Toward a Developmental Evolutionary Psychology:

Genes, Development, and the Evolution of the Human Cognitive Architecture

Steven R. Quartz[1]
Division of Humanities and Social Sciences, and
Computation and Neural Systems Program
California Institute of Technology

Citation: Quartz, S.R. (2002). Toward a Developmental Evolutionary Psychology: Genes, Development, and the Evolution of the Human Cognitive Architecture. In *Evolutionary Psychology: Alternative approaches*, Scher, S. and M. Rauscher (Eds). Kluwer.

1. Rethinking the Relationship Between Development and Evolution

1.1 The Modern Synthesis and What Was Left Behind

After a century of intermittent dialogue between psychology and evolutionary biology, the outline of a synthesis between the two disciplines now appears to be emerging. The current form of this synthesis, known as narrow evolutionary psychology (Barkow et al., 1992; Buss, 1999), is the union of two specific frameworks from evolutionary biology and psychology. Specifically, narrow evolutionary psychology brings together the Modern Synthesis of evolutionary biology, which views evolutionary change primarily in terms of changes in gene frequency, with a nativist cognitive psychology, which views the mind as a collection of relatively autonomous, specialized processors, or modules (Hirschfeld & Gelman, 1994). As I outline in more detail below, both strands of evolutionary psychology are largely adevelopmental. There is accumulating evidence, however, that both evolutionary and psychological theory must incorporate a developmental perspective in order to construct successful theory. For example, it is now well established that a major route to evolutionary change is via alterations in developmental programs. If this is indeed the case, then evolutionary change must act in accordance with the range of possible changes to these programs, which in the case of behavior and cognition involves alterations to the development of the brain. From a psychological perspective, it is increasingly clear that ontogeny plays a far more central role in shaping behavior and cognition than its marginalization in nativist cognitive psychology allows (Quartz & Sejnowski, 1997). Any evolutionary psychology integrative framework must therefore take development seriously.

In recent years, new evolutionary and psychological frameworks have emerged that are thoroughly developmental in perspective. In evolutionary theory, this developmental perspective is known as evolutionary developmental biology (Raff, 1996; Arthur, 1997; Hall, 1998); in psychology, this developmental perspective is known as developmental cognitive neuroscience (Elman et al., 1996; Quartz & Sejnowski, 1997) .

To date, evolutionary developmental biology and developmental cognitive neuroscience have not been brought together to construct an alternative evolutionary psychology framework that places development at its center to explicitly account for the evolution of cognition in terms of developmental alterations to neural structures. In this chapter, I present the preliminary outlines of such a view, which I refer to as "developmental evolutionary psychology." From this perspective, I will suggest that failing to take into account how evolution acts on developmental programs to regulate alterations in brain structure and function has resulted in a seriously distorted view of the evolution of cognition and the resulting human cognitive architecture; it has also contributed to a mischaracterization of the role of culture in human development and evolution. In contrast, I will suggest that developmental evolutionary psychology suggests an alternative view of the human cognitive architecture that replaces the highly modular view of narrow evolutionary psychology with a behavioral systems view. As I explore below, this perspective views the human cognitive architecture as a hierarchically organized control structure, where this hierarchical organization is evident both evolutionarily and developmentally. Additionally, this perspective provides a principled means of incorporating recent results from cognitive neuroscience, which have tended to be marginalized in narrow evolutionary psychology. This, then, furthers the naturalistic stance narrow evolutionary psychology advocates but falls short of achieving. Finally, I will present evidence from paleoclimatology that suggests the environment of evolutionary adaptation (EEA) was markedly different from the account most prevalent in narrow evolutionary psychology. This evidence suggests that the selective forces underlying the evolution of the human cognitive architecture were critically connected to highly unstable climes, as there is an intriguing temporal coincidence of rapid hominid encephalization and increasing ecological instability during the Middle Pleistocene (600-150 kyr BP; Ruff et al., 1997). Based on these considerations, I suggest that an important feature of hominid evolution was a process I have referred to as progressive externalization (Quartz & Sejnowski, 1997; Quartz, 1999), whereby the brain's development became increasingly regulated by extrinsic factors, likely mediated by heterochronic changes in neural development. I suggest that this process allowed for flexible prefrontally mediated cognitive function, particularly in the social domain, and

underlies the capacity for rapid changes in social structure that was a response to the need for buffering ecological instability (see also Potts, 1996). The upshot of this process was symbolic culture, which plays a central role in shaping the structures underlying human cognition (Tomasello, 1999).

1.2 Taking Development Seriously

Both components of narrow evolutionary psychology, evolutionary biology and nativist cognitive psychology, are largely adevelopmental. Regarding the latter, Chomsky's work on language acquisition has been highly influential in the field of cognitive development. Chomsky's principal argument concerned the relative impoverishment of the environment as an informational source, which he argued was too barren to provide sufficient information for a child equipped only with general learning mechanisms to converge on a grammar that would underlie language competence (see Cowie, 1998). Aimed against B.F. Skinner's behaviorist analysis of language use, Chomsky argued that the child must bring certain knowledge of grammar to the task of language acquisition *a priori*. This knowledge took the form of a language organ, a structure containing domain-specific knowledge.

Although Chomsky hypothesized such a specialized structure only for language, his arguments proved compelling and resulted in a widespread adoption of domain-specific organs, or modules, for a variety of other cognitive capacities, such as social cognition (Brothers & Ring, 1992) and numerical cognition (Dehaene et al., 1999). This view has led to a widely adopted modular view of the mind (Fodor, 1983; Hirschfeld & Gelman, 1994). This, in turn, has had major consequences for the specific proposals of narrow evolutionary psychology regarding the structure of the mind and its development. In particular, Tooby and Cosmides'(1992, pp. 93ff.; Cosmides and Tooby, 1994) critique of the Standard Social Science Model echoed many of Chomsky's arguments against behaviorism's general learning strategies (for an evaluation of many of these arguments in their evolutionary context, see Fodor, 2000).

Although nativist cognitive psychology has been largely adevelopmental, the rise of cognitive neuroscience in recent years has spawned a growing interest in development.

This work has in turn given rise to a growing developmental cognitive neuroscience (Elman et al., 1996; Johnson, 1997; Quartz, 1999). In contrast to nativist cognitive psychology, the cornerstone of developmental cognitive neuroscience is a series of new experimental results; the findings range from developmental neural plasticity at the systems level to single cell physiology, as I explore in more detail below. Together, these results indicate that human development is both more protracted and more sensitive to environmental signals than nativist cognitive psychology supposed; this makes it important to understand the implications of these results for an evolutionary psychology integrative framework.

As stated above, there is another source of narrow evolutionary psychology's adevelopmental perspective: the Modern Synthesis in evolutionary biology. Although embryology and evolution were considered together in the late 19th century, by the 1920s and 1930s Morgan, Dobzhansky, and others provided powerful arguments for why evolutionary biology should move away from the ties to embryology that Haeckel, Weissmann and others emphasized (for a review, see Gilbert et al., 1996). In place of embryology, the shapers of the Modern Synthesis argued that transmission genetics provided the most appropriate coupling with natural selection, culminating in the view that evolution is essentially changes in gene frequency. Despite the attempts of Goldschmidt in the 1930s, and Waddington in the 1940s and 1950s, to combine evolution and development, little progress was made integrating development into evolutionary theory until recently. Only with major advances in the molecular genetics of development, beginning in the 1980s, have inroads been made regarding adding a developmental perspective to evolutionary theory. Specifically, this work has begun to demonstrate the "deep structure" of development, beginning with fundamentally important insights into the homologous developmental pathways underlying a variety of embryonic processes from drosophilia to mammals. The cornerstone result of this work was the discovery of homeobox genes and their striking conservation (reviewed in Hirth & Reichert, 1999; Reichert & Simeone, 1999). Given the enormous differences in neuroanatomy between vertebrates and invertebrates, their brains were long thought to be unrelated with little obvious homology. However, at a deeper, molecular level they are remarkably similar in that homologous regulatory genes have been identified that control

regionalization, patterning, and identity in embryonic brain development. So striking are these new results that it now appears unlikely that successful evolutionary theory can be constructed that does not have a prominent place for development. Based on such observations, the nascent field of evolutionary developmental biology has emerged (for a review, see Raff, 2000).

