Innate Modules vs Innate Learning Biases Denise Cummins and Robert Cummins
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ABSTRACT

Proponents of the dominant paradigm in evolutionary psychology argue that a viable evolutionary cognitive psychology requires that specific cognitive capacities be heritable and "quasi-independent" from other heritable traits, and that these requirements are best satisfied by innate cognitive modules. We argue here that neither of these are required in order to describe and explain how evolution shaped the mind.

Since the cognitive revolution over thirty years ago, psychologists have embraced an implicit assumption that cognition and intelligence can be understood in the abstract, without benefit of reference to the biological peculiarities or evolutionary histories of the organisms in which they were instantiated. More often than not, this assumption has been severely challenged by pointedly disconfirming evidence, such as the Garcia effect's impact on theories of conditioning, or the wealth of early emerging cognition that disproved the tabula rasa view of the infant mind. As neuroscience and developmental psychology continues to inform our view of how evolution shaped the mind/brain, contemporary psychologists have come to appreciate that a complete explanation of cognition requires a synthesis of cognitive psychology, cognitive development, neuroscience, and evolutionary science. Each informs the other.

In recent years, a new subdiscipline called *evolutionary psychology* has emerged, populated by psychologists who recognize the necessity of reinstating biological processes in psychological theories. The dominant paradigm in this subdiscipline, however, rests upon assumptions that we have argued are not required in order to understand and explain how evolution shaped the mind. In this paper, we present an alternative view of the impact of evolutionary processes on the mind/brain which, unlike the dominant paradigm, does not preclude domain-general intellectual capacities.

I. The Dominant Paradigm in Evolutionary Psychology

The basic principles of evolutionary psychology *as stipulated by the dominant paradigm* can be summarized as follows:

> *"Principle 1. The brain is a physical system. It functions as a computer. Its circuits are designed to generate behavior that is appropriate to your environmental circumstances.*
>
> *Principle 2. Our neural circuits were designed by natural selection to solve problems that our ancestors faced during our species' evolutionary history. Principle 3. Consciousness is just the tip of the iceberg; most of what goes on in your mind is hidden from you. As a result, your conscious experience can mislead you into thinking than our circuitry is simpler that it really is. Most problems that you experience as easy to solve are very difficult to solve -- they require very complicated neural circuitry.*
>
> *Principle 4. Different neural circuits are specialized for solving different adaptive problems. Principle 5. Our modern skulls house a stone age mind."*

(Cosmides & Tooby, November 6, 2002) These principles imply a strong claim concerning the evolutionary design of the brain and the cognitive architecture it manifests, namely, that *the mind/brain is a collection of independent*

modules that were designed by natural selection to solve adaptive problems faced by our hunter-gatherer ancestors. As Tooby and Cosmides (1995) put it: *[O]ur cognitive architecture resembles a confederation of hundreds or thousands of functionally dedicated computers (often called modules) designed to solve adaptive problems endemic to our hunter-gatherer ancestors. Each of these devices has its own agenda and imposes its own exotic organization on different fragments of the world. There are specialized systems for grammar induction, for face recognition, for dead reckoning, for construing objects and for recognizing emotions from the face. There are mechanisms to detect animacy, eye direction, and cheating. There is a 'theory of mind' module .... a variety of social inference modules .... and a multitude of other elegant machines.* (Tooby and Cosmides, 1995, pp. xiii-xiv)[1]

The motivation for this particular characterization of evolution's impact on neural and cognitive architecture comes from three sources, two empirical and one theoretical. First, the adult brains of many species admit of functionally and neurologically distinct structures and pathways whose destruction impairs some cognitive capacities while sparing others. For example, damage to Broca's area, the hippocampus, the amygdala, and prefrontal cortex impairs (respectively) language production, short term memory formation, processing of threat stimuli, and the processing of socio-emotional aspects of stimuli. In each case, however, overall intellectual function remains unimpaired. In some cases, the destruction of neural pathways results in

"double dissociation" of impaired functions. A classic example is the "what vs where" pathways in visual cortex (Farrar, 1989; Ungerleiter & Mishkin, 1982). The former subserves object recognition while the latter subserves locating objects in space. Damage to the "what" (ventral) pathway produces a syndrome in which the patient cannot identify objects but can reliably identify their location. Damage to the "where" (dorsal) pathway produces the reverse syndrome in which the patient can reliably identify objects but cannot reliably identify their location in space. Function-specific deficits like these are consistent with a modularized view of the brain, a neural and cognitive architecture that consists of independent modules devoted to processing specific stimuli.

