Modularity of Mind and Language

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Abstract

The concept of Modularity, i.e., the degree to which the lexicon, syntax, and other neurocognitive domains operate independently of one another, has played an important role in theorizing about brain architecture and function, both in development and in adulthood. In this chapter, we present an overview of the theoretical and empirical arguments relevant to three parameters of modularity: localization, universals (ontogenetic and pathological), and domain specificity. Converging evidence from several research areas on language and other domains, including acquired brain damage and developmental disorders, lends little credence to the notion of prespecified modules, but rather supports a dynamic, experience-dependent view of the progressive localization and specialization (i.e., progressive modularization) of brain regions across developmental time.

Parameters and Definitions

Modularity is an important concept relevant to a number of central debates in the cognitive and neuro-sciences, including theories of the structure of the mind/brain. Broadly speaking, modularity concerns the degree to which cognitive domains can be thought of as separable, i.e., whether they function independently of one another. Exactly what constitutes a module varies widely across disciplines and theoretical approaches.

The most explicit definition of modularity comes from Fodor (1983). In Fodor's view, a module is a perceptual input system that has the following criteria: (1) It is *informationally encapsulated;* other parts of the mind/brain cannot interfere with the internal functioning of a module. (2) The operations within a module are *unconscious* and not available for reflection. (3) Modules have *shallow outputs*, i.e., the intervening operations that give rise to output are not apparent from that output. (4) The operation of a module is mandatory - *obligatory firing*. Four more criteria apply, and are exclusive to the notion of 'innate' modules. Innate modules are (5) *localized* in particular brain areas, common to all individuals. They exhibit (6) *ontogenetic universals*; their development is bound to a given time schedule. There are characteristic ways in which modules break down, giving rise to (7) *pathological universals*. Finally, innate modules are (8) *domain specific*; they operate exclusively on certain types of input, in Fodor's terms, modules have proprietary inputs.

Albeit the most explicit, Fodor's vision of a module is by no means unchallenged. Importantly, different definitions of what constitutes a module may include various combinations of these criteria (Elman, Bates, Johnson, Karmiloff-Smith, Parisi & Plunkett, 1996). Also, some criteria may be more important than others, depending on the level of description: anatomical structure, computation, function, or knowledge. It is generally accepted that some form of modularity exists in the human mind/brain, but there is little agreement on what exactly that is, nor on the degree of fractionation. For example, there is little controversy that highly specialized areas of the visual cortex

selectively process specific dimensions of the visual experience of colour and orientation. However, for higher-level cognition, whether a specific brain region can be thought of as the 'language module' or the 'face module' involves more controversial questions.

Bearing that in mind, we find it more useful to think of Fodor's criteria as separable hypothetical parameters - as opposed to necessary criteria - that are important in theorizing about the architecture of the mind/brain. For the purposes of this chapter, we will focus on three crucial questions in the modularity debate that correspond to some of the parameters discussed. These questions are by no means exhaustive, but they capture key elements that are relevant to the field as a whole:

I. The question of localization: At what level is the brain modular?

II. The question of ontogenetic and pathological universals: When, and how, do certain functions become modularised?

III. The question of domain specificity: Are modules independent of one another?

The theory and methodology of modularity: logic and assumptions

Before discussing the key questions, it is important to present the methodological framework most widely used in investigating modularity. As a theoretical construct, modularity is tightly linked to the field of adult neuropsychology, and can be thought of as one of its axioms. The main objective of this discipline has been to use patterns of impairment in the adult to arrive at a theory of normal cognitive processes and brain structure. Modularity is also an important construct in the more recent field of developmental neuropsychology, where the objective is to arrive at a model of the normal chid state from patterns of developmental impairment. Of course, both adult and child neuropsychology share another important and practical objective of diagnosis and symptomology for clinical populations.

The single most important piece of evidence for functional modularity is the existence of double dissociations (henceforth, DD), of which different versions share an underlying logic.

The adult and child brain damage version: After brain injury, Patient A loses the capacity to perform task X but can still perform task Y. Another Patient, B, shows the opposite pattern, where he can perform Y but not X. In this case, researchers infer that functions X and Y are doubly dissociated. A further inference might also be made, i.e., that the sites of lesion in Patients A and B are causal for functions X and Y respectively. Although worded to fit the single case, the logic of DD applies to group studies as well. There is considerable debate on whether single-case or group studies are more appropriate in investigating modularity (McCloskey, 1993; Robertson, Knight, Rafal, & Shimamura, 1993; Shallice, 1988). Nevertheless, most of the issues discussed here apply equally to single-case and group studies.