The emergence of developmental cognitive neuroscience and evolutionary developmental biology in recent years suggests a possible major reorientation of evolutionary psychology. As I have indicated, the major shortcoming of narrow evolutionary psychology is its marginalization of development, whose incorporation now appears essential to any satisfactory account of an evolutionary framework for human cognition and behavior. In what follows, I will sketch the outlines of such an approach that begins to integrate developmental cognitive neuroscience and evolutionary developmental biology into a view I refer to as developmental evolutionary psychology. As I explore, this approach adds an additional important constraint. Whereas both the Modern Synthesis and nativist cognitive psychology largely treat the brain as a black box, both evolutionary developmental biology and developmental cognitive neuroscience explicitly address the issue of neural structures and mechanisms. This, then, allows developmental evolutionary psychology to further the naturalistic perspective narrow evolutionary psychology advocates but falls short of by relegating neuroscience to a minor role (e.g., Tooby & Cosmides, 1992). Because developmental evolutionary psychology is explicit about the mechanisms and structures underlying cognition and behavior, it examines the nature of the deep structure in the fundamental patterning of vertebrates that both constrains possible evolutionary changes and facilitates morphological alterations along certain routes. The central questions developmental evolutionary psychology asks, then, are, what alterations in developmental mechanisms and processes underlie the evolution of the structure and function of the brain and sensory systems, how are these reflected in the organization of the human cognitive architecture, and how do these generate human behavior and cognition?

In what follows, I sketch a response to these central questions that incorporates the insights of comparative neuroanatomy and the molecular genetics of development. Based on these results, I will suggest that narrow evolutionary psychology's model of the

human cognitive architecture, one based on massive modularity, is inconsistent with the permissible mechanisms underlying evolutionary alterations to neural structures. I then present an alternative, hierarchical behavioral systems view of the evolved human cognitive architecture that is based on integrating developmental cognitive neuroscience and evolutionary developmental biology into a developmental evolutionary psychology. More specifically, in section 2.1, I consider shortcomings in the trait-level analyses that have played a prominent role in adaptive thinking and the problematic inference to cognitive modules based on that level of analysis. To make this concrete, I examine Lovejoy et al.'s (1999) analysis of the hominid pelvis. This example demonstrates that relatively simple alterations in developmental programs can have a cascade effect and thereby alter systemic properties, such as the entire pelvic field, casting doubt on the sufficiency of trait level analyses. These considerations suggest that alterations in genomic regulatory systems is a key mediator of evolutionary change. Next, in section 2.2, I consider this insight from the perspective of brain evolution. In particular, I examine the striking finding that, despite a 10,000-fold range in neocortex size across mammals, the relative size of many brain structures is highly correlated. I review evidence indicating that heterochronic changes in the duration of neurogenesis result in the coordinated pattern of brain size across a variety of mammalian species. These results suggest that neural systems highly covary with one another as a consequence of the restricted range of permissible alterations that evolutionary change can act upon. This makes the massive modularity hypothesis of narrow evolutionary psychology untenable. In section 2.3, I then turn to consider whether these heterochronic changes may themselves reflect a deeper structure of biological design. I review evidence that demonstrates that much of the diversity of mammalian brains reflects the spatial organization of neural tube. This suggests that the range of permissible changes to the relative size of brain structures must reflect this deep structure of neural development.

Based on this evidence, I conclude that, since evolutionary changes involve heterochronic alterations to developmental programs that result in systemic changes throughout the brain, narrow evolutionary psychology's modular account of the human cognitive architecture should be replaced by one that views the brain as a collection of behavioral systems. In section 3, I present a proposal for one such behavioral systems

view that is based on comparative work that identifies common design principles across a wide variety of nervous systems. This behavioral systems view places development at its center, suggesting an alternative evolutionary psychology framework that integrates developmental cognitive neuroscience and evolutionary developmental biology. In section 4, I consider recent work in paleoclimatology that suggests that the ecological conditions that may have driven hominid brain evolution were markedly different from the proposals of narrow evolutionary psychology and accords better with the model of cognitive architecture I present. In section 5, I examine this issue in more detail and consider how alterations in development may underlie the capacity for complex cultural learning that was a response to ecological instability.

2. Building Brains: Development and the Units of Selection

2.1. Traits, Genes, and the Morphogenetic Field

In narrow evolutionary psychology there is a deep connection between modules and genes (e.g., Pinker, 1997, p.32). An informal criterion of a module is that it has a semi-independent evolutionary account, which involves an analysis of genetic transmission under natural selection. As Sterelny and Griffiths (1999) note, narrow evolutionary psychologists follow the strategy of adaptive thinking in attempting to identify modules and their function. That is, a solution is inferred from the structure of a historical problem, which requires reconstructing the evolutionary context, or environment of evolutionary adaptation. This trait-level analysis of behavior depends on identified traits being separately heritable, corresponding to a module, so that evolution can act on the basis of that trait's variation. Such a view makes strong predictions regarding permissible evolutionary alterations to the substrates of cognition. Indeed, it is somewhat surprising that this modular account of the human cognitive architecture, and the evolutionary path to it via semi-autonomous selection of modules, has not been considered in terms of whether it is consistent with the emerging understanding of the paths to evolutionary change in nervous systems. There have, however, been many more

general cautions regarding the functional identification of a trait and its putative separate heritability (Dobzhansky, 1956; Gould and Lewontin, 1979). Without additional constraints, a behavioral analysis can lead to behaviors being seen as composed of numerous characters, or modules; such trait atomization relies on the assumption of particulate inheritance. Put another way, the capacity to functionally dissect behaviors into component parts in no way entails that those components are mediated by modules with separable heritability.

Recently, Lovejoy et al. (1999) analyzed the mammalian postcranium from a developmental perspective and demonstrated that trait atomization could lead to a serious distortion of cladistic analyses. Their specific example involved the transformation of the common ancestral pelvis into that of early hominids. They suggest that the evolution of the pelvis may have involved the modification of the geometry of pattern formation, such as a progressive increase in the slope of molecular gradients in the limb bud, initiating a developmental cascade that would alter the entire pelvic field. Thus, although it is possible to identify separable traits at the morphological level – sacrum, platypelloid birth canal, pubic symphysis, superior and inferior pubic rami, obturator foramina – none of these may have a unique evolutionary history nor be under separate selective pressure. Rather, the entire pelvis may be systemically modified as a function of alterations in development.

Gilbert et al. (1996) similarly suggest that incorporating results regarding the deep structure of developmental programs results in a major modification of the units of selection. Rather than the gene, they suggest that the morphogenetic field is the basic unit of ontogeny whose alterations mediate evolution. Such a perspective leads naturally to the view that changes in genomic regulatory systems are the mediators of evolutionary change (Davidson, 2001). This observation merits further investigation, as it is of central importance to an evolutionary psychology perspective. That is, what is the range of permissible evolutionary alterations to nervous systems, and does analysis at a behavioral trait level result in a distorted account?

