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Assesing some Evolutionary Assumptions in Modular Theories of Mind

Introduction

There are three common ways to investigate human cognition and the neural mechanisms that underlie such cognitive processes: (1) analyzing and comparing conceptual or theoretical approaches in philosophy of mind; (2) studying specific brain damage associated to cognitive impairments in patients, usually by means of a dissociation of functions method¹; (3) using functional neuroimage techniques (PET, fMRI) in order to register selectively activated (localized) brain areas when a cognitive task is performed. Anyhow, at some point these investigation strategies (conceptual analysis, dissociation, or neuroimage) have to confront their assumptions about how cognitive architecture and/or brain are organized, most of the times they entail a modular view.

Modularity of mind is one of the most influential approaches to how human cognitive architecture is assembled. It claims that the architecture of mind is, to some important extent, made up of modules, broadly understood as mechanisms dedicated to specific functions. Despite its conceptual complexity and the lack of consensus in its characterization, modularity matches with many common sense premises about how our mind as well as our brain is organized.

Modular assumptions are crucial in many respects. Modularity, by any name, is crucial in order to disentangle the theoretical and technical impasses faced by many fields in cognitive science when one is trying to understand how the mind and the brain work. Examples abound, just to mention some cases: Patterson & Kay (1982) postulated a reading module; Dehaene & Cohen (1995) claimed we have a brain module for analogue quantity processing, located in the angular gyrus of the parieto-inferior cortex; Parsons & Osherson (2001) reported that deduction activates the inferior frontal and middle temporal lobes of the right hemisphere; they speculated that these regions correspond to a deductive reasoning module.²

¹ Dissociation is a method based in the study of cognitive impairments in patients, those that result either from a neurodevelopmental deficit, or from a brain lesion. Single dissociation occurs when after a damage in the brain region 'x' (let's say 'Rx') the patient S1 has a cognitive deficit in the task 'tA', but not a cognitive deficit in the task 'tB'. A double dissociation occurs when there's another patient S2 with brain damage in 'Ry' associated to a cognitive deficit in the task 'tB', but not a cognitive deficit in the task 'tA'. Double dissociation is very useful in neuropsychology in order to study or discard crossover interactions between cognitive tasks; its main goal is to establish functional independence of cognitive processes. Nonetheless, it has been demonstrated that double dissociation is a tool, not a proof of separated cognitive functions.

² Other authors have allocated the "deduction module" in other brain areas; or have denied the existence of a reasoning module claiming that for human reasoning it is required several different cognitive-processing components, which are dynamically configured depending on the type of task and specific environment with which the subject is faced (Grafman & Goel, 2003, p. 879; Goel, 2007, p. 440). This lack of consensus in the localization of a specific brain functions is usually ascribed to mistakes in the measure techniques, as failures

In short, modularity is important not only because it is an influential theory of mind, but it is also a vivid presupposition in neuropsychology and neuroimaging. It recovers some intuitions according to which mind and/or the brain is separated into components that codify for/or carry out highly specialized and specific functions.

In this work, I will examine some traditional evolutionary arguments aiming to support a modular architecture of mind, some of which are leading not only to sterile (only terminological) research programs, but also to a lack of capacity to advance on how brain and mind work. I will then focus on a different adaptive resource, which is perhaps the most adaptive brain mechanism we inherited: the ability to cope with different types and ever changing environments: brain plasticity. This resource can fuse together anatomical modularity and cognitive modularity.

Where does (conceptual) modularity come from?

Modularity of mind or peripheral modularity started with Jerry Fodor rather as a terminological debate (Fodor 1983 & 2000). It claimed that we only have modules in the periphery of our mind; the rest is a central processor where complex cognitive processes take place. This formulation established that the mind is made of domain specific, encapsulated, autonomous processes, among six other characteristics.³ The Fodorian formulation was criticized by a second version, i. e., the *massive* modularity version (MM). Massive modularity is the thesis according to which human mind is massively structured by modules. MM emphasizes innateness as the basic characteristic of modularity. In general, MM aims to merge massive modularity with Darwinism, claiming that our cognitive architecture is an evolutionary adaptation.

