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Constructive criticism: An evaluation of Buller and Hardcastle's genetic and neuroscientific arguments against Evolutionary Psychology

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David Buller and Valerie Hardcastle have argued that various discoveries about the genetics and nature of brain development show that most "central" psychological mechanisms cannot be adaptations because the nature of the contribution from the environment on which they are based shows they are not heritable. Some philosophers and scientists have argued that a strong role for the environment is compatible with high heritability as long as the environment is highly stable down lineages. In this paper I support this view by arguing that the discoveries Buller and Hardcastle refer to either do not show as strong a role for the environment as they suggest, or these discoveries show that the brain's developmental process depends in many cases on input from the environment that is highly stable across generations.

Keywords: Brain Development; Evolutionary Psychology; Neuroscience; Philosophy of Cognitive Science

1. Introduction

David Buller and Valerie Hardcastle (Buller, 2005a, 2007; Buller & Hardcastle, 2000)[1] have argued that various discoveries about the genetics that underlie the brain's development, its plastic response to severe damage and disruption, and the causes that lead to its being wired up correctly into systems that instantiate psychological mechanisms show that the "central" psychological mechanisms that make up the mind are not heritable, and hence cannot be adaptations. For Buller, these arguments are part of a larger project (see especially Buller, 2005a; but also Buller, 2005b) where

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he argues that the "Santa Barbara" school of Evolutionary Psychology is essentially a pseudoscience: he argues that the central work of Evolutionary Psychology on such topics as social exchange (Cosmides, 1989; Cosmides & Tooby, 1992), mating strategies (Buss, 2000), and step-parental infanticide (Daly & Wilson, 1988) is fundamentally flawed, and that some of these flaws can be partly explained in terms of some of the problematic commitments of the Evolutionary Psychologists.

One of these commitments is to what Samuels (1998a) calls the "massive modularity hypothesis," that is, the claim that the human mind is composed largely or entirely of many innate, domain-specific psychological mechanisms, which are adaptations in response to specific adaptive problems that humans faced in their "environment of evolutionary adaptation" (EEA; Cosmides & Tooby, 1994; Tooby & Cosmides, 1990). This view of modularity is weaker than the original idea found in Fodor (1983): for example, Evolutionary Psychologists are not committed to the idea that modules are informationally encapsulated, nor that only input and language mechanisms are modular; many so-called central processing mechanisms are, too (Barrett, 2006). Buller and Hardcastle's arguments from the neuroscience evidence are designed to respond to the component of the massive modularity hypothesis on which all the Evolutionary Psychologists' work relies, i.e., that human psychological mechanisms are *adaptations*, and that consequently, scientists can use information about the EEA to predict what those mechanisms will be like. Specifically, Buller and Hardcastle argue that most human "central" psychological mechanisms cannot be adaptations because the nature of the genetic and developmental processes that are responsible for them shows that they are not *heritable* in the right way. However, this does not mean that Buller and Hardcastle do not think that the human mind is modular in a weaker sense. What they accept is something closer to Karmiloff-Smith's (2000) view of modularity—that the brain has domain-*dominant* areas, and that this dominance emerges from an interaction between a few minor innate biases and the developmental environment (Buller, 2005a, pp. 135–136; Buller & Hardcastle, 2000, p. 313).

So what exactly do Buller and Hardcastle mean when they say that many central psychological mechanisms are not heritable, and why is this important for their claim that such mechanisms cannot evolve by natural selection? There are multiple meanings of heritability (for a discussion, see Mameli & Bateson, 2006); in the simplest sense, that a trait is heritable simply means that offspring tend to have the same variant of that trait as their parents. However, the meaning of heritability that Buller and Hardcastle are using is roughly "broad sense heritability" in the classical population genetics sense, i.e., that a trait T is heritable when most of the variation in T in a population is due to variation in genes, rather than variation in the environment (Buller & Hardcastle, 2000, p. 319). The reason why high heritability in Buller and Hardcastle's sense is supposed to be necessary for natural selection to act on T is that when there is selection for certain variants of T, individuals with those variants will leave more offspring than individuals with other variants. Because genes are transmitted across generations, these offspring will, on average, have the same genes as their parents. If it is differences in genes that make most of the difference in what