2.2 Scaling Brains

The first question to ask is, what processes underlie changes in the size of brain structures? The organization and neuropharmacology of the brain stem, which mediates basic homeostatic functions, appear to be highly conserved across species (Ross et al., 1984). In contrast, there is a 10,000-fold range in neocortex size across mammals. A uniquely mammalian structure, neocortex occupies a disproportionate percentage of total brain mass in anthropoid primates (monkeys, apes, and humans), from 60-80% of the total (Nieuwenhuys et al., 1998). The disproportionate increase in neorcortex size in anthropoid primates is believed to reflect important cognitive and behavioral skills that underlie complex social and cognitive functions. Indeed, this disproportionate increase is referred to as encephalization and is the basis for the important view that anthropoid evolution is in part characterized by the increasing cortical mediation of complex behavior and cognition.[2]

Given the enormous range of neocortex size across mammals in terms of both relative and absolute sizes, it is important to consider what mechanisms and processes determine these differences and along what dimensions these differences lie. Neocortex is not unconstrained to change across all dimensions. Indeed, the thickness and the general organization of neocortex differs relatively little across species.[3] Rather, neocortex across species is organized into radially oriented, vertically interconnected columns, and shares a horizontal organization into layers designated I-VI. Cortical circuitry also shares basic themes, with deep layers (VI and V) sending efferent to subcortical and cortical structures, the middle layer (IV) receiving afferents from the thalamus, and with the upper layers (II and III) integrating information within the cortex. Phenotypic variability in the size, number, and interconnectedness of cortical areas thus underlie species differences in behavior and cognition.

Alterations in the size of neocortex appear to be mediated primarily by the number of neurons and their supporting elements. It is possible to increase the size of neurons, however this would require a complex recalibration of their physiological properties, which in turn would require novel biophysical mechanisms that instead appear to be highly conserved. Thus, while larger brains do tend to have larger neurons, this increased size is not highly significant and does not account for differences in brain size.

Based on this and other considerations, a possible route to neocortical diversity is via evolutionary modifications of the program of cortical development. The fact that neocortex does not vary across all dimensions, but retains common organizational themes such as radial and laminar organization, suggests that only a portion of cortical development programs differ, making it possible to use a comparative methodology across species to assess alterations in cortical development programs. Combined with other experimental techniques, such as gene knockout experiments, it is possible to identify the processes underlying neocortical diversity.

One process underlying alterations in the size of body parts, including the brain, involves alterations to cell proliferation. The three-dimensional neuronal organization of the neocortex develops from a two-dimensional sheet of proliferating cells during a restricted period of early development (McConnell, 1995; Rakic, 1988). During early gestation, the anterior-most end of the neural tube expands outwards, forming a pair of telencephalic vesicles that become the cerebral hemispheres. Neurons are not generated in the region they will occupy in the mature organism. Instead, they are generated in the ventricular zone (VZ), a primitive epithelial sheet of dividing cells that line the cerebral ventricles. Prior to neurogenesis, these progenitor cells divide symmetrically to establish a precursor pool. The onset of neurogenesis is marked by the first postmitotic cells leaving the ventricular zone and migrating along radial glial fibers, eventually forming a structure known as the cortical plate. As more postmitotic cells migrate out into the cortical plate, they do so in an inside-out temporal sequence, generating the layers of the neocortex, with later migrating cells forming the more superficial layers.

One potential route to building specialized brain structures, such as modules, would be through modulating the process of neurogenesis, whereby specific precursor populations would be generated and migrate to a specific neocortical site. For such a strategy to be feasible, the process of neurogenesis would have to be dissociable and thus restricted phases of neurogenesis would be under natural selection. It is possible to investigate this possibility through a comparative method, as it makes a strong prediction: if natural selection operates on neurogenesis in this way, then the size of some individual brain structures in different species should diverge from the relative size of other

structures. That is, the relationship among brain structures across species should not show strong signs of linked regularities.

Heinz Stephan and colleagues published a series of volumetric data sets for eleven brain divisions and for more discrete nuclei and zones for a large sample of insectivores, prosimians, simians, and bats (Stephen et al., 1981; Frahm et al., 1982) that has become a widely used dataset to analyze comparative brain structures. Using a factor analytic approach, Finlay and Darlington (1995) found that the size of various brain structures across 131 mammalian species was highly correlated, with the primary exception of the olfactory bulb. In general, the relative proportions of different brain structures can be highly predicted by overall brain size. Put another way, mammalian brains appear to scale in a highly coordinated fashion. These highly predictable relationships between the sizes of major brain structures indicate that diverse brains derive from a highly conserved homeotic starting point. Differences across species stem from alterations in global proliferative processes: heterochronic changes in the duration and/or rate of neurogenesis that result in linked regularities among brain structures. This suggests that restricted phases of neurogenesis are not independently under selective pressures. Further evidence to support this possibility stems from the striking finding that the order of neurogenesis—the order in which neuronal populations give rise to various brain structures – appears to be highly conserved across species (reviewed in Finlay et al., in press). During neurogenesis, many progenitor cells continue dividing symmetrically, producing one postmitotic cell and one progenitor cell. Thus, the pool of progenitor cells grows exponentially. This suggests that later generated structures will get proportionally larger, largely as a consequence of the exponential nature of symmetric cell division, as indeed the linked regularities discussed above confirm. That is, Finlay and Darlington (1995) found that greater durations of neurogenesis were correlated with a proportional increase in overall brain size and differential effects on the size of brain components, with later generated structures growing in a predicable fashion. As Finlay and Darlington (1995) put it, late makes large.

2.3 Heterochrony, Segmental Models, and the Deep Structure of Development

Given the coordinated, predictable shift in relative neural structures, a key concept that emerges is that of heterochrony, the phylogenetic variation in the relative timing of major developmental events (for a recent review of the notion of heterochrony, see Gould, 2000). Does the fact that heterochronic changes in the duration of neurogenesis result in the coordinated pattern of brain size across a variety of mammalian species reflect something about the deep structure of development? These linked regularities might indeed reflect a deeper structure of development, whereby the highly conserved order of neurogenesis reflects the spatial organization of the neuroaxes of the neural tube, a highly conserved organization that likely precedes vertebrates. One of the most important insights from the molecular genetics of development regards the fact that conserved regulatory genes have highly restricted spatial patterns of activity, underlying the segmental patterning of body plans. Segmentation is best understood in *Drosophilia*, where the basic segmented body plan is specified by positional information laid down in the early embryo by an interacting group of regulatory genes (reviewed in Pick, 1998; for a computational analysis, see Reinitz et al., 1998). Under the sequential, hierarchical action of these genes, the embryo is subdivided into increasingly specified body regions along the anterior-posterior axis. Morphogenesis is thus specified by a progressively restricted subdivision of the embryo, including the action of homeotic genes that assign an identity to established regions.

The central nervous system is composed of four major subdivisions: the spinal cord, hindbrain, midbrain, and forebrain. The forebrain (prosencephalon) mediates most higher cognitive functions, and includes such structures as neocortex, archicortex, and thalamus. The forebrain was long thought to be an exception to segmental models, as its topography appeared non-segmental, making it unclear how topographically organized developmental programs would operate. Rubenstein and colleagues (Rubenstein et al., 1994) suggested that the vertebrate forebrain does follow a segmental model, and postulated that dorsoventral (D/V) and anteroposterior (A/P) patterning mechanisms subdivide the embryonic forebrain into longitudinal and transverse domains. According to their prosomeric model, the embryonic forebrain is a neuromeric structure subdivided into a grid-like pattern of histogenic domains defined by longitudinal and transverse

boundaries, and so follows many of the deep developmental themes found in other segmental models.

Recently, Finlay et al. (1998; 1999) examined whether there was any relation between the conserved ordered of neurogenesis and the prosomeric model of brain organization. They found a strong relationship between position on the prosomeric axes and duration of neurogenesis. Specifically, more ventral and anterior regions have a more protracted period of neurogenesis, illustrating that the coordinated scaling of the relative size of brain structures is in part a reflection of the spatial organization of the neuraxes. This suggests that the range of permissible changes to the relative size of brain structures must reflect this deep structure of neural development. In particular, the exponential growth of neocortex relative to the rest of the brain may therefore be in part a consequence of its prosomeric location.