The developmental disorders versions: The extension of the adult DD logic to development is somewhat curious since most conditions of developmental disorders are not associated with frank neurological lesions (despite differences in brain anatomy and physiology being extremely common). In the developmental case the logic is modified: Child A has learned skill X but not skill Y. Another Child, B, has learned Y but not X. On those bases, it is inferred that skills X and Y are dissociated over developmental time.

Yet another, stronger version of DD is used to support a particular claim, namely, genetic modularity: Due to genetic impairment, Child A fails to learn skill X but learns skill Y. Another Child, B, shows the opposite pattern, where he learns Y but not X. In this case, researchers infer that skills X and Y map onto the impaired gene or specific set of genes in each case. The child is deemed to be missing the necessary module to develop the skill in question, as illustrated in the following claim:

> "…Overall, the genetic double dissociation is striking… The genes of one group of children [Specific Language Impairment] impair their grammar while

> sparing their intelligence; the genes of another group of children [Williams syndrome] impair their intelligence while sparing their grammar."
>
> (Pinker, 1999, p. 262).

The different versions of the modularity claim, as applied to the language domain, will be presented in detail in subsequent sections of this chapter. However, Frequently overlooked in these applications are the critical assumptions embedded in the logic of DD and whether these assumptions hold in each version.

There are at least four critical assumptions: (1) that specific brain substrates underlie dissociable functional components - the *dissociability assumption*; (2) that there is a direct mapping between behavioural impairment and functional components – *the transparency assumption;* (3) that lesions or disorders cause the subtraction of the affected module, reflected in functional impairment on tasks relevant to that module, while all other functions operate normally - the *subtractivity or residual normality assumption*; finally, (4) that all cognitive systems are similar across individuals where individual differences are non-contributive - *the universality assumption*. In sum, a direct mapping between modules and behaviours is assumed.

The extension of the DD logic to cases of developmental or acquired disorders has a number of strong implications: (1) that the behavioural patterns in the adult case and in the child case share the same underlying cause, namely the defective module in question; (2) that a static model can explain patterns of behavioural outcomes, deeming the developmental process itself to be a minor contributor; and (3) that pure cases, exhibiting selective deficit as in the adult, can be found in infancy and childhood. These assumptions and implications shape the kinds of inferences that are made from the data and will thus be evaluated in detail when we turn to the three main questions of this chapter.

Localization: At what level is the brain modular?

A certain function or domain is said to be localized if it is represented and/or processed in circumscribed regions of the brain. At the other end of the continuum is distributed processing, where multiple, potentially discontinuous brain regions are involved in the representation or processing of a given function. Localization is not necessarily held as a criterion for modularity. Some theorists distinguish between cognitive modularity (arising from cognitive neuropsychology) and neural modularity (arising from classical neuropsychology) where each is of more relevance to cognitive and neural theories respectively. In some strong versions of this distinction, the neural substrates of cognitive modules are deemed irrelevant (see Robertson et al., 1993, for discussion).

It is fairly widely accepted that adult neurological patients exhibit deficits that can be described as selective. The accumulation of a significant body of data from brain damage, and more recently from imaging, has helped elucidate what the components of cognition might look like. Classic findings point to breakdown within the language domain, including grammatical processing in Broca's area, and comprehension and word-finding in Wernicke's area. Face processing is another domain, claimed to be carried out uniquely in specific regions of the brain, i.e., in the fusiform gyrus.

The logic and assumptions of DD face a number of theoretical challenges. Modular analysis lends itself to tremendous flexibility in the face of inconsistent data; a modular framework can, without fail, carve existing modules into increasingly smaller components or keep adding new modules to accommodate a given behavioural pattern. At the extreme, multiple modules the size of each tiny concept could be hypothesized (Sperber, 2002). This infinite decomposition is best exemplified by the modular analysis of language function, where linguistic knowledge is separated from general cognition (Pinker, 1994), and grammar from the lexicon (Pinker, 1999). Some theorists have carved the lexicon into components for nouns and verbs, and nouns into

separate modules for tools vs. utensils (Tranel, Logan, Frank, Damasio, 1997). This seemingly infinite refinement of cognitive architecture raises the possibility that it is simply the task demands themselves that can account for what is deemed to be a module, shedding doubt on the falsifiability of modular theories.