variants of T individuals have, then in the offspring's generation, T will increase its representation in the population. The idea is that since the environment individuals face can change, the same will not necessarily be true if differences in T are mainly due to differences in the environment.[2] When Buller and Hardcastle assert that the neuroscientific evidence shows that most "central" human psychological mechanisms are not heritable and therefore cannot be subject to natural selection (Buller & Hardcastle, 2000, p. 319), they mean that this evidence reveals that most variation in central human psychological mechanisms in human populations is due to variations in the environment[3] (Buller, 2005a, p. 141) because information from the environment is the critical resource in those mechanisms' development.[4]

Some philosophers and scientists have responded to Buller and Hardcastle's arguments (Barrett, 2006; Barrett & Kurzban, 2006; Sarnecki, 2007) or to others making similar kinds of developmental arguments against Evolutionary Psychology (Lickliter & Honeycutt, 2003; see for responses Bjorklund, 2003; Krebs, 2003; Tooby, Cosmides, & Barrett, 2003). These authors argue that traits can be broad sense heritable even when their development relies heavily on environmental resources. All that is required is that those environmental resources be stable and not vary within lineages. Sarnecki (2007) uses as an example the Jack Pine tree, which has a hard waxy coating on its pine cones that prevents the seeds from being released until the coating is melted in a forest fire. Because such trees have faced such fires reliably at least once a generation, the necessity of the fire for reproduction does not prevent the tree from developing reliably and its traits being subject to natural selection. In such a case, because the environment does not vary much, the importance of the environmental resources is still compatible with the Jack Pine's traits being broad sense heritable.

However, the problem for these responses is that it is an empirical matter whether in fact the crucial environmental resources in brain development are stable in this way, and Buller and Hardcastle think that the developmental evidence shows that they are not: while they accept that there has been some consistency in the environment in human history leading to some consistency in developmental outcomes (Buller & Hardcastle, 2000, p. 317), any variation in "central" psychological mechanisms of the sort that would need to be present for natural selection to act is largely or entirely due to environmental, and not genetic, variation (Buller, 2005a, p. 141). They believe that the evidence from neuroscience shows that the contribution of the environment is of this sort. While some authors have tried to show that Buller and Hardcastle's interpretation of some parts of this evidence is wrong (e.g., plasticity, or gene shortage), none have addressed it completely, especially the most compelling part of Buller and Hardcastle's evidence, evidence of the role of neural activity in development. In this paper, then, I want to go after Buller and Hardcastle's evidence in detail, and argue that it does not show that central psychological mechanisms have low heritability. Instead, their evidence suggests that the environmental resources in the development of psychological mechanisms are often of a sort that would only make a significant difference in what developed when they varied a great deal, or else are often of a sort that one could expect to have been very stable during human evolutionary history.

The arguments that Buller and Hardcastle offer are of three types: arguments from genes; the argument from plasticity; and arguments from the role of neural activity in brain development. Where a couple of these arguments (versions of the argument from gene shortage and the argument from plasticity in particular) have been addressed in some form or another in other work I will try to be brief, but for the sake of completeness I want to show either specifically how the standard responses to such arguments bear on the question of the heritability of psychological mechanisms as such, or to show that there are interpretations of Buller and Hardcastle's arguments not yet considered and that need to be addressed. I will address each set of arguments in turn in sections 2, 3, and 4. I will then raise some general concerns about the type of evidence to which Buller and Hardcastle are appealing in section 5. In section 6, I will sum up the reasons for rejecting their arguments.

