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# Biological preparedness and evolutionary explanation

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**Abstract**

It is commonly supposed that evolutionary explanations of cognitive phenomena involve the assumption that the capacities to be explained are both innate and modular. This is understandable: independent selection of a trait requires that it be both heritable and largely decoupled from other 'nearby' traits. Cognitive capacities realized as innate modules would certainly satisfy these contraints. A viable evolutionary cognitive psychology, however, requires neither extreme nativism nor modularity, though it is consistent with both. In this paper, we seek to show that rather weak assumptions about innateness and modularity are consistent with evolutionary explanations of cognitive capacities. Evolutionary pressures can affect the degree to which the development of a capacity is canalized by biasing acquisition/learning in ways that favor development of concepts and capacities that proved adaptive to an organism's ancestors. 

*Keywords:* Evolutionary psychology; Cognition; Development; Plasticity; Modules; Innate

## 1. Introduction

Evolutionary explanations of cognitive phenomena are often thought to imply that the cognitive capacities targeted for evolutionary explanation are *innate* and *modular*. We argue that neither of these implications is necessitated by evolutionary explanations of particular cognitive effects. Instead, we argue that issues of innateness should be conceived in terms of *canalization*, i.e the degree to which the development of a trait is robust across normal environmental variations (Ariew, 1996; McKenzie &

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O'Farrell, 1993; Waddington, 1975). Evolutionary pressures can affect the degree to which the development of a trait is canalized. High canalization can be the consequence of biasing learning/acquisition processes in ways that favor the development of concepts and cognitive functions that proved adaptive to an organism's ancestors. The end result of these biases is an adult organism that exhibits a number of highly specialized cognitive abilities that have many of the characteristics associated with modules: functional specialization, reliable emergence in spite of considerable environmental variability, and some degree of informational encapsulation.

This perspective makes it evident that criticisms of innate cognitive modules are not *ipso facto* criticisms of evolutionary explanations of cognitive capacities. Since evidence for modularity in the developed organism is compatible with a high degree of neural placticity in the early stages of development, it is possible to have an evolutionary explanation of cognitive modules that does not assume these modules to be innate in the sense in which this means unlearned or present at birth or coded in the genes. Of course, an evolutionary approach to cognition is compatible with modules that are innate in this sense. Our point is simply that it need not presuppose them.

We begin by discussing two factors that appear to be prominent in motivating interest in evolutionary approaches to cognition. We then characterize how evolutionary explanations of cognitive phenomena that appeal to innate modules are typically interpreted. We then review some of the criticisms that have been levelled against this approach. Finally, we expound and defend a conception of the relation between natural selection and cognitive development that is responsive to worries about innate modules yet compatible with an evolutionary explanation of specialized and relatively independent cognitive mechanisms in adult organisms.

## 2. The motivation for an evolutionary approach to cognition

Two factors appear to be prominent in motivating researchers to adopt an evolutionary approach to cognition. The first is simply that cognitive psychologists are in the business of explaining cognition in biological organisms, and biological organisms are the product of evolutionary forces. To put it more succinctly:

- If you are a materialist, then you are committed (at least implicitly) to the view that *The mind is what the brain does.*

That is, our cognitive and emotional functions are instantiated as neurological processes. Unless you are a creationist, you are also committed to the view that

- *The brain (like all other organs) was shaped by evolution.*

If you accept these two premises, you are also committed to accepting their logical conclusion, namely, that

- *The mind was shaped by evolution.*

This much we believe is uncontroversial.

A second factor is the need to account for domain-specificity effects in cognition, their early emergence in development, and their apparent adaptiveness. As illustrations, consider the following three episodes in the recent history of psychology.

