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Cognitive Modularity, Biological Modularity, and Evolvability

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There is an argument that has recently been deployed in favor of thinking that the mind is mostly (or even exclusively) composed of cognitive *modules*; an argument that makes use of some ideas and concepts of evolutionary and of developmental biology. In a nutshell, the argument concludes that a mind that is massively composed of cognitive mechanisms that are cognitively modular (henceforth, c-modular) is more evolvable than a mind that is not c-modular (or that is scarcely c-modular), since a cognitive mechanism that is c-modular is likely to be biologically modular (henceforth, b-modular), and b-modular characters are more evolvable (e.g., Sperber 2002; Carruthers 2005). In evolutionary biology, the evolvability of a character in an organism is understood as the "organism's capacity to facilitate the generation of non-lethal selectable phenotypic variation from random mutation" with respect to that character (Gerhart and Kirschner 2003).

Here I will argue that the notion of cognitive modularity

needed to make this argument plausible will have to be understood in terms of the biological notion of *variational independence*; that is, it will have to be understood in such a way that a cognitive feature is c-modular only if few or no other morphological changes (cognitive and not) are significantly correlated with variations of that feature arising in members of the relevant population as a result of ontogeny.[1] I will also argue that all–except for (possibly) one—of the connotations contained in a cluster of notions of cognitive modularity widely accepted in some of the mainstream currents of thought in classical cognitive science, are simply *irrelevant* to the argument. In order to argue for this, I will have to examine the question as to whether there are any strong theoretical connections between (1) those connotations and (2) notions of modularity accepted in biology, specially in evolutionary and in developmental biology, that are thought to be most relevant to arguments to the effect that biological modularity enhances evolvability.

1. Cognitive Modularity <A>

In the contemporary literature in evolutionary psychology, cognitive ethology, developmental psychology, cognitive neuropsychology, and other cognitive disciplines, one can often find a discussion as to whether—and how many, and in what sense—the cognitive mechanisms that constitute the minds of animals and of humans are c-*modular.* It is virtually true, however, that no

two authors in these cognitive disciplines handle the same notion of cognitive modularity.

The most influential notion of c-modularity was initially introduced by Fodor (1983) who proposed that a classical computational mechanism is c-modular when (and to the extent that) it has one or more of the following characteristics[2]:

(a) it is domain specific; i.e., it admits inputs only from a certain informational domain;

(b) its operation is mandatory since its operation (or its ceasing to operate) does not depend upon the subject deciding or wanting the mechanism to operate (or to cease to operate);

(c) other parts of the mind have limited access to the representations or information that the mechanism is using to compute its output;

(d) the mechanism operates very fast;

(e) it is informationally encapsulated since its has no (or limited) access to most of the information that is accesible to other parts of the mind;

(f) it has “shallow” outputs that are informationally poor;

(g) it is associated to a fixed neural architecture;

(h) it exhibits specific breakdown patterns; and

(i) it is innate.

Fodor thought that, in some ways, he was following an older school of thought in psychology, which fell into disrepute for a long time and which he dubbed “vertical faculty psychology,” initiated

by Franz Joseph Gall (1758-1828), and which, according to Fodor, defended that there is no such a thing as a general faculty of understanding; rather, “the intellectual aptitudes … are distinguished by their subject matter” (Fodor 1983: 15)–or, to put it in Fodor’s terminology, each aptitude is subserved by a distinct, domain specific faculty. Fodor thought that his proposal that our sensory capacities and at least some aspects of the linguistic capacity are modular, is partially within the spirit of Gall’s views.

But other authors after Fodor have emphasized, rejected or added other characteristics as interestingly associated to c-modularity; they also have differed from him in the extent to which they think that the mind is modular. Those authors who think that the mind is mostly (or even exclusively) c-modular are called “massive modularists.” For example, Cosmides and Tooby (1997: 80, 92, 93) are massive modularists who emphasize domain specificity, and add functional specialization and genetic specification as necessary conditions for c-modularity, while they tend reject or ignore the rest. Annette Karmiloff-Smith is probably not inclined to be a massive modularist, and thinks that domain specificity, mandatoriness, encapsulation and speed are necessary conditions of c-modularity (Karmiloff-Smith 1992: 4-6), while rejecting that innateness is a necessary condition because she thinks that certain abilities can become modularized as a result of learning. Additionally, although Sperber (1994: 48), a massive modularist, used to think that domain specificity, encapsulation, and genetic specification were necessary conditions of c-modularity, recently

he seems more inclined to reject domain specificity and to think that encapsulation and functional specificity (or specialization) are the only key elements required to talk about c-modularity in the cognitive sciences (Sperber 2002). Carruthers (2005), another massive modularist, agrees that domain specificity is not a necessary condition of c-modularity, while claiming that encapsulation, independent operation, and functional specialization are some of the necessary conditions.

Here I will not examine the debates concerning which characteristics one should include in a theoretically interesting and useful notion of c-modularity.[3] Instead, I will examine the question as to what notion of c-modularity is needed to make plausible the aforementioned argument for the view that the mind is mostly c-modular.

Now, as we have seen so far, the consensus among massive modularists tends to be that one should understand c-modularity in terms of one or more of the following characteristics: *encapsulation, domain specificity, functional specialization, innateness, and the presence of specific patterns of cognitive breakdown*.[4] Thus one should ask the question as to whether massive modularists are understanding c-modularity in a way that is conducive to making plausible the argument for the evolvability of c-modules –an argument that, according to at least some of them, is key to upholding the massive modularity hypothesis (Sperber 2002; Carruthers 2005).