The second source comes from cognitive and developmental studies of cognition that are best described in terms of domain-specific, or content-based, effects on performance. Even a cursory review of the literatures on developmental cognition and reasoning performance shows that certain types of knowledge and reasoning strategies come "on line" earlier than others, are acquired more readily than others, or are performed less effortlessly and more accurately than others (see Cummins, 1996a, 1998, in press for a summary of these phenomena).

The third motivational source is theoretical in nature. According to proponents of the dominant paradigm, a viable evolutionary cognitive psychology requires that specific cognitive capacities be heritable and 'quasi-independent'.[2] They must be heritable, because there can be no evolutionary response to selection for traits that are not. They must also be quasiindependent—i.e., there must be additive genetic variance– because adaptive variations in a specific cognitive capacity could have no distinctive consequences for fitness if effecting those

variations required widespread changes in other traits and capacities as well. Since what is innate is heritable, the requirements of heritability and quasi-independence would be satisfied by innate cognitive modules on the plausible assumption that modules are relatively decoupled from each other and from other traits.

By 'innate', proponents of the dominant paradigm do *not* mean 'present at birth', but rather, encoded in the genome. For example, secondary sex characteristics are innate in that their development is encoded in the human genome but they are not present at birth. The relevant notion of a cognitive module derives from Fodor (1983). But, whereas Fodor held that modules were largely peripheral mechanisms, the modules at issue here know no such boundaries. Nor are all of Fodor's characteristics always, or even typically, assumed. Rather, the key features are (1) domain specificity, both informationally and computationally, (2) universality—i.e., present in every normal mind in the species (assuming it has gone to fixation), and (3) relative encapsulation—insensitivity to collateral information[3].

Let us be clear that this is a theoretical position whose primary purpose is to support an evolutionary psychology *as defined by the dominant paradigm.* It is entirely possible for selection to operate on more domain-general capacities, such as greater overall intelligence as long as variation in this trait occurred within a population of agents who compete for the same resources. If, for example, the emergence of a *theory of mind* in young children is simply the consequence of the operation of a general purpose learning device, then the adaptive consequences of having a theory of mind (assuming there are some) would simply be a factor in the selection of the general purpose learning device in question. While this sort of relation between evolution and cognition is theoretically possible, it would hardly justify a new sub

discipline of cognitive evolutionary psychology. Instead, it would serve as motivation for any cognitive psychologist to take evolutionary biology and neuroscience more seriously when developing explanations of cognitive phenomena, a view we strongly embrace.

II. Two Problems with "Innate Modules" or the "Swiss Army Knife" Mind/Brain

A number of arguments have been advanced against "innate modules", and concomitantly, against the dominant view of evolutionary psychology (see e.g., Cheng & Holyoak, 1989; Samuels, Stich, & Tremoulet, 1999). For our purposes, only three (which seem to carry the most weight) will be discussed here.

IIa. The gap between genetic specification and neural connectivity. A direct genetic specification of an innate module would, as far as we know, require specifying synaptic connections in the cortex. (It might require more than this—e.g., neuron differentiation—but it will surely require at least a specification of synaptic connections.) Even if one supposes with Fodor and Pylyshyn (1988) that the level of synaptic connections is not the appropriate level for the specification of cognitive function, cognitive capacities still must be implemented as patterns of synaptic connectivity. If cognitive capacities are specified in the genome, the genome must encode for specific patterns of synaptic connections.

The problem is that the human genome does not appear to have the resources to directly specify a significant amount of cortical connectivity. It is now known that human genotypes contain many fewer genes than previously thought (around 30,000-40,000 instead of a 100,000; International Human Genome Sequencing Consortium, 2001). Among these, it is estimated that from 20-30% (Wills, 1991) to perhaps as many as half (Thompson, 1993) may be implicated in

brain development. However, our brains literally contain trillions of synaptic connections and 5,000 to 15,000 genes are clearly insufficient to directly encode all of these (Churchland, 1995; Buller and Hardcastle, 2000 – see also McCullough 1951). Moreover, it seems that *very few of the genes involved in brain development are concerned with cortical development.* Most of the genes involved in brain development are dedicated to making sure our sensory transducers are properly hooked-up. Winberg and Porter (1998) report that fully 4% of them are concerned with the sensory cells located inside our nose! If there are innate *cognitive* modules, they surely will be found in the cortex.