3. From Modules to Behavioral Systems: The Hierarchical Organization of Behavior and its Evolution

3.1 Common Nervous System Design Principles

The above considerations demonstrate that it is infeasible to view the neocortex as a collection of relatively autonomous modules. Rather, evolutionary changes in neural structures involve heterochronic alterations to developmental programs that result in systemic changes throughout the brain. As Finlay et al., (in press) state, “natural selection does not do its work on some equipotent substrate, but on a complex mechanism with a history of previous change that makes some adaptations more "workable" than others.” For these reasons, an analysis of the neural mediation of behavior, and the evolutionary paths available to alter such structures, requires abandoning a modular, trait atomistic view of the human cognitive architecture. In its place, I outline a model that is consistent with the results presented above. Specifically, I present a behavioral systems model that regards the brain as a hierarchical control structure, where this hierarchical organization is evident both developmentally and evolutionarily. This behavioral systems model

places a premium on the complex interaction between developmental mechanisms and a structured environment, and, therefore, rests on the second component of developmental evolutionary psychology, namely developmental cognitive neuroscience.

The existence of highly conserved nervous system developmental mechanisms suggests that nervous systems, despite their apparent diversity, share a deep structure, or common design principles, just as the fact that two million distinct species share only 35 major body plans suggest that body plans share many common design principles. Even the simplest motile organisms require control structures to regulate goal-directed behavior necessary for survival in a variable environment (for discussion, see Allman, 1999). For example, although the bacterium *E. coli* does not possess a nervous system, it does possess control structures for sensory responses, memory, and motility that underlie its capacity to alter behavior in response to environmental conditions. The capacity to approach nutritive stimuli and avoid aversive stimuli in the maintenance of life history functions is the hallmark of behavioral systems across phyla. Whereas chemotaxis in bacteria involves a single step from sensory transduction to motor behavior, some multicellular organisms embody control structures that involve intercellular communication via hormonal signaling, while others possess nervous systems with control structures that add layers of mediating control between sensory transduction and motor behavior.

There are several alternative design possibilities for biological control structures. One is to make a closed system, in the sense of linking fixed behavioral patterns between internal goal states and their environmental targets. Although there are many examples of this strategy (Gallistel, 1992), there are more powerful and flexible control structures. One such strategy involves leaving the path from internal goal state to target state open and discoverable via learning. Principal among this latter design strategy are reinforcement-based systems that are capable of learning an environment's reward structure.[4]

3.2 The Ubiquity of Reward Structures in Nervous Systems

A variety of experimental techniques, ranging from psychopharmacology to neural imaging, has demonstrated the striking ubiquity and conservation of reward structures across species. At virtually all levels of the human nervous system, for example, reward systems can be found that play a central role in goal-directed behavior (Schultz, 2000). Here, I focus on one such system, the midbrain dopamine system (Figure 1). The midbrain dopamine system projects principally from the ventral tegmental area to the nucleus accumbens and the temporal and frontal cortex. Studies utilizing self-stimulation paradigms revealed that activation of this system was highly reinforcing, often with laboratory animals preferring to self-stimulate this system than eat or copulate with a receptive partner (reviewed in Wise, 1996). Most addictive substances involve this system, giving rise to the hedonic theory of dopamine as the signal underlying pleasure (though see Garris et al., 1999).

Given what I have previously stated regarding the possibility that control structures are highly conserved, it is interesting to note, as Figure 1 illustrates, the striking homology between the dopamine system in humans and a reward system in the bumblebee. The bumblebee suboesophogeal ganglion contains an identified neuron, VUMmx1, which delivers information about reward during classical conditioning experiments via the neurotransmitter, octopamine, which is similar in molecular structure to dopamine (Hammer, 1993).

Both experimental and computational work on the role of VUMmx1 in bumblebee foraging has provided important insights into the signal carried by octopamine and the system's functional significance (Real, 1991; Montague et al., 1995). Rather than simply carrying information regarding reward, it appears that octopamine signals information regarding prediction errors. Whereas reward is traditionally a behavioral notion, prediction is a computational notion. The difference between certain rewarding outcomes and their predictions can be used to guide adaptive behavior. A system that learns through prediction learning need not have the path from goal to reward specified, in contrast to fixed behavioral patterns, such as stimulus-response learning. Instead, the path from goals to rewards may be left open and discoverable via learning, resulting in flexible action. Evolution, then, may shape the pattern of basic rewards

animals are motivated to obtain, but the behavioral path is left open to discovery, as are more complex relations among predictors. In this sense, brains are prediction machines that use information gathered from past experience to predict future events important for survival (reviewed in Montague and Quartz, 1999).

Experiments utilizing neurophysiological recording in behaving monkeys by Schultz and colleagues demonstrate that the midbrain dopamine system plays an important role in prediction learning in the mammalian brain (Schultz et al., 1993). When these monkeys were presented with various appetitive stimuli, dopaminergic neurons responded with short, phasic activations, which typically lasted for only a few repeated presentations. In an important finding, however, Schultz and colleagues found that when the rewarding stimuli was preceded by an auditory or visual cue, dopamine neurons changed their time of activation to just after the time of cue onset. In contrast, when the reward did not follow the conditioned stimulus, dopamine neurons were depressed below their basal firing rate exactly at the time the reward should have occurred. These results indicate that the dopamine signal encodes expectations regarding the delivery of reward. That is, the output of dopamine neurons code for an error between the actual reward received and predictions of the time and magnitude of reward. Like the octopamine signal in the bumblebee, the dopamine signal codes a prediction error that can be used in learning and in action selection. This mode of action is equivalent to Temporal Difference learning, a thoroughly examined form of reinforcement learning (Sutton and Barto, 1998) that learns the predictive structure of an environment. Simulations demonstrate that despite the apparent simplicity of this model, it is a very powerful learner, capable of learning master level backgammon, for example (Tesauro, 1995).

A variety of evidence supports the notion that this system works in a similar fashion in humans (though it is important to point out that this in no way is meant to be the exclusive locus of behavioral choice). For example, it is possible to design reward functions where the computational model of dopamine will pursue sub-optimal strategies. Montague and Quartz (1999) found that human choice behavior in a simple two-card task followed these sub-optimal strategies when faced with these anomalous reward functions. Berns et al (2001) have recently examined prediction learning directly with functional imaging, essentially replicating Schultz's monkey experiments in humans, and have

found activation of the midbrain dopamine system. These results suggest that the midbrain reward system in the human brain shares common functional properties with homologous reward systems across a diverse array of species.

3.3 The Hierarchical Structure of the Human Behavioral System

It is deeply intriguing to note where the midbrain dopamine system projects to in the human brain. In particular, what is most intriguing is the fact that it projects to dorsolateral prefrontal, premotor, and parietal cortex, which are structures believed to mediate goal representations, and the orbitofrontal cortex, which is believed to mediate the representation of relative reward value and reward expectation (for a review, see Schultz, 2000). A great deal of attention has centered on the dorsolateral and orbitofrontal prefrontal cortex as structures implicated in crucial components of human cognition, particularly social cognition and theory of mind (Stone et al., 1998), symbolic learning (Deacon, 1997), representations of self (Craik et al., 1999), and executive function and behavioral inhibition (Norman & Shallice, 1986).

In an evolutionary context, it is important to ask, what is the functional significance of the fact that a phylogenetically old part of the brain projects to a relatively phylogenetic newcomer? According to the view of developmental evolutionary psychology, these structures constitute a hierarchically organized control structure, where additional layers of control have been added to the evolutionarily conserved dopamine system and where this hierarchical organization is evident developmentally as well. To see how, it is important to examine the developmental links between these components, as I explore in more detail below.