Let us now examine the notions of modularity that have appeared in different fields of biology and the related thesis to

the effect that biologically modular characters are more evolvable.

2. Modularity in Biology <A>

Although notions of modularity as such are relatively recent in biology, some conceptual antecedents to these notions exist in a number of authors since the 1930s. One of these conceptual antecedents can be found in the concept of dissociability as used by developmental biologists such as Needham (1933) who noted that certain developmental processes can occur relatively independently from others. Later on, Berg (1959: 171) proposed to interpret the presence of what Terentjev called "correlation pleiades" as an indication of "the increasing independence of certain developmental processes with respect to environmental factors, *including the influences exerted by the other parts of the same organism.*" Correlation pleiades are correlations that exist between some quantitative characteristics—e.g., between dimensions of certain parts of an organism—and, at the same time, the absence of correlations between these and other parts. The idea of developmental independence which Berg mentioned, is one of the central intuitive notions underlying many of the concepts of modularity in developmental biology. In the 1970s, Rupert Riedl and S. J. Gould both pointed to similar ideas in connection with developmental and evolutionary phenomena. Riedl suggested that the "adaptability of functionally independent characters–adaptive

freedom, so to speak—will require independently changeable genetic information" (Riedl 1977: 360). He proposed a theory of "genome systemization," which "demands feedback loops of cause and effect both from the genome to the phenome and in the *reverse* direction. Such feedbacks can accelerate adaptation only in the direction in which gene interactions have imitated the patters of functional interactions in the phenome" (Riedl 1977: 361).

On the other hand, Gould (1977) suggested that dissociable developmental processes are necessary in order for heterochronic change to take place; that is, change in the relative rates or timing of development of different cell lines. Later, Bonner (1988: 174) coined the concept of *gene net*, which stands for "a grouping of a network of gene actions and their products into descrete units during the course of development." He thought that the existence of these nets during ontogeny–especially the ontogeny of complex organisms—is what makes possible both the success of the process of development as well as constituting an explanation as to how "complex developing organisms can change in evolution" (175). As we shall see, Wagner and others have in some form or another taken these ideas—but especially the idea of gene nets—in order to formulate notions of b-modularity that can be relevant to the evolvability of certain biological traits.

Different versions of the view that b-modularity enhances evolvability have been recently discussed in some evolutionary biological circles (to mention just a few: Wagner 1995; Wagner and Altenberg 1996; Raff and Raff 2000; Hansen 2003; Welch and Waxman 2004; Altenberg 2005; Eble 2005). The intuitive idea behind many

of the notions of modularity in biology is that a biological system that is built out of a number of modular systems each of which, *qua* modular, enjoys a certain autonomy from the rest, is more evolvable since evolution can work on each of its autonomous and simpler parts one at a time, without the changes on each part having to affect much the other parts of the system in complex and unmanageable ways. Indeed, this idea seems pre-theoretically very plausible.

There are, however, various recent theoretical proposals to characterize different notions of b-modularity that are attempts to capture the diverse intuitions which underlie that seemingly simple idea in such a way that the thesis that b-modularity somehow enhances evolvability is validated. Nonetheless, we can say that one of the most influential notions of b-modularity to be widely discussed in evolutionary biological circles is the one proposed by Wagner and Altenberg (1996). This is a notion that applies to the genotype-phenotype map–i.e., the map that depicts the manner in which a set of genes map onto a phenotypic character (or complex of characters). Thus, according to Wagner and Altenberg (1996: 971):

> Independent genetic representation of functionally distinct character complexes can be described as modularity of the genotype-phenotype mapping functions. A modular representation of two character complexes C1 and C2 is given if pleiotropic effects of the genes fall mainly among members of the same character complex, and are less frequent between members of different complexes.[5]

The central idea of this proposal is to think of these modules as "clustered pleiotropic mappings … that 'align' genotypic and phenotypic space" (Eble 2005: #). Roughly speaking, when the pleiotropic effects (i.e., effects on more than one character or character complex) of the group of genes that influence a character complex tend to cluster around that complex and not around other character complexes, then it will be said that the genotype-phenotype map for that complex is modular. I shall call this "*pleiotropic* modularity" (p-modularity).[6] Thus, if a complex of characters of an organism O is p-modular (in this sense), then it is both (so to speak) genetically integrated and genetically independent, both to a certain extent. As such, p-modularity presumably enhances evolvability since any change in a complex of characters C that is p-modular would tend to be independent from changes in other characters (or character complexes) in such a way that a change in C would not be correlated with many (or any) changes in other characters (or character complexes) and, other things being equal, the probability that these isolated changes in C are selectable is higher than the probability that more systemic or holistic changes are selectable.

Yet other ways to characterize b-modularity have been proposed. Consider the characterization of modularity discussed by Thomas Hansen (2003). This form of modularity is understood in terms of variational independence. A character (or a functionally distinct complex of characters) is said to be highly variationally independent in a population when variations of it arise in that population without at the same time appearing (many) changes in

other characters (or character complexes). For example, the lens of an eye (of members of a population) would be variationally independent of other characters if variations in the shape, structure or position of the lens occur in that population without any other character showing any change as a result.[7] I shall call this "*variational* modularity."[8] The idea, then, is that a high degree of variational modularity in a character (or character complex) enhances the *evolvability* of that character (or complex of characters): if changes in a character (or complex of characters) C in a population occur with no or only a few changes in other characters (or character complexes) occurring as a result, then this means that the changes in C in this population tend to be less systemic or holistic, and more local.