IIb: <u>The plasticity of cortical tissue</u>. There is sufficient neuroscientific data indicating that the human cortex is deeply immature at birth, leading some to claim that we are not really born with a cortex but with a proto-cortex (O'Leary 1989, 1997). If the pattern of connectivity in some area of the cortex were genetically pre-specified, then it should be impossible to transplant it to another area without bizarre consequences. If, for example, cells in the visual cortex are pre-programmed to finely connect themselves so as to subserve specifically visual processing, then surely they could not function properly when transplanted to the auditory cortex. But it has been shown that such transplants are possible, and that visual cortex cells transplanted to the auditory cortex do connect themselves in a manner that is relevant to auditory processing (O'Leary, Schlaggar, & Stanfield, 1992; see Samuels 1998 for a critique).

It is also well-established that aphasias don't occur in children, suggesting that damage to areas that normally subserve particular cognitive functions (such as language) does not preclude the development of those functions. Other areas of the cortex appear to take over the function

normally handled by the damaged areas, and, in fact massive reorganization of brain structure can occur (see Banich, 1997, pp. 496-503.) Recovery of function can also occur in adults who sustain brain damage, although not to the same extent as children (see Banich, 1997, pp. 489-496).

Finally, there is sufficient evidence of domain general cognitive capacities, such as learning and reasoning, to mitigate against the strong thesis of massive modularity of cognitive function. Although it is true that we reason better about certain domains than others (and that these domains often turn out to be ones that played a significant role in our evolutionary history), it is not the case that we can reason *only* about those domains. Similarly, it is not the case that we can learn only about domains that loomed large in our ancestral past. If that were the case, we would have no disciplines of theoretical physics or computer science.

III. Learning biases and canalization as an alternative view.

These criticisms have been cited as evidence that an evolutionary cognitive psychology is not viable. But a viable evolutionary psychology does not require the existence of such modules in order to satisfy the heritability and 'quasi-independence' constraints. These requirements could also be satisfied by *heritable learning biases*, perhaps in the form of architectural or chronotopic constraints, that operated to increase the *canalization* of specific cognitive capacities in the ancestral environment (Cummins and Cummins, 1999). Chronotropic constraints are constraints on the time course of development (Elman et al, 1996). A trait is said to be more or less canalized as its development is more or less robust across environmental variations (See Ariew, 1996; McKenzie & O'Farrell, 1993; Waddington, 1975). As an organism develops, cognitive capacities that are highly canalized as the result of heritable learning biases might

produce an organism that is behaviorally quite similar to an organism whose innate modules come 'on line' as the result of various environmental triggers.

Nor does the existence of domain-specific or double-dissociation effects imply the absence of domain-general cognitive capacities. Indeed, the case against the view that domain specific knowledge/processors are genetically encoded (a view sometimes referred to as 'representational nativism') is becoming increasingly strong (Elman et al., 1996; see also Samuels, 1998, for a critique; Karmiloff-Smith et al. 1998 for an answer). Conceptualizing matters in terms of learning-bias-induced canalization of domain/task specific cognitive capacities, however, offers the promise of extending a interactionist perspective to cognition—a perspective that takes the effects of genetic endowment, development, and learning to be essentially interdependent. One might ask of a jointly authored paper who wrote which sections, or paragraphs, or even sentences. But it could happen that both authors are jointly responsible for every sentence, with the *degree* of responsibility varying from place to place. This suggests thinking of all of knowledge as co-authored. It suggests that the question is not which concepts (or rules or algorithms) are contributed by the genes and which by learning, but how canalized the development of a given concept or body of knowledge happens to be (Cummins and Cummins, 1999).

To illustrate the LBC view, five examples are presented of capacities that differ in terms of learning bias and degree of canalization.

III.a. Limb development. Limb development is an example of a highly canalized trait with virtually no learning bias involvement. Limbs develop during gestation, and, barring genetic

abnormalities or environmental insult, the developmental "program" proceeds with little required from outside sources (other than, of course, nutritional support). It is not perfectly canalized, however, as evidenced by the tragic history of thalidomide use during gestation.

