forced to conclude that undirected or broadly but not specifically directed growth followed by pruning is the necessary principal mechanism of neural selection. The theory presented by Q&S, once shorn of its unsupportable suggestions of directed mutation, differs from earlier theories in its emphasis on a closer temporal coupling of growth and selection processes, but it is otherwise substantially the same theory distinguished with a new name.

3. Summary. Q&S make a valuable contribution by reminding us that postnatal development leads to increased anatomical complexity of the nervous system which must be taken into account in constructing theories of learning. They have also very properly emphasized the importance of forming mappings with local coherence, which are generally lacking in connectionist networks. However, they have taken a "directed mutation" stance in regard to the mechanisms for formation of new anatomical diversity, which essentially defines away the very real problem of how the system can "discover" appropriate behaviors in previously unexperienced situations. This approach exaggerates the differences between their theory and earlier selectionist approaches.

The right way, the wrong way, and the army way: A dendritic parable

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Abstract: We suggest that neither selectionism nor constructivism alone are responsible for learning-based changes in the brain. On the basis of quantitative structural studies of human brain tissue it has been possible to find evidence of both increase and decrease in tissue mass at synaptic and dendritic levels. It would appear that both processes are involved in the course of learning-dependent changes.

The neurosciences have seen more than their share of impassioned conceptual dualities. Reticularism versus neuronism and "soup versus spark" synaptic transmission dynamics are two among many that come to mind. It is interesting to recall that neural reality was finally determined to encompass both poles of each duality. Neurons were indubitably separate entities but in the case of gap junctions, virtually continuous through the agency of connexions establishing structural continuity for ion flow. Neurons clearly communicate through the agency of neurotransmitter release but "electrical" transmission remains a reality at gap junctions. I would suggest that we may be dealing with another impassioned duality in the matter of "selectionism versus constructivism."

Quartz & Sejnowski (Q&S) have presented powerful evidence supporting environmentally engendered dendritic and axonal growth. Ideationally implicit in the work of Ramon y Cajal (1952) and further explored at the conceptual level by Hebb (1949), the conjectures became reality at the chemical level in the studies of Krech et al. (1960) and, at the structural level, in the extended series of studies by Diamond and her collaborators (1964; 1975; 1988) and in a number of other findings. Our own quantitative histological studies of human cerebral cortex argued strongly for causal links between computational complexity and structural complexity (Scheibel et al. 1990). Thus dendritic elaboration in the primary sensory cortical representational area for hand and fingers was significantly greater than that in the adjacent area for trunk representation. Furthermore, there were "suggestive associations between the complexity of dendrite systems of the hand-finger zone of the primary receptive area and the nature of the work with which the individual had been associated during his/her working life" (Scheibel et al. 1990, p. 85). Furthermore the conjoint development of language facility and waxing dendrite elaboration in Broca's area of the language-dominant hemisphere (Simmonds & Scheibel 1989) provided correlative if not causal relations between escalating cognitive demands and expanding neuropil.

Arguments can also be advanced for selectionism, however. In several series of electron microscope studies performed on rodents, measurable and significant decreases in the number of synaptic terminals in cortical axo-spino-dendritic synapses accompanied exposure to enriched environments (e.g., Mollgard et al. 1971). Individual synaptic terminals showed significant increase in the length of the postsynaptic thickening, thereby suggesting the presence of fewer, but larger and more effective synapses in environmentally enriched animals. Further analysis of these changes indicated that the effects of enriched environmental input as expressed in loss of synaptic terminals and enlargement of the remainder actually increased with age (Diamond et al. 1975). And the enriched rats were quicker maze-learners than their non-enriched mates (Diamond 1988)!

