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Evolutionary Psychology, Meet Developmental Neurobiology: Against Promiscuous Modularity

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Abstract. Evolutionary psychologists claim that the mind contains “hundreds or thousands” of “genetically specified” modules, which are evolutionary adaptations for their cognitive functions. We argue that, while the adult human mind/brain typically contains a degree of modularization, its “modules” are neither genetically specified nor evolutionary adaptations. Rather, they result from the brain’s developmental plasticity, which allows environmental task demands a large role in shaping the brain’s information-processing structures. The brain’s developmental plasticity is our fundamental psychological adaptation, and the “modules” that result from it are adaptive responses to local conditions, not past evolutionary environments. If different individuals share common environments, however, they may develop similar “modules,” and this process can mimic the development of genetically specified modules in the evolutionary psychologist’s sense.

Key words: adaptation, brain development, domain specificity, evolutionary psychology, modularity, plasticity

Evolutionary psychologists¹ claim that the human mind is a network of distinct, yet interacting, psychological adaptations. Summarizing this view, Pinker says, “the mind is organized into modules or mental organs, each with a specialized design that makes it an expert in one arena of interaction with the world. The modules’ basic logic is specified by our genetic program. Their operation was shaped by

¹ The term “evolutionary psychology” is ambiguous in common usage. It sometimes refers to a *field of inquiry* encompassing a range of work so broad that it is united only by a desire to understand the evolution of the human mind. More frequently, however, the term is used in a more specific sense, designating only work conducted within a particular set of theoretical and methodological commitments shared by a prominent and highly influential group of researchers (most notably, David M. Buss, Leda Cosmides, Martin Daly, Steven Pinker, Donald Symons, John Tooby, and Margo Wilson). In this narrower sense, “evolutionary psychology” designates a Kuhnian *paradigm* – a shared and unquestioned framework of theory, methodology, and *exemplars* (specific explanations that serve as models for further scientific research). Our focus in what follows is this paradigm, which has become very prominent and has received the most attention of all the work on the evolution of human psychology.

natural selection to solve the problems of the hunting and gathering life led by our ancestors in most of our evolutionary history” (Pinker, 1997, p. 21).

We do not doubt that an adult human brain is at least partially modularized. However, in what follows, we contend that the brain is not modularized in the ways that evolutionary psychologists claim; we do not have lots of “genetically specified,” domain-specific, informationally encapsulated, cognitive processing streams. To see why this is true, one must understand a bit about brain plasticity and development. But first a summary of evolutionary psychology’s modular view of the mind.

1. The Modularity of the Adapted Mind

It was once widely accepted that the mind comes equipped with just a few general cognitive procedures, which are employed in learning everything we come to know about the world. These few procedures were considered *domain general*, in that they were assumed to be applicable to any problem domain that might be encountered – everything from the acquisition of language or mathematical skill to the ability to play chess or ride a bicycle. In this view, the mind doesn’t bring any specific knowledge of a particular problem domain *to* the process of learning in that domain. Rather, all the information the mind possesses about a particular problem domain is extracted from the world by its few domain-general procedures.

Evolutionary psychologists reject this in favor of a view of the mind as consisting of numerous *modules*, which are characterized by a few special properties (Cosmides and Tooby, 1987, 1994; Pinker, 1997; Tooby and Cosmides, 1992; Symons, 1987). First, a module is *domain specific* – that is, it is specialized to deal only with a restricted problem domain. As such, its information-processing procedures are activated only by information about a particular aspect of the world, and it is unresponsive to information about other aspects of the world, in much the way the ear is responsive only to specific vibratory frequencies (Cosmides and Tooby, 1987; Pinker, 1997).

Further, according to evolutionary psychologists, a module develops in the absence of any explicit instruction in the problem domain with which it is specialized to deal (Cosmides and Tooby, 1994; Pinker, 1997; Tooby and Cosmides 1992). Their view is not, however, that a module develops in the absence of *any* environmental input; for they claim that some kind of triggering stimulus is typically required during a *critical period* of development in order to bring a module on line (Tooby and Cosmides, 1992). Their view, rather, is that the environmental inputs that trigger development of a module always contain far less information than is present in the fully developed mechanism. This is because a module comes equipped with certain “innate knowledge” about the problem domain with which it is specialized to deal and an “innate” set of procedures for applying that knowledge to solve problems in its special domain. That is, rather than needing to extract all its information about its problem domain from the world during the course of an

individual's life, a module contains "unlearned" information and procedures about its problem domain, which it employs in its problem solving (Cosmides and Tooby, 1987, 1994; Pinker, 1997; Tooby and Cosmides, 1992).

Finally, though infrequently acknowledged by evolutionary psychologists, their account of the properties and behavior of modules requires that each module is, to some degree, *informationally encapsulated*. That is, a module doesn't have access to the full range of information available in an organism's brain, even when some of that information is relevant to solving problems in its specialized problem domain (Cosmides, 1989; Tooby and Cosmides, 1992). Rather, a module tends to have access only to the outputs of other psychological mechanisms and not to the information employed by those mechanisms in generating their outputs. This, to some extent, is simply domain specificity functioning internally: Not only are modules activated by and responsive to restricted ranges of information in the environment, "screening out" all other environmental information, but they access only restricted ranges of information in the brain as well, screening out all other mental information.