III.b. Binocular columns. Because the eyes occupy different positions in the face, the brain receives two different images of the same visual scene simultaneously, one offset relative to the other. The brain resolves the binocular disparity to produce a single image, using disparity as cue for depth. This resolution, however, depends on binocular columns in the visual cortex, which are not there at birth; they develop shortly after birth in response to visual stimulation (Banich, 1997, p. 472). Like limb development, development of binocular columns is highly canalized, but not perfectly so: Binocular columns will not develop in the case of extreme exotropy, where the images received by the brain are too disparate to resolve coherently.

III.c. Cortical cell response biases. The previous two cases are examples in which developmental biases exist implicitly in terms of where and how a certain section of the anatomy will develop. But the brain also shows developmental biases that manifest in terms of learning biases. At birth, cells in visual cortex evidence response biases to lines of different orientations (Blakemore, 1974; Blakemore & Cooper, 1970; Hirsh & Spinelli, 1970). These biases are diffuse in that the cell responds to all line orientations, but shows a preference (in terms of heightened firing rate) for vertical orientations. With visual experience, these diffuse biases are sharpened. Now, the cell responds maximally to vertical lines and virtually not at all to lines of other orientations. If the developing animal is raised in abnormal visual environments (e.g., environments in which it is surrounded only by vertical lines), cells that originally show biases

for other orientations will switch their preferences, firing maximally to vertical lines. There is a limit, however, to how much they can adapt; those with preferences for horizontal or near-horizontal line orientations will "go silent", that is, they will cease responding to visual inputs. In this case, the firing preferences of cortical cells begin as biases and require normal environmental input to develop fully. We can say, then, that with respect to edge detection, cortical development is biased but less canalized than limb development. Orientation preferences are present at birth in terms of diffuse biases, but environmental input is required to develop the kind of sharp orientation preferences observed in the mature brain.

III.d. Phonetic contrasts. An even more striking example of content-based developmental biases can be found in early language learning. At birth, human infants can distinguish among all phonetic contrasts that occur in any human language (Eimas, 1975). This ability is lost within the first six months of life as a result of exposure to a language community, and only phonetic contrasts occurring in the language community can be distinguished (Kuhl, 1987). This indicates that language acquisition depends in part on biases that are apparent at birth. The fact that these biases change dramatically in response to environmental input shows that language learning is only moderately canalized. Or to put it differently, the capacity to learn some language or other is highly canalized, but learning a particular language is only moderately canalized.

III.e. Garcia effect. Nervous tissue is conditionable, that is, it evidences the capacity to become habituated to repeated stimulation and to form associations between contingent stimulus events. The development of the capacity to be conditioned is therefore highly canalized. Unlike

language acquisition, however, biases exist that influence which type of associations can be readily learned. The most celebrated demonstration of these learning biases is the Garcia effect (Garcia & Koelling, 1966). Garcia and Koelling allowed rats to drink bitter-tasting water while lights were flashing and are then either shocked them or irradiated them to produce nausea. Contrary to learning theories of the time which rested on the assumption of equipotentiality of conditionable associations, the rats were found to have formed selective associations. Those who were shocked avoid drinking while lights are flashing but are indifferent to the taste of the water they drink, while those who were irradiated avoid drinking bitter water but were indifferent to drinking while lights flash. This result has been replicated numerous times in a variety of species, including humans (Bernstein & Borson, 1986; Etscorn & Stephens, 1973; Garcia, Brett, & Rusiniak, 1989; Logue, 1988).

Humans and other animals also are more likely to develop phobias to stimuli that constituted a threat during the species' evolution (e.g., spiders and snakes) than to other stimuli (e.g., rabbits, flowers or spatulas) (Cook & Mineka, 1989 and 1990; Öhman, 1986; Seligman, 1971). This is not to say that fear responses cannot be induced to otherwise non-threatening stimuli—they can. But doing so requires more repeated pairings of stimuli, and the pairings must occur very close in time. The capacity to be conditioned, therefore, is highly canalized but admits of biases which make some associations more readily acquired than others. These favored associations typically are ones that have biological/evolutionary roots, as is apparent in the described effects.