2. Arguments from Genes

Buller and Hardcastle's first argument is that if most central psychological mechanisms are to be heritable adaptations, then there must have been variation in those mechanisms in the evolutionary past, and most of that variation must be due to differences in the genes involved in the development of all those mechanisms. However, Buller and Hardcastle claim, there simply aren't enough genes to account for all that variation given the number of mechanisms proposed—the human genome only contains about 30,000 genes and only about 50% of these are involved in the development of the nervous system (this has since been revised down to about 20,000–25,000 since the publication of Buller and Hardcastle's 2000 paper; International Human Genome Sequencing Consortium, 2004). Very similar arguments exist elsewhere in the literature and are often referred to as "arguments from gene shortage" (see, for example, Ehrlich, 2000, p. 124). Buller and Hardcastle's second "argument from genes" is what I call the "argument from the cellular environment"—that the way that individual cells are wired together is primarily caused by features of the cellular environment (Buller & Hardcastle, 2000, pp. 314–315), not genes. Buller and Hardcastle claim that developing neurons are able to position and connect themselves correctly due to such things as neurotrophic factors, exploratory fibers, and cell competition and death. Where differences in psychological mechanisms are due to differences in such resources, the differences concerned are differences in the environment, not differences in genes, and hence the variant mechanisms would not be heritable.

Responses to the "argument from gene shortage," either Buller and Hardcastle's or other versions, exist elsewhere in the literature (Barrett, 2004; Hagen, 2005; Mameli & Papineau, 2006; Marcus, 2004; Plaisance, Reydon, & Elgin, 2012; Sarnecki, 2007). While I agree with these authors' conclusions about the argument, I do think it is worth being precise about why the argument from gene shortage does not show that psychological mechanisms cannot be *heritable* as such. The problem is that the concept of heritability relies on the classical Mendelian concept of genes (alleles)

whereas gene counting measures are based on counts of coding sequences in DNA. The problem for the heritability of psychological mechanisms, then, is whether we have enough differences in "classical genes" to account for all the proposed variation in psychological mechanisms in the past. Buller and Hardcastle's answer is no, presumably because they are taking differences in classical genes to correspond straightforwardly to differences in coding sequences.

However, this reading has two problems. First (as Plaisance et al., 2012 suggest), it is at odds with how the concept of a classical gene gets interpreted in molecular terms by biologists and philosophers of biology. Second, the concept of heritability refers to genetic differences because genes are the developmental resources that are transmitted across generations: it seems reasonable to count as "allelic differences" differences in products of the genetic system that could be difference-making in development and that are reliably transmitted. Biologists and philosophers of biology do not think "differences in (classical) genes" should only be interpreted in molecular terms as differences in coding sequences. Instead, at the molecular level, differences in "genes" can also be differences, for example, in how the products of individual coding sequences are manipulated: they are often "snipped" into multiple mRNA or protein products; multiple coding sequences contribute "bits" to the same functional product; and there can even be sense and antisense readings of the same DNA sequence (see for a discussion Griffiths & Stotz, 2007; Kitcher, 1984).

Moreover, coding genes and their (very) various products produce physiological structures as a hierarchy in which genes at the top change the expression of those lower down. Coding genes can be promoted or down-regulated by DNA binding proteins (among other things) which bind to regulatory regions of DNA near the gene in question. These DNA binding proteins are themselves the products of other genes; in many cases they are generated by a chemical cascade consequent on other proteins called "growth factors" binding to receptors on the outside of the cell. These growth factors are themselves produced by genes switched on in other cells, and growth factor genes are themselves often ultimately regulated by other growth factors. The placement of many of the structures in organisms' bodies relies on gradients or combinations of gradients of growth factors formed because the growth factor spreads out from the emitting cells by osmosis, with a change in concentration of the growth factor as a function of distance from the emitting cell. Combinations of these gradients can, effectively, give a cell information about where it is in the developing organism; and since genes can have multiple promoters or inhibitors, individual genes can get turned on in cells in specific places. This hierarchy of response means that the same coding genes can get reused in different combinations for multiple purposes at different places or points in development (via, for example, different receptors responding to different growth factors, or different consequent chemical cascades; for a discussion, see Marcus, 2004).