Consider first simple inductive learning processes. Early learning theories rested on the assumption that an association could be made between any two stimuli through repeated pairings, yet it subsequently became apparent that some associations were learned more readily than others. This 'fast-tracked' learning typically involved contingencies that had significant survival advantages during an organism's evolutionary history. Humans (and other primates) appear predisposed to acquire fear responses to classes of animals that proved dangerous to our ancestors, such as spiders and snakes (Cook & Mineka, 1989; Cook & Mineka, 1990; Öhman, 1986; Seligman, 1971). It is also notoriously easy to acquire taste aversions to foods that make us ill even if the time between ingestion and illness is quite long (Bernstein & Borson, 1986; Etscorn & Stephens, 1973; Garcia, Brett & Rusiniak, 1989; Logue, 1988). Perhaps the most dramatic demonstration is the oft-replicated Garcia effect. If animals are allowed to drink quinine-adulterated water in a room with flashing lights, those subsequently shocked will avoid drinking while the lights are flashing but are indifferent to bitter-tasting water, while those subsequently irradiated to produce nausea will avoid bitter-tasting water but are indifferent as to whether lights are flashing while they drink (Garcia & Koelling, 1966). As Hilgard and Bower (1975) (p. 574) put it: 'One might say that the animal is innately preprogrammed to see certain cues and responses as 'naturally fitting' together, so that they are readily learned'. These favored associations often appear to be ones that have adaptive value.

As a second example, early theories of cognitive development proposed during the 1950s rested on the assumption that infants were little more than sensory-motor systems, and that complex concepts were constructed from these simple building blocks through experience with the environment (Piaget, 1952). But the last two decades of research on infant cognition has forced developmental psychologists to re-examine their assumptions about the infant mind. Some types of domain-specific knowledge appear to emerge quite early in infancy, before infants have had sufficient time to induce this knowledge through experience. These data seem to indicate that infants are cognitively predisposed to interpret the world in terms of agents and objects whose behaviors are constrained by different sets of principles (e.g. Leslie & Roth, 1994; Spelke, 1994).

A third example comes from research on higher cognition. During the 1970s, theories of human reasoning were proposed in which reasoning was presumed to be a content-free process, sensitive only to syntactic properties of reasoning problems. Subsequent research reported such robust domain-specific effects that even the staunchest proponents of the syntactic view of reasoning began incorporating domain-specific parameters in their models (Braine & O'Brien, 1991; Rips, 1994). Many of these 'privileged' domains turn out to be ones that developmentalists identified as 'early emerging' and that can plausibly assumed to have had adaptive value, such as causality, frequency, ontological category, and certain social

reasoning strategies (Cosmides, 1989; Cosmides & Tooby, 1992; 1994; Gigerenzer & Hug, 1992; Cummins, 1996a,b,c,d; 1997; 1998a,b,c, 1999a,b,c,d).

In each case, psychologists had to re-think their theories in order to account for biases in learning and cognition that are apparent in their data. In the case of biological organisms, a plausible interpretation is that early-emerging, domain-specific, adaptive capacities are the result of evolutionary forces.

## 3. Characterization of the innate modules view

According to some researchers, the early emergence and domain-specificity of many cognitive capacities is evidence that evolution has produced a mind best characterized as a collection of innate and independent modules, each of which arose in response to environmental pressures during a species' evolution.

- Our 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 & Cosmides, 1995) (pp. xiii–xiv).
- We argue that human reasoning is guided by a collection of innate domain-specific systems of knowledge. Each system is characterized by a set of core principles that define the entities covered by the domain and support reasoning about those entities. Learning, on this view, consists of an enrichment of the core principles, plus their entrenchment, along with the entrenchment of the ontology they determine. In these domains, then we would expect cross-cultural universality; cognitive universals akin to language universals (Carey & Spelke, 1994) (p. 169).
- I have argued that the normal and rapid development of theory-of-mind knowledge depends on a specialized mechanism that allows the brain to attend to invisible mental states. Very early biological damage may prevent the normal expression of this theory-of-mind module in the developing brain, resulting in the core symptoms of autism (Leslie, 1992) (p. 20).