In Cosmides & Tooby MM formulation, evolution by natural selection favored the development and fixation of innate cognitive systems or modules, which were selected for processing adaptive information. They claim that:

...selection... is the only known account for the natural occurrence of complexity organized functionally in the inherited design of undomesticated animals (Cosmides & Tooby 1992, p. 53).

In the same vein, Sperber declares he has been convinced by Cosmides & Tooby that:

...we (*they*) know enough about evolution and cognition to come up with well motivated assumptions as when to expect modularity, what properties to expect of modules, and even what modules to expect (Sperber, 1994, p. 42).

in the hemodynamic response in the fMRI; poor experimental design or control; mistaken statistic analysis; etc. It has been noticed that activations from no two studies are identical (Goel, 2007, p. 437 y ss.), i.e., different studies report different brain regions for deductive reasoning.

³Modular cognitive systems, according to Fodor (1983) can have the following characteristics: domain specificity, mandatory operation, limited access to other processes operations, they are fast, they are informationally encapsulated, they produce shallow outputs, they are associated to a neural architecture, they exhibit typical breakout patterns, its ontogeny exhibits a characteristic pattern (they might be innate).

Carruthers (2006, p. 16), another adaptive modular theorist, echoing Dawkins (1986) considers that evolution by natural selection remains the only explanation for the organized functional complexity we have:

Any complex phenotype structure, such as the human eye or the human mind, will require the cooperation of many thousands of genes to build it. And the possibility that all of these thousands of tiny genetic mutations might have occurred all at once by chance, or might have become established in sequence (again by chance), is *unlikely* in the extreme... We can be *confident* that each of the required small changes, initially occurring through chance mutation, conferred at least some minor fitness-benefit on its possessor, sufficient to stabilize it in the population, and thus providing a platform on which the next small change occur... the more complex the organization of the system, the *more implausible* it is that it might have arisen by chance macro-mutation or random genetic walk.” (Carruthers 2006, pp. 16-17).

Although this debate between Fodor’s Modularity and MM can be tagged as terminological or conceptual debate, it can be enriched by empirical evidence.

There are two possibilities to explain cognitive and brain segregation of functions (i.e., modularity): as innately or genetically transmitted; or as a result of qualitative changes shaped by environmental experience.

I will firstly examine some evolutionary-nativist assumptions in favor of a massively modular architecture of mind (the first possibility), and posit, as many others before, that the attempt to trace back an adaptive value for almost every cognitive task is a mistake. This will take us, subsequently, to examine the second possibility.

Genetic Nativism in Massive Modularity and some of its Problems (1)

A reason for adopting evolutionary considerations

No matter if you are a modular theorist or not, a critical question in current cognitive science is to account for the origins and fixation of the specialized cognitive mechanisms we have. Massive Modularity explained the origin of those mechanisms as innate products of natural selection.

These explanatory patterns are taken, among others, from conclusions coming from developmental biology, according to which a modular organization is a prerequisite of evolvability (West-Eberhard 2003). Since any module’s properties are regularly independent of one another, both the modules and the developmental pathways that lead to them can have distinctive effects on the organism overall fitness. By the same token, MM theorists argue that since modules are separately modifiable, natural selection can act on one part without altering all the system. They claim that only a modular organization can enable this to happen, from which it is appropriate to think of the mind as a *biological* system, subject to the same evolvability requirements as any other such system.

The typical critics to Darwinian Suppositions

It is common ground that not every property of the mind is an adaptation, for some might be by-products of those traits that are adaptations. It remains possible that some properties of the mind might be ‘spandrels’ of some other selected-for traits (in the sense of Gould and Lewontin, 1979).

It has also been claimed (Buller, 2008) that to discover why any trait evolved, we need to identify the adaptive functions it served among early humans, for which we have little evidence.⁴

Additionally, it has been noticed (Fodor, 2000) that the important question in order to know if our mind is an adaptation is the degree of genotypic alteration required for evolving from our farthest ancestor to us, or what is similar, the environmental pressures that our ancestors had to overcome in order to develop the characteristics we possess at present. In this respect, it is agreed that we know almost nothing about the cognitive architecture of the ancestral ape. In Fodor’s formulation:

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. And, since nothing at all is known about *how* the architecture of our cognition supervenes on our brains’ structure, it’s entirely possible that quite small neurological reorganizations could have effected wild psychological discontinuities between our minds and the ancestral ape’s...The little we do know is that our brains are very similar to those of apes; but our minds are very different. It’s not hard to see that relatively small alterations to the neurology must have produced very large discontinuities in cognitive capacities in the transition from the ancestral apes to us. If that’s right then there is no reason at all to believe that our cognition was shaped by the gradual action of Darwinian selection on pre-human behavioral phenotypes. In particular, the (presumed) fact that our minds are complex and conducive to fitness is no reason to believe this. (Fodor 2000, p. 88).