Differences in how the products of coding genes are manipulated and in how coding genes are regulated (e.g., whether the product is turned off or on, or at different levels, or produced at different times during the developmental process) can both make a difference in development and be reliably transmitted across generations. This means

regarding these as "genetic differences" also makes sense given the classical concept of heritability to which Buller and Hardcastle appeal. There are many more such potential differences than just differences in coding sequences alone. So while whether or not there could *actually* have been enough and appropriate differences in alleles to permit most central psychological mechanisms to be heritable is not yet known, it is clear that *coding* gene-counting alone will not decide the question (and most authors responding to the gene counting argument agree, e.g., Plaisance et al., 2012).

What's more, the nature of gene regulation also makes clear what is wrong with Buller and Hardcastle's other argument from genes, the "argument from the cellular environment." Buller and Hardcastle imply that the role of the cellular environment in development means that it is the larger environment that is the main difference maker in the development of the mechanisms of the brain, rather than the genetic and epigenetic system. However, it should be obvious from the above that one of the main ways that the genetic and epigenetic system works in development is by releasing proteins into the cellular environment—i.e., growth factors—that then change gene regulation. And indeed, all of the cellular environmental causes that Buller and Hardcastle describe as part of brain development are the direct products of, or have as important causes, hierarchical gene expression. For example, the "neurotrophic factors," which Buller and Hardcastle describe as being what really controls neuron growth, are just growth factors for neurons. The "feeling" axons employed by some neurons are "feeling" for or being repulsed by gradients of growth factors emitted by genes in other neurons (Gierer & Muller, 1995). Consequently, differences in the nature of these causes (which neurotrophic factors there are, or which molecular gradients there are and where, and so on) is a matter, in large part, of differences in coding genes, their regulation, and their products.

3. Arguments from Plasticity

Another argument Buller and Hardcastle make is the argument from plasticity, i.e., the brain's capacity to change in response to damage or disruption. Buller and Hardcastle make this argument primarily as a response to the view that most psychological mechanisms are domain-specific, but also that because "our functionally specialized cortical circuits are plastic and environmentally shaped," we can conclude that "they simply are not biological adaptations" (Buller, 2005a, p. 140). In other words, plasticity indicates that brain structures are not heritable because it indicates that these structures vary within a population more with the environment than they do with genes (Buller & Hardcastle, 2000, p. 319). Buller and Hardcastle cite a number of different studies in support of the view that brain processing areas are highly plastic. For example, when some animals have digits or limbs removed, the neurons responsible for controlling the missing limb or digit have been shown to become responsive to other limbs or digits (Clark, Allard, Jenkins, & Merzenich, 1988; Merzenich et al., 1984). Buller and Hardcastle also discuss the way that the brains of animals are able to compensate for vestibular disturbances in the form of loss of

information from the semicircular canals. Animals recover their equilibrium very quickly after these events (too quickly for neuronal rewiring to be taking place), and the suggestion seems to be that the vestibular system receives input from both the inner ear and the eyes, but that the visual information is "cloaked" until there is damage to the inner ear system (Berthoz, 1988; Hardcastle & Stewart, 2007). In other words, different areas of the brain are capable of computing information from multiple sources or adapting to new functions (Buller & Hardcastle, 2000, pp. 312–313). The point is that plasticity of this sort shows that the environment "participates heavily" in the formation of what mechanisms we have (Buller, 2005a, p. 139). If our circuits change in response to the environment not only in development, but throughout life, then they cannot be adaptations (Buller, 2005a, p. 140).