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, and (3) relative encapsulation – insensitivity to collateral

information. This characterization differs somewhat from the 'Darwinian module' typically ascribed to evolutionary psychology.

- To sum up, a (prototypical) Darwinian module is 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 (Samuels, Stich & Tremoulet, 1999).

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'. We do not include being naturally selected, since the origin of such modules, if there are any, is largely what is at issue.

Part of the motivation for the innate modules view is that, without the assumption of innate modules, there seems little latitude for evolutionary explanations of cognitive phenomena. For example, if there is no innate theory of mind module, it might seem the adaptive consequences of having a theory of mind could have no specific effect on selection. It could only have the indirect effect of reinforcing whatever general purpose architecture makes a theory of mind learnable in the environments in which our ancestors found themselves. While not utterly trivial, this is certainly not the basis for a new subdiscipline, and certainly not for evolutionary psychology as currently practiced. The innate modules view, on the other hand, *seems* to be just what is needed to ground a rich evolutionary cognitive psychology. If there *is* a theory of mind module, and it is heritable, then it might have spread through the population because it was adaptive.

The underlying line of thought here seems to be this: for an evolutionary explanation of a cognitive capacity to be viable, we must assume (a) that the capacity is specified in the genes, since the genes are the mechanism for the inheritance of evolved traits, and (b) that it is modular, since the independent evolution of specialized capacities requires that these be largely decoupled from other independently evolved systems. We have not seen this argument explicitly advanced by evolutionary cognitive psychologists. We offer it here as a plausible explanation of the link between evolutionary cognitive psychology and the assumption of innate modules.

To sum up: there appear to be two basic lines of argument for the innate modules view. One is that the existence of innate modules would explain the well-documented domain specificity and early emergence of many cognitive capacities. The other is that the evolution of cognition seems to require an architecture of relatively independent and heritable capacities.

## 4. Objections to innate modules

The objections to the innate modules view divide into two classes. The first and most fundamental consists of arguments from neural plasticity. The second consists of arguments defending the sufficiency of a few general-purpose learning mechan-

quate information in the environment (e.g. the Garcia effect and other domain-specific effects), etc. Replies are therefore attempts to show that one or another learning architecture is actually up to the job, or that the opposition has underestimated the available information or resources.

We suggest that for higher-level cognitive behaviors, most domain-specific outcomes are probably achieved by domain-independent means. (Elman et al., 1996) (p. 359).

...the general framework for induction proposed by Holland, Holyoak, Nisbett and Thagard (1986) stresses the importance of constraints of various degrees of generality in determining whether and how readily knowledge about a regularity in the environment will be induced. Two of the most general constraints they proposed involve the role of failed expectations concerning goal attainment in triggering inductions, and the role of knowledge about variablility of classes of objects and events in determining the propensity to generalize. Within this framework, it is clear that pragmatically useful inductions will often be triggered... (Cheng & Holyoak, 1989) (p. 308).

Our principle criticism of [domain specific] approaches put forward to account for biases and content effects is that they lack the generality of our model... Domain-specific knowledge may influence the parameters in our model, and the utilities subjects use... (Oaksford & Chater, 1994) (p. 626).We do not propose to rehearse this debate here. We merely remind the reader that these arguments need to be made case by case, and that a sound case against an innate module for some cognitive capacity is not *ipso facto* a case against selection of that capacity.

The argument from neural plasticity and the criticisms of poverty of stimulus style arguments address innateness, not modularity. Of course, if cognitive capacites are not innate, they are not innate and modular. Still, there is a close connection between these criticisms of innateness and wariness about modularity. If general purpose learning mechanisms account for cognitive capacities, it would be somewhat surprising if these capacities were highly modular. Not that general learning mechanisms could not produce modules – they surely could – but it is not clear why they would. Any argument against domain specific learning would therefore appear to be also a *prima facie* argument against domain specific computational mechanisms, and hence against encapsulation.

