



Nativism and Neuroconstructivism in the Explanation of Williams Syndrome

PHILIP GERRANS

Philosophy Department

University of Adelaide

Adelaide SA 5005

Australia

E-mail: Philip.gerrans@adelaide.edu.au

Abstract. Nativists about syntactic processing have argued that linguistic processing, understood as the implementation of a rule-based computational architecture, is spared in Williams syndrome, (WMS) subjects – and hence that it provides evidence for a genetically specified language module. This argument is bolstered by treating Specific Language Impairments (SLI) and WMS as a developmental double dissociation which identifies a syntax module. Neuroconstructivists have argued that the cognitive deficits of a developmental disorder cannot be adequately distinguished using the standard gross behavioural tests of neuropsychology and that the linguistic abilities of the WMS subject can be equally well explained by a constructivist strategy of neural learning in the individual, with linguistic functions implemented in an associationist architecture. The neuroconstructivist interpretation of WMS undermines the hypothesis of a double dissociation between SLI and WMS, leaving unresolved the question of nativism about syntax. The apparent linguistic virtuosity of WMS subjects is an artefact of enhanced phonological processing, a fact which is easier to demonstrate via the associationist computational model embraced by neuroconstructivism.

Key words: nativism, neuroconstructivism, Williams syndrome specific language impairment

Introduction

Recent advances in molecular genetics have identified the genetic origin of a rare developmental disorder known as Williams syndrome (WMS), namely, a deletion of some 17 contiguous genes on the chromosome 7q11.23. WMS has also been the subject of a large body of experimental research designed to probe the behavioural and cognitive phenotype with the consequence, as Bellugi and Gallaburda put it, that

WMS provides a unique opportunity to study the brain in a condition where the genetic basis is understood and the cognitive profile is highly distinctive, and may represent a prototype cognitive disorder for the

study of the relationship between gene and behaviour (Gallaburda and Bellugi 2001: 123).

In effect WMS is an opportunity to evaluate nativist claims that certain cognitive traits are genetically-specified. We should note however that nativism about *cognitive* traits is nativism about computational architecture *implemented* by the brain, rather than about the brain itself. Hence the discovery, in the absence of a theory of the relation between neuroanatomy and cognition, of more detail about the link between genes and neuroanatomical structure would not advance our understanding of cognition. A mapping from gene to brain is not a full explanation of cognition without explanation of the way neuroanatomical structures, which express genes, implement cognitive functions. Thus, as stated, the following quotation from Gallaburda and Bellugi mistakes the discovery of correlations between genetic neuroanatomical, cognitive and behavioural abnormalities in WMS for an explanation of their developmental relationship.

Anatomy is the logical link between genes and behaviour. The purpose of our research on the neuroanatomy of WMS is to help link the anatomical findings to the genetic/molecular disorder on the one hand and the behavioural disorder on the other, thus helping to link the genes to cognition (Gallaburda and Bellugi 2001: 124)(My emphasis).

In the rest of this paper I shall discuss the extent to which evidence about WMS is good evidence for nativism about aspects of language processing which develop abnormally in WMS. I shall show that the issue depends not on anatomy but on the adequacy of different cognitive models adopted by nativists and their opponents. Anatomy can have a confirming role to play, however, in cases where strong genetically specified structure-function correspondences have been independently established. For example there is a large body of independent evidence that the left and right hemispheres implement functional components of face recognition and hence that the abnormalities of face processing in WMS can be plausibly explained in terms of abnormal RHS development.

The nativist approach to WMS has two aspects. Firstly, it identifies cognitive function, including syntactic processing in a standard neuropsychological way via patterns of association and dissociation among deficits (Shallice 1988). Secondly it endorses a classical theory of cognition in which syntactic processing is understood as the implementation of a rule-based computational architecture. Thus the nativist takes WMS to be part of the evidence for the genetic specification of a module which implements a rule based syntactic processor. Neuroconstructivists argue that both aspects of the nativist's approach are inappropriate in the case of developmental disorders

such as WMS. Firstly, cognitive functioning in developmental disorders cannot be identified using standard neuropsychological tests and, secondly, the neuroconstructivist argues that the evidence can be explained if neuroanatomical structures are understood as implementing an associationist rather than a rule-based architecture (Thomas and Karmiloff-Smith, in press).