Evolutionary psychologists have tended to ignore this property of modules, or even to downplay its significance. As Pinker says: "Modules are defined by the special things they do with the information available to them, not necessarily by the kinds of information they have available" (1997, p. 31). But their account requires some degree of modular informational encapsulation, for at least two reasons. First, only some degree of informational encapsulation accounts for the specific kinds of cognitive "misfiring" that are prevalent in evolutionary psychologists' explanations of aspects of human behavior. For example, although evolutionary psychologists claim that human males have evolved modules regulating sexual behavior so as to promote reproductive success, those modules fail to cause men to donate to sperm banks in large numbers, even when men know that doing so would promote their reproductive success. The reason offered is that those modules are unresponsive to information acquired through cognitive channels other than those narrowly involved in their own functioning (Buss, 1995). Similarly, in Cosmides' (1989) work on social cognition, she hypothesizes that the module subserving reasoning about cheater detection systematically causes logically fallacious inferences under certain circumstances, even when subjects have received training in formal logic. Again, the reason is that the cheater-detection module is unresponsive to knowledge of logic, which is acquired through cognitive channels external to the cheater-detection module's functioning. If the modules postulated by evolutionary psychologists weren't informationally encapsulated to some degree, then these sorts of cognitive misfiring wouldn't occur. For an unencapsulated "module" would simply incorporate the information available through the other channels and condition its outputs on that information.

Second, if putative modules had access not only to one another's outputs, but to the information processed in generating those outputs as well, then the mind wouldn't be truly modular. For a mind composed of "separate mechanisms" that

made use of the information available to all the other mechanisms would just be a general-purpose mind. To put this another way, domain specificity and “innate” knowledge in themselves don’t necessitate modularity – that is, they don’t require *separate*, functionally specialized psychological mechanisms (see Samuels, 1998 and Fodor, 2000). For a non-modular mind equipped with just a few general-purpose cognitive procedures, which were applied in all learning and problem solving, could still perform domain-specific cognitive processing: If it were solving problems about cows, it would draw on and process specialized knowledge about cows, rather than information about desktops (unless, of course, it was figuring out how to get the cows off the desktops). And such a general-purpose mind could still possess “innate” knowledge about special problem domains. So domain-specific processing and “innate” knowledge are compatible with a non-modular, general-purpose mind. *Modules* only emerge when domain-specific processing and bodies of “innate” knowledge are contained within informationally encapsulated mechanisms; for only then are the mechanisms truly separate and functionally specialized (Fodor, 2000).

Evolutionary psychologists frequently support their modular view of the mind with the following argument. They point out that the adaptive problems faced by our Pleistocene ancestors were very diverse in character, ranging from identifying edible plant matter and avoiding deadly predators to selecting a reproductively valuable mate and cooperating with others in a status hierarchy. They then hypothesize that, given the diverse characters of these problems, what constitutes a successful solution to one problem is very different from what constitutes a solution to another. They then infer that no domain-general problem-solving strategy could have successfully solved the full range of adaptive problems faced by our Pleistocene ancestors, but that each adaptive problem would have required its own domain-specific problem-solving strategy. As Symons says: “It is no more probable that some sort of general-purpose brain/mind mechanism could solve all the behavioral problems an organism faces (find food, choose a mate, select a habitat, etc.) than it is that some sort of general-purpose organ could perform all physiological functions (pump blood, digest food, nourish an embryo, etc.)” (1992, p. 142). Thus, evolutionary psychologists conclude, humans could not have evolved minds consisting of a single all-purpose problem-solving mechanism; rather, they must have evolved distinct domain-specific mechanisms, each executing a program devoted to solving a specific adaptive problem.

Thus inspired, evolutionary psychologists postulate modules for incest avoidance, sexual attraction, mate choice, jealousy, mate retention, allocation of parental resources, kin relations, alliance formation, aggressive threat, danger avoidance, food preferences, habitat choice, and so on for all manner of complex cognitive and behavioral functions (Tooby and Cosmides, 1992, p. 110). Indeed, Tooby and Cosmides claim that “our cognitive architecture resembles a confederation of *hundreds or thousands*” of modules (1995, p. xiii; emphasis added). They contend that “the brain must be composed of a large collection of circuits, with

different circuits specialized for solving different problems. One can think of each specialized circuit as a minicomputer that is dedicated to solving one problem” (Cosmides and Tooby, 1997, p. 81). “Over evolutionary time, the brain’s circuits were cumulatively added because they reasoned or processed information in a way that enhanced the adaptive regulation of behavior” (Cosmides and Tooby, 1997, p. 78). Consequently, they conclude, “what is special about the human mind is not that it gave up ‘instinct’ in order to become flexible, but that it proliferated ‘instincts’ – that is, content-specific problem-solving specializations” (Tooby and Cosmides, 1992, p. 113).