Diamond and colleagues (reviewed in Diamond, 1998) have demonstrated that a functional midbrain dopaminergic system is necessary for normal development of prefrontal functions. The most compelling evidence regarding this developmental dependence stems from studies of Phenylketonuria (PKU). Patients suffering from PKU do not naturally produce a particular enzyme, phenylalanine hydroxylase, which converts the essential amino acid phenylalanine to another amino acid, tyrosine, the precursor of dopamine; when untreated, PKU leads to severe mental retardation.

Diamond and colleagues found that lowered levels of tyrosine uniquely affect the cognitive functions dependent on prefrontal cortex because of the special sensitivity of prefrontally projecting dopamine neurons to small decreases in tyrosine. In a 4-year longitudinal study, they found that PKU children performed worse than matched controls, their own siblings, and children from the general population on tasks that required the working memory and inhibitory control abilities dependent on dorsolateral prefrontal cortex. In contrast, these PKU children performed well on control tasks that were not mediated by prefrontal cortex (Diamond et al., 1997).

The hierarchical organization of the control structures that constitute the human cognitive architecture is apparent developmentally, with human cognition and behavior becoming increasingly mediated by frontal structures. In contrast to the early functional involvement of midbrain dopamine systems, prefrontal structures develop relatively late and exhibit a protracted development that continues into adolescence. Thus, behavior and cognition increasingly comes under the mediation of frontal structures from subcortical structures across development, a process sometimes referred to as frontalization of behavior (Rubia et al., 2000). For example, executive function is a control mechanism that guides, coordinates, and updates behavior in a flexible fashion, particularly in novel or complex tasks (Norman and Shallice, 1986). This requires that information related to behavioral goals be actively represented and maintained so that these representations may guide behavior toward goal-directed activities. In humans, executive function follows a special developmental trajectory, reflecting an evolutionary reorganization of prefrontal structures and their development. Between 7 ½ and 12 months of age, infants show a developmental progresson on A-not-B (Diamond, 1985), delayed response (Diamond and Doar, 1989), and object retrieval tasks (Diamond, 1988). There is substantial evidence that these tasks are mediated by dorsolateral prefrontal cortex and rely on working memory, neural representations of goal-related information, and behavioral inhibition (Goldman-Rakic, 1990; Petrides, 1995). Further, various sources of evidence indicate that dopamine is necessary for successful performance on these tasks (Sawaguchi and Goldman-Rakic, 1994).

3.4 Computational Links

Although there is strong evidence that an intact dopamine system is necessary for the developmental emergence of prefrontal functions, a largely unresolved question concerns the specific nature of this developmental link. One particularly intriguing possibility is that the dopamine signal serves as a learning signal that guides the construction of prefrontal structures during development. Computational work on the midbrain dopamine system suggests such a learning role with strong analogies to temporal difference learning, a form of reinforcement learning (Sutton & Barto, 1998). A key notion underlying reinforcement learning is that of learning through interacting with one's environment. For example, a major source of knowledge stems from an infant's interactions with its environment, which produces a wealth of information about cause and effect, about the consequences of actions, and about what to do in order to achieve goals—all without the need for an explicit teacher. Of course, Piaget also emphasized the central importance of the developing child's agency and active exploration with its environment in his constructivist theory of cognitive development.

Learning through interacting with one's environment requires structures that direct the system to its environment. According to the view I have been outlining here, this is mediated in part by the midbrain dopamine system. One clue for this role derives from studies of the neurobiology of personality, which view personality as deriving from motivational systems. From this perspective, the midbrain dopamine system constitutes a behavioral facilitation system that underlies fundamental properties of personality, specifically extraversion and positive emotionality (Depue & Collins, 1999). From a developmental perspective, this behavioral facilitation system appears to be operative at an early age and likely underlies major dimensions of temperament, along with other diffuse ascending systems, such as noradernergic and serotonergic systems. Thus, given this system's computational properties and its role as a behavioral facilitation system early in postnatal development, this system is ideally situated to be involved in the reinforcement or self-supervised construction of prefrontal structures underlying complex behavioral control.

This computational role can be illustrated by comparing reinforcement models of learning to models of self-organization, or unsupervised learning. The best-known account of unsupervised learning is Hebbian learning, which in its simplest form is:

$$\Delta w_{kj}(t) = \eta y_k(t) x_j(t)) \tag{1}$$

where a synaptic weight w_{kj} of neuron k with presynaptic and postsynaptic signals denoted by x_j and y_k respectively are altered at time step t and where η is a positive constant that determines the rate of learning. Algorithms such as equation 1 and a variety of modifications essentially find efficient representations of salient environmental information by implementing such data reduction strategies as principal component analysis. Such algorithms can be modified to become reinforcement learning algorithms by making weight updates dependent on the Hebbian correlation of a prediction error and the presynaptic activity at the previous timestep. This takes the following form:

$$w(i,t-1)_{\text{new}} = w(i,t-1)_{\text{prev}} + \eta x(i,t-1)\delta(t) \tag{2}$$

where $x(i, t-1)$ represents presynaptic activity at connection i and time $t-1$, η is a learning rate, and $w(i, \text{t} -1)_{\text{prev}}$ is the previous value of the weight representing timestep $t-1$. The term $\delta(t)$ is a prediction error term (see Figure 2) and is the difference between a prediction of reward and the actual reward, represented as the output of the dopaminergic projection to cortex in the simulation framework. The addition of this term changes the Hebbian framework to a Predictive Hebbian one (Montague & Sejnowski, 1994) and is the essential computed differential in the temporal differences method of reinforcement learning (Sutton & Barto, 1998) with close connections to dynamic programming (Bellman, 1957).

The developmental link between the midbrain dopamine system and prefrontal structures suggests that an explicit account of the developmental trajectory of cognitive skills is necessary; an account based on innately-specified modules is inadequate. According to this view, complex developmental skills decompose into developmental precursors, which may often be mediated by structures that are distinct from those

mediating the mature state. For example, face processing is believed to be mediated by subcortical structures during early postnatal development, but it subsequently shifts to cortical sites (reviewed in Johnson, 1997). The model I have outlined above suggests a possible way of bootstrapping a system into such complex representations by biasing development by making the system selectively attentive to faces. An economical means of implementing such a strategy would be by making faces, or primitive template representations of them, rewarding to the system, thereby designing a system that preferentially attends to faces. It is clear that human infants possess such behavioral biases, which may be implemented through projections to midbrain dopamine systems that constitute unconditioned stimuli.

3.5 Protracted Development and Constructive Learning

These considerations suggest another important evolutionary alteration to developmental programs with important consequence for the evolution of human cognition. Above I highlighted evolutionary alterations in the duration of neurogenesis that appears to account for many aspects of increases in brain size. In addition, it appears that the human brain's development is also more protracted than other anthropoid apes. For example, the chimpanzee brain reaches 95% of its final mass by age two, whereas the human brain does not reach this milestone until the age of five. On many accounts, the protracted nature of human development has mainly negative consequences, such as extending the period of heightened vulnerability. Such accounts often regard protracted human development as a side effect of the constraints bipedalism placed on the design of the female pelvis, and thus on limits to the size of the birth canal.

The interpretation of protracted development as a liability stems in part from a view of development as largely a process of intrinsic maturation. Protracted neural development need not be viewed simply as a cost to the organism. Instead, under certain conditions extending development can result in powerful learning strategies. There are two requirements: first, development must not be simply a process of intrinsic maturation. Instead, it must be sensitive to environmental structure, in that activity emanating from the environment must play a role in the construction of neural circuits.