There are three possible ways to interpret what Buller and Hardcastle are doing here; two of these already have some responses in the literature. The first is to treat Buller and Hardcastle's argument as simply about cases of mature plasticity, and to argue that evidence of mature plasticity need not be evidence of low heritability but of the presence of facultative adaptations that permit response to damage or other environmental causes (Hagen, 2005; Marcus, 2004). This can be the case even where the mechanism is domain-specific (so long as the change isn't outside of the domain of the mechanism; Hagen & Hammerstein, 2005). Evidence such as the cross-modal processing in the vestibular system only seems to suggest that the damaged vestibular mechanism can compensate for changes, not that the mechanism itself changes in any interesting sense (the visual circuit seems to take up the slack from the semi-circular canal circuit, but both have always been present). The cases where brain areas change which digits or limbs they respond to again seems to be a matter of neurons in those areas responding to inputs from other digits that were always there but only uncovered by the loss of original digits. Even if that view of what is happening is incorrect, these cases still don't involve *new* mechanisms arising; instead, they seem to involve an expansion of the area of the cortex used by the mechanism responsible for an already existing digit or limb.

The second alternative is to take Buller and Hardcastle to also mean to refer to *developmental* plasticity. Considerable evidence has shown that in humans and other mammals, where or how cortical mechanisms develop can change significantly if development is disrupted by extreme environmental manipulations. Some of the neuroscientists Buller and Hardcastle cite favorably make this argument. For example, Elman et al. (1996) cite Webster, Bachevalier, and Ungerleider (1995), who show that infant monkeys who have parts of their temporal lobes removed are able to regain most of their capacity to recognize objects; O'Leary and Stanfield (1989), who show that brain cells that are transplanted from one region of cortex into another develop much the same way as cells in the area into which they are transplanted; and so on. In this case, if plasticity means that the brain can generate different mechanisms when the developmental environment is variously changed, that suggests those mechanisms are not heritable. However, as Sarnecki (2007) and Samuels (1998b) argue, developmental plasticity only seems to involve changes in *where* the mechanisms in question form under extreme experimental manipulation, not *which ones* form (e.g., in the studies above, the monkeys with the damaged temporal lobes still had the same mechanisms,

but the mechanisms were instantiated in different brain areas; in the transplant case, the transplanted cells took on the preexisting functions of the regions they were transplanted into). Evidence that such mechanisms still form even when the developmental environment is extremely disrupted is, if anything, evidence for *high* heritability, not low heritability.

However, I think that there is another, better interpretation of Buller and Hardcastle's appeal to plasticity not yet considered. It is not that they take these plasticity studies to be evidence of new mechanisms forming in response to changes in the environment, but instead they take it to be evidence of the *type of process* that forms these mechanisms. Plasticity shows that the creation of brain mechanisms is a process of *activity-dependent construction*, such that if the environment were right, genuinely different mechanisms would form; the main reason the same mechanisms form in new places in plasticity cases is because the environmental stimulus that forms the new mechanisms after the initial mechanisms have been damaged is much the same as the old one. And indeed, there is some evidence that plasticity is achieved by activity dependence (von Melchner, Pallas, & Sur, 2000). If this is the correct interpretation of Buller and Hardcastle, then the only way to fully address their argument from plasticity is to address their claims about the role that neural activity plays in brain development. I will tackle these claims in the next section.

4. Arguments from the Role of Neural Activity in Development

The third set of arguments Buller and Hardcastle present are those that appeal to the role of neural activity in shaping the wiring of the areas and mechanisms of the brain, and thus the psychological mechanisms which those brain mechanisms and areas instantiate. When the brain is developing, the wiring is formed by axons from each neuron seeking out and forming a synapse with a dendrite on another neuron. Buller and Hardcastle point out that there is some evidence that the exact patterns of synapses that form in the brain are highly dependent on activity traveling between neurons. Buller and Hardcastle cite a lot of different studies in favor of their view, but don't really discuss the content of most of these. Instead they focus on discussing the much more controversial view that brain development is Darwinian, that is, that most or all neural structure is formed by a process where neurons form many connections with other neurons—there is "exuberant branching"—and then levels of activity determine which connections are retained and which are dropped—or "pruned"—the most active connections are retained at the expense of the less active ones (Buller & Hardcastle, 2000, p. 316). Neural activity is largely or entirely responsible, consequently, for which wiring pattern in the cortex gets retained over time.