Since each of these many modules is purportedly designed by selection to solve its own adaptive problem, evolutionary psychologists are committed to the claim that each module evolved independently of the others (though many may have evolved during the same time period). Further, evolutionary psychologists claim that modules are “complex adaptations,” which require “hundreds or thousands of genes to regulate their development” (Tooby and Cosmides, 1992, p. 78). Consequently, the evolution and adaptive shaping of each encapsulated module would have required numerous mutations over evolutionary time, each of which added or modified a functionally specialized “brain circuit,” and all of which were preserved by selection as the gene complex that builds that module during development – or, in Pinker’s words, as that part of the “genetic program” that “specifies” the “basic design” of that “mental organ” (1997, pp. 21 and 32).

2. Informational Encapsulation and Domain Specificity

Brain plasticity belies the idea of encapsulated modularity, for our information-processing streams are not really separate streams at all. There is much informational overlap between what are normally thought of as distinct processing areas. In other words, whatever modules one might want to identify in the brain are not as distinct, or informationally encapsulated, as evolutionary psychologists typically imply.

Charting the variety of brain plasticity has become a cottage industry in neuroscience. Despite the popular press’s pronouncements that brains don’t or can’t change much after our first few years – and that the changes in the first few years are absolutely crucial (hence the need for black and white mobiles for infants and the White House’s tremendous interest in early childhood education) – our brains are changing all the time, and changing quite rapidly and profoundly. We now know that the number of neurons in prefrontal, inferior temporal, and posterior parietal cortex increases throughout life (Gould *et al.*, 1999). In addition, not only do our brains continue to grow (at least in some places), but they are continually reorganizing themselves in response to environmental demands. If we lose a finger, the brain region that used to respond to its input in somatosensory cortex will decrease in size and the neighboring regions will expand until nothing is left of the functional brain area at all (Merzenich *et al.*, 1983). The converse holds true as well. If we

overstimulate a digit for a while, its corresponding area in cortex increases in size. It is important to note that these changes occur outside of any "critical" period for learning and can occur within a matter of hours or days. Indeed, it appears that our brains change enough over our lifetimes such that, by the time we are old, we use regions in our brains that are different from the ones we used as young adults to accomplish the same tasks (MacIntosh *et al.*, 1999).

How can our brains maintain a plasticity that allows for changes of function within hours? It now looks as though areas or regions once thought to be dedicated to one processing task are actually receiving inputs from more than one processing stream. When the median nerve of the hand is severed in adult owl or squirrel monkeys, areas 3b and 1 in somatosensory cortex (areas that normally respond to medial nerve stimulation) begin almost immediately to respond to inputs from other nerves in the hand (Merzenich *et al.*, 1983; see also Clark *et al.*, 1988). Our best explanation of the rapidity of the response is that silencing the medial nerve inputs "unmasks" secondary inputs from other afferent nerves.

fMRI studies of human somatosensory cortex, human behavioral studies of phantom limb patients, and animal studies all indicate that the brain maintains overlapping dynamic representations (Doetsch *et al.*, 1983; Dostrovsky *et al.*, 1976; Merrill and Wall, 1978; Metzler and Marks, 1979; Ramachandran, 1994; Sanes *et al.*, 1995; Wall and Egger, 1971). In each case, we see areas of cortex allegedly dedicated to processing one sort of information process very different sorts of information as well. And these areas change as the environmental stimuli change. We are misled by our own localizationist methodologies to a certain extent; we expected the one area-one function rule to apply in the brain before we ever started recording neural activity. However, what we expected to find and what we are in fact finding are two entirely different things.

Perhaps the most striking research that supports this blurring of processing streams concerns how our brains compensate for vestibular disturbances. If we ablate the semicircular canals in our ear so that our vestibular system no longer receives any orientation information, we recover at least our static vestibular responses very quickly, much faster than we could grow new synaptic connections. Single-cell recordings show that vestibular processing is not rerouted elsewhere in the brain; the same neurons in the brain stem that respond to normal vestibular inputs are also used in recovery. Obviously, they must be getting orientation information from somewhere other than the (now ablated) semicircular canals. Some other sensory system must already be feeding into the vestibular system. One hypothesis is that brains use a form of sensory substitution to compensate for the vestibular-ocular reflex (Berthoz, 1988; Miles and Lisberger, 1981). In this case, the brain would use internally generated signals from the visual or somatosensory systems to compensate for the vestibular loss. It would substitute computations from the saccadic or a visual pursuit system (both of which probably reconstruct head velocity internally) for vestibular throughputs. Perhaps as animals try to orient toward targets, error signals from the retina help the vestibular system compute

head location. Currently ongoing work is directed toward exploring this possibility (Stewart, personal communication).