Second, this developmental strategy can be enhanced if neural development is not concurrent across different regions of the cortex, but instead follows a hierarchical scheme. Viewed instead as a kind of learning, its protractedness takes on special, positive qualities. In previous work I outlined a view I refer to as neural constructivism whereby the functional properties of cortex are built from the dynamic interaction between neural growth mechanisms and environmentally-derived neural activity, acting hierarchically at the regional level and with high specificity at the cellular level (Quartz & Sejnowski, 1997; Quartz, 1999). Neural constructivism suggests that the interaction between processes traditionally described cognitively as learning interact in complex ways with their neural substrates to construct neural circuits.

The starting point for this work was an investigation into the relationship between developing neural structures and the learning properties of cortex. Since its beginnings, developmental neurobiology has been embroiled in debate over whether development is a progressive increase in neural structures or whether it essentially involves a selective elimination of exuberant structures (see Purves et al., 1996 for a summary of this debate). As this question has important consequences for the learning properties of cortex, I examined the developmental time course of synaptic numbers, axonal processes, and dendritic arbors and concluded that the bulk of the evidence favors progressive increases in these measures during development. In addition, I reviewed neurobiological results spanning over thirty years that support the role for activity-dependent mechanisms in the progressive construction of neural circuits. On the basis of this work, I suggested that cortical development is not characterized by an early overproduction of neural elements followed by selective elimination, nor is it one exhausted by mechanisms of selective elimination operating on transient, exuberant structures. Rather, neural development during the acquisition of major cognitive skills is best characterized as a progressive construction of neural structures, in which environmentally-derived activity plays a role in the construction of neural circuits. This revised view of the role of activity in the construction of neural circuits forms the basis for neural constructivism, From the perspective of cognitive development, I suggested that this far-reaching interaction between neural growth and environmentally-derived neural activity undermined the distinction between biological maturation and learning. In place of this dichotomy, I

articulated a theory of "constructive learning" and suggested that it possesses more powerful acquisition properties than traditional accounts of cognitive development assumed.

In more recent work (Quartz, 1999; Quartz & Sejnowski, 2000) I have investigated the implications of two important advances for neural constructivism. Recent longitudinal studies of brain development using MRI have demonstrated pre-adolescent increases in cortical gray matter in all cortical lobes (Giedd et al., 1999; Thompson et al., 2000). In addition, this growth is heterochronic; that is, regions of cortex develop at different rates (Thompson et al., 2000). This is extremely significant for theories of cognitive development for the following reason. Although many features of Piaget's developmental view have come under extensive criticism, the core idea that development involves the expansion of hierarchically organized sequential operations, beginning with perceptual and sensorimotor functions and becoming more combinatorially complex, remains popular. These studies and others suggest that the brain develops hierarchically, with early sensory regions developing prior to more complex representations in association areas (Quartz, 1999). Given the influence of activity in this construction, it suggests a powerful hierarchical construction process whose acquisition properties remain essentially unanalyzed. Although MRI studies lack the spatial resolution to identify the cellular components of neuropil – neural processes and non-neuronal cells, or glia – responsible for increases in cortical gray matter, recent advances in microscopy that allow the continuous monitoring of cellular components at high resolution (Maletic-Savatic et al., 1999; Engert & Bonhoeffer, 1999; reviewed in Wong and Wong, 2000) have revealed a highly dynamic view of development at the cellular level. In particular, these studies demonstrate that activity is not simply permissive in its regulation of development. Rather, temporally correlated activity between pre- and post-synaptic elements that induces long-term potentiation results in the local sprouting of dendritic elements, in agreement with Hebb's original postulate in its developmental context (Hebb, 1949). These results are highly significant for theories of cognitive development, as they indicate that environmentally-derived patterned neural activity plays an instructive role in the construction of neural circuits, both within unsupervised and self-supervised modes.

Although prefrontal function has traditionally been most closely associated with purely cognitive functions, its central involvement in social cognition has become increasingly apparent in recent years. Indeed, one potential reason for protracted development lies in the difficulty of developing the social competence necessary for complex social life. There is now good evidence to indicate that one component of social competence, theory of mind, depends at least in part on the appropriate social exposure for its development, as many deaf children show delays on theory of mind tasks (Peterson & Siegal, 1995; Russell et al., 1998). This is believed to be due to the fact that parents of deaf children are typically naïve signers, and so household social interactions are limited by communicative ability.

Human social behavior becomes increasingly sophisticated over the developmental timecourse. In particular, over development individuals become increasingly skilled at reading subtle social cues and adjusting their behavior accordingly by applying appropriate behavioral schemes and norms to rapidly shifting contexts. Increasingly mature forms of social cognition involve a cognitive flexibility and the ability to match behavioral strategies with the contingencies of various situations. The developmental frontalization of behavior that underlies these capacities reflects a process I have referred to previously as progressive externalization (Quartz & Sejnowski, 1997). Progressive externalization refers to the process whereby neural development becomes regulated by environmental influences over longer periods of postnatal exposure. This emphasis on behavioral plasticity contrasts with the emphasis on evolutionarily-encoded behavioral strategies. Under what conditions did such capacities emerge?

4. The Adaptive History of Hominid Evolution: Rethinking the Environment of Evolutionary Adaptation

4.1 Beyond Directional Selection

A crucial assumption of any evolutionary psychology integrative approach is that human cognitive and behavioral capacities reflect our lineage's history. Without this assumption, there would be little impetus to understand the relationship between

evolution and psychology. An analysis of the environment of evolutionary adaptation (EEA) plays an especially prominent role in narrow evolutionary psychology, as its adaptive thinking places a premium on inferring the mind's Darwinian algorithms from the nature of the enduring challenges confronting our ancestors.

The most prominent scenarios of hominid adaptation are habitat-specific. That is, a specific, stable ecological context is typically identified as the EEA. The most popular such account is the savanna hypothesis, according to which our ancestors lived as hunter-gatherers on the African savanna. According to some interpretations of this EEA, the ecological challenges confronting our ancestors were relatively minor, making the social environment the primary selective force, where a Machiavellian intelligence was most adaptive.

The notion that the EEA was characterized by a stable ecological context is complicated by recent work in paleoclimatology. Through painstaking analysis of ice cores, deep ocean cores, and land and lake sediments, climate scientists are piecing together a surprising history of the earth's climate (for a review, see Potts, 1996; Bradley, 1999). This research reveals that the last million years was a time of jarring climatic changes, the greatest period of climatic fluctuation since Lucy walked the planet 3.5 million years ago, and could be the period of the greatest climatic fluctuations ever registered on the planet. Often within the span of a decade, climates underwent dramatic alterations, from rain forest to arid savanna to steppe. The pressures ecological instability placed on species is evident by the pronounced reduction in biodiversity during this period, particularly with regard to species that were highly specialized for particular ecologies (Potts, 1996).

The notion of a long enduring EEA that remained stable enough for its problems to act on hominids over an evolutionary timescale is also complicated by the fact that for most of hominid evolution there was a statis in relative brain size. Indeed, between 1.8 and .6 million years ago, the brain scaled essentially as a straightforward function of body mass. Hominid encephalization appears to have occurred mostly within the last 600,000 years (Ruff et al., 1997).

It is intriguing to note that this fairly recent process of encephalization coincided with the period of unprecedented climatic instability I mentioned above. Although

ecological instability certainly does not exclude a wide variety of enduring problems that likely remained stable across varying climes, it suggests that solutions to such problems alone cannot account for the evolution of human cognition. Rather, it suggests that human cognitive evolution was driven in part by environmental variance and the challenges such instability presented. This suggests a basic adjustment in how we ought to view the evolutionary pressures that helped shape human cognition. There are two possible responses to ecological instability. A species may attempt to track its preferred habitat, as appears to have been the case with chimpanzees, who might have taken shelter in rain forest refugia during glacial periods. An alternative response is open to those species that possess enough behavioral flexibility to adapt to differing ecological contexts. Potts (1996) contrasts the selective pressure of adapting to multiple ecological contexts, a pressure he refers to as variability selection, with the more traditional notion of directional selection, and suggests that variability selection was a major force in human origins.

