, and cognition.

ACKNOWLEDGMENTS

I am grateful to Gary Marcus, Uta Frith, and Sarah White for comments on this paper.

NOTES

1. Note that this is a completely different issue from that of the phonological versus surface subtypes advocated by Castles and Coltheart (1993) and discussed at length by T&K-S. It is now quite clear to most dyslexia researchers that the phonological and orthographic reading routes cannot really develop independently and, therefore, that selective damage to one of them is unlikely in developmental dyslexia (e.g., Zabell & Everatt 2002). I therefore generally agree with T&K-S's criticism of this hypothesis. However, this is orthogonal to the question of RN outside dyslexics' phonological and reading systems.

2. According to Stein and Walsh (1997), the motor part of the syndrome can be explained from there by the massive inputs from the magnocellular system via the posterior parietal cortex to the cerebellum.

3. It is well established from studies of autoimmune mice that cortical ectopias are of genetic origin (Sherman et al. 1990).

Are developmental disabilities the same in children and adults?

Paula Tallal

Center for Molecular and Behavioral Neuroscience, Rutgers University, Newark, NJ 07102. **Tallal@axon.rutgers.edu**

Abstract: Thomas & Karmiloff-Smith (T&K-S) raise an issue of considerable theoretical importance: Are developmental disorders like cases of adult brain damage? However, a related question: Are developmental disabilities the same in children and adults? is rarely addressed. Failure to consider the cumulative and differing effects of aberrant development across the life span confounds the current literature on both developmental dyslexia and Specific Language Impairment.

The role of rapid auditory processing (RAP) constraints in the origin of language development and disorders is a hotly debated is-