This debate over WMS is important not only in its own right but because evolutionary psychologists and nativists such as Stephen Pinker (1999) and Neil Smith (1999) have recruited WMS as evidence for nativism about syntactic processing and, by analogy, as support for the idea that other high level cognitive processes such as those involved in theory of mind could be subject to strong innate constraints. (Baron-Cohen 2000; Leslie 1992; Gerrans, in press). An alternative view, congenial to neuroconstructivists, is that the appearance of modularity in high-level cognition is the result of developmental processes whose only innately specified constraints are very low-level structure function-correspondences.

The cognitive profile of WMS

WMS is a developmental disorder which affects approximately 1 in 20,000 live births. The physical phenotype is characterised by, facial dysmorphology (WMS is sometimes called “elfin face syndrome” because of the pixie-like appearance of subjects) and physical abnormalities including supra-valvular aortic stenosis, hyperacusis and dental hypoplasia (Jones and Smith 1975; McKusick 1988).

It is the cognitive profile of WMS which is of greatest interest here: *prima facie*, WMS subjects have a fascinating pattern of associations and dissociations of cognitive abilities. Some aspects of language and social cognition appear to be spared (or enhanced in the case of sociability), as is face recognition, while spatial cognition, numerosity, planning and problem solving are impaired. The most striking aspect of WMS is the apparent linguistic fluency and depth of vocabulary *despite an IQ generally in the 50–65 range* (Bellugi et al. 2000; Karmiloff-Smith 1998).

Thus a WMS sufferer asked to describe cartoon of a boy signalling recent discovery of a frog will say

And he was looking for the frog. What do you know? The frog family!
Two lovers. And they were looking. And then he was happy ‘cause they had a big family. And then said “good bye” and so did the frog. “Ribbit”

A Downs syndrome (DNS) subject of comparable IQ described the scene this way.

There you are. Little frog. There another little frog. They in that . . . water thing. That's it. Frog right there (Bellugi et al. 2000: 49).

This disparity between the vocabularies of WMS subjects and others of the same IQ in WMS is displayed in a number of tests. When asked to match words with images, in searches for primary and secondary homonyms, in providing definitions and listing things of the same category WMS sufferers display a much richer repertoire than DNS counterparts matched for IQ. To give just one example, when asked to name all the animals she could think of in a minute a WMS subject said “tiger, owl, sea lion, zebra, hippopotamus, turtle, lizard, reptile, frog, beaver, giraffe, chihuahua” another said “. . . ibex, whale, bull, yak, zebra, puppy, kitten, tiger, koala, dragon.” A typical DNS response is “goats, rabbits, bunnies, horsey, French fries . . .” (Bellugi et al. 2000: 49). Interestingly this is not an example of developmental precocity since vocabulary in WMS is delayed. However, once the acquisition spurt arrives, it follows a different path to that of normal and DNS children (Laing et al. 2002).

This apparent linguistic virtuosity together with the low IQ initially suggests both that language ability is not affected by the developmental consequences of the genetic deletion and that it is independent of general cognition. Hence it has been recruited by nativists as evidence for the presence of an innately-specified language module. In using the presence of selective deficit to define a cognitive entity, these nativists are extending the standard neuropsychological approach to acquired disorders (Shallice 1998) to a developmental disorder.

Modularity and multiple realisability

The inference from deficit to modular cognitive structure is an inference to the best explanation which depends on a computational model of normal function. That model can then be invoked to explain the deficit in question. For example some Parkinson's patients repeat verb suffixes. “I decided . . . ed . . . ed . . . ed . . .” This might be simply a motor problem. Perhaps the efferent copy of the motor instruction to produce the phoneme has been incorrectly generated or suppressed so that there is no signal for completion. Alternatively it may be a linguistic problem, generated by malfunction of the system which links stems to suffixes (Pinker 1999). Answering this question depends on a detailed model of the way linguistic competence regulates speech production and a theory of its neural implementation. Such problems can be approached using lesion, deficit and imaging studies. We might find, for example, that the motor cortex is unaffected but areas responsible for

language production are abnormal. This is the classic neuropsychological approach to disorder which maps functional to neuroanatomical modularity.

However the *cognitive* part of cognitive neuropsychology does not depend on a one-to-one mapping from cognitive function to brain region. This is because cognitive function is identified by the pattern of association and dissociation among deficits *independently of implementation in a neural substrate*. Someone who has prosopagnosia has a cognitive deficit in face recognition, irrespective of how or where the brain implements the computations necessary to recognise faces. It may be the case that different individuals recognise faces in different ways using different algorithms constructed over possibly idiosyncratic developmental histories. This point simply reflects a point about the characterisation of function inherited by cognitive neuropsychology: it abstracts from physical implementation. Just as video can be beta or VHS or a telephone digital or analog the brain may perform a function in more than one way. Abstraction from physical realisation in the characterisation of a function is called *multiple realisability* (MR).