Even more problematic for modularity is whether it can really be considered a data-constrained model at all. Finding the doubly dissociated components requires a model that defines a priori what the components are. Furthermore, it requires a model of how the tasks used to assess the integrity of a given component relate to that module. Shallice (1988) points out the circularity inherent in such an analysis:

> "An argument to justify [modular analysis] could ... proceed in the following way: 'If modules exist, then, … DD are a relatively reliable way of uncovering them. DD do exist. Therefore modules exist.' Presented in this form, the logical fallacy is obvious." (Shallice, 1988, p. 248)

According to Shallice, the validity of this claim rests on the assumption that dissociations can only arise from damage to modular systems. Yet, findings from neural network lesioning clearly demonstrate that selective deficit can arise, not only from a modular structure, but also from a distributed neural network architecture (for example, Plaut, 1995). In fact, Shallice (1988) presents a number of alternative theoretical possibilities of non-modular architectures that would also give rise to double dissociations after impairment.

On empirical grounds, the classic conclusion that damage in specific areas produces selective functional deficit has been countered by numerous findings. In language, aggrammatic patients show some preservation of grammatical judgment albeit at a fragile level (Wulfeck & Bates 1991). Conversely, clinical populations with damage in areas other than Broca may exhibit aggrammatic symptoms (Dick, Bates, Wulfeck, Utman, Dronkers, & Gernsbacher, 2001). More generally, the standard association between damage to these areas and patterns of language breakdown does not consistently hold, i.e., damage to Broca's or Wenike's areas is neither necessary nor

sufficient to produce the classic symptoms (Goodglass, 1993; Dronkers, 2000). Similar conclusions come from reviews of brain imaging studies of phonological speech processing where, across different studies, little overlap was found in the activated regions (Poepel, 1996).

Turning to the developmental literature, we observe that in default developmental circumstances, brain regions become progressively specialized in a relatively specific manner. On the other hand, many have argued that cases of acquired lesions in infancy and childhood pose a major problem to claims of prespecified modular architecture. Although reorganization post injury is not viewed as an important factor in adult cases, plasticity and reorganization are the rule rather than the exception in childhood cases. For example, work with vertebrate brains has elucidated the extent to which reorganization and compensation are possible (reviewed in Johnson, 1996; Quartz & Sejnowski, 1997). Experience-dependent plasticity appears to be a hallmark of our species' brains.

Some general trends come from large longitudinal studies of individuals who suffered focal brain lesions before six months of age (Reilly, Bates & Marchman, 1998; Thal, Marchman, Stiles, Aram, Trauner, Nass & Bates, 1991). Despite their lesions, the majority of these children still attain normal language skills. As a general trend, the effects of lesions in children were consistently different from the effects of acquired lesions in similar locations in adults. This challenges the notion that a missing or damaged module, in both the adult and child cases, is causal for behavioural components such as language. This does not imply, however, that language developed in the normal fashion in these atypical circumstances. In fact, there are clear demonstrations that this is not the case. For example, in the majority of children with focal brain lesions the language acquisition process is delayed. Another interesting example comes from patterns of language specialization when children who had suffered focal brain damage reach adulthood. There does not seem to be a uniform solution that the brain employs after damage; some individuals exhibit specialization

for language production in the left hemisphere, others in the right hemisphere, and others bilaterally (Satz, Strauss, & Whitaker, 1990).

For decades, much of neuropsychology has focused on where functions/behaviors are localized. Indeed, far more emphasis has been given to the *where* question at the expense of the *why* question. Beyond claims of genetic specification, very little is understood about why in default circumstances a region takes on certain functions and not others. An argument can be made that in normal circumstances, Broca's area may become specialized for language processing, not because it is specifically designed for language, but partly because it is the area with the computational characteristics that are particularly well suited to deal with the requirements of this domain (Elman et al., 1996). In other words, a "domain-relevant" region becomes domain-specific over developmental time (Karmiloff-Smith, 1998). Thus, many regions may initially compete for the processing of given inputs, with the special computational properties of one region ultimately winning out. However, a full specification of what those computational properties are, is as yet largely unknown. We return to these questions in the final section on domain specificity.

Ontogenetic and Pathological universals: When and how does the brain become modular?

Thus far, the application of modular analysis to adult and child acquired disorders does not necessarily imply genetic specification of a modular architecture. This is even accepted by those who are proponents of the applicability of the adult neuoropsychological approach to the child (e.g., Temple, 1997). On the other hand, cases of developmental disorders with genetic aetiology have been used to support claims of genetic modularity. The critical assumptions in the adult model, namely *dissociability*, *transparency*, *and subtractivity* (*residual normality*) are relevant here, too. The universality assumption has a stronger version, where the child's modular architecture is considered to be not only universal but also *prespecified*. The problem is

seen as one of simply finding out what the structure is. Furthermore, the same implications of the extension of the adult model to the child case hold here, namely, *shared causes*, *appropriateness of static models*, and *the existence of pure cases*.