Assuming that a complex interweaving of dendritic/synaptic gain and loss are involved in the maturation-learning process, a third mechanism seems intertwined with these two, adding to the richness and subtlety of the process. Quantitative comparisons of dendritic tissue in Broca's area of left and right hemispheres revealed an unexpected result (Scheibel et al. 1985). There was no significant difference between the total dendritic length of neurons on either side. What did differ was the amount of dendritic length 'invested' in various portions of each dendritic tree. On the right, the non-language-dominant side, most of the dendrite length was involved in the first three orders of dendrite branching. On the language-dominant side, a much greater proportion of dendrite length was devoted to the outer branches (fourth, fifth, sixth order dendrite branches, etc.). Note that the inner, lower order branches developed earlier in the developmental history of the individual, while the outer branching segments developed later. Thus both *temporal patterns* of development and *position* on the dendrite tree were significant parameters in CNS growth and maturation. Note also, that successive additions to the periphery of the dendrite ensemble should (at least theoretically) not affect the more central parts of the dendrite system where synaptic patterns had presumably already been established. However, more than a tidy "add-on" effect was noted here. Our data (Simmonds & Scheibel 1989) strongly suggested that along with the pattern of use-dependent centrifugal *growth* there was also a related (and presumably use-dependent) partial *resorption* of lower order branches more centrally located within the dendrite ensemble. Simultaneous involvement of cortical dendritic tissue gain and loss during the maturation-learning process argues for the inextricable combination of constructivist and selectionist processes.

Neural constraints on cognitive modularity?

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Abstract: Is (some) innate *cognitive* modularity consistent with a lack of innate *neural* modularity? Quartz & Sejnowski's (Q&S's) implicit negative answer to this question fuels their antinativist and antimodular cognitive conclusions. I attempt here to suggest a positive answer and to solicit discussion of this crucial issue.

Cognitive development figures prominently in the title and concluding paragraphs of this target article, but receives too little discussion in the middle. I would like to solicit a more explicit discussion of the precise relationship between the neural evidence and cognitive conclusions proffered by Q&S. The burning question: *Is (some) innate cognitive modularity consistent with a lack of innate neural modularity?* The answer, for Q&S, appears to be (a quite implicit) "no." I would like to question this assumption.

The essence of modularity is a restriction on information flow. A function is modular to the degree that it is "informationally

encapsulated" – such that some of the information outside the module is not accessible from within. *Cognitive* modularity then concerns the impenetrability of the function by outside cognitive influences (e.g., Pylyshyn 1985). The converse may also hold: some of the "inter-levels" within the module may not be available from the outside. These restrictions on information flow engender several other symptoms: modules are typically fast, unconscious, obligatory, computationally shallow, and possessed of characteristic breakdown patterns. Some writers have suggested that the conjunction of all these symptoms is required for modularity (e.g., Garfield 1994), but this is not an apt characterization. Fodor is sometimes ambiguous on this question, but it seems clear that "informational encapsulation is . . . the essence of . . . modularity" (Fodor 1983, p. 71). [See also *BBS* multiple book review of Fodor's *The Modularity of Mind BBS* 18(1) 1985.]

Cognitive modularity thus imposes no constraints on how these patterns of information flow are realized in the underlying biological architecture (neural or otherwise): the relevant cognitive functions could be neurally implemented in a highly distributed fashion, even though they comprise a module at the cognitive level. This is not to say that cognitive modules *cannot* be neural modules – indeed, that they are often seems to be the case, and neural localization may often be useful evidence *for* cognitive modularity. I am instead trying to emphasize our vast ignorance concerning the mechanics of the biological implementation of cognition by pointing out that cognitive modules *need not* be neurally localized. This is often recognized by cognitive researchers. Segal (1996), for example, comments in a discussion of the modularity of "theory of mind" that "it's at least *a priori* possible that distributed, global characteristics of the brain, rather than [neurally] modular ones, realise computational or intentional modules" (pp. 145–146).

Q&S fail to address such considerations. They suggest that "the view that strong, domain-specific knowledge is built into cortical structures runs into severe difficulties from developmental neurobiological evidence" (sect. 4.1.1, para. 2). This depends, I think, on an overly restricted view of how exactly this "knowledge" could be realized neurally. Q&S do not discuss precisely what they mean by modularity (though they cite Fodor 1983), but they have elsewhere clarified that from their perspective "the modularity thesis becomes a question of whether some cortical regions are prespecified for domain-specific knowledge that determines *a priori* the computation a region may perform" (Quartz & Sejnowski 1994, p. 726).