There are two ways a function can be multiply realisable: Physically or cognitively. In the first case, two physically different systems may perform the same function using the same representations and algorithms. They are alternative physical implementations. Someone might do base 10 arithmetic on an abacus or using a pencil and a piece of paper for example. Alternatively the same function may be performed using different algorithms. For example a mathematical function can be computed in base 10 or base 2. In this case we have alternative algorithmic implementations of the same function and the function is *cognitively as well as physically multiply realisable*. Face recognition is such a function. Faces can be recognised feature by feature, or configurally, in virtue of the relationships features bear to one another. If the neuropsychological approach is to disclose those alternative cognitive strategies we would need, either to discover individuals with patterns of deficits reflecting those different structures, or to design experimental tasks which yielded different outcomes according to the different cognitive strategies being deployed (Deruelle et al. 1999; Karmiloff Smith et al. 1998; Mills et al. 2000).

The relevance of this point to the neuropsychological investigation of cognition is clear. Firstly, explanation is not exhausted until the possibility of multiple cognitive realisability is exhausted. This requires discrimination of subtle deficits using non-standard tests. Secondly, neuropsychology is blind to *physical* multiple realisability.

The distinction between physical and cognitive multiple realisability is especially important when we come to consider the relationship between acquired and developmental deficits. In the case of an acquired deficit, say

a case of acquired prosopagnosia immediately following stroke, we are examining only a single person whose cognitive and physical architecture is past the developmental phase and is therefore more or less invariant. Hence the assumption that such deficits “spare” or leave intact normal functioning in areas which do not test abnormal.

Of course this assumption can be made only if the brain has not cognitively reorganised itself to cope with the consequences of the deficit. The fact that some functions are multiply realisable makes this an open possibility. The face recognition system of someone who had previously recognised faces relationally *might* shift to parsing them feature by feature, in which case the deficit would be invisible to an investigation which did not discriminate the two possibilities. This would exploit cognitive MR. Another possibility exploiting physical MR is that the brain might recruit some redundant processing capacity or retrain another neural assembly to perform the same task.

Residual Normality (RN) is the idea that the deficit confronting the neuro-psychologist is selective and not compensated for by a strategy which exploits cognitive MR (Thomas and Karmiloff Smith, in press). The explanation of acquired deficits can invoke NR because of a hypothesis that mature cognitive function depends on a stable modular architecture, given the lack of cognitive and neural plasticity in the adult brain (the extent of plasticity is an empirical matter with serious consequences for rehabilitation. People *do* recover from strokes, which argues for some neural plasticity in the adult brain).

The assumption of RN is more problematic in the case of developmental disorders because of the greater cognitive and neural plasticity of the developing brain. A genetic deletion or early developmental traumatic event may be compensated for in different ways over a protracted developmental history in which the brain is at its most plastic. As a consequence, the end state or behavioural phenotype of someone with a genetic defect may be very similar to that of a normal subject, but the developmental route, cognitively and neurally, may be very different. The face recognition of WMS subjects is an example. Bellugi and collaborators who investigated WMS in the early nineties using standard tests of face recognition concluded that it was “spared” in WMS. “Spared” in this context means that the subject tests within the range of normal function on the relevant tasks, here facial recognition. Against the assumption of RN the interpretation must be that the face recognition module is ‘intact’. Bellugi, for example concludes her introduction to WMS by remarking on the characteristic pattern of dissociations including “impaired spatial cognition, remarkably spared face processing” (Bellugi et al. 2000: 37).

However the inference from normal results on standard tests to normal cognitive function may be misleading if the assumption of RN cannot be sustained. According to Donnai and Karmiloff-Smith this assumption is misplaced in the case of WMS

whereas normal controls use predominantly configural or holistic processes to recognise faces, people with WS tend to use predominantly componential or featural processes (Deruelle et al. 1999; Karmiloff-Smith et al. 1998). The tendency to use featural rather than configural processes in WS, as compared to patients with Downs syndrome, is seen not only with respect to faces, but also in other visuo spatial tasks (Donnai and Karmiloff-Smith 2000).