## 5. Objections to general-purpose learning

The widely recognized difficulty with a general-purpose learning approach is that it does not explain the 'biases' that are plainly evident in the newborn brain. Although it is possible, for example, to force auditory cortex to acquire the capacity for visual processing, the result is not normal vision (Roe, Pallas, Kwon, & Sur, 1992). Similarly, it is highly unlikely that the hippocampus is suited to do either visual or auditory processing. Thus, there are neurological biases present at birth, and these are the result of millions of years of evolution operating on the ontogeny of

the modern mammalian brain. This means that the developing brain is not entirely plastic.

Throughout development, however, non-plasticity is also a hallmark of the brain. For example, early in gestation undifferentiated precursor cells become fated to express the characteristics of the brain region where they migrate to and remain. Thus, plasticity and non-plasticity occur during prenatal development (Gazzaniga, Ivry & Mangun, 1998) (p. 485).

Moreover, biases seem to exist not just with respect to sensory/perceptual functions, but with respect to cognitive development as well. As mentioned earlier, the explosion of data on infant cognition that has come about in the last decade indicates that the infant mind is cognitively predisposed to interpret the world in terms of agents and objects whose behaviors are constrained by different sets of principles. With respect to agents, they appreciate the inherently reciprocal nature of social interactions (Vandell & Wilson, 1987), and the meaning of emotional facial expressions (Campos & Stenberg, 1981; Stenberg & Hagekull, 1997). With respect to objects, they appreciate that objects are permanent entities that cannot occupy the same space at the same time (Baillargeon, 1987; 1994; Spelke, 1994) whose movements are constrained by physical causality (Leslie, 1987; Leslie & Keeble, 1987) and principles of biomechanical movement (Bertenthal, 1984; 1985). They also appreciate the abstract concept of number and arithmetic operations (Starkey, Spelke & Gelman 1990; Wynn, 1992). A purely general-purpose learning account of human development would be faced with the unwelcome task of explaining data such as these as biases in the environment that are exploited by the learner. This is reminiscent of the behaviorist tendency to posit histories of reinforcement required by their learning theories without any direct evidence that such histories existed, or of the tendency of neo-Gibsonians to posit affordances when confronted with perceptual capacities their theories could not otherwise explain.

## 6. A third interpretation: evolution affects degree to which cognitive traits are canalized

As diametrically opposed as these positions seem to be, there in fact exists a common ground which they occupy and upon which a coherent evolutionary psychology can be founded. The following quotations exhibit this common ground.

There can be no question about the major role played by our biological inheritance in determining our physical form and our behaviors. We are not empiricists. What troubles us about the term innate is that, as it is often used in cognitive and developmental sciences, it suggests an overly simplistic view of how development unfolds. To say that a behavior is innate is often taken to mean – in the extreme case – that there is a single genetic locus or set of genes which have the specific function of producing the behavior in question, and only that behavior. (Elman et al., 1996) (p. 357)

...a better way of thinking about it is that the brain has to be assembled, and the assembly requires project scheduling over an extended timetable The timetable does

not care about when the organism is extruded from the womb: the installation sequence can carry on after birth. The process also requires, at critical junctures, the intake of information that the genes cannot predict. (Pinker, 1997) (p. 238).

In other words, rather than assume that early emerging and specialized cognitive capacities are either innate or learned, we may suppose instead that organisms do not inherit modules fully formed, but have a *biological preparedness* (Seligman, 1971) to very quickly develop specialized cognitive functions for solving classes of problems that were critical to the survival and reproductive success of their ancestors. Conceiving of cognitive functions in this way puts them on a par with other biological traits that can differ in their degree of *canalization*, that is, in the degree to which the environment plays a role in their expression (Waddington, 1975; McKenzie & O'Farrell, 1993; Ariew, 1996).