Of course, it is not the case that everything is connected to everything else. It might be that our vestibular system is only connected to retinal error detection signals and nothing else. In that sense, we might maintain that various processing streams are isolated from other processes. But, if our alleged topographical maps in somatosensory cortex contain secondary connections for other inputs, if brain representations and processing fundamentally overlap one another, if even “modality-specific” processing receives inputs from other modalities, there is little sense in which we can maintain that even our most basic processes are distinct in the way that evolutionary psychology’s modularity hypothesis implies. And, if this is the case for our most basic cognitive processes, it likely holds as well for the “higher” cognitive processes involved in adaptive problem solving of the sort that interests evolutionary psychologists.

Research in neural plasticity also dovetails with recent data that highlight cross-modal connections. For example, if you touch someone’s body on the same side and at the same time as you present a visual stimulus to her, activation in the lingual gyrus is significantly more than when the visual stimulus is presented alone. Imaging studies of this phenomenon indicate that the somatosensory areas of cortex project back to the visual areas, thus telling the visual cortex about tactile stimuli received (Macaluso *et al.*, 2000). The extent of crossmodal communication between and among our alleged sensory “modules” is still a matter of investigation, though we do know that our auditory and visual areas exchange lots of information regarding speech perception and that there are considerable crossmodal connections between thalamic nuclei and cortex.

This degree of informational overlap also shows that our brain systems are not domain specific; rather, they are domain *dominant*. One sort of processing in a brain region may be more prominent than others, but other processing is still occurring. Our “modules” are not so specialized that they deal only with restricted domains. Instead, they deal *mostly* with particular domains, and do so only contingently; the dedication of a brain system to a particular task domain is subject to change as the inputs to that brain system change. Despite evolutionary psychologists’ claims to the contrary, the need for specialized problem solving does not prevent our various brain areas from talking to one another.

3. From Genes to Thought

Now consider the following facts about brain development. From the time the human brain begins to develop in utero, at about 25 days gestation, it increases by a remarkable 250,000 cells per minute (Thompson, 1993). No doubt much of this development is “hardwired” to some degree, for all the major areas of the brain are essentially the same across mammals. However, how we go from our genetic endowment to a fully developed human brain is a complicated story of feedback

loops piggybacking on environmental contingencies, not a story of a developmental process unfolding in accordance with a “genetic program.”

The total number of genes in human DNA is currently estimated at around 80,000 (Gerhart and Kirschner, 1997, p. 121). As much as 50% of these may be concerned with our brain (Thompson, 1993), yet we have literally trillions of synaptic connections in our head. There is no way even 40,000 genes could code for that exactly. Only in very simple creatures, such as the nematode *C. elegans* (who has 302 neurons with about 7,000 neuronal connections), might we even hope to find relatively direct genetic control of nervous system development. (But we may not even find genetic control of development in them; for work on mapping the genome of *C. elegans* led even the early proponents of the notion of a “genetic program” controlling development to abandon it as a hopelessly inadequate model of development (de Chadarevian, 1998).)

This picture is even more bleak for those who wish to find genetic programs for our higher cognitive functions, which are located primarily in cortex, for it appears that most of the genetic “specification” for our brain concerns the more peripheral structures. Our genes do not seem to worry much about how to create most of our brain and are instead dedicated to making sure our sensory transducers are constructed properly. Fully 4% of the genes concerned with brain development (roughly 1,600) are concerned with building the sensory cells located inside our nose (Winberg and Porter, 1998).²

Moreover, if our genes programmed for the development of the cortical structures involved in higher cognition, we would expect *some* positive correlation between genome size and the degree of complexity of higher cognitive functions. But the comparative data instead reveal a “relative constancy of genome size across vast differences in brain size and correspondingly astronomical differences in connections” (Deacon, 1997, p. 197). Indeed, despite our vastly more complex brains, humans have roughly the same number of genes as the house mouse, *Mus musculus* (Gerhart and Kirschner, 1997, p. 121).

For much of the brain, the immediate cellular environment, and not our genes, determines the formation of brain structure. Epithelial cells later destined to be a brain first grow in a neural plate, which later folds into a neural tube. As the cells stop dividing, they begin migrating toward their final destination. Exactly how neurons know to stop dividing or how they know where to go in the cranium is still a mystery, but scientists have uncovered at least three relevant factors. First, there are chemical signals, neural trophic factors, that force certain neurons to grow toward particular target cells. Second, some neurons, such as the Purkinje cells in the cerebrum, send out exploratory fibers they later migrate up. Third, and most important for our purposes, there is cell competition and cell death (Gierer and Muller, 1995).

² If genetic “specification” is an essential property of modules, then, Fodor’s (1983) minimalist modularity hypothesis, which counts only the sensory input systems (and language input systems) as modules, appears far closer to the truth than the evolutionary psychological modularity hypothesis.