I think this is simply incorrect. The modularity thesis (in this sense) concerns whether there exists domain-specific knowledge, *period* – and imposes no constraints on how that knowledge might be implemented in the underlying neural architecture. (Actually, that's not quite right either. Cognitive modules are also often domain-specific, but this need not always be the case: domain specificity refers to the sorts of inputs a system can process, while informational encapsulation refers to the information that the system can make use of whilst processing. This misreading of modularity will not matter here, since the same considerations about implementation apply whether you're talking about encapsulation or domain-specificity.) In any case, I think that in characterizing modularity this way, Q&S greatly diminish the impact of their arguments for the many cognitive scientists who use different notions of modularity. Q&S manage to draw anti-modular cognitive conclusions from their neurobiological evidence, but only because the relevance of that neural evidence crept illicitly into their initial (and implicit) conception of cognitive modularity in the first place.

Now, as Karmiloff-Smith (1992) and others have stressed, it is conceivable that some sorts of cognitive modularity may actually be acquired, without any innate predisposition. [See also *BBS* multiple book review of Karmiloff-Smith's *Beyond Modularity BBS* 17(4) 1994.] My comments thus far have been agnostic with regard to this issue. I have been suggesting that some cognitive modularity – however acquired – may be consistent with a lack of neural modularity. The same sorts of considerations, however, may also bear on the question of innate cognitive structure more generally. Q&S marshal an impressive array of evidence that cortical development can be affected by the environment in all sorts of ways (see their Table 4). Is this consistent with a degree of innate *cognitive* structure? Q&S require a negative answer, which again is simply assumed. But the fact that particular cells' functions depend on their interaction with the environment (see their sect. 2.3.3) may have no implications at all for how these cells will end up being used by innately determined *cognitive* processes. In any case, the specific types of environmental effects matter crucially here. No nativist ever denied the importance of environmental impact – witness appeals to triggering, imprinting, parameters, critical periods, and so on. To address nativism about cognitive structure, neurobiological evidence would have to demonstrate that bona fide *learning* was involved (see Pylyshyn 1985, especially pp. 409–14).

It remains an open question whether neurobiological equipotentiality is relevant to the evaluation of theories of cognitive modularity: at this point, we simply haven't a clue how cognition is built into the underlying, implementing biological architecture. At bottom, therefore, I think many cognitive scientists and neuroscientists simply assume different answers to the burning question above, in pursuing their research. This issue is still quite controversial, even within cognitive neuroscience (cf. Sarter et al. 1996), and this may be the ideal forum in which to devote some explicit discussion to these assumptions. Quartz & Sejnowski suggest that their theory "provides a meeting ground for cognitive scientists and neuroscientists" (sect. 5, para. 2). Maybe so; I would like to suggest the issues raised here as among the first items on the meeting's agenda.

Deconstructing neural constructivism

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Abstract: Activity-dependent processes play an active role in shaping the structure of neuronal circuitry and therefore contribute to neural and cognitive development. Neural constructivism claims to be able to account for increases in the complexity of cognitive representations in terms of directed growth of neurons. This claim is overstated, rests on biased interpretations of the evidence, and is based on serious misapprehensions of the nature of somatic variation and selection.

Much evidence points toward the importance of correlated neuronal activity in forming and maintaining neuronal architecture. Such correlations occur throughout an animal's lifetime, in parallel with developmental processes such as structural regression and growth of neural connectivity. Correlated neural activity may reflect important aspects of the statistical structure of the environment and is thought to be a key factor in neural plasticity. In their target article, Quartz & Sejnowski (Q&S) review a variety of experimental and computational results and link structural neuronal plasticity to learning theory. Their aim is a comprehensive account of cognitive development in terms of environmentally guided directed growth of neurons and connections. This so-called "neural constructivism" is counterposed to what is called the dominant or popular view: selectionism. Q&S's reasoning is flawed, however, and the synthesis they attempt to reach ultimately eludes their grasp.

Q&S's "selectionism" is a caricature at best, a straw man deliberately erected only to be knocked down. No one has ever proposed that all of brain development can be subsumed in a two-stage process, initial overproduction and subsequent elimination. Quite the contrary, according to selectionism (Edelman 1987; 1993; Sporns & Tononi 1994; Sporns 1997a; 1997b), the basic processes of selection in the nervous system overlap temporally throughout