### *6.1. Nature, nurture and canalization*

The nature-nurture debate in cognitive psychology is generally a debate about what knowledge (rules, theories, concepts) is innate, and what is learned. Couching the issue in terms of canalization or biological preparedness, however, allows us to see things quite differently. Consider a jointly authored paper. We might ask who authored which sections or paragraphs or even sentences. This is how people tend to think of the nature vs. nurture issue in the cognitive realm. But it could also happen that both authors are responsible for every sentence, with the *degree* of responsibility varying from sentence to sentence, or section to section. The suggestion is that we should think of our cognitive abilities as all thoroughly co-authored. From this perspective the question is not *which* concepts or capacities are contributed by the genes, and which by learning, but rather how canalized the development of a given concept or cognitive capacity is: how much variability in the learning environment will lead to the same developmental end-state? An advantage of this way of thinking is that we see at once that little or nothing in development is inevitable, even though it may be (nearly) universal. And when we investigate things in this light, we are led to ask which variations in the learning environment will divert the stream into a different and perhaps preferable canal. (See Lewontin, 1974 for a similar analysis of the contributions of genes and environment).

This perspective does not rule out innate concepts (representational nativism) or innate computational modules, but neither does it require them. Our concern, to repeat, is to articulate a framework for an evolutionary cognitive psychology that is maximally flexible. Evolutionary cognitive psychology requires relatively independent heritable cognitive traits. These could result from innate modules, but they could also result from developmental/learning biases that interact with the environment in such a way as to yield highly canalized cognitive traits.

### *6.2. Two examples of canalization*

Consider first the neurological changes that subserve the development of vision and language. Binocular columns (used in depth perception) are not present at birth, but appear in the visual cortex during a critical period after the infant has received

visual input (Banich, 1997) (p. 472). Other visual cortical cells show diffuse line orientation 'preferences' at birth, firing maximally to lines of a particular orientation (e.g. vertical), but responding to lines of other orientations as well, albeit to a lesser degree (Hubel, 1988). After receiving visual input, however, these cell preferences are sharpened so that they respond maximally *only* to lines of a particular orientation (Blakemore, 1974). Further, if visual input is restricted to only a single orientation (e.g. the animal is exposed only to lines of vertical orientation), the majority of cells will shift their preferences to match their visual experiences, responding maximally to lines of vertical orientation even if their initial preferences were for lines of other orientations (Blakemore & Cooper, 1970; Hirsh & Spinelli, 1970). The animal, in short, is blind to all line orientations except that to which it was exposed during this critical period. Development of visual cognitive functions depends on tightly coupled transactions between neurological predispositions and environmental inputs. Under normal circumstances, binocular columns will form in a particular area of visual cortex, and initial diffuse biases in visual cortical cells will sharpen into definite response preferences as a result of environmental stimulation during a critical period of development. The neurological predispositions are there at birth, but require an environmental 'co-author' to fully develop into functions that subserve visual cognition.

Next, consider language development. Like vision, language development also shows a complex pattern of interplay between innate biases and environmental input. Deaf babies will begin to babble vocally just as hearing babies do, but their babbling declines and eventually ceases, presumably because they don't receive the auditory feedback hearing babies do (Oller & Eilers, 1988). Babbling deaf babies are practicing sounds that they have never heard, a phenomenon that is perhaps best explained as the unfolding of a biological program that requires environmental feedback to fully develop. Infants are also born with the capacity to hear all phonetic contrasts that occur in human communicative systems, yet within the first year of life they lose the capacity to distinguish among phonemes that are not marked in their language community (Eimas, 1975; Kuhl, 1987). Thus, they initially exhibit an auditory bias in processing speech sounds that treats the phonemes of human language as signal and everything else as noise, and subsequent language inputs modify this bias to include as signal only the phonemes of the child's native tongue. There also appears to be a critical period for language acquisition that ends approximately at puberty: children who do not acquire their first language during this critical period fail to acquire the rules governing the use of grammatical morphemes and the syntactic constraints necessary for forming grammatical sentences (Curtiss, 1977; Pinker, 1994). Further, the ability to extract the grammatical rules of a natural language is selectively impaired in certain genetic disorders (Gopnik, 1990a,b).