Notice that some biologists are inclined to think that notions of modularity in biology must include suitable notions of both independence as well as integration (Winther 2001). In other words, b-modularity must somehow insure both tight "internal" integration and loose "external" dependence. If so, then it may be necessary to add to Hansen's notion of variational modularity (as independence) some notion of variational *integration or unity.* Such a notion, as applied to complexes of characters, would roughly go as follows:

> A functionally distinct complex C of characters, C1, …, Cm, is *variationally integrated* to degree *n* in population P if and only if variations of C in P are strongly correlated with variations of at least *n* of the Ci (i = 1, …, m and n≤ m) in P.

We could then say that a character or complex of characters is variationally modular if and only if it is variationally independent and (when relevant) variationally integrated. However, at present there does not appear to be a good rationale for the idea that variational integration is likely to enhance evolvability–while, *prima facie*, such a rationale exists for variational independence. If so, there appears to be no reason to include variational integration in the characterization of a notion of cognitive modularity that could make plausible the argument for the evolvability of cognitive modules that we are considering in the present article.

Furthermore, we must note that it is very likely that Wagner's notion of p-modularity and the idea of variational modularity discussed by Hansen are *strongly connected although not identical.* For one, if a complex of characters C in an organism of a population is p-modular, then C has the *disposition* to exhibit in that population (and in certain conditions) what Hansen calls "variational modularity"–but this is not a foregone conclusion. Other factors may intervene to prevent this outcome. What p-modularity ensures is only that C in P has the *disposition* to be variationally modular—a disposition that may or may not be actualized. What we can say, though, is that p-modularity is likely to be the underlying genotype-phenotype pattern typically associated with the occurrence of variational modularity in a population.

But there are other biological entities and processes that have been and are considered as modular by a variety of biologists

(Raff and Raff 2000)—entities and processes that do not neatly fall under the characterizations listed above. For example, apart from the kinds of modules already mentioned, Eble talks about three other kinds of b-modules:

(a) *structural* modules, which are basically modular by virtue of their spatial geometrical properties, e.g., having discrete boundaries, or different shapes;

(b) *ontogenetic* modules, such as morphogenetic fields, i.e., distinct regions of the body in which the cells have sufficient information to form a specific structure –regions which thus appear to have a certain degree of developmental autonomy in relation to a certain feature, e.g., a limb (Carlson 2003);[9] and

(c) *functional* modules, or functional units.

Concerning functional modularity, we must note that, in biology, there are at least two distinct notions that are commonly associated to this idea:

(a) *Functional integration*, which refers to the functional unity of the parts constituting an organ or a mechanism (as the case may be) in the undertaking of a certain function that the organ or mechanism in question as a whole undertakes (Eble 2005: #-#); thus, an organ or mechanism M of an organism O is functionally integrated with respect to a function F of M *to degree n* when there are *n* functional proper parts (or subsystems) of M each of which is such that for M to undertake F, the proper part in question has to undertake at least one of the functions (distinct from F) it can undertake—in other words, the more the parts of M must contribute functionally to the undertaking of function F of M, the more

functionally integrated M will be with respect to its function F; and

(b) *Functional independence*: roughly, an organ or mechanism of an organism is functionally independent (with respect to one of its functions) when it can undertake that function without any other organ or mechanism of the organism undertaking any of its functions. More precisely, a certain organ or mechanism M1 of O is functionally independent from a distinct organ or mechanism M2 of O[10] (with respect to a certain function F that M1 undertakes) when the undertaking of F by M1 does not require the undertaking by M2 of any of the functions, G1, …, Gn, that M2 can undertake. We can then characterize the following notion: a certain organ or mechanism M is functionally independent *to degree n* (with respect to a certain function F that M undertakes) when there are *n* other distinct organs or mechanisms of O with respect to which M is functionally independent (in relation to F of M).

Note that these are two different notions: an organ O may be functionally very integrated (with respect to function F) in such a way that, say, all of its parts functionally contribute to the F-functioning of O, but O itself may not be very functionally independent (with respect to F) from most other organs of the organism because their performing some of their functions may actually be required for O to perfom F. This is possible because functional integration refers to the functional relationships that the parts constitutive of an organ (or mechanism) have to *have* among them, while functional independence points to the functional relationships that that organ must *lack* with other organs of the

individual. Thus, and for similar reasons, an organ may be functionally very independent from most other organs of an organism but not be very functionally integrated.

Returning to our question concerning the existence of connections between notions of b-modularity and evolvability: if functional modularity is understood as integration, then there appears to be no reason to think that functional modularity enhances evolvability; i.e., we have no reason to think that an organ's being functionally integrated has anything to do with improving the organism's ability to produce more heritable selectable variations with respect to that organ –indeed, in connection with functionally integrated units, Schwenk (2001: 176) rightly notes that

> in the vast majority of cases we have no knowledge of the genetic architecture underlying the functional unit, nor of its heritability; nor do we know how the genetic variance-covariance structure of the characters might change in different environments.