III.f. Social norms. Finally, early emerging biases are apparent with respect to higher

cognitive capacities, such as reasoning. At birth, humans show a distinct preference for looking at faces when compared to other equally complex stimuli (Goren, Sarty, & Wu, 1975). We also can distinguish among basic emotional facial expressions within the first few weeks of life (Entremont & Muir, 1997). These early emerging biases for attending to social stimuli appear to facilitate the learning of social norms. Reference to social norms appear in the speech of children as young as 24 months of age (Dunn, 1988), and children as young as three years of age reason as indistiguishably from adults about social rules on age-appropriate versions of the Wason card selection task (Cummins, 1996b). These phenomena suggest that the acquisition of social norms (the rules that constrain behavior in one's social group and elicit context-sensitive expectations concerning behavior) is similar to language acquisition. It is facilitated by early emerging biases that allow contrasts in socio-emotional stimuli to be distinguished, but the nature of the rules learned depends on the particular social environment in which learning occurs. In this way, acquisition of social norms and the reasoning strategies that subserve them is similar to language acquisition—highly canalized in terms of a general capacity for these cognitive skills but only moderately so with respect to which rules are induced.

IV. The LBC view, heritability, and quasi-independence.

LBC does not require a genetically problematic representational nativism, yet it does appear to satisfy the two fundamental criteria set out above for the viability of evolutionary cognitive psychology: heritability and quasi-independence. We discuss these in turn.

IV.a Heritability. The LBC framework assumes that there was variability in learning biases in ancestral populations. A learning bias makes acquisition of a cognitive trait more likely.

That is, it can increase the canalization of a cognitive trait. When such a trait proves adaptive, there will be a tendency for selection to spread the bias(es) responsible for increased canalization through the population. The end result is a population with a highly canalized cognitive trait.

An example of such a learning bias is infants' preference for looking at facial stimuli and their ability to appropriately distinguish among certain canonical emotional expressions. Entraining attention on facial stimuli in this way would constitute a learning bias that facilitated acquisition of social norms: If you're spending a good deal of time watching the faces of behaving agents around you, you will receive ample opportunities to notice which behaviors are appropriate (and expected) in which circumstances.

IV.b. Quasi-Independence. If we assume that learning biases are not themselves representationally implemented, then, as remarked above, they will not be intrinsically specific to particular learning tasks, and this will impact the degree to which the cognitive traits they enable can evolve independently. This is both good news and bad news. The good news is that a demonstrable coupling between cognitive traits would provide strong evidence for a bias that grounded both, since the existence of such a bias would immediately explain the coupling. The hypothesis thus has the capacity to generate some interesting and falsifiable predictions. The bad news is that, as we noted in the introduction, some degree of independence among cognitive traits seems required to make evolutionary analysis a useful and interesting tool in cognitive psychology. It is worth noting that, even if learning biases proved to be quite general, it would not follow that they could not give rise to a modular functional architecture. A single bias could canalize development of several domain specific capacities. For instance, Jacobs, Jordan, & Barto

(1991) have shown 'how mixtures of expert networks exposed to a what/where problem […] will always assign the 'where' task to the expert network which possesses a linear activation function. The implication is that networks do not necessarily need to be designed to carry out particular tasks. Rather, the task will select the network which has the appropriate (i.e. innate) computational properties'. (McCleod, Plunkett, & Rolls, 1998)

V. Conclusion.

The LBC framework has a long an venerable history in psychology, reaching back at least as far as the "biological preparedness" proposed by Seligman and others to account for domain-specific effects in otherwise domain-general conditioning, such as the Garcia effect. Historically, psychological theories have often fallen short when evolution and biology have not been taken into account (see Cummins, in press, for the details of this argument). This is where the dominant paradigm gets it exactly right: A complete explanation of cognition requires a synthesis of cognitive psychology, neuroscience, and evolutionary science. Each informs the other.

Unlike the dominant paradigm, however, the LBC view does not hamstring psychological theories with a view of the mind/brain that makes no room for domain-general intellectual capacities. If the mind/brain truly is a "Swiss army knife", it is not clear how phenomena such as domain-general learning and reasoning or the high degree of cortical plasticity observed during development can be explained. Cognitive capacities such as memory formation and retrieval, which seem to be dispersed throughout the brain (involving both cortical and subcortical structures) also become difficult to explain. Instead, it perhaps may be more fruitful to think of evolutionary forces as shaping both domain-general and domain-specific cognitive capacities, a scenario that is indeed possible as long as genetic variation responsible for these traits exists.