For a simple example of the latter, consider the development of motor neuron axons (see, e.g., Thompson, 1993, pp. 307–308). Each motor neuron is connected to at least one, though usually more than one, muscle fiber, yet each muscle fiber is innervated by only one motor neuron. But we do not find this one-many connectivity pattern in the developing embryo. Instead, we find many motor neurons connected to many muscle fibers. Over time, the ends of most of the motor neurons retract until only one neuron controls each muscle fiber. Our best hypothesis for the mechanism for this sort of neuronal pruning is that motor neurons compete with one another for sole activation rights (Changeux and Danchin, 1976). The neuron with the strongest activation wins, and the penalty for losing this fight is death.

Two types of innervation are relevant in the competition for cellular survival. Both spontaneous endogenous firing and the activity produced by sensory inputs shape our final brain structures (Cramer and Sur, 1995; Innocenti, 1995; Katz and Shatz, 1996; Penn and Shatz, 1999; Wong, 1999). Indeed, without both of these sorts of activity, the relevant neural structures simply do not develop. If we block spontaneous retinal ganglion signaling using a chemical intervention, then the lateral geniculate nucleus does not develop its normal layered structure (Shatz, 1996). If we keep one eye closed as our visual system is being pruned so that our brain receives little input from one retina, we will end up functionally blind in that eye. Even though the retinal ganglia produce normal inputs, the areas in cortex to which they feed will no longer respond to visual inputs appropriately (Penn and Shatz, 1999). Other brain regions are similarly dependent on environmental or endogenous stimuli, with cortical regions being largely dependent on innervation from other brain cells (Deacon, 1997). If we block auditory inputs, infants will not learn how to process speech or understand language without some sort of special intervention (using a signed language, for example, or artificially stimulating central auditory cortex) (Sinninger *et al.*, 1999).

What do these facts tell us about evolutionary psychology's modularity hypothesis? No one expects psychological modules to be cleanly drawn in the brain, not even evolutionary psychologists. As Pinker says, modules probably look "like roadkill, sprawling messily over the bulges and crevasses of the brain" (1997, p. 30). Nor do evolutionary psychologists think that environmental inputs are not relevant to the development of modules. Indeed, they frequently repeat the truism that all traits, including psychological modules, are a product of causal interaction between genes and environment. However, evolutionary psychologists treat environmental factors as "triggers" that activate the development of a module in accordance with a "developmental program" that is coded in the genes (Tooby and Cosmides, 1992, pp. 82–87). The picture is that genes encode the information for constructing a module, but that they await a cue from the environment telling them when to begin constructing the module (or, in the case of a conditional genetic program, that they await a cue from the environment telling them which of a limited number of "settings" to apply in constructing the module).

But environmental inputs and endogenous innervations do not simply “trigger” the formation of various processing modules. Instead, during development we find a diffuse proliferation of connectivity, which later brain activity, guided by interaction with the environment, sculpts into its final form (cf., Penn and Shatz, 1999). Brain functions in infants appear to be widely distributed across a variety of neural areas, and as they mature these same functions become localized to particular regions (Goldman-Rakic *et al.*, 1983; Webster *et al.*, 1995). In this process, neurons compete with one another for the sort of processing structure they are going to be, and *brain activity* determines which neurons win this competition, hence which processing roles neurons end up playing. The processing roles of neurons are not laid down in advance by a “genetic program.” Genes play a role, of course, in synthesizing the proteins that construct the initial proliferation of neurons and their connections. But gene expression is not involved in sculpting, and therefore it cannot explain, the “final” form that brain structures take.

To make these points intuitive, consider the analogy of scarring. Scarring is a paradigm process of gene-environment interaction: Scar tissue will not form without both a trauma of environmental origin and the synthesis of specific proteins (which is genetically coded). If we are interested in explaining merely the micro-level process by which cells form scar tissue, appeal to genes may play a heavy role in the explanation, since the explanation will “background” information about the specific type of environmental trauma that initiates the process. If, on the other hand, we are interested in explaining the particular *pattern of scars* on a particular individual’s body (that is, explaining the number of scars, their location, and their particular shapes), our explanation will be entirely in terms of the history of environmental traumas to which that individual has been subjected. This is by no means to deny that genes play a role in the formation of scar tissue. But that role could never explain a particular pattern of scars.

Similarly, although genes clearly play a role in the formation of brain structures (in particular, in the construction of neurons and their connections), the “shape” of those brain structures is explained by the patterns of environmental and endogenous innervation of brain cells, not by any process “programmed” by the genes. So little of our brain structure depends on information provided from the DNA and so much turns on brain/world interaction that our psychological structures, modularized or not, are simply not “genetically specified.” If we need stimulus inputs and endogenous innervations not only to create the structures in the first place, but to maintain them in their current form (and their current form evolves as innervation patterns change), then it is impossible to maintain that our processing streams are “genetically specified.” For the role of genes in the process of brain structure formation is grossly insufficient to explain the (potentially changing) shape that brain structure takes.