In brief, in evolutionary descriptions a linear relation between alteration of some physiological parameter and the alteration of a creature’s fitness is often assumed. However, in a theory of mind it is unknown whether the relations between alterations of brain structures and alterations of cognitive structures are incrementally linear. We do not know enough about the laws according to which cognition supervenes on brain structures, or even about on which brain structures it is that cognition supervenes.⁵ Therefore, it is highly risky to hypothesize adaptationism about cognitive matters.

⁴ A widespread and easy formulation for this issue is in: David J. Buller (2008). The first fallacy he points out is that the analysis of Pleistocene adaptive problems doesn’t yield clues to the mind’s design. He claims that those descriptions are purely speculative as long as we have little evidence of the conditions under which early human evolution occurred. According to him it is necessary in the first place to know our ancestor’s psychological traits.

For a more in depth formulation see his (2005).

⁵ Fodor adds: “Make an ancestral ape’s brain just a little bigger (or denser, or more folded- or, who knows, grayer) and it’s anybody’s guess what happens to the creature’s cognitive-cum-behavioural repertoire. Maybe the ape turns into us... Likewise, nothing we know impugns the possibility that quite small changes in a creature’s cognitive *structure* may produce very large reorganizations of its cognitive capacity. (Fodor 2000, p. 89-90).

Furthermore, we are not justified in putting together a selectional history of a human trait and a theory of mind. We have no solid evidence that they are mutually explanatory. It is reasonable to bear in mind that we cannot validly adopt them as “the” methodological or explanatory principle. Theory of mind is in need of help coming from selectionist considerations, or wherever, as an important reference,⁶ but not as its main dictum. Note that I’m not claiming that evolutionary assumptions are all false or unnecessary, but they are insufficient to understand cognition in its broad sense. It is feasible that some (few) cognitive modules can be explained as a product of adaptation, but is quite unlikely that most of them are explainable by only such resource.

Several critics have been formulated against the *(ab)use* of the notion of adaptation and natural selection in MM formulations, but I won’t examine them here. So far, we have reviewed the first possibility to explain cognitive and brain segregation and/or differentiation of functions (i.e., modularity) as innately or genetically transmitted.

Setting aside the question of to what extent evolution could help us to explain the factual role of our present cognitive processes, and bearing in mind their obvious limitations; some other research perspectives should be considered. Let’s now focus on the second possibility, that our cognitive abilities and segregation of functions are results of qualitative changes shaped by environmental experience.

Environment, another consideration to incorporate (2)

It would be rare to find someone who denies that a large amount of our brain formation systems are genetically transmitted. The fact that such systems provide the basic brain architecture we actually have is not impugned; notwithstanding, it is dubious that we have inherited cognitive mechanisms as ready-made products.

There are some reasons that cast doubt on the cognitive architecture postulated by MM, according to which cognition is massively composed of ready-made cognitive mechanisms that are innate or genetically transmitted. Such reasons come from some questions that have not been properly answered by MM theorist:

- (1) How could our cognitive architecture be massively made up by segregated (innate) mechanisms; and minimally or vacuously made up by plastic mechanisms, i.e., the ones that can cope with an ever-changing environment?
- (2) How a mind massively loaded with innate cognitive processes could be able to face new environmental challenges?
- (3) How cognitive *information*, contingent and recent in evolutionary terms, can be genetically stored and then transmitted from one generation to the next?

⁶ As Sperber (2002) puts it: “...there is nothing obvious about the organization of the mind/brain, so any evidence is welcome as it is the case of evolutionary perspective. This perspective also allows us a wider and comprehensive account for modularity”.

Taken together these questions give us enough reasons to explore a way out beyond the massively modular explanation for the origin of the cognitive architecture we currently possess. Bear in mind that the key issue here is that at some point in our brain phylogenetic and ontogenetic development, a functional specialization emerges. It is the nature of such a phenomenon what we want to find out.