Some of these claims seem to be supported by evidence from childhood disorders. For example, Temple (1997) describes the similarity between behavioural profiles of adults and children with prosopagnosia, dyscalculia, and certain subgroups of dyslexia, where in some instances, the behavioural symptoms are indistinguishable in the child and adult cases. Interestingly, such similarities are clearest with respect to school achievement skills, like reading and mathematical abilities (Temple, 1997).

Additional claims for genetic modularity come from the study of Williams syndrome and Specific Language Impairment (SLI). According to some authors, in WS, we find 'striking preservation' of language and face processing alongside severe visuospatial deficit (Bellugi, Lichtenberger, Jones, Lai, & George, 2001). In some forms of SLI, grammar is severely impaired whereas all other functions are claimed to be unaffected (van der Lely, Rosen & McClelland, 1998). This has led to the conclusion that behavioural components in these cases are indeed dissociable (*dissociability*) and that performance in the unaffected domains is normal (*residual normality*).

In a more strict application of modular analysis, SLI and WS have been taken as a doubly dissociated pair of the rule-based vs. associative memory language systems, implying genetically specified modules for these two domains (Pinker, 1999). Evidence for this claim comes from investigation of English past-tense formation. Children with SLI have difficulties in past-tense formation and crucially do not exhibit an advantage for regular over irregular past-tense formation (Ulman & Gopnick, 1999). WS children, on the other hand, are claimed to display a particular difficulty in generating irregular past-tense forms whereas the regular past-tense forms are unproblematic (Clahsen & Almazan, 1998).

The vast majority of claims pertaining to genetic modularity have been challenged, particularly with respect to the utility of applying a strictly modular approach in

investigating developmental disorders (Karmiloff-Smith, 1998; Karmiloff-Smith, Scerif & Ansari, 2003). There are, for example, some general arguments against the view of genetic modularity based on our current understanding of how genes and the brain work. If modularity is genetic, then genes would have to specify the representational content of each module - an unlikely claim. Genes obviously contribute to behavioural outcomes, but it is more likely that the mapping between these is indirect and highly experience-dependant. Instead of specifying domain-specific knowledge, genes contribute to the formation of domain-relevant learning mechanisms, which are gradually refined by the ontogenetic process (Karmiloff-Smith, 1998; Karmiloff-Smith & Thomas, in press).

In addition to these arguments, specific claims of *pure cases and residual normality* have been refuted by studies, for instance, of Williams Syndrome. First, once fine-grained methods are used to assess participants, clear-cut selective patterns of impairment and sparing are no longer found. For example, Thomas and colleagues found subtle deficits in WS for both regular and irregular past tense formation (Thomas, Grant, Barham, Gsödl, Laing, Lakusta, Tyler, Grice, Paterson & Karmiloff-Smith, 2001). Second, equivalent levels of performance may be driven by atypical processing strategies. For example, face processing in WS seems to be driven by a featural processing strategy (Deruelle, Mancini, Livet, Casse-Perrot & de Schonen 1999; Karmiloff-Smith, 1997; Karmiloff-Smith, Thomas, Annaz, Humphreys, Ewing, Brace, van Duuren, Pike, Grice & Campbell, in press). Moreover, different brain structures than those found in normal circumstances are involved in the albeit proficient face processing in WS (Grice, Spratling, Karmiloff-Smith, Halit, Csibra, de Haan & Johnson, 2001). Similar analyses have been conducted for SLI, which despite its name of **Specific** Language Impairment, consistently displays subtle deficits in domains other than language (Chiat, 2001; Hill, 2001; Leonard, 1998).

Further arguments against genetic modularity can be made when we simultaneously consider typical and atypical language development. While it is true

that there are clear developmental milestones in language acquisition (*ontogenetic universals*), there is also substantial variability in the rate and style or strategy of acquisition (see Bates, Thal, Finlay & Clancy, in presss, for a review). If a defective grammar module explained SLI as well as individual differences in normal language learning, then we should see a bimodal distribution of language abilities, with genetic differences accounting for the separation between the impaired and unimpaired groups. But the distribution is normal not bimodal (Bates et al., in press). Furthermore, if grammar and the lexicon were separate modules (Pinker, 1999), it would be difficult to explain the strong correlations between early grammar and early lexical ability where growth curves for late and early talkers show that the level of grammatical complexity reflects lexical abilities and not chronological age (Bates & Goodman, 1997).