We may, nonetheless, have been faced with recurrent adaptive problems throughout our evolutionary history, and our brains may have indeed recurrently produced information-processing solutions to these problems. But separate,

distinct, genetically specified, “brain circuits” were not required to solve these recurrent adaptive problems. Our brains hit upon a different solution: general plasticity that allows particular environmental demands to participate heavily in tailoring the responses to those very demands. This process can produce relatively stable brain structures that specialize primarily in particular information-processing tasks, and these relatively stable structures can be produced with some regularity across populations and down lineages. However, the degree to which the outcomes of human brain development have been regular throughout some of our evolutionary history is due to the fact that generally plastic human brains have encountered recurrent environmental demands throughout that history. It is not due to the presence of a “genetic program” that “specifies” recurrent developmental outcomes. Armchair reasoning leads evolutionary psychologists astray: Our ancestors may have encountered diverse problems, but the brain evolved a general solution to those problems.

Such a general solution, by the way, is not unique to the brain. The immune system constantly faces threat from a structurally diverse array of antigens. Like evolutionary psychologists, we could reason a priori that the immune system must have evolved separate “immuno-modules,” each of which is specialized to solve the adaptive problem posed by a particular antigen. But the immune system has, in fact, hit upon a general solution to the multitude of specific problems posed by antigens. Through a single, elegant process B cells assemble antibodies in response to each invading antigen, without the benefit of a “gene for” each different antibody (Clark, 1995). If we were to look at the antibody population in any given adult human, we would find a dazzling variety of antibodies, each specialized at attacking a specific antigen. But this “structure” within the antibody population of a mature adult has very little to do with genes, having instead been shaped by interaction between the B-cell antibody-assembly process and the antigenic environment to which that individual has been exposed. The “structure” is not a product of gene expression, but of immune system/environment interaction.³

4. What Is Old Is New Again?

In concluding, we would like to address a few objections that may have accumulated in response to the foregoing.

Evolutionary psychologists subscribe to the view, common among cognitive scientists, that cognitive processes can be described at both an information-processing level (which specifies the information and procedures, or algorithms,

³ The analogy with the immune system may bring to mind Edelman (1992), who maintains that the brain and the immune system are both instances of a *recognition system* and that, consequently, their dynamics are in accordance with the same set of principles. We are neutral with respect to these metaphysical views. Our point is simply that, since the immune system evolved a single, general solution to a plethora of diverse adaptive problems, there can be no a priori reason why the brain *could not* have evolved a single, general solution to the plethora of diverse adaptive problems it faced.

involved in performing certain tasks) and a physical level (which specifies “the interaction of neurons, hormones, neurotransmitters, and other organic aspects” in which the information processing is instantiated) (Tooby and Cosmides, 1992, p. 64; see also Cosmides and Tooby, 1987). In short, they maintain a distinction between describing the “software,” or programs, that constitute cognition and describing the “hardware” in which those programs are implemented. Further, according to evolutionary psychologists, an accurate description of information-processing procedures can be provided “without reference to the exact neurophysiological processes that perform these tasks” (Tooby and Cosmides, 1992, p. 64). Indeed, they claim, “knowledge of this hardware . . . is not necessary for understanding the programs [that constitute cognition] as information-processing systems” (Tooby and Cosmides, 1992, p. 65).

The evolutionary psychological concept of a module is clearly a “software” concept, which individuates modules in terms of the information they deal with and the procedures they employ in dealing with it. So, for example, evolutionary psychologists might not be impressed that our speech areas receive inputs from both auditory and visual areas and that how much input we receive from each modality depends on how we are getting linguistic information. (Are we seeing it or are we hearing it?) They wouldn’t be impressed because they could say that our language module is *information specific*. It processes linguistic information, regardless of modality. They could respond that, since all of our arguments are cast at the “hardware” level (concerning the neurological details of brain development and organization), our arguments are not relevant to whether the brain exhibits the evolved *information-processing modularity* they hypothesize. They could, that is, argue that all the points we’ve made concern mere implementation details, and that their modularity hypothesis is neutral with respect to such details. What matters, they could claim, is not *how* the brain succeeds in developing and organizing itself into modules, but only *that it succeeds* in doing so, even if the method it employs in doing so is proliferate-and-prune. As long as domain-specific mechanisms, which function to solve adaptive problems, *somehow* emerge during the course of development, then, even if they overlap one another at the implementation level (so that the same neurons or neural groups process inputs for more than one module), such domain-specific mechanisms could still be psychological adaptations, having been tailored by natural selection to solve their respective problems. For “it is primarily the information-processing structure of the human psychological architecture that has been functionally organized by natural selection, and the neurophysiology has been organized insofar as it physically realized this cognitive organization” (Tooby and Cosmides, 1992, p. 66). Thus, evolutionary psychologists might conclude, by focusing strictly on the “physical level,” our arguments fail to disprove their modularity hypothesis.