Furthermore, Wagner and Schwenk (2000) identify a very important type of functional unity, which they call "evolutionarily stable configuration (ESC)," and which is characterized as a set of characters which are functionally very integrated (with respect to what Millikan [1984] called a "proper function"). Schwenk and Wagner argue that the functional integration of an ESC enhances its evolutionary *stability.* But if they are right, then functional integration will likely work in the direction *opposite* of evolvability, that is, in the direction opposite of an enhanced ability to produce variations of certain sorts.

On the other hand, functional independence comes in two varieties: it can arise as a result of ontogeny or not. For example, some of the factors that contribute to the functional independence of an organ may not be purely ontogenetic. In cases like this, it cannot be plausibly thought that functional independence enhances evolvability. But what about the functional independence that arises purely as a result of ontogeny? If functional modularity is understood in terms of this sort of functional independence, then here we may have a slightly improved case for saying that functional modularity enhances evolvability. At present, however, there is no evidence to think that this type of functional independence is strongly correlated with *variational* independence –which is, after all, the property that is most straightforwardly related in a clear manner to evolvability. Indeed, it still remains to be seen whether, say, an organ's being functionally independent with respect to other organs in the individual organisms belonging to a certain population, is strongly correlated with there being certain patterns of variation with respect to that organ in the population to which those individuals belong –patterns that are indicative of variational independence.

Additionally, as Eble rightly notes, although being what I call a "pleiotropic module" or being a variational module is not necessarily incompatible with being a functional module or a structural module, nonetheless their relationship "may not be straightforward" (Eble 2005: #). One striking example of this occurs in a case mentioned by Raff and Raff (2000: 236): the tail of a mouse is a structure that has a topologically coherent structure

(i.e., it is a *structural module*) that is made up of *different variational modules*; thus when directional selection is made for greater tail length, one may get either vertebra size increase or else increase in the number of vertebrae developed. Furthermore, the tail is a functionally integrated unit and is thus, in this sense, a *functional module*. Here then we have a character that is structurally and functionally modular but not variationally modular and probably not pleiotropically modular either.

The mouse mandible is another example of the way in which the same feature can be b-modular in some senses but not in others. This mandible is a single structural unit that is functionally integrated, arising in ontogeny through several morphogenetic fields, but which consists of two pleiotropic modules (Schwenk 2001). Here then we have a case of a structural and functional module (in the sense of integration) consisting of several ontogenetic modules and only two pleiotropic modules.

Thus we can now appreciate that there is an ample variety of notions of b-modularity that are used, characterized, and discussed in various fields of biology. Most of these discussions are linked to the ongoing controversy in evolutionary biology concerning the evolvability of a character or complex of characters. And not only is it true that there is no single biological notion of modularity but an exhuberant and increasing variety of such notions; it is also true that there is an ongoing discussion—*that is by no means settled among biologists of any sort*—concerning whether and which notion or notions of biological modularity are involved in an empirically testable and acceptable

assertion to the effect that biological modularity enhances evolvability, indeed, whether any such assertion can be made. For example, Wagner's hypothesis to the effect that genotype-phenotype maps that are very modular (in his sense) are probably very evolvable, is still considered as tentative—most of its empirical backing is yet to come (see, e.g., the discussions by Hansen 2003 and Altenberg 2005; cf. Wagner and Wagner 2003: 30).

However, even if it were generally accepted that a correlation between b-modularity and evolvability existed, the relevance of this result to the discussion in cognitive science would still be unclear. Yet, some cognitive scientists write as though the existence of a connection between c-modularity and b-modularity was obvious. Sperber (2002: 3), for example, simply mentions Wagner's work on pleiotropic modularity and its presumed connection to evolvability, and adds: "In psychology this suggests that the two notions of a mental module and of a psychological adaptation (in the biological sense), thought definitely not synonymous or coextensive, are nevertheless likely to be closely related." What Sperber suggests is that we will likely find an interesting correlation between something's being a cognitive module and its being a biological adaptation (i.e., the result of evolution by natural selection), through the connection between cognitive modularity, biological modularity and evolvability.

In addition, Carruthers (2005: 12) hints at this claim when, as part of an argument in favor of massive (cognitive) modularity, he says: "Evolution needs to be able to tinker with one function in response to selection pressures without necessarily impacting

any of the others." Now, as we shall briefly see next (and as we can already begin to appreciate), Carruthers is wrong in thinking that the notion of cognitive modularity is sufficient to ensure that evolution favored modular cognitive mechanisms because they were more independently "tinkerable" by it, is a notion that includes only functional specialization (which, as we shall see, is neither independence nor integration) and informational frugality (or what he called "wide scope encapsulation)."[11] At present there is no reason to think that either of these characteristics is in any way correlated to the independent sort of "tinkerability" that is relevant to enhanced evolvability.

Indeed, we will show that, at present, there is reason to think that there is a connection *only* between two notions of b-modularity (i.e., variational and pleiotropic modularity) and one of the characteristics normally associated to cognitive modularity, i.e., cognitive dissociability, and only when this one is understood in a specifically biological manner.