There are two important lessons to be drawn from these familiar examples. The first is that biological preparedness comes in degrees, and is probably best conceived in terms of canalization. A combination of genetic and environmental factors cause development to follow a particular pathway, and once begun, development is more or less likely to achieve a particular end-state depending on the type and amount of

environmental stimulation the organism receives. Limb development is highly canalized in humans (humans everywhere grow limbs in the same way) but not perfectly so, as the example of Thalidomide shows. Language is highly canalized, though not so highly as limb development. Tennis and chess are comparatively low on the canalization scale.

The second lesson to be drawn from the examples lately rehearsed is that the environment can influence trait development in many different ways. The most interesting of these to the psychologist is learning. It is important to keep in mind that learning can affect the development of even highly canalized traits. Thus language, though highly canalized, is still learned. Biology puts strong constraints on what properties a language must have to be learnable (as a first language), and it virtually guarantees that language will be learned in a huge variety of environments. This is what is meant by the claim that language acquisition is highly canalized. Few doubt that the high canalization of language acquisition is to be explained by a specific biological preparedness for language acquisition. But our genetic endowment does not determine which language we will acquire or even whether we will acquire any. This is determined largely by the learning environment.

The important point is this: As long as we continue to pose the question 'Which cognitive traits are learned and which are innate', we will continue to run the risk of misconceiving the issue. Maybe everything is some of each, the question being how much. In the next section, we provide an illustration of how this could work for 'higher' cognition.

## 7. How preparedness and environmental input can constrain higher cognition

As an example of how genetically encoded biases and environmental input can combine to channel the development of higher cognitive functions, consider the development of social reasoning. Newborns (no more than a few minutes old) show a distinct bias for looking at faces as compared to other equally complex stimuli (Goren, Sarty & Wu, 1975). Ten-week-old infants have been found to distinguish among emotional facial expressions (Entremont & Muir, 1997). Within the first year of life, they also engage in *social referencing*. looking at their caregivers' reactions to novel stimuli (e.g. Stenberg & Hagekull, 1997). By 2 years of age, they can succeed at tasks that require them to grasp another's goals, desires, or preferences (e.g. Bartsch & Wellman, 1989; Flavell, Favell, Green & Moses, 1990; Meltzoff, 1995), and can readily identify violations of arbitrary social rules (Cummins, 1999b). By 3 years of age, children spontaneously adopt a violation detection strategy when attempting to determine whether or not a social rule is being followed, but not when attempting to determine whether a conditional utterance is true or false, and the magnitude of this reasoning bias is equivalent to the magnitude found in the adult literature (Cummins, 1999b). Children also find it easier to recognize instances of cheating than instances that prove a rule false (Cummins, 1999a; Harris, 1996).

These early-emerging and robust domain-specific effects can be explained as the

result of a biological preparedness to (a) distinguish agents from other objects, (b) entrain one's attentions on facial expressions, and (c) attempt to engage in reciprocal interactions with agents as opposed to objects. This cluster of social cognitive biases ensures that infants will be provided ample opportunity to notice contingencies between agents' actions and their consequences, and, hence 'fast track' the induction of social norms and the development of agent models necessary for complex social interaction. Just as there is a biological predisposition to acquire language, but which language is acquired depends on the surrounding language community, so too does there seem to be a biological predisposition to acquire social norms (i.e. the rules or conventions that constrain social behavior), but which norms are acquired depends on the surrounding social environment.