While certainly *some* neural structure is formed by exuberant branching and subtractive processes, there is no particular reason at this point in time to think that *all* or even *most* of it is (Purves, White, & Riddle, 1996; for a discussion see also Machery & Barrett, 2006). Buller and Hardcastle try to support the view that most or all neural connections are formed by subtractive, activity-dependent pruning by making the

strange claim that the adult brain contains fewer cells *and* fewer connections than the infant brain (Buller, 2005a, p. 132). If this were the case then the brain would not increase in mass during childhood. Instead, what appears to be the case is that adult brains contain about the same amount of neurons as infant brains, and contain neurons which are larger and have more and more elaborate connections than do neurons in infant brains (Purves et al., 1996). In which case, development during childhood must involve additive as well as subtractive processes.

There *is* good evidence, however, for the weaker view that neural activity has an important role in the development of the cortex. The reason why an important role for activity is supposed to support Buller and Hardcastle's argument that environmental information shapes cortical mechanisms is that neural activity can *derive from the environment via the senses*, and hence changes in what is coming in from the senses can change the pattern of neural activity the cortex experiences, and therefore which mechanisms develop. Buller and Hardcastle note, correctly, that both endogenous activity (i.e., activity that is a consequence of the nature of the neurons and their structure) and exogenous activity (derived from the environment) play a role in shaping the cortex (Cramer & Sur, 1995). However, Buller and Hardcastle argue that since endogenous activity is shaped by structures which are in turn shaped by exogenous processes, the whole system indirectly relies on exogenous activity (Buller, 2005a, p. 133). Buller and Hardcastle further argue that since most *cortical* mechanisms either consist of connections dependent on neural activity or at least are larger scale structures made out of connections dependent on neural activity, most *psychological* mechanisms have to be dependent on activity, too (Buller & Hardcastle, 2000, p. 316). This suggests that it is differences in the environment that must account for most or all of the differences in psychological mechanisms in a population, and therefore that most psychological mechanisms must not be heritable. So what I want to do is describe a few of the studies that are described in the reviews that Buller and Hardcastle cite, those that present the best evidence for their view that environmentally derived activity is very important in forming the cortex.

One set of studies that Buller and Hardcastle mention obliquely (Buller, 2005a, p. 133; Buller & Hardcastle, 2000, p. 315) are the classic experiments on ocular dominance columns. Ocular dominance columns form in the input layer of the visual cortices of most mammals (similar columns also form in the part of the thalamus which transfers the signals from the retina to the visual cortex). These are stripes of cells where the cells in some stripes only respond to the left eye and the cells in others only to the right. A wide variety of studies suggest that if various animals (including cats, ferrets, and mice) have the signals from the retina of one or both eyes blocked or stopped during a critical period of their development, the columns only respond to the open eye or fail to form at all (for the seminal studies on ocular dominance columns see Hubel & Wiesel, 1962, 1963; Wiesel & Hubel, 1963; for a review of the very extensive literature, see Katz & Crowley, 2002). Blocking of the retinal input to just one eye can lead to all the cells in question becoming responsive to the open eye (Hubel & Wiesel, 1962, 1963; Wiesel & Hubel, 1963) and the animal becoming functionally blind in the closed eye.