3. Biological and Cognitive Modularity <A>

We have seen so far that there are multiple (not necessarily related) notions of biological modularity; that, furthermore, the idea that there is a correlation between any of these notions of b-modularity and a theses about the evolvability of b-modular characters, is still considered somewhat controversial. The question now is whether, even granting the empirical plausibility of such

assertions linking b-modularity to evolvability, something follows concerning the evolvability of c-modular cognitive characters. That is, the question must be answered as to whether it is likely that if a cognitive mechanism is cognitively modular (i.e., either domain specific and/or encapsulated and/or functionally specialized, etc.), then it is b-modular in some sense of b-modularity which allows at least a *prima facie* plausible link to evolvability. However, here I will argue that there seem to be no reasons at present to suspect the existence a link between c-modularity and b-modularity, except for one limited case. For obvious limitations of space, here I will consider only three notions of b-modularity, to wit, Wagner and Altenberg's, Hansen's, and functional modularity—keeping in mind that others have been proposed. However, I think that my discussion concerning these three notions can give the reader an idea as to how the corresponding argument concerning other notions of b-modularity may go.

3.1 Variational Modularity and C-Modularity <B>

As we saw, according to Hansen (2003) the variational modularity of a trait in a population is given by its degree of variational independence; i.e., a character C is variationally modular in members of a population P if, when variations of M arise in some members of P, none (or very few) other morphological changes occur in those members of P. On the other hand, we also saw that cognitive modularity is usually understood in terms one or more of the following notions: domain specificity, encapsulation, adaptive

function distinctness (or functional specialization), psychological function distinctness, or specific patterns of cognitive breakdown.

Let us now turn to the question concerning how likely it is that a c-modular mechanism is also variationally modular. First, neither domain specificity nor encapsulation in a cognitive mechanism make it more likely that the mechanism in question is variationally modular: that a cognitive mechanism M of the members of a population P only admits certain tokens or types of information as inputs or in its database does not appear to make it more likely that the variations V_1, …, V_n of the mechanism that arise in that population are such that the occurrence of a variation V_i ($i = 1$, …, n) in a member of P does not have as a result the occurrence of other morphological changes in that member of P. For similar reasons, I do not think that (psychological and/or adaptive) functional specificity in a mechanism raise the likelihood that the variations of that mechanism arising in a population do not result in additional morphological changes in the relevant organisms.

Does the presence of a specific breakdown pattern make it more likely that the mechanism is variationally modular? Now, one can understand the phrase “specific breakdown pattern” in at least two different ways:

First, one may mean that the breakdown of the cognitive capacity that is subserved by the mechanism in question has some distinctive set of symptoms from which the type of breakdown can be diagnosed, a set of symptoms that is not substantially shared by other cognitive disfunctions. This is not the sense of “specific breakdown pattern” commonly associated to cognitive modularity since

the breakdown of a domain general mechanism can also have its own set of distinctive symptoms.

Second, “specific breakdown pattern” may mean that the breakdown of the cognitive capacity that is subserved by the mechanism in question does not affect (or does not affect greatly) the functioning of other cognitive mechanisms—the mind can be selectively impaired, so to speak.

It is the second sense that is thought to be a strong indicator of cognitive modularity. But can it also be an indicator of variational modularity? Well, it depends upon whether the impairments under study happen as a result of ontogeny or not. If they do, then this is non-conclusive evidence that they are variations of a cognitive mechanism arising in the population, and since their occurrence is not causally correlated with changes in the functioning of other cognitive mechanisms, we can say that this is some form of variational independence—one could call it “cognitive variational independence”—which may indicate at least a degree of variational modularity. Of course, one has to assess the variational independence of a cognitive mechanism not only in relation to other cognitive mechanisms, but also with respect to all other morphological traits of the organism, say, the shape or function of other organs. The more variationally independent M is with respect to more morphological traits, the more variationally modular M will be said to be.

Additionally, there are other types of variations in a cognitive mechanism M –apart form cognitive *impairments*—that are relevant to determining the degree of variational modularity of M in population

P—to wit, *cognitive excellences*; cases where the mechanism works much better than average. Indeed, if a cognitive excellence of a mechanism M in a population P arises as a result of ontogeny, and if this excellence is not significantly correlated with changes in other morphological traits in P, then this also is *prima facie* evidence of a high degree of cognitive variational independence of M in P.

On the other hand, if either the impairments or the excellences in a cognitive mechanism M do not occur as a result of ontogeny—but, e.g., as a result of an accident—then the degree of selectiveness of the impairment (or of the excellence) says nothing about the degree of variational modularity of the mechanism, since in this case the impairments are not likely to be *variations* of the mechanism in a sense that is relevant to the discussion concerning evolvability.

3.2 Pleiotropic Modularity and C-Modularity <B>

Previously we saw that, according to Wagner and Altenberg (1996), the p-modularity of a complex of characters in a population P occurs when the genes that have effects on that complex tend to have pleiotropic effects clustered around the characters belonging to that complex, and have no (or few) pleiotropic effects across other complex of characters of members of P. Let us now turn to the question concerning how likely it is that a cognitively modular mechanism is also p-modular.

First, does domain specificity and/or encapsulation in a cognitive mechanism make it more likely that the mechanism in question is p-modular? The answer appears to be negative. That a

cognitive mechanism M of the members of a population P only admits certain tokens or only certain types of information as inputs or in its database does not appear to make it more likely that the set of genes on which its development depended had pleiotropic effects clustered in the manner described above.

But what about psychological and/or adaptive function distinctness (i.e., functional specialization)? Does the fact that a mechanism has a distinct (either psychological or adaptive) cognitive function make it more likely that the mechanism is p-modular? I do not see why: functional specialization can conceivably happen even in a mechanism that is not p-modular—in a cognitive mechanism which is such that the set of genes on which its development depended did not have pleiotropic effects clustered around that cognitive mechanism and had many pleiotropic effects across other complex of characters (cognitive or not). The example of the mice's tail goes to show that you can have a structure that is functionally specialized –since the tail performs a function that no other organ of the body undertakes—and that is nonetheless pleiotropically unmodular.