This is explained plausibly as a case of neural and cognitive reorganisation in early development. Lacking the capacity for holistic processing, the WMS subject mobilises another cognitive strategy, substituting featural for configural processing. In a face matching task (matching upright to inverted faces), Mills et al. (2001) found that WMS subjects showed a very different pattern of ERP in the right hemisphere to normal subjects (hypothesised to be involved in configural processing). They concluded that this result was consistent with the hypothesis which “*suggested abnormal cerebral specialisation for spared cognitive functions in individuals with WMS*” (72). Given the importance of the human face to infant development this is unsurprising. On the neural constructivist framework, the developing brain will reorganise itself as far as possible to perform this essential function in the absence of normal RHS function. The result is that faces are processed on a feature-by-feature basis, recruiting available left hemisphere resources to the task. Of course abnormal cerebral specialisation *alone* does not indicate abnormal cognition. It may be an alternative physical realisation of a normal cognitive function. However, in the case of WMS, it does seem that both the anatomical structure and cognitive processing of faces are unusual.

Thus the idea of ‘spared’ cognitive function here is misleading *if interpreted as evidence for RN*. Face processing is multiply realisable both cognitively and physically and both aspects are exploited by the WMS subject who reorganises her developing brain to produce a behavioural phenotype which is not distinguishable from the normal one by standardised tests. The hypothesis that faces are processed in the same way by the WMS and normal subject is clearly not licensed.

WMS and nativism about syntax

Language processing in WMS raises similar issues. On an assumption of NR the behavioural phenotype of WMS syndrome might be taken as evidence that language processing is “spared”, i.e., the same as that of normal subjects. In particular nativists in the Chomskian tradition such as Stephen Pinker and Neil Smith (Smith 1999: 24, 25) have argued that WMS and a disorder known as Specific Language Impairment (SLI) constitute a *developmental double dissociation*. That is to say a genetically-specified module for grammatical processing develops normally in WMS and fails to develop in SLI.

. . . overall, the genetic double dissociation is striking The genes of one group of children [SLI] impair their grammar while sparing their intelligence; the genes of another group of children [WS] impair their intelligence while sparing their grammar (Pinker 1999: 262).

Pinker and Gary Marcus (2000) defend the view that in normal humans and WMS subjects whose syntactic processing is “spared”, genes build a neural syntactic processor which cognises the linguistic domain according to rules which are innate. Not innate because genes encode syntactic information but because genes build a language module which because of its physical architecture can only implement a rule based cognitive architecture. Thus all competent speakers of English end up marking the past tense of regular verbs according to the rule “stem+ed” because their language module is pre-sensitised to detect syntax.

On this theory, regular verbs are cognised by a rule-based module and irregular verbs by a non rule-based associative memory system. The two systems are modelled computationally in a dual route network with two components. One implements a rule based architecture and the other a typical associationist form of pattern recognition by statistical inference. The validity of the computational model is tested by training the dual route network on inputs which mimic the primary linguistic data of human development. One can then damage one or other component and compare the results to the performance of WMS and SLI subjects. Namely

The first group of [SLI] children rarely generalise the regular pattern [stem+ed]; the second [WMS] group of children generalise it freely (Pinker, 1999).

The nativist explanation is that, due to genetic abnormalities characteristic of the different syndromes, WMS spares a rule-based syntactic processing route which is impaired in SLI. The converse pattern of impairments affects an associationist route in both disorders, which accounts for the differences with respect to marking the past tense of irregular verbs.

This inference is made on the basis of standardised tests and is subject to the same type of criticisms we noted above. This does not mean that Pinker is wrong, just that the inference requires us to rule out the possibility that the “normal” results attained by WMS subjects on standardised tests are not achieved using abnormal cognitive strategies.

As with the case of face processing, closer attention to the behavioural phenotype and the use of non standard tests by Karmiloff Smith and collaborators shows that the language processing of WMS individuals develops along a different trajectory and exploits different cognitive strategies to that of the normal subject. WMS subjects point protodeclaratively *after* the typical vocabulary spurt, *reversing* the normal sequence of development. With regard to syntax, the core of the nativist explanation of WMS, Karmiloff Smith and her collaborators concluded that WMS subjects learnt inflectional morphology [stem+ed] but, crucially, did not generalise to novel cases. It is this type of generalisation on which nativism depends, since it cannot be explained by associative memory. Thus Donnai and Karmiloff-Smith reached the opposite conclusion to the above-mentioned nativists

It is questionable whether any aspect of language – syntax, semantics, phonology or pragmatics – is intact in WS despite claims to the contrary (Thomas et al., Donnai and Karmiloff Smith: 9).