Modularization processes based on experience can account for independent and functional specialization in some cognitive processes. A very sensible supposition could be the simpler: brain's developmental blueprint is genetically transmitted, brain plasticity⁷ (the ability of our brain to be adapted to our particular environment) is also inherited, but cognitive architecture is shaped by the subject's environment.

Brain specialization can be seen as an adaptive response to a particular environment. It can also be hypothesized that plasticity was the ability selected by evolution as long as it favors adaptation and survival. So, our cognitive architecture might not be an assembly of ready-made cognitive mechanisms, but an assembly of specialized cognitive functions according to a particular environment. I will call this the Environmental Plasticity Modular Hypothesis (or EPMH).

There's plenty of neuroscientific evidence compatible with the EPMH, according to which experience does lead to changes in qualitative and quantitative neuronal organization (Ptito & Desgent, 2006; among many others). There are several authors who have argued for the environmental modularization of cognitive functions. Polk & Hamilton (2006) have studied functional specialization and localization of cognitive functions in reading, writing and arithmetic. Similarly, Ptito & Desgent (2006); Röder (2006); have studied brain's responses to peculiar environmental inputs in cases of abnormal sensory deprivation or lesions.

Genetic Nativism or Brain's Plasticity? Which can explain cognitive specialization better?

In order to account for the cognitive functions and brain specialization we currently have, i. e., segregation of functions or modularity, we can either: 1) take into consideration nativist assumptions, as MM does, or; 2) try to explain segregation of functions as a result of brain's adaptation to the environmental challenges, as in the EPMH. I will defend this last option.

If we are truly believers in selectionism and MM, we should focus on the characteristics of the brain that are inheritable, such as the cortical organization in humans and other mammals. A part of the cortical organization that is genetically mediated and constrained by evolution is the *visual cortex*: It is thought to be one of the most ancient senses in evolutionary terms, since we share it with other mammals; its topographic distribution is almost invariant in mammals; their developmental patterns seem to follow an intrinsic

⁷ Plasticity can be minimally defined as an adaptive phenomenon where structural changes in the brain occur due to the environmental pressures faced by the subject; despite the fact that brain plasticity decreases with age, human cortex is always trying to cope with the environmental circumstances.

program, such as the development of the ocular dominance; we have two eyes arranged in a similar distribution; visual system development seems to be independent of any instruction or learning; there's evidence according to which some minimal enough-for-survival activity shapes the initial configuration of the visual cortex, probably based on some genetic transformations of the most basic function; there is also evidence of different specific molecules to the left eye and to the right eye brain regions, each of which have a role in the initial structure of the primary visual cortex. All those facts seem to reflect the genetic codification of the visual system.

Despite the fact that visual system is genetically and deeply rooted, it is also highly plastic at birth, during the critical periods of development when visual cortex is settled. There's evidence indicating that without visual experience to stimulate or alter the synaptic connections, gene expression is not possible, and visual cells will not develop normally.

Visual system in the human cortex is one of the most studied senses. Several thinkers have studied how brain development depends on the interaction between the basic components of the nervous system (nature) and the stimulating environment (nurture). There's plenty of evidence of visual system compatible with the environmental plasticity modular hypothesis (or EPMH). Studies on plasticity in the visual system in animal and human models show how the visual system's interaction with the environment produces not only permanent and extensive modifications of cortical organization, but also changes in the (cognitive) function. The visual system is also an excellent case of study for our purposes as long as the sight is critical for many brain processes and for complex cognitive processes.

There are plenty of results showing how the brain reacts in conditions of sensory deprivation, abnormal gene expressions, or injuries. In such conditions, cortical reorganization occurs; which means that morphological changes in the visual cortex are driven by cognitive processing demands.

There are studies of rats raised in enriched housing environments reporting 25% more dendritic space for synapses (Johansson & Belichenko, 2002). Similarly, there have been reported changes in the primary somatosensory cortex, specifically a sharper topographic organization, in rats raised in enriched environments (Coq & Xerri, 1998).