5.Progressive Externalization and The Ontogenic Role of Culture

5.1 The Progressive Externalization of Development

This perspective places a premium on behavioral flexibility. I have suggested that this behavioral flexibility is mediated by a human cognitive architecture that is a hierarchically-organized control structure, and which displays a developmental trajectory whereby behavior is increasingly mediated by prefrontal structures. Based on an increased encephalization, which appears to be a fairly recent process, it appears to be the product of heterochronic alterations in development that result in both increased neocortical volume and protracted development, reflecting a process I have referred to as progressive externalization.

Ecological instability suggests another possible response: the construction of buffers that make one less vulnerable to the immediate environment. One such buffer is novel forms of social organization, and ultimately symbolic culture. The cognitive

structures that I have emphasized in developmental evolutionary psychology's model of the human cognitive architecture are those necessary for complex social life and symbolic culture. The process of progressive externalization, mediated in part by heterochronic changes in neural development, whereby the development of cognitive structures became increasingly dependent on prolonged environmental interaction, may thus have been the route to designing a cognitive architecture capable of the highly flexible and context-sensitive behavior necessary for participation in a complex culture. Symbolic culture, then, plays a central role in constructing the structures that make it possible.

References

Allman, J. (1999). *Evolving Brains.* New York: Freeman.

Arthur, W. (1997). *The origin of animal body plans : a study in evolutionary developmental biology*. Cambridge, U.K. ; New York: Cambridge University Press.

Barkow, J.H., Cosmides, L., & Tooby, J. (Eds.). (1992). *The adapted mind: Evolutionary psychology and the generation of culture*. New York, NY, US: Oxford University Press.

Bellman, R.E. (1957). *Dynamic Programming*. Princeton, N.J.: Princeton University Press.

Berns, G.S., McClure, S.M., Pagnoni, G., & Montague, P.R. (2001). Predictability modulates human brain response to reward. *Journal of Neuroscience,* 21: 2793-2798.

Bradley, R.S. (1999). *Paleoclimatology : reconstructing climates of the Quaternary* (2nd ed.). San Diego: Academic Press.

Brothers, L., & Ring, B. (1992). A neuroethological framework for the representation of minds. *Journal of Cognitive Neuroscience,* 4: 107-118.

Buss, D.M. (1999). *Evolutionary psychology: The new science of the mind*. Needham Heights, MA.: Allyn & Bacon, Inc.

Cosmides, L., & Tooby, J. (1994). Origins of domain specificity: The evolution of functional organization, *Mapping the mind: Domain specificity in cognition and culture.* (pp. 85-116). New York: Cambridge University Press.

Cowie, F. (1998). *What's within? : nativism reconsidered*. New York: Oxford University Press.

Craik, F.I.M., Moroz, T.M., Moscovitch, M., Stuss, D.T., Winocur, G., Tulving, E., & Kapur, S. (1999). In search of the self: A positron emission tomography study. *Psychological Science,* 10: 26-34.

Darlington, R.B., Dunlop, S. A., & Finlay, B.L. (1999). Neural development in metatherian and eutherian mammals: variation and constraint. *Journal of Comparative Neurology,* 411: 359-368.

Davidson, E.H. (2001). *Genomic regulatory systems : development and evolution*. San Diego: Academic Press.

Deacon, T. W. (1997). *The symbolic species : the co-evolution of language and the brain*. New York: W.W. Norton.

Dehaene, S., Spelke, E., Pinel, P., Stanescu, R., & Tsivkin, S. (1999). Sources of mathematical thinking: Behavioral and brain-imaging evidence. *Science,* 284: 970-974.

Depue, R.A., & Collins, P.F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral & Brain Sciences,* 22: 491-569.

Diamond, A. (1985). Development of the ability to use recall to guide action, as indicated by infants' performance on AB. *Child Development,* 56: 868-883.

Diamond, A. (1998). Evidence for the importance of dopamine for prefrontal cortex functions early in life, *The prefrontal cortex: Executive and cognitive functions.* (pp. 144-164). New York: Oxford University Press.

Diamond, A., & Doar, B. (1989). The performance of human infants on a measure of frontal cortex function, the delayed response task. *Developmental Psychobiology,* 22: 271-294.

Diamond, A., Prevor, M.B., Callender, G., & Druin, D.P. (1997). Prefrontal cortex cognitive deficits in children treated early and continuously for PKU. *Monographs of the Society for Research in Child Development,* 62: 1-205.

Elman, J.L. (1996). *Rethinking innateness : a connectionist perspective on development*. Cambridge, Mass.: MIT Press.

Engert, F., & Bonhoeffer, T. (1999). Dendritic spine changes associated with hippocampal long-term synaptic plasticity. *Nature,* 399: 66-70.

Finlay, B.L., & Darlington, R.B. (1995). Linked regularities in the development and evolution of mammalian brains. *Science,* 268: 1578-1584.

Finlay, B.L., Darlington, R. B., & Nicastro, N. (in press). Developmental Structure in Brain Evolution. *Behavioral & Brain Sciences*.

Finlay, B.L., Hersman, M.N., & Darlington, R.B. (1998). Patterns of vertebrate neurogenesis and the paths of vertebrate evolution. *Brain, Behavior and Evolution,* 52: 232-242.

Fodor, J.A. (1983). *Modularity of mind : an essay on faculty psychology*. Cambridge, MA.: MIT Press.

Fodor, J.A. (2000). *The mind doesn't work that way: The scope and limits of computational psychology*. Cambridge, MA.: The MIT Press.

Frahm, H.D., Stephan, H., & Stephan, M. (1982). Comparison of brain structure volumes in Insectivora and Primates. I. Neocortex. *Journal fur Hirnforschung,* 23: 375-389.

Gallistel, C.R. (1990). *The organization of learning*. Cambridge, Ma.: MIT Press.

Garris, P.A., Kilpatrick, M., Bunin, M.A., Michael, D., Walker, Q.D., & Wightman, R.M. (1999). Dissociation of dopamine release in the nucleus accumbens from intracranial self-stimulation. *Nature,* 398: 67-69.

Giedd, J.N., Blumenthal, J., Jeffries, N.O., Castellanos, F.X., Liu, H., Zijdenbos, A., Paus, T., Evans, A.C., & Rapoport, J.L. (1999). Brain development during childhood and adolescence: a longitudinal MRI study. *Nature Neuroscience,* 2: 861-863.

Gilbert, S.F., Opitz, J. M., & Raff, R.A. (1996). Resynthesizing evolutionary and developmental biology. *Developmental Biology,* 173: 357-372.

Goldman-Rakic, P.S. (1990). Cortical localization of working memory, *Brain organization and memory: Cells, systems, and circuits*. (pp. 285-298). New York: Oxford University Press.

Gould, S.J. (2000). Of coiled oysters and big brains: how to rescue the terminology of heterochrony, now gone astray. *Evolution and Development,* 2: 241-248.

Gould, S.J., & Lewontin, R.C. (1979). The spandrels of San Marco and the Panglossian paradigm: a critique of the adaptationist programme. *Proceedings of the Royal Society of London. Series B: Biological Sciences,* 205: 581-598.

Hall, B.K. (1998). *Evolutionary developmental biology*. New York: Chapman & Hall.

Hammer, M. (1993). An identified neuron mediates the unconditioned stimulus in associative olfactory learning in honeybees. *Nature,* 366: 59-63.

Hebb, D.O. (1949). *The organization of behavior; a neuropsychological theory*. New York: Wiley.

Hirschfeld, L.A., & Gelman, S.A. (Eds.). (1994). *Mapping the mind: Domain specificity in cognition and culture*. New York: Cambridge University Press.