The ongoing discussion demonstrates that the use of developmental disorders as evidence for genetic modularity is flawed, and that the role of genes in the specification of brain architecture and behaviour is far more complex than direct mapping between genes and behaviour.

Domain Specificity: Are modules independent?

The domain specificity question concerns the extent to which the operations of a given module such as syntax are special and exclusive to that domain. It is critical that the question is phrased in a relative, as opposed to a dichotomous manner, since it is clear that at some level of processing, the brain integrates information from various sources to produce coherent experience. Elman et al. (1996) point to a number of broad notions of domain-specificity. Domains can be specific because they have specific input/output systems: visual cortical areas receive input form the retina, whereas auditory cortical areas receive input from the ears. Specificity may also arise because different problems require different behavioural solutions and/or different computational mechanisms. It is, then, hardly surprising that, because a particular domain requires specific kinds of representations or behavioural solutions, domain

specificity would seem to exist. The important question, however, is the extent to which computational mechanisms of a given domain are exclusive to that domain, i.e., modular, throughout development or in adulthood.

In a narrow interpretation of domain-specificity, Fodor (1983) claims that modules operate using specialized mechanisms, dedicated to handle specific types of input, what he calls 'proprietary inputs'. For example, speech perception is distinguished from general problem-solving ability, where speech perception involves computing what is heard, with little reflection or analysis of the problem domain of language in which it is used. Moreover, any module deemed to be innate is, by definition, domain specific. However, converging evidence from different areas of research reveals a more complex pattern of how different domains interact and become specialized:

(1) Both infants and adults show remarkable skills in discovering patterns and regularities, not only in linguistic input, but also in the general auditory and visuospatial domains (Newport & Aslin, 2000). While we still have little understanding of how these are used in learning and processing language, and importantly, of the constraints on this type of learning, it does indicate that domain-general abilities can play a crucial role in organizing linguistic input.

(2) Increasingly, evidence points to the involvement, in other more general skills, of the areas classically thought of as uniquely housing domain-specific operations. With respect to language, Broca's area has also been implicated in a number of non-linguistic processes including musical syntax (Maess, Koelsch, Gunter, & Friederici, 2001), general selection mechanisms (Kan & Thompson-Schill, 2003), and imitation (Heiser, Iacoboni, Maeda, Marcus & Mazziotta, 2003).

(3) In certain cases, dedicated mechanisms appear to be the result of experience with a given class of stimuli. For example, behavioural and brain imaging studies show that the experience of experts with another class of stimuli (e.g., dog judges), or training with an artificial class of stimuli ('greebles') results in activation of areas in the

fusiform gyrus, previously though to be activated exclusively for faces (Gauthier, Behrmann, & Tarr, 1999; Gauthier, Skudlarski, Gore, & Anderson, 2000). So, additional factors, like level of categorization and expertise, also modulate the involvement of the fusiform gyrus in processing visual objects.

The question of whether domain specificity is prespecified in the human brain as a result of evolution or arises over ontogenesis from experience-dependent processes, coupled with self-organizing cortical mechanisms, is still an open one. What is clear, though, is that patterns and regularities in the input can quickly and efficiently lead to progressive specialization of the brain. Evolution is more likely to have given rise to greater flexibility of learning than to increasingly complex presepecified modules (Karmiloff-Smith, 1992).

In developmental cases, the domain specificity question has also been extensively discussed. In the genetic modularity view, genes code for a specific representations that are domain-specific. The alternative view is that domain-relevant computations early in development are better able to handle certain forms of input, and those become progressively specialized in close interaction with the environment (Karmiolff-Smith, 1992; Karmiloff-Smith & Thomas, in press). The idea of progressive modularisation forcefully challenges the notion that a static model can be used to explain development in both the typical and atypical case. On the contrary, the developmental process itself is a major contributor to behavioural outcomes (Karmiloff-Smith, 1998). In support of this claim, brain imaging data from infants and toddlers show that there is a pattern of progressive specialization of important functions for our species, like face processing (de Haan, Pascalis, & Johnson, 2002) over developmental time. In atypical cases, the origins of the behavioural profiles found later in development may stem from differential processing of input and utilization of different strategies that begins early in infancy.

A related hypothesis is that deficits in 'low-level', perceptual mechanisms is a contributing factor in developmental disorders. Some possible mechanisms are: rapid

auditory processing impairment in Specific Language Impairment (Temple, Deutsch, Poldrack, Miller, Tallal, Merzenich & Gabrieli, 2003), magnocellular system impairments in dyslexia (Stein, 2001), and attentional abnormalities Williams Syndrome (Brown, Johnson, Paterson, Gilmore, Gsödl, Longhi, & Karmiloff-Smith, 2003).