There are (at least) two problems with this response. First, it is simply false that knowledge of our “hardware” is not necessary for understanding our cognitive “programs,” as long as understanding our cognitive programs means *getting it right*

about the information processing that takes place in the mind. Standard methods in cognitive science for (dis)confirming computational models are measures of reaction times of human subjects and relative complexity profiles of computational models, e.g., number of computational steps (see Pylyshyn, 1984). But reaction times are measures of *physical* processes, while relative complexity profiles concern the abstract formal properties of a model. To compare the relative complexity profiles of competing computational models with human reaction times, in order to determine which model (more) accurately portrays human information processing, assumptions must be made about precisely how the competing computational processes might be realized in human brains. In short, computational models *must* be hypotheses about the information processing occurring in identified processes in the brain. If they are strictly abstract models, which attempt to remain neutral about implementation, no possible empirical evidence can (dis)confirm them (see Buller, 1993 for the full argument; see also Hardcastle 1995 and Schweizer, forthcoming). Thus, an understanding of cognitive processing precisely *is* an understanding of the *neurophysiological* processes in the brain.

Second, the response presupposes that selection can build adaptations at an abstractly functional level, without bothering with the details about how to build the structures that implement the functions. But this gets things precisely backwards. By definition, adaptations are traits that have a history of preservation and modification under selection in the direction of greater functional effectiveness. And selection occurs only when three conditions are met: (1) there must be variation in traits; (2) this variation must produce fitness differences; and (3) the variation must be *heritable* (Endler, 1986; Ridley, 1996; Futuyma, 1998). Heritability, in turn, requires that differences in the traits responsible for differential fitness be due to *genetic* differences. Thus, if a trait is an adaptation, we know three things about it. (1) At some point in the evolutionary history of the lineage with that trait, there was variation with respect to the possession of the trait; some individuals had it and some didn't. (2) The individuals with that trait had higher fitness, on average, than individuals without. And, most importantly, (3) the difference between having and not having the trait was due to a *genetic* difference between individuals with and without the trait (see also Brandon, 1990, pp. 165–174). This is why evolutionary biologists standardly say: "Adaptive evolution is caused by natural selection acting on genetic variation" (Futuyma, 1998, p. 227). In short, adaptations must have a "genetic basis."

Genes, however, do nothing but code for the proteins that make up an organism's body (Godfrey-Smith, 1999, 2000). So the genetic basis of an adaptation can affect its development only by affecting its structure. That is, while adaptations are (typically) selected because of the functions they perform, the genetic basis of an adaptation can only developmentally produce that function by producing a structure that performs it. Thus, the genes for cognitive adaptations can affect information processing ("software") only by affecting brain processes ("hardware"). Genes cannot possibly affect information processing without affecting the neural

substrate in a way that determines its functional properties (cf., Deacon, 1997, pp. 332–333). Consequently, selection has shaped human cognition *only by* altering the neurophysiology of the brain over evolutionary time; so whatever psychological adaptations humans possess are the indirect result of direct modifications to the so-called “hardware” of the brain. Or, if you like yours with metaphysical jargon, as Fodor says: “Since psychological structure (presumably) supervenes on neurological structure, genotypic variation affects the architecture of the mind only via its effect on the organization of the brain” (2000, p. 88). In contrast to the above passage from Tooby and Cosmides, then, it is primarily the neurophysiology of the brain that has been organized by natural selection.

Perhaps it’s worth clarifying a couple of points here. First, this bit of standard evolutionary biology has nothing to do with the so-called “units of selection problem.” That problem concerns which entities in a hierarchy running from genes to organisms to groups benefit from adaptations (see Sober, 2000, p. 90). Saying that adaptations must have a “genetic basis” is wholly neutral with respect to this issue, since it’s wholly compatible with organisms’ being the recipients of the benefits of an adaptation. Second, it also has nothing to do with what there is “selection for.” In particular, it does not entail that there is only selection for genes. *Obviously* there is selection for adaptive phenotypes; this follows straightforwardly from the definition of “selection for” (see Sober, 1984, pp. 97–102). To say that adaptations must have a “genetic basis” is merely to say that the difference between having (had) and not having (had) an adaptive trait must be due to a genetic difference between individuals with and without the trait. Differences between individuals that are due strictly to environmental differences cannot form the basis of adaptive evolution; so traits that are environmentally induced are not adaptations (see Brandon, 1990, pp. 165–174). Indeed, the standard definition of “evolution” in evolutionary biology is that evolution is change in gene or genotype frequencies across generations in a population (Ridley, 1996; Futuyma, 1998; Sober, 2000). So phenotypic changes across generations that are due to environmental changes (e.g., the increase in average height in the western world during the 20th century, which was due to improved nutrition) are not even biological evolution, let alone adaptive evolution. Only phenotypic changes that are driven by underlying genetic changes can constitute adaptive evolution.

The fact that psychological adaptations are the result of genetic modifications to the brain means that so-called “implementation details” are absolutely central to issues about cognitive adaptation. Furthermore, the neural details can often refute what psychologists take to be clearly and distinctly true. Let us return to the example of the information-specific language module, which had been taken as more-or-less gospel in psychology for decades. Once neurophysiologists began to look at how our auditory system actually works, they came to learn that our brain processes speech and non-speech acoustic information in essentially the same way along the same processing tracts running from the inferior colliculus to the medial geniculate nucleus to primary and secondary auditory cortex. Our brains simply

don't segregate speech from non-speech information.⁴ And there are two morals to be drawn from these facts.