Hirth, F., & Reichert, H. (1999). Conserved genetic programs in insect and mammalian brain development. *Bioessays,* 21: 684.

Johnson, M.H. (1997). *Developmental cognitive neuroscience: An introduction*. Malden, MA.: Blackwell Publishers Inc.

Lovejoy, C.O., Cohn, M.J., & White, T.D. (1999). Morphological analysis of the mammalian postcranium: a developmental perspective. *Proceedings of the National Academy of Sciences of the United States of America,* 96: 13247-13252.

Maletic-Savatic, M., Malinow, R., & Svoboda, K. (1999). Rapid dendritic morphogenesis in CA1 hippocampal dendrites induced by synaptic activity. *Science,* 283: 1923-1927.

McConnell, S.K. (1995). Constructing the cerebral cortex: neurogenesis and fate determination. *Neuron,* 15: 761-768.

Montague, P.R., Dayan, P., Person, C., & Sejnowski, T.J. (1995). Bee foraging in uncertain environments using predictive hebbian learning. *Nature,* 377: 725-728.

Montague, P.R., & Quartz, S.R. (1999). Computational approaches to neural reward and development. *Mental Retardation & Developmental Disabilities Research Reviews,* 5: 86-99.

Montague, P.R., & Sejnowski, T.J. (1994). The predictive brain: temporal coincidence and temporal order in synaptic learning mechanisms. *Learn Mem, 1*(1), 1-33.

Nieuwenhuys, R., Donkelaar, H.J.T. & Nicholson, C. (1998). *The central nervous system of vertebrates*. New York: Springer.

Norman, D.A., & Shallice, T. (1986). Attention to Action: Willed and Automatic Control of Behavior. In R. J. Davidson, Schwartz, G.E. , Shapiro, D. (Ed.), *Consciousness and Self-Regulation* (pp. 1-18). New York: Plenum Press.

Peterson, C.C., & Siegal, M. (1995). Deafness, conversation and theory of mind. *Journal of Child Psychology and Psychiatry,* 36: 459-474.

Petrides, M. (1995). Functional organization of the human frontal cortex for mnemonic processing: Evidence from neuroimaging studies, *Structure and functions of the human prefrontal cortex.* (pp. 85-96). New York, NY, US: New York Academy of Sciences.

Pick, L. (1998). Segmentation: painting stripes from flies to vertebrates. *Developmental Genetics,* 23: 1-10.

Pinker, S. (1997). *How the mind works*. New York: Norton.

Potts, R. (1996). *Humanity's Descent*. New York: William Morrow.

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

Quartz, S. R. (1999). The Constructivist Brain. *Trends in Cognitive Sciences, 3*(2), 48-57.

Quartz, S.R. & Sejnowski, T.J. (2000). Constraining constructivism: Cortical and subcortical constraints on learning in development, *Behavioral and Brain Sciences*, 23: 785-791.

Raff, R.A. (1996). *The shape of life: genes, development, and the evolution of animal form*. Chicago: University of Chicago Press.

Raff, R.A. (2000). Evo-devo: the evolution of a new discipline. *Nature Reviews Genetics,* 1: 74-79.

Rakic, P. (1988). Specification of cerebral cortical areas. *Science,* 241*:* 170-176.

Real, L.A. (1991). Animal choice behavior and the evolution of cognitive architecture. *Science,* 253: 980-986.

Reichert, H., & Simeone, A. (1999). Conserved usage of gap and homeotic genes in patterning the CNS. *Current Opinion in Neurobiology,* 9: 589-595.

Reinitz, J., Kosman, D., Vanario-Alonso, C.E., & Sharp, D.H. (1998). Stripe forming architecture of the gap gene system. *Developmental Genetics,* 23: 11-27.

Ross, C.A., Ruggiero, D.A., Park, D.H., Joh, T.H., Sved, A.F., Fernandez-Pardal, J., Saavedra, J.M., & Reis, D.J. (1984). Tonic vasomotor control by the rostral ventrolateral medulla: effect of electrical or chemical stimulation of the area containing C1 adrenaline neurons on arterial pressure, heart rate, and plasma catecholamines and vasopressin. *Journal of Neuroscience,* 4: 474-494.

Rubenstein, J. L., Martinez, S., Shimamura, K., & Puelles, L. (1994). The embryonic vertebrate forebrain: the prosomeric model. *Science,* 266: 578-580.

Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S.C.R., Simmons, A., Andrew, C., & Bullmore, E.T. (2000). Functional frontalisation with age: Mapping neurodevelopmental trajectories with fMRI. *Neuroscience & Biobehavioral Reviews,* 24: 13-19.

Ruff, C.B., Trinkaus, E., & Holliday, T.W. (1997). Body mass and encephalization in Pleistocene Homo. *Nature,* 387: 173-176.

Russell, P.A., Hosie, J.A., Gray, C.D., Scott, C., Hunter, N., Banks, J.S., & Macaulay, M.C. (1998). The development of theory of mind in deaf children. *Journal of Child Psychology and Psychiatry,* 39:, 903-910.

Sawaguchi, T., & Goldman-Rakic, P.S. (1994). The role of D1-dopamine receptor in working memory: Local injections of dopamine antagonists into the prefrontal cortex of rhesus monkeys performing an oculomotor delayed-response task. *Journal of Neurophysiology,* 71: 515-528.

Schultz, W. (2000). Multiple reward signals in the brain. *Nauret Review Neuroscence,* 1: 199-207.

Schultz, W., Apicella, P., & Ljungberg, T. (1993). Responses of monkey dopamine neurons to reward and conditioned stimuli during successive steps of learning a delayed response task. *Journal of Neuroscience,* 13: 900-913.

Stephan, H., Frahm, H., & Baron, G. (1981). New and revised data on volumes of brain structures in insectivores and primates. *Folia Primatologica,* 35: 1-29.

Sterelny, K., & Griffiths, P.E. (1999). *Sex and death : an introduction to philosophy of biology*. Chicago: University of Chicago Press.

Stone, V.E., Baron-Cohen, S., & Knight, R.T. (1998). Frontal lobe contributions to theory of mind. *Journal of Cognitive Neuroscience,* 10: 640-656.

Sutton, R.S., & Barto, A.G. (1998). *Reinforcement learning: an introduction*. Cambridge, MA.: MIT Press.

Tesauro, G. (1995). Temporal difference learning and TD-Gammon. *Communications of the ACM, 38*: 58-68.

Thompson, P.M., Giedd, J.N., Woods, R.P., MacDonald, D., Evans, A.C., & Toga, A.W. (2000). Growth patterns in the developing brain detected by using continuum mechanical tensor maps. *Nature,* 404: 190-193.

Tomasello, M. (1999). *The cultural origins of human cognition*. Cambridge, MA.: Harvard University Press.

Tooby, J., & Cosmides, L. (1992). The psychological foundations of culture, *The adapted mind: Evolutionary psychology and the generation of culture.* (pp. 19-136). New York, NY, US: Oxford University Press.

Wise, R.A. (1996). Addictive drugs and brain stimulation reward. *Annual Review of Neuroscience,* 19: 319-340.

---

[1] This research was supported by National Science Foundation Career Grant # 0093757.

[2] Although encephalization may underlie important cognitive differences among anthropoid primates, in general it is difficult to relate differences in brain size to cognitive differences across species; see Kaas (2000).

[3] There are some important exceptions. For example, Allman has recently demonstrated that anthropoid primates possess a special class of cells, known as spindle cells, whose physiology might underlie important elements of higher cognition. In addition, cortical thickness is twice as thick in human neocortex as it is in mice neocortex, largely a consequence of the increased need for connectivity.

[4] Although this strategy emphasizes learning, it is important to bear in mind that it requires a primitive set of target states that have intrinsic reward value to the organism (classically known as unconditional stimulus).