Some have challenged the view that low-level impairment can be viewed as causal in many of these developmental conditions (Ramus, Rosen, Dakin, Day, Castellote, White & Frith, 2003; Rosen, 2003), based on the fact that such impairments are not found in all cases. The validity of this claim is difficult to assess for a number of reasons. First, many developmental disorders are diagnosed based on behavioural impairment in a specific area, shedding doubt on whether it is necessary to look for impairments in other domains, after having a priori excluded those whose impairment less marked. Second, low-level problems need not exist throughout the entire lifespan. The crucial point is that their presence, **early** in development, may trigger cascading effects, the **indirect** results of which are found later in development. Third, even if not taken as singular explanatory factors, low-level impairment may play an important role in altering the experience of the child in her environment, providing a possible mechanism by which developmental outcomes are achieved.

In sum, converging evidence suggests that modules are the final outcomes of the developmental process as opposed to prerequisites to it. Furthermore, the progressive development of modules, both in infancy and adulthood, is tightly bound to experience. Even in adulthood, experience continues to play a role in forming brain architecture and processing.

Concluding Remarks

We started this chapter with three questions relevant to the modularity debate. First, the question of localization: at what level is the brain modular? Second, the question of ontogenetic and pathological universals: when and how do certain

functions become modularised? Third, the question of domain specificity: are modules independent of one another? We aimed to provide insights into each of these questions, but current understanding of how the human brain organizes itself over time remains relatively limited. However, the substantial body of evidence in support of dynamic interactions in development, as well as plasticity, experience-dependency, and indirect gene-behaviour mapping, enables us to arrive at useful reformulations for future modularity debates.

First, with respect to localization, the important question for future theorizing will be why is it that certain areas take on the functions that they do in default circumstances, and why can other areas support the same functions with a fair degree of success in certain atypical circumstances but not others? With respect to ontogenetic and pathological universals, we need to ask: how can we characterize the developmental process in each case? How do altered sensitivities affect the developmental process and give rise to different phenotypic outcomes? With respect to domain-specificity, future theorizing should focus on the following issues: are there general principles by which our species handles and organizes input to produce coherent units and learn regularities? And finally, how do these domain-relevant mechanisms progressively give rise to the specialized and complex adult brain? In our view, these types of questions will further the modularity debate on the relations between syntax, the lexicon, and other neurocognitive domains, beyond the claims of adult neuropschological models, and where the gradual process of development is centre stage.

References

Bates, E., & Goodman, J. (1997). On the inseparability of grammar and the lexicon: Evidence from acquisition, aphasia and real-time processing. In G. Altmann (Ed.), Special issue on the lexicon, *Language and Cognitive Processes, 12(5/6)*, 507-586.

Bates, E., Thal, D., Finlay, B.L., & Clancy, B. (in press). Early language development and its neural correlates. To appear in F. Boller & J. Grafman (Series Eds.) & I. Rapin & S. Segalowitz (Vol. Eds.), *Handbook of neuropsychology, Vol. 7: Child neurology* (2nd ed.). Amsterdam: Elsevier.

Bellugi, U., Lichtenberger, L., Jones, W., Lai, Z., George, M. S. (2001). The neurocognitive profile of Williams syndrome: A complex pattern of strengths and weaknesses. In Bellugi, U. (Ed), St. George, M. (Ed). *Journey from cognition to brain to gene: Perspectives from Williams syndrome.* Cambridge, MA, US: The MIT Press.1-41.

Brown, J., Johnson, M.H., Paterson, S., Gilmore, R., Gsödl, M., Longhi, E., & Karmiloff-Smith, A. (2003). Spatial Representation and Attention in Toddlers with Williams Syndrome and Down Syndrome. *Neuropsychologia, 41,* 1037-1046.

Clahsen, H., & Almazan, M. (1998). Syntax and morphology in Williams syndrome. *Cognition, 68,* 167-198.

Chiat,S. (2001). Mapping theories of developmental language impairment: premises, predictions and evidence. *Language and Cognitive Processes, 16,* 113-142

de Haan, M., Pascalis, O., & Johnson. M.H. (2002). Specialisation of neural mechanisms underlying face recognition in human infants. *Journal of Cognitive Neuroscience, 14,* 199-209.

Deruelle, C., Mancini, J., Livet, M. O., Casse-Perrot, C. & de Schonen, S. (1999). Configural and local processing of faces in children with Williams syndrome. *Brain & Cognition, 41*, 276-98.