First, even if brain development results in modularization, the process by which this occurs does matter with respect to determining whether those modules are adaptations, as evolutionary psychologists claim. As mentioned earlier, evolutionary psychologists claim that modules have evolved under selection independently of each other – that there has been independent selective shaping of, for example, the alleged module for cheater detection and the alleged module for mate preference. For each module, this requires the presence of numerous genes that have been preserved by selection because of their role in the developmental construction of that module. For all the reasons we have offered, however, the facts don't support the idea of there being such “genes for” all our many relatively special-purpose brain structures. Those structures, instead, are environmentally induced by the processes we have described. They are not “specified” by gene complexes that were selected during human evolutionary history. (This too would explain why our auditory-processing tracts are virtually identical to those found in non-human species who have no linguistic capacities.)

Second, it is a mistake to seek adaptations among the *products* of brain development – that is, among the relatively special-purpose brain structures that emerge during the course of brain development. Those products are highly plastic responses to environmental inputs. Our cognitive adaptation is, instead, the *process* that continually generates and modifies special-purpose brain structures. That is, with the possible exception of our sensory transducers, it is not the contingently stable brain structures in an adult's brain that are adaptations; rather, the brain's very *developmental plasticity* is the adaptation (Deacon, 1997), and the relatively stable structures are byproducts of that adaptation's functioning in a particular environment. Similarly, it is not each and every antibody in an adult's system that is an evolutionary adaptation (although they are *adaptive* in the adult's current environment); rather, the evolutionary adaptation is the process by which the immune system is capable of generating a virtually endless array of antibodies in response to a virtually endless (and evolutionarily novel) array of antigens.

At this point, evolutionary psychologists may object that we have simply come full circle and returned to good-old-fashioned domain-general psychology, which they claim to have soundly refuted. But we haven't exactly, although that may be hard to tell. The reason it may be hard to tell is that evolutionary psychologists conflate two different domain-general conceptions of the mind/brain. That conflation is evident in typical characterizations of the good-old-fashioned view. That view, according to Tooby and Cosmides, maintains that “any evolved component, processing, or mechanism must be equipotential, content-free, content-independent, general-purpose, domain-general, and so on In short, these mechanisms must be constructed in such a way that they can absorb any kind of cultural message or environmental input equally well” (1992, p.

⁴ We thank an anonymous referee for this example.

29). In addition, according to the old view, they say, “natural selection removed ‘genetically determined’ systems of behavior [in humans] and replaced them with general-purpose learning mechanisms or content-independent cognitive processes” (1992, p. 34). But, “equipotential, content-free, content-independent, general-purpose, domain-general” *mechanisms* are one thing; “equipotential, content-free, content-independent, general-purpose, domain-general” *cognitive processes* are quite another. We believe that evolutionary psychologists (though not only evolutionary psychologists) have been right to argue that a mind equipped solely with content-independent, domain-general cognitive learning rules (such as trial-and-error induction) “cannot unassisted perform at least most and perhaps all of the tasks humans routinely perform and need to perform” (Tooby and Cosmides, 1992, p. 39). It is no doubt true that content-independent, domain-general *rules* could never acquire all the information and domain-specific problem-solving procedures that are necessary for speaking a language, say, or detecting cheaters in social exchanges. From this, however, it doesn’t follow that no content-independent, domain-general *mechanism* is capable of producing a structure that is specialized at solving problems in such narrow domains. Indeed, we have shown that the very complex adaptive mechanism by which our brains structure themselves *is* equipotential, content free, content independent, general purpose, and domain general. That general-purpose adaptive mechanism, however, gives rise to domain-dominant processing regions, which in turn give us rich, content-filled, and highly specialized cognitive processes. Our point is that you don’t need (and in fact don’t have) “genetically specified” cognitive modules, which evolved under selection, in order to get cognitive processes tailored to meet highly specific environmental task demands. Though our cognitive processes are highly specific indeed, our brains *learned* how to produce them – not in the sense of acquiring them from antecedently possessed, domain-general cognitive rules, but in the sense of allowing environmental demands to shape a structure that executes those cognitive processes.

We can think of our mind/brains as similar to impressionistic paintings. When we look at them from a distance, we can discern definite shapes, texture, and colors, united into distinct objects existing against a specific background. From a distance, impressionistic paintings look modular, with each object possessing its own unique characteristics. Up close, however, we can see that the oh-so-definite objects are nothing but carefully placed blobs of paint, blobs not distinguishable in terms of any unique characteristics. A background blob is just like an object blob.

Evolutionary psychologists expect our mind/brains to be a paint-by-the-numbers, with all the objects and the scene set by the machinery that cranks out such toys well before any painters arrive to follow the directions. They are wrong; our mind unfolds in an elegant dance, fueled by its environment and its internal dynamics, determined by nothing more than its immediate and more distal surroundings.

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