Finally, does the presence of a specific breakdown (excellence) pattern in a cognitive mechanism make it more likely that the mechanism is p-modular? Again, as in the previous section, this will depend upon whether the pattern of cognitive dysfunction (or of excellence) arises as a result of ontogeny. If it does, then the presence of this selective pattern of dysfunction (or of excellence) could be indicative of a certain degree of p-modularity with respect to other cognitive traits of members of P.

3.3 Functional modularity and c-modularity <B>

One question that immediately arises in connection with biological notions of functional modularity is whether there are strong conceptual links between such notions and the idea of functional specialization associated to c-modularity. Are they similar notions? Are they interestingly connected? As we saw, there are at least two distinct notions that are commonly associated to an idea of functional modularity in biology:

(a) *Functional integration*: roughly, the more the parts of an organ or mechanism M must contribute functionally to the undertaking of function F of M, the more functionally integrated M will be with respect to its function F; and

(b) *functional independence*: roughly, the more an organ or mechanism M can undertake its function without other organs or mechanisms undertaking any of their functions, the more functionally independent M will be with respect to its function F.

On the other hand, the notion of c-modularity in the cognitive sciences usually incorporates a different functional notion; to wit, that of *functional specialization*, which refers to the uniqueness of the function that the organ or mechanism undertakes in comparison with the functions that can be undertaken by other organs or mechanisms belonging to the same individual. This notion is also characterizable in terms of degrees:

(c) *Functional specialization:* To characterize this notion we first specify that the less functionally non-specialized an

> organ or mechanism of an organism is, the more functionally specialized it is. Secondly, we stipulate that an organ or mechanism M of an organism O is functionally non-specialized *to degree n* with respect to function F that M undertakes as a whole when there are *n* organs or mechanisms of O (distinct from M) each of which has at least one function G that is identical to F of M.

Note that the notions of functional integration and of independence are each distinct from the notion of functional specialization. For example, an organ may be functionally specialized but not functionally integrated; thus, it may undertake a function that no other part of the organism undertakes and at the same time contain parts that contribute nothing to the functioning of the entire organ. Conversely, an organ may be functionally integrated but not very specialized since it may undertake a function that other organs of the body also can undertake. Similarly, an organ O may be functionally specialized but not functionally independent since it may undertake a function F that is distinct from all the functions undertaken by other organs, and yet the functioning of these other organs may be causally required for O to undertake F.

Thus it would seem that functional modularity as it is understood in biology (either as integration or as independence) has not much in common with functional specialization which is the functional notion usually included in the concepts of cognitive modularity used by massive modularists.

Now, we have seen already that, as far as we know, neither functional integration nor functional independence appear to enhance

evolvability. But perhaps functional specialization itself does. I do not think so. The functional specialization of an organ or system of an organism refers to the fact that the organ in question undertakes a function that few or no other organ of that organism can undertake. But this does not appear at all to be correlated with that organ's showing patterns of variation (in the population to which the organism belongs) that are indicative of variational modularity –indeed, it is likely that there are many organisms with at least one organ which is functionally very specialized but whose variations in the relevant population have systematic morphological consequences in those organisms. Furthermore, as far as I know, no developmental or evolutionary biologist defends the idea that functional specialization by itself if likely to be strongly correlated either directly or indirectly with evolvability.[12]

4. Cognitive Modularity, Double Dissociations, and Evolvability <A>

What I have argued so far is that selective impairments and excellences (occurring as a result of ontogeny) ought to be considered as the key characteristics associated to the notion of c-modularity when trying to argue that c-modularity enhances evolvability.

I am not suggesting, however, that the level at which the selectiveness of the impairments/excellences ought to be described has to be neurological or some other level more basic than the cognitive level. In particular, I am not saying that one can consider a certain cognitive mechanism as selectively

impaired/excelled (in the relevant sense) only when one can show that there is a specific part of the brain where the impairments/excellences are “located”. On the contrary, my view is that showing that a certain mechanism M displays a selective pattern of impairments/excellences at the cognitive level in a certain population—without there being specific brain locations associated with those patterns—is strong evidence in itself to postulate a degree of cognitive variational independence.

I have also said that the study of patterns of cognitive impairments/excellences displayed by a cognitive mechanism in a population P is relevant to attempts to determine the degree of variational independence/dependence of that mechanism in P only when the impairments/excellences in question arise in the members of P *as a result of ontogeny*. But it must be added that I am talking about the study of the presence or absence of those selective patterns of cognitive variation in the *adult* (part of) population P. Indeed, the patterns of variation of a cognitive mechanism in a population should be measured only in those individuals in which ontogenetic development with respect to all cognitive capacities is basically through. To see why, let us first look at the following results obtained from studies in people with Williams Syndrome (WS) and with Down Syndrome (DS). Toddlers with WS and with DS “both show equal language delay, despite the WS adult language being significantly better than the DS adults’” (Karmiloff-Smith et al. 2003: 162). Furthermore, toddlers with WS appear to develop normally up to a point with respect to sensitivity to changes in number, whereas toddlers with DS perform worse at this stage; yet by adulthood DS

people perform much better than people with WS on number relevant tasks (ibid. and Oliver et al. 2000). What these results indicate is that a measurement of variation in members of a population with respect to a certain cognitive mechanism M—a measurement that includes those members still undergoing cognitive development—will probably give a misleading picture of the actual pattern of variation of M in that population.