REFERENCES

Ariew, A.: 1996, "Innateness and Canalization", *Philosophy of Science, 63,* S19-S27.

Banich, M.T. (1997) *Neuropsychology: The neural bases of mental function.* Boston: Houghton-Mifflin.

Blakemore, C. (1974) Developmental factors in the formation of feature extracting neurons. In F.G. Worden and F.O. Smith (eds.), *The Neurosciences, 3rd Study Program.* Cambridge, MA: MIT press. Blakemore, C., & Cooper, G.F. (1970) Development of the brain depends on visual environment, *Nature, 228,* 477-478.

Campos, J.J., & Stenberg, C. (1981) Perception, appraisal, and emotion: The onset of social referencing. In M. Lewis and L. Rosenblum (eds.), *Infant social cognition: Empirical and theoretical considerations.* Hillsdale, NJ: Erlbaum. Carey, S., & Spelke, E. Domain-specific knowledge and conceptual change. In L.A. Hirshfeld &

S.A. Gelman (eds.), *Mapping the mind: Domain specificity in cognition and culture.* Cambridge: Cambridge University Press. Cook, L.M., & Mineka.S. (1989) Observational conditioning of fear to fear-relevant versus fear-irrelevant stimuli in rhesus monkeys, *Journal of Abnormal Psychology, 98,* 448-459.

Cook, L.M., & Mineka, S. (1990) Selective associations in the observational conditioning of fear in rhesus monkeys, *Journal of Experimental Psychology: Animal Behavior Processes, 16,* 372-389.

Cummins, D.D. (1996a) Evidence for the innateness of deontic reasoning. *Mind & Language, 11,* 160-190.

Cummins, D.D. (1996b) Evidence of deontic reasoning in 3- and 4-year-olds. *Memory & Cognition, 24,* 823-829.

Cummins, D.D. (1998). Social norms and other minds: The evolutionary roots of higher cognition. In D.D. Cummins & C. Allen (eds.), *The evolution of mind* (pp. 30-50). New York: Oxford University Press.

Cummins, D.D. (in press) The evolution of reasoning. In J. Leighton & R. Sternberg (eds.), *The Nature of Reasoning,* Cambridge University Press.

Cummins, D.D., & Cummins, R.C. (1999). Biological preparedness and evolutionary explanation. *Cognition, 73,* B37-B53.

Dunn, J. (1988) *The beginnings of social understanding.* Oxford: Basil Blackwell.

Eimas, P. D. (1975) Speech perception in early infancy. In L.B. Cohen and P. Salapafek (eds.), *Infant perception,* New York: Academic Press.

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

Entremont, B. & Muir, D. W. (1997) Five-month-olds attention and affective responses to still faced emotional expressions. *Infant Behavior & Development, 20,* 563-568.

Etscorn, F., & Stevens, R. (1973) Establishment of conditioned taste aversions with a 24-hour CS-US interval. *Physiological Psychology, 1,* 251-253.

Farah, M. (1989) The neuropsychology of mental imagery. In J.W. Brown (ed.),

Neuropsychology of visual perception. Hillsdale, NJ: Erlbaum.

Fodor, J.A. (1983) *The modularity of mind: An essay on faculty psychology.* Cambridge, MA: Bradford/MIT Books.

Garcia, J., & Koelling, R.A. (1966) The relation of cue to consequence in avoidance learning. *Psychonomic Science, 4,* 123-124.

Garcia, J., Brett, L.P., & Rusiniak, K.W. (1989) Limits of Darwinian conditioning. In S.B. Klein and R.R. Mowrer (eds.), *Contemporary learning theories: Instrumental conditioning theory and the impact of biological constraints on learning,* Hillsdale, NJ: Erlbaum.

Goren, C.C., Sarty, M., & Wu, P.Y.K. (1975) Visual following and pattern discrimination of face-like stimuli by newborn infants. *Pediatrics, 59,* 544-549. Hirsh, H.V.B., & Spinelli, D.N. (1970) Visual experience modifies horizontally and vertically oriented receptive fields in cats. *Science, 168,* 869-871.

Jacobs, R.A., Jordan, M.I., & Barto, A.G. (1991). Task decomposition through competition in a modular connectionist architecture: The what and where vision tasks. *Cognitive Science, 15,* 219-250.