Dick, F., Bates, E., Wulfeck, B., Utman, J. A., Dronkers, N., & Gernsbacher, M. A. (2001). Language deficits, localization, and grammar: evidence for a distributive model of language breakdown in aphasic patients and neurologically intact individuals. *Psychological Review, 108,* 759-788.

Dronkers, N. F. (2000). The pursuit of brain-language relationships. *Brain & Language, 71*, 59-61.

Elman, J. L., Bates, E., Johnson, M.H., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness: A connectionist perspective on development*, Cambridge, MA: MIT Press.

Fodor, J. A. (1983). *Modularity of Mind.* Cambridge, MA: MIT Press.

Gauthier, I., Behrmann, M., & Tarr, M. J. (1999). Can face recognition really be dissociated from object recognition? *Journal of Cognitive Neuroscience, 11, 349-370.*

Gauthier, I., Skudlarski, P., Gore, J. C., & Anderson, A. W. (2000). Expertise for cars and birds recruits brain areas involved in face recognition. *Nature Neuroscience, 3*, 191-197.

Goodglass, H. (1993). *Understanding Aphasia.* San Diego: Academic Press.

Grice, S., Spratling, M.W., Karmiloff-Smith, A., Halit, H., Csibra, G., de Haan, M., & Johnson, M.H. (2001). Disordered visual processing and oscillatory brain activity in autism and Williams Syndrome. *Neuroreport, 12,* 2697-2700

Heiser, M., Iacoboni, M., Maeda, F., Marcus, J., & Mazziotta, J. C. (2003). The essential role of Broca's area in imitation. Eur J Neurosci, 17(5), 1123-1128.

Hill, E. L. (2001). Non-specific nature of specific language impairment: a review of the literature with regard to concomitant motor impairments. *International Journal of Language and Communication Disorders, 36,* 149-171.

Johnson, M. (1996) *Developmental cognitive neuroscience*. Blackwell.

Kan, I. P. & Thompson-Schill, S. L. (2003). Effect of name agreement on prefrontal activity during overt and covert picture naming. *Cognitive, Affective & Behavioral Neuroscience*, *4*, 43-57.

Karmiloff-Smith, A. (1992). *Beyond Modularity: A Developmental Perspective on Cognitive Science.* Cambridge, Massachusetts: MIT Press.

Karmiloff-Smith, A. (1997). Crucial differences between developmental cognitive neuroscience and adult neuropsychology. *Developmental Neuropsychology, 13*, 513-524.

Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences, 2,* 389-398.

Karmiloff-Smith, A., G. Scerif, & Ansari, D. (2003). Double dissociations in developmental disorders? Theoretically misconceived, empirically dubious. *Cortex*, *39*, 161-3.

Karmiloff-Smith, A., & Thomas, M. (in press) Can developmental disorders be used to bolster claims from Evolutionary Psychology? A neuroconstructivist approach. In J. Langer, S. Taylor Parker & C. Milbrath (Eds.) *Biology and Knowledge Revisited: From Neurogenesis to Psychogenesis.*

Karmiloff-Smith, A., Thomas, M., Annaz, D., Humphreys, K., Ewing, S., Brace, N., van Duuren, M., Pike, M., Grice, S., & Campbell, R. (in press). Exploring the Williams Syndrome Face Processing Debate: The importance of building developmental trajectories. *Journal of Child Psychology and Psychiatry.*

Leonard, L. B. (1998). *Children with specific language impairment.* Cambridge, MA, US: The MIT Press.

Newport, E.L., & Aslin, R.N. (2000). Innately constrained learning: Blending old and new approaches to language acquisition. In S.C. Howell, S.A. Fish, and T. Keith-Lucas (Eds.), *Proceedings of the 24th Annual Boston University Conference on Language Development.* Somerville, MA: Cascadilla Press.

Pinker, S. (1994). *The language instinct.* Penguin Books.

Pinker, S. (1999). *Words and rules.* London: Weidenfeld & Nicolson.

Maess, B., Koelsch, S., Gunter, T. C., & Friederici, A. D. (2001). Musical syntax is processed in Broca's area: an MEG study. *Nature Neuroscience, 4*, 540-545.

McCloskey, M. (1993). Theory and evidence in cognitive neuropsychology: a "radical" response to Robertson, Knight, Rafal, and Shimamura (1993). *Journal of Experimental Psychology: Learning, Memory, and Cognition, 19,* 718–734.

Plaut, D. C. (1995). Double dissociation without modularity: Evidence from connectionist neuropsychology. *Journal of Clinical and Experimental Neuropsychology, 17,* 291–321.