This is not simply a marginal comment: both cognitive psychologists and neuropsychologists have long used a method of research known as the "Double Dissociation Method" which comprises an experimental design and a statistical tool that is used to infer the existence of selective cognitive impairments in certain developmental disorders, such as WS, DS, Specific Language Impairment (SLI), autism, and others. From what we have seen in the previous sections, it should be clear by now that Double Dissociation studies in certain kinds of developmental disorders are *potentially relevant* to the study of patterns of cognitive variation in a population, and thus to the notion of cognitive modularity relevant to evolvability. However, Double Dissociation studies often arrive at conclusions concerning selective impairments drawn from comparisons of groups that show an atypical cognitive profile in adults and a typical cognitive profile in children; comparisons, for example, between a group of adults with WS and a control group of typically developing children that match the WS group in what is known as "mental age (MA)". Clearly, if what we have said so far is correct, conclusions drawn from comparisons between cognitive profiles in adults and in children are not relevant to determining

the cognitive variational patterns found in the relevant population—in determining whether these patterns are variationally independent or not.

In fact, many cognitive psychologists and neuropsychologists acknowledge that conclusions concerning double dissociations drawn from comparisons of groups of organisms at different stages of their ontogenetic development are at best misleading and at worst unwarranted (Karmiloff-Smith 1998; Karmiloff-Smith et al. 2003b; Oliver et al. 2000; Vicari et al. 2005). Additionally, there is a lively ongoing discussion in these fields concerning what the Double Dissociation Method is, what it does and does not show, whether and how it should be modified, and whether it can by itself show the existence of at least two underlying symbol manipulating cognitive systems that process only specific types of information (i.e., that are domain specific).[13] In fact, the consensus among cognitive psychologists and neuropsychologists of any persuasion is that the method in question does not by itself warrant an inference to the existence of two such domain specific classical systems.

The reason for this consensus lies in the fact that some of those who embrace connectionist models of the mind have obtained certain results that suggest a different possible explanation of the data obtained by the application of the Double Dissociation Method: they trained a single network with two types of connections, A and B, to perform two distinct tasks, T1 and T2, one of which, T1, tended to depend more on connections of type A in the network, while T2 depended more on connections of type B. Furthermore, they showed that impairments to connections of type A would produce an impaired

performance on task T1 but not a significant impairment on task T2, while impairments to connections of type B resulted in impaired performance of the net on task T2 but not on T1 (Plaut 1995). What this shows is that all of the experimental data obtained by a careful application of the Double Dissociation Method in a particular case is compatible with either of the following two distinct interpretations:

(a) there are *two* distinct specialized symbol manipulating classical systems each of which processes information from a distinct domain; or

(b) roughly, there is a *unique* connectionist system which processes two distinct informational tasks, and where different parts of this system differentially devoted to each one of these tasks.

Which of these interpretations is chosen (if any) will depend upon ones's own previous commitments to either the classical or the connectionist approach to cognitive science (Dunn and Kirsner 2003; Chater 2003; Juola and Plunkett 2000).

However, for our present purposes, this is of no consequence. The kind of independence that interests us—i.e., the kind that goes to show that, as Carruthers puts it, evolution can tinker with certain systems one at a time—is variational independence; and it may well turn out to be the case that, although the mind is composed of some connectionist networks, nonetheless, evolutionarily speaking, it is composed of more variational modules—just like the mouse mandible is one functional module but at least two variational modules. Alternatively, it may also be the case that the massive

modularist is right in saying that the mind is composed of a great number of classical cognitive systems each of which processes information from a distinct and relatively small informational domain, but this does not mean that it is necessary *or even likely* that each of these systems will be a distinct variational module in its own right.

One more point: all I have said so far also suggests that something like the Double Dissociation Method (properly understood and applied) should be used to study the cognitive profiles of gifted people—i.e., of people who markedly excel at one or more cognitive tasks. Do people who are extraordinary at, say, numbers, also highly excel at other cognitive tasks? Studies such as these are potentially relevant to the determination of the patterns of variational independence that interest us here. Furthermore, here is where we can locate the relevance of studying those rare cases of autistic people with isolated yet extraordinary cognitive gifts.

5. Conclusions <A>

I have argued that the notion of cognitive modularity required to make plausible the argument to the effect that c-modular cognitive mechanisms are more *evolvable* than mechanisms which are not c-modular (because the former are b-modular) will have to be understood (roughly) as follows:

> A cognitive mechanism M of members of a population P is very *c-modular* only if few or no other morphological changes are

> significantly correlated with variations of M arising in members of P as a result of ontogeny.[14]

In other words, c-modularity will have to be understood, at least partially, in terms of variational independence. If so, then cognitive scientists trying to determine whether a cognitive mechanism M is modular and to what degree *–with an eye to determining the evolvability of* M—will have to study not only the developmental cognitive impairments of M in the adults of the relevant population and the degree to which they are variationally independent (in a cognitive *and non-cognitive sense*), but also the degree of variational independence of the cognitive *excellences* with respect to M that appear in the adults of the corresponding population as a result of ontogeny. Both types of research appear to be relevant in a study of the modularity of a cognitive mechanism in so far as such a study can make any legitimate claims towards determining the evolvability of that mechanism.