On the contrary, results from poor visual environment experiments are often quoted. One of them is the report of kittens reared with exposure to one single orientation stripes where, as a consequence, kittens visual cells developed a preference for the experienced stimulus, i. e., one orientation stripes, being almost unable to recognize stripes running in the opposite orientation (Blakemore & Cooper, 1971). There are also old experiments of complete visual deprivation. Wiesel & Hubel (1965) reported a comparison of effects in the cortex after unilateral and bilateral induced blindness. They produced blindness during the critical periods of development in kittens, suturing the lid of one eye. They found that the deprived eye had a reduction in the number of cells, as well as a smaller ocular dominance column in the primary visual cortex; whereas the untreated eye showed anatomical dominance in the visual cortex. Similarly, recent studies, by Berardi, Pizzorusso, Ratto & Maffei (2003), have showed some important changes that occur if an eye is deprived during the critical

periods of development, such as: an irreversible reduction in visual acuity, an atypical pattern of distribution of the cortical neurons, and ocular dominance of the untreated eye.

These experiments suggest that in subjects raised in a poor environment, visual connections in the cortex do not consolidate normally and visual acuity does not develop properly; hence, a poor or a rich environment produces anatomical changes in the sensory cortex.

I have mentioned the case of the visual system, one of the most genetically driven (more modularized), in order to illustrate how environmental modifications in the form of specific inputs do modify cortical structures, i.e., how specific environmental inputs lead to a rearrangement of the brain architecture. The rearranged brain (sensory) structures process cognitive functions in order to adapt to its environmental processing demands as best as it can. If this environmental modification occurs within one of the most genetically driven or modularized of our senses, what can we expect to happen to the less modularized cognitive process, such as those domains that are recent developments on an evolutionary time scale?

Conclusion

The visual system is one of the most genetically rooted senses; it is also a prime candidate for anatomical and cognitive modularity. There are reasons to believe that some minimal enough-for-survival activity shapes the initial configuration of the visual cortex, with undeniable genetic bases, as most evolutionary believers (including MM theorists) claim. Thus, visual system must be one of the best cases of study for MM thinkers, who contend that (most) modular systems have genetic basis. Notwithstanding, there is evidence according to which we didn't inherited ready-made brain mechanisms, as the visual system empirical evidence can show us. We can expect this last to be true for cognitive systems too, once that senses as the visual system are the basic components of many cognitive processes. As a result, we need another resource to explain segregation of brain and cognitive functions, functional specialization (modularity). Such a useful explanatory resource might be emphasizing brain plasticity as one of the most efficient adaptive mechanisms.

We can thus opt for the EPMH explanation resource to account for segregation of functions, and claim, contrary to massive modularity thesis, that we didn't inherited ready-made cognitive mechanisms; instead, it is feasible to assume that, through the senses adaptation to the environment throughout life, cognition relies more on its experience than in its genetic basis.

Segregation of functions (modularity) based minimally on genetic basis and mainly in functional specialization dependent of the subject's environment, is also consistent with evidence from acquired cognitive functions and neural specialization in reading, writing and other cognitive processes, as Polk & Hamilton (2006) described. They focus on experience-dependent neural modularity in reading, writing and arithmetic, and they provide support for the claim that experience can lead to the creation of new functional and anatomic modules in the human brain.

Similarly, Petersson & Reis (2006) provide cognitive, neuroanatomic, and functional neuroimaging results indicating that formal education modularizes the brain, in such a way that adults with formal education develop neural modules (forced by explicit and systematic instruction in the form of specific environmental task demands) dedicated to specific cognitive functions, such as reading and writing. Notoriously, the neuroimages they provided show that illiterate subjects process reading and writing skills with different brain areas than literate subjects. PET study indicated that in the literate group there was a more prominent left-sided inferior parietal activation (Brodmann's area 40 <BA 40>), in the anterior insular cortex (BA 14/15) bilaterally and in the right inferior frontal opercular cortices (BA 24/32), left basal ganglia, midline anterior thalamus/hypothalamus, and midline cerebellum; whereas in the illiterate group, a significant activation was only observed in the right middle frontal/frontopolar region (BA 10).⁸

Taking into consideration this kind of evidence of functional specialization and environmental influence, could be of much help to explain the cognitive abilities or modules in which massive modularity theorists are interested in, as the ones listed above.

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⁸ The study compared with PET immediate verbal repetition of words and pseudo words in two groups that did not correlate with the pattern of brain activations in either group (Petersson & Reis, 2006, pp. 295-296).

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