A neuroconstructivist alternative

Neuroconstructivism is the view that cognitive architecture emerges as the brain organises itself in response to environmental and internal (such as metabolic or structural) contingency rather than according to a program encoded as a genetic blueprint (Quartz and Sejnowski 1997; Elman et al. 1996). Interestingly neuroconstructivism shares with Bellugi and collaborators and nativists about syntax a concentration on the ontogenetic aspects of neuroanatomy as a key to understanding cognition. Neuroconstructivism, however supplements the anatomical description with computational (typically, although not exclusively connectionist) models of cognition whose computational architecture is not built following a genetic blueprint but is best thought of as a diachronic adaptation of neuroanatomical structure to environmental contingency.

Traditionally neuroconstructivists have argued that the genotype’s contribution to cognition is in specifying minimal initial constraints on the construction of a highly plastic learning device (modelled by their connectionist network) which can follow many developmental paths to a given end state. Hence there is no one to one mapping from genotype to phenotype. Neuroconstructivism thus shifts the understanding of cognition away

from synchronic identification of high-level cognitive function abstracted from neurobiological realisation towards the understanding the diachronic development of neurobiological assemblies which perform those functions (Karmiloff Smith 1998; Karmiloff Smith et al., in press; Quartz, this volume).

Note that for both nativists and neuroconstructivists the claim that the genotype strongly constrains neural structure is not the same as the claim that it constrains cognitive structure *unless there is very strong structure-function correspondence* (Quartz, this volume). The extent of such correspondence cannot however be determined simply by pointing to typical and atypical structure-function correspondences (whether or not they are localised or distributed) in the absence of a theory of the way in which particular neuroanatomical structures realise cognitive functions. As we saw above nativists couch their theory in terms of a dual route model.

By contrast Karmiloff Smith and collaborators explain the appearance of intact or enhanced language ability in WMS in terms of increased sensitivity to sound patterns in a *single route associative memory network*. The WMS subject remembers and reproduces instances of the past tense because she is especially sensitive to phonology not syntax (note that if this is correct we should expect the WMS subject not to exhibit standard forms of overregularisation, such as “she runned to the shops” since they depend on syntactic processing). Thus they propose an alternative cognitive theory of the way in which WMS subjects process language.

This debate cannot be resolved by appeal to anatomy in the absence of a cognitive model. We need to test different cognitive architectures under constraints which approximate human development and determine which most closely approximates normal and WMS human performance. (Marcus 2000; Plunkett and Marchman 1993). At present testing of such theories takes place in simulated “neural networks” whose very basic information processing features (connections between individual nodes in the network) hypothesised to approximate those of actual neural assemblies. If nativists are right any system able to acquire natural language syntax with the same facility as humans must have some elementary rules of syntactic processing “wired in” at the outset. This initial sensitivity to grammatical structure in the acoustic stream accounts for the subsequent development of syntactic processing.

A neuroconstructivist alternative is to construct a single associative network, vary its input properties, weightings and learning algorithms and see whether outputs approximate the more fine-grained data about WMS obtained when language performance is assessed on non standard tests. Thomas and Karmiloff-Smith, for example, achieved a good match by

making the network more sensitive to phonology, supporting the idea that WMS subjects are regularising on the basis of phonology not syntax.

Interestingly, making a network less sensitive to phonology produced a pattern of regularisation (more accurately lack of regularisation) characteristic of SLI. If it is genuinely the case that the symptoms of SLI and WMS can be accounted for by differential phonological processing in a single associationist network then claims that they represent a double dissociation of rule-based syntactic processing are undermined. And in a way this is not surprising since closer attention to the difference between WMS subjects and normal would have shown differences in the ability to extend to novel cases, the property which is agreed on all sides to distinguish rule-based from associationist systems.

One might argue that such findings do not refute nativism about syntactic processing. They just refute the idea that *WMS is evidence for nativism about syntactic processing*. After all, WMS subjects are not doing syntactic but phonological processing and the latter is not simply an alternative cognitive realisation of the former since it cannot explain generalisation to novel cases. The nativist then has to say that syntactic processing is impaired in WMS. But in that case why WMS do they not resemble more closely the linguistic profile of SLI cases? It is likely then that SLI and WMS are not a developmental double dissociation of the type most useful to the nativist about syntax.

This does not mean that there is no genetic contribution to syntactic processing, just that the evidence from WMS has not helped us identify it, for reasons identified by Thomas and Karmiloff-Smith and the neuroconstructivists.