Finally, much more can be said with respect to the experimental and statistical tools which will have to be brought into the study of patterns of cognitive variation in animal and human populations. Indeed, much more will have to be said concerning the application of ideas and concepts borrowed from evolutionary and developmental biology—e.g., the concepts of character, variation, ontogeny, function, etc.—into the experimental design and understanding of empirical data obtained in fields such as: cognitive ethology, cognitive psychology, developmental psychology, neuropsychology, and others.

Notes

1. The notion of variation I have in mind here—to which I refer in talking about "variations that arise as a result of ontogeny"—is part of the notion that, in evolutionary biology, is called "genetic variation" (Futuyma 1998: 267ff).

2. By a "classical computational mechanism" I mean a cognitive mechanism which can be described as a Turing Machine. Note that cognitive modularity, as Fodor understands it, is a matter of degree; and most everybody else in the cognitive sciences agrees with him. Furthermore, the same can be said of at least two of the notions of biological modularity examined here; to wit, pleiotropic, functional, and variational modularity.

3. Carruthers (2005: 20-21) distinguishes between wide scope and narrow scope encapsulation, as follows: (a) A mechanism is wide-scope (WS) encapsulated at time t when it is necessary that there is some (usually much) information available to other systems of the mind, that is not available for M's use at t; and (b) on the other hand, a mechanism is narrow-scope (NS) encapsulated when, at all times, there is some specific type or types of information that the mechanism cannot use. Narrow scope encapsulation is what Fodor, Cosmides and Tooby, Karmiloff-Smith, the old Sperber, the cognitive ethologists like Peter Marler, and many other evolutionarily inclined cognitive scientists had in mind when they talked about encapsulation and about modularity. Wide-scope encapsulation—Carruther's own notion—is more general than the classical, narrower notion. He coins this new notion because he wants to marry two different lines of research in cognitive

psychology—i.e., evolutionary psychology whose paradigm members are Cosmides and Tooby, and the simple heuristic movement headed by Gerd Gigerezer (Gigerenzer 1991, 2001; Gigerenzer and Hug 1992). A wide enough notion of encapsulation—and thus of modularity—would be helpful to Carruthers in this respect. However, the conclusions at which we arrive here do not depend upon whether encapsulation is understood in a narrow or in a wide manner.

4. As we shall see, most modularists understand the phrase "specific patterns of cognitive breakdown" to mean cognitive dissociability: a cognitive mechanism is said to be dissociable from other such mechanisms when it can break down without those other mechanisms breaking down as a result.

5. Note that this passage talks about the modularity of "functionally distinct" character complexes –this, as we shall see, refers to what cognitive scientists, but not biologists, call "functional modularity." Functional modularity in biology, on the other hand, is understood as either functional independence or functional integration. Below we characterize all of these functional notions in more precise terms, and clarify their differences.

6. Note that what I here call "pleiotropic modularity" is what Wagner and Wagner (2003) call "variational modularity." I use "pleiotropic modularity" because, as we shall see, I call "variational modularity" something somewhat different to the Wagner's notion, more akin to what Hansen (2003) suggests.

7. As it turns out, the refraction of the lens of an eye is not very variationally independent of other functions of an organism.
8. The notion of a *variational* module is closely related to a notion of a *biological character*, to wit, to the notion of a unit of independent variation in organisms that are related. The central idea behind the notion of a variational module is that of variational independence; and a variational module will be that unit which tends to vary in ways that are independent from other units. Yet there are two ways in which the notion of a biological character has been understood in biology:

(a) As characters that are "inferred from the observation of correlations among units of description and quantification," such as morphometric variables. In these cases, variational modules will be inferred, when appropriate, from their "actual dissociability in collections of organisms that are treated as contemporaneous" (Eble 2005: #-#); in other words, for X and Y to count as two distinct variational modules, in this sense, it must be the case that they can actually be dissociated in actual populations;

(b) As the characters of phylogeny which are considered as the "stable units of evolutionary variation across species." In this case, a module is a unit corresponding to a distinct historical event, which suggests its "potential dissociability over evolutionary time" (Eble 2005: #). In this sense, if X and Y have two different evolutionary histories, then they will be two variational modules, even in those cases where X and Y are somehow presently correlated or have coevolved.

Here I cannot undertake the discussion as to which of these notions of a biological character is the one that is most relevant to the notions of variation and of variational independence that are ultimately relevant to a characterization of variational modularity in connection with a claim concerning evolvability.

9. Calling these developmental modules “ontogenetic” is part of my own terminology.

10. Of course, one must add here the requirement that M2 is not a proper part (or subsystem) of M1.

11. See note 2 above.

12. One more interesting point to make concerning functional independence: those studies in neurocognitive science that establish the presence of selective cognitive impairments in adults can reasonably be interpreted as evidence of the functional independence of the underlying cognitive mechanisms—whether or not those impairments occur as a result of ontogeny—precisely because talking about the functional independence of a mechanism implies nothing concerning its underlying causes–whether they all are plausibly “biological” or not.

13. All of volume 39 of the journal *Cortex* (2003) is devoted to these discussions. See also Gerrans (2003).

14. To my mind, this characterization of c-modularity implies that a c-modular mechanism has to be innate in a sense of innateness I proposed and defended elsewhere; to wit, an innate feature is one which arises in ontogeny as a result of causal factors some of which involve genetic expression and all of which are "typical" in the sense of having a certain phylogeny. For more details see my (2005) <MISSING FROM LIST>.

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