It seems necessary to posit a biological component to account for the acquisition of these aspects of social cognition because certain aspects of social cognition seem to depend on having the right neurological substrates. If there is a failure of biological preparedness, e.g. if a neurological impairment produces failure to attend to social stimuli, then impairments in social learning and social reasoning will occur. Turner syndrome is a genetic abnormality in which a female lacks all or part of one X-chromosome. Individuals with only a maternally-inherited X-chromosome show marked social difficulties, particularly on measures of social insight and adeptness (McGuffin & Scourfield, 1997). Autism is a neurodevelopmental syndrome whose most vivid impact at the cognitive level is an impaired ability to reason about social stimuli (e.g. Leslie & Roth, 1993; Baron-Cohen, 1995). The selective impairments in social reasoning seen in Turner syndrome and autism may occur because the neurological substrates necessary for detecting and attending to social stimuli are congenitally absent or fail to develop normally.

That specialized pathways develop as a result of this 'nature-nurture' interaction is perhaps best supported by the selective cognitive impairments reported in syndromes such as prefrontal lobe syndrome. Prefrontal lobe syndrome is a pattern of impaired reasoning performance that results from bilateral damage to the ventromedial prefrontal cortical lobes. In humans, this syndrome is characterized by an impaired capacity to reason effectively about socio/emotional stimuli while leaving other types of intelligent reasoning virtually untouched (Damasio, 1994). Monkeys with bilateral prefrontal ablations (both ventromedial and dorsolateral) show diminished self-grooming and reciprocal grooming behavior, greatly reduced affective interactions with others, diminished facial expressions and vocalizations, and sexual indifference (Damasio, 1994) (pp. 74–75). They can no longer relate properly to others in their troop and others cannot relate to them. Damage to other sections of the cortex – even those resulting in paralysis – do not impair these social skills. The selective impairment of social reasoning that characterizes prefrontal lobe syndrome suggests that neural substrates exist whose primary purpose are the processing and integration of social reasoning functions.

Biological preparedness makes acquisition of reasoning skills and norms specific to the social domain nearly inevitable in normal environments when the neurological substrates are intact. But, although social reasoning and norm acquisition is highly canalized, *which* types of social skills and social norms a normal infant

will acquire depends on the social stimuli to which it is exposed. Rhesus monkeys are notorious for their aggressive natures, while stump-tail monkeys typically are characterized by cohesive group life, high social tolerance, and frequent reconciliation after fights. Co-housing juveniles between the two species, however, produces a dramatic shift in social interaction strategies among the rhesus (de Waal & Johanowicz, 1993.) Those co-housed with stump-tailed macaques adopt many of the cooperative and conciliative behaviors typically seen only in stump-tails. As this example shows, social mammals are biologically predisposed to acquire the social norms that exist within their troops. That is the aspect of their cognition that is canalized. Which norms are acquired, however, depends on the social environment they find themselves in.

Acquisition of social reasoning skills and norms, then, is the result of a complex interaction of learning and innate components. But these innate components are not usefully conceptualized as innate modules, or as innate rules or theories or concepts. They are more usefully conceptualized in terms of biases in learning, especially in categorization and attention, that function to canalize the development of a specialized social reasoning system whose form is relatively invariant, but whose specific content tends to reflect the individual's socializing group. Because these biases are heritable, are relatively decoupled neurologically from other cognitive traits, and lead to highly canalized adaptive abilities, it is plausible to propose that they were selected for.

## 8. Closing comments

By invoking the concepts of biological preparedness and canalization, one can readily explain how a highly plastic developing brain could end up like a Swiss Army knife. Highly specialized functions need not be present at birth. Instead, the majority of comparative, developmental, and neuroscientific evidence weighs in on the side of fast-track learning through biological 'biases' or predispositions that entrain the focus of our attention on the environmental stimuli and contingencies that really mattered to the survival and reproductive success of our ancestors. Our biological predispositions impose the framework that is necessary to learn the things most vital for survival in a complex social environment, while neurological plasticity allows our actual environmental experiences the final say in whether and how those predispositions are expressed.

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