References

- Baron-Cohen, S. and Tager-Flusberg, M. (eds): 2000, *Understanding Other Words. Perspectives from Developmental Cognitive Neuroscience*, OUP, Oxford.
- Bellugi, U. and St George, M. (eds): 2000, *Journey from Cognition to Brain to Gene. Perspectives from Williams Syndrome*, MIT Press, Cambridge, MA.
- Bellugi, U., Lichtenberger, W., Jones, W., Lai, Z. and Marie St George: 2000, 'The Neurocognitive Profile of Williams Syndrome: A Complex Pattern of Strengths and Weaknesses', in U. Bellugi and M. St George (eds) *Journey from Cognition to Brain to Gene. Perspectives from Williams Syndrome*, MIT Press, Cambridge, MA.
- Deruelle, C., Mancini, J., Livet, M.O., Casse-Perrot, C. and de Schoen, S.: 1999, 'Configural and Local Processing of Faces in Children with Williams Syndrome', *Brain and Cognition* **41**, 276–298.
- Donnai, D. and Karmiloff-Smith, A.: 2000, 'Williams Syndrome: From Genotype Through to Cognitive Phenotype', *American Journal of Medical Genetics: Seminars in Medical Genetics* **97**, 164–171.

- Galaburda, A. and Bellugi, U.: 2000, 'Cellular and Molecular Cortical Neuroanatomy in Williams Syndrome', in U. Bellugi and M. St George (eds), *Journey from Cognition to Brain to Gene. Perspectives from Williams Syndrome*, MIT Press, Cambridge, MA.
- Gerrans, P.: 2002, 'Theory of Mind and Evolutionary Psychology', *Biology and Philosophy* **17**, 305–321.
- Jones, K.L. and Smith: 1975, 'The Williams Elfin Faces Syndrome: A New Perspective', *Journal of Paediatrics* **86**, 718–723.
- Karmiloff-Smith, A.: 1998, 'Development Itself Is the Key to Understanding Developmental Disorders', *Trends in Cognitive Sciences* **2**, 389–398.
- Karmiloff-Smith, A., Scerif, G. and Ansari, D.: in press, 'Double Dissociations in Developmental Disorders. Theoretically Misconceived, Empirically Dubious', *Cortex*.
- Laing, E., Butterworth, G., Ansari, D., Gsödl, M., Longhi, E. Panagiotaki, G., Paterson, S. and Karmiloff-Smith, A.: 2002, 'Atypical Development of Language and Social Communication in Toddlers with Williams Syndrome', *Developmental Science* **5**, 233–246.
- Leslie, A.: 1992, 'Pretense, Autism and 'Theory of Mind' Module', *Current Directions in Psychological Science* **1**, 18–21.
- McKusick, V.: 1998, *Median Inheritance in Man: Catalogs of Autosomal Dominant, Autosomal Recessive and X-Linked Phenotypes*, Johns Hopkins University Press, Baltimore.
- Mills, D. Alvarez, T., Marie St George, Appelbaum, L., Bellugi, U. & Neville, H.: 2000, 'Neurophysiological Amrkers of Face Processing in Williams Syndrome', in U. Bellugi and M. St George (eds), *Journey from Cognition to Brain to Gene. Perspectives from Williams Syndrome*, MIT Press, Cambridge, MA.
- Oyama, S., Gray, R. and Griffiths, P. (eds): 2001, *Cycles of Contingency. Developmental Systems and Evolution*, MIT Press, Cambridge, MA.
- Pinker, S.: 1999, *Words and Rules*, Weidenfield and Nicholson, London.
- Quartz, S.R. and Sejnowski, T.J.: 1997, 'The Neural Basis of Cognitive Development: a Constructivist Manifesto', *Brain and Behavioural Sciences*.
- Samuels, R.: in press, 'Nativism in Cognitive Science', *Mind and Language*.
- Shallice T.: 1988, *From Neuropsychology to Mental Structure*, Cambridge University Press, Cambridge.
- Thomas, M. and Karmiloff-Smith, A.: in press, 'Are Developmental Disorders Like Cases of Adult Brain Damage? Implications from Connectionist Modelling', *Behavioural and Brain Sciences*.
- Thomas, M.S.C., Grant, J., Barham, Z., Gsödl, M., Laing, E., Lakusta, L., Tyler, L.K., Grice, S., Paterson, S. and Karmiloff-Smith, A.: 2001, 'Past Tense formation in Williams Syndrome', *Language and Cognitive Process* **16**, 143–176.