Poeppel, D. (1996). A critical review of PET studies of phonological processing. *Brain and Language, 55*, 317-351.

Quartz, S. R. & Sejnowski, T. J. (1997). The neural basis of cognitive development: A constructivist manifesto, *Behavioural & Brain Sciences, 20*, 537–596.

Ramus, F., Rosen, S., Dakin, S. C., Day, B. L., Castellote, J. M., White, S., & Frith, U. (2003). Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults. *Brain, 126*, 841-865.

Reilly, J., Bates, E.., & Marchman, V. (1998). Narrative discourse in children with early focal brain injury. In M. Dennis (Ed.), Special issue, Discourse in children with anomalous brain development or acquired brain injury. *Brain and Language, 61,*335-375.

Robertson, L. C. K., Knight, R. T., Rafal, R., Shimamura, A. P. (1993). Cognitive neuropsychology is more than single-case studies. *Journal of Experimental Psychology: Learning, Memory, & Cognition, 19*, 710-717.

Rosen, S. (2003) Auditory processing in dyslexia and specific language impairment: Is there a deficit? What is its nature? Does it explain anything? *Journal of Phonetics, 31*, 509-527.

Satz, P., E. Strauss, and H. Whitaker (1990). The ontogeny of hemispheric specialization: some old hypotheses revisited. *Brain and Language, 38*, 596-614.

Shallice, T. (1988). *From neuropsychology to mental structure*. New York, NY: Cambridge University Press.

Sperber, D. (2002). In defence of massive modularity. In E. Dupoux: *Language, Brain and Cognitive Development: Essays in Honor of Jacques Mehler*. Cambridge, Mass. MIT Press. 47-57

Stein, J. (2001). The magnocellular theory of developmental dyslexia. *Dyslexia, 7*, 12-36.

Temple, E., Deutsch, G. K., Poldrack, R. A., Miller, S. L., Tallal, P., Merzenich, M. M., & Gabrieli, J. D. (2003). Neural deficits in children with dyslexia ameliorated by behavioral remediation: evidence from functional MRI. *Proceedings of the National Academy of Science USA,* 100, 2860-2865.

Thal, D., Marchman, V., Stiles, J., Aram, D., Trauner, D., Nass, R., & Bates, E. (1991). Early lexical development in children with focal brain injury. *Brain and Language, 40,* 491-527**.**

Thomas, M. S. C., Grant, J., Barham, Z., Gsödl, M., Laing, E., Lakusta, L., Tyler, L. K., Grice, S., Paterson, S. & Karmiloff-Smith, A. (2001). Past tense formation in Williams syndrome. *Language and Cognitive Processes*, *16 (2/3)*, 143-176.

Tranel, D., Logan, C. G., Frank, R. J., Damasio, A. R. (1997). Explaining category-related effects in the retrieval of conceptual and lexical knowledge for concrete entities: Operationalization and analysis of factors. *Neuropsychologia, 35, 1329-1339.*

Ullman, M.T., & Gopnik, M. (1999). Inflectional morphology in a family with inherited specific language impairment. *Applied Psycholinguistics*, *20*, 51–117.

van der Lely, H. K., Rosen, S., & McClelland, A. (1998). Evidence for a grammar-specific deficit in children. *Current Biology*, *8*, 1253-1258.

Wulfeck, B., & Bates, E. (1991). Differential sensitivity to errors of agreement and word order in Broca's aphasia. *Journal of Cognitive Neuroscience,3*, 258-272.

Further Reading

Elman, J. L., Bates, E., Johnson, M.H., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness: A connectionist perspective on development*, Cambridge, MA: MIT Press.

Fodor, J. A. (1983). *Modularity of Mind.* Cambridge, MA: MIT Press.

Johnson, M.H. (2001). Functional brain development in humans. *Nature Reviews Neuroscience*, *2*, 475-483.

Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences*, *2*, 389-398.

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Professor Annette Karmiloff-Smith is Head of the Neurocognitive Development Unit, Institute of Child Health, London where she runs a team examining typical and atypical infant and child development, her main interest being how basic deficits in infancy impact differentially on a developing system to result in domain-specific outcomes. A "Doctorat en Psychologie Génétique et Expérimentale" from the University of Geneva, she was awarded, in 1995, the British Psychological Society's Book Award for her book *Beyond Modularity* (MIT Press, 1992). She recently published with her daughter, Kyra Karmiloff, *Pathways to Language: From foetus to adolescent* (Harvard University Press, 2001).