Karmiloff-Smith, A. (1992). *Beyond modularity.* Cambridge, MA: MIT Press. Kuhl, P.K. (1987) Perception of speech and sound in early infancy. In L.B. Cohen and P. Salapafek (eds.), *Infant perception,* New York: Academic Press. Leslie, A.M. (1992) Pretense, autism, and the "Theory of Mind" module. *Current Directions in Psychological Science, 1,* 18-21. Lewontin, R.C. (1978). Adaptation. *Scientific American, 239,* 157-169.

Logue, A.W. (1988) A comparison of taste aversion learning in humans and other vertebrates: evolutionary pressures in common. In R.C. Bolles and M.D. Beecher (eds.), *Evolution and Learning,* Hillsdale, NJ: Erlbaum. Öhman, A. (1986) Face the beast and fear the face: Animal and social fears as prototypes for evolutionary analysis of emotion. *Psychophysiology, 23,* 123-145. O'Leary, D.D.M. (1989). Do cortical areas emerge from a protocortex? *Trends in Neuroscience, 12,* 400-406.

O'Leary, D.D.M. (1997). Area specialization of the developing cortex: Differentiation, developmental plasticity, and genetic specification. In Magnusson et al., (eds.), *The lifespan development of individuals: Behavioral, neurobiological, and psychosocial perspectives-A syntthesis,* (pp. 23-37). Cambridge: Cambridge University Press.

O'Leary, D.D.M., Schlaggar, B.L., & Stanfield, B.B. (1992). The specification of sensory cortex: The lessons from cortical transplantation. *Experimental Neurology, 115,* 121-126. Roe, A. W., Pallas, S. L., Kwon, Y. H., Sur, M. (1992) Visual projections routed to the auditory pathway in ferrets: Receptive fields of visual neurons in primary auditory cortex.

Journal of Neuroscience, 12, 3651-3664. Samuels, R. (1998). What brains won't tell us about the mind: A critique of the neurobiological argument against representational nativism. *Mind and Language, 13,* 548-570.

Samuels, R., Stich, S.P., Tremoulet, P.D. (1999). Cognitive science and human rationality: From bleak implications to Darwinian modules. In E. LePore & Z. Pylyshyn (eds.), *Rutgers University Invitation to Cognitive Science.* Oxford: Blackwell.

Seligman, M.E.P. (1971) Phobias and preparedness. *Behavior Therapy, 2,* 307-320.

Stenberg, G. & Hagekull, B. (1997) Social referencing and mood modification in 1-year-olds, *Infant Behavior & Development, 20,* 209-217.

Tooby, J., & Cosmides, L. (1995) Foreword. In S. Baron-Cohen *Mindblindness*, xi-xviii.

Ungerleiter, I.G., & Mishkin, M. (1982). Two cortical visual systems. In D.J. Ingle, MA., Goodale, & R.J.W. Mansfield (eds.), *Analysis of visual behavior* (pp. 549-586). Cambridge, MA: MIT Press. Waddington, C. H.: 1975, *The evolution of an evolutionist*. Ithaca, NY: Cornell University Press.

Footnotes

[1] Cosmides and Tooby are not the only proponents of this view. See also, e.g., Carey & Spelke (1994) and Leslie, 1992.

[2] The term is Lewontin's (1978). 'Quasi-independence means that there is a great variety of alternative paths by which a given characteristic may change, so that some of them will allow selection to act on the characteristic without altering other characteristics of the organism in a countervailing fashion; pleiotropic and allometric relations must be changeable.' The point is that, from a genetic point of view, an intermediate grade of trait interaction/independence is required: Too much interaction (universal pleiotropy) means that there is basically one evolutionary unit that must evolve as a unit; too little interaction (complete additivity) blocks the developmentally concerted integration of complex phenotypes because the component genes/effects are always being divided up in reproduction. In between is the realm of quasi-independence.

[3] This characterization differs somewhat from the 'Darwinian module', ascribed to evolutionary psychology by Samuels, Stich and Tremoulet (1999). They define a Darwinian module as 'an innate, naturally selected, functionally specific and universal computational mechanism which may have access (perhaps even unique access) to a domain specific system of knowledge of the sort we've been calling a Chomskian module.' Encapsulation is not mentioned in this quote, but we retain this characteristic from Fodor's original formulation because, without it, it is difficult to distinguish a module from a mere 'subroutine'.