Other studies lead Buller and Hardcastle to conclude that while brain structure appears to be under "rigid genetic control," creation of the wiring and circuits requires the presence of activity (Buller, 2005a, pp. 132–133). One reason neuroscientists think this is true is that while most large-scale structures in the brain form well before activity begins, many of the fine-grained structures do not develop until they receive their incoming connections (Pallas, 2001). This could be because it takes activity to form these fine-grained features properly. Buller and Hardcastle cite a review by Cramer and Sur (1995) which mentions a series of lesion experiments performed by Sur's laboratory on the development of the ferret's visual cortex and thalamus. In a recent version of this type of experiment, von Melchner et al. (2000) were able to encourage some of the connections from the retina of ferrets to wire to their medial geniculate nucleus (MGN) and therefore in turn to the auditory cortex, instead of the lateral geniculate nucleus (LGN), their normal target. What was really impressive about this study was that after the rewiring was complete the auditory cortex as a whole began to take on structural properties similar to (although not identical to) those of the visual cortex; von Melchner et al. were also able to show that the ferrets in question responded to the input to the auditory cortex as if it were visual input. It should be noted that while the ferrets were able to receive visual input to their auditory cortex and respond to it appropriately, their powers of discrimination were limited; the representational structures the auditory cortex took on were also not entirely like those of the visual cortex (which may explain certain limitations in the ferrets' discriminatory powers).

5. Response to Buller and Hardcastle's Arguments from Neural Activity

There are two possible ways to respond to Buller and Hardcastle's arguments from neural activity. One way is to challenge their claims about the role that neural activity derived from the environment plays in the formation of most *cortical* mechanisms. The other is to argue that, given that neural activity is important in the appropriate way, the nature of the instantiation of *psychological* mechanisms in cortical mechanisms means that the presence and properties of those psychological mechanisms is indifferent to variations in that activity.

5.1 The Role of Activity in Cortical Development

First, then, let's address the studies I described above as evidence for the role of activity in cortical development. The evidence for the importance of activity is somewhat mixed, both in how far and in what type of activity is important. For example, there are a number of studies which suggest that removing the possibility of activity (either by preventing neurotransmitter release, dark rearing, and so forth) still results in brain systems that form normally or mostly normally (Crowley & Katz, 2002; Verhage et al., 2000). In other cases, cortical mechanisms and representations *partially* form in the absence of neural activity (Callaway & Katz, 1991; Ruthazer & Stryker, 1996; Sengpiel,

Stawinski, & Bonhoeffer, 1999). In the Hubel and Wiesel case, while blocking one eye meant ODCs formed only for the open eye, stopping input to both eyes resulted in *normal* ODCs. Some of the changes induced by activity (or lack of activity) in these cases may be due to a separate mechanism that permits the organism to respond adaptively to injury or developmental disruption. For example, one interpretation of the work on ocular dominance columns is that their basic structure develops robustly and early, but later, during a critical period, becomes sensitive to environmentally induced activity from the senses which can disrupt the original pattern (see, for example, Hubel, 1988; Marcus, 2001, 2004)—i.e., the kittens' ODCs develop normally with environmentally derived activity blocked, but if their ODCs are exposed to disruptive activity during a particular point in development, the ODCs do not form normally. This view is consistent with the data so far accumulated (Katz & Crowley, 2002). The timing and nature of the sensitive period itself may be dependent on a set of specific regulatory genes, whose activity seems to limit when the systems in question can be modified (Majdan & Shatz, 2006).

Second, as I mentioned earlier, there is more than one kind of activity involved in the development of the cortex, and not all of these would support Buller and Hardcastle's contention that cortical mechanisms are constructed from experience. Neuroscientists make two main distinctions between types of cortical activity involved in cortical development. The first distinction is between endogenous and exogenous activity. The second, according to Crair (1999), is between two roles that that activity can take in forming the developing cortex; the first is a *permissive* role for activity—that is, the activity needed to correctly form the system can be of any kind—versus a role for *instructive* or *patterned* activity—the activity needed to correctly form the system must take a particular form or pattern. For example, Shah and Crair (2008) have shown that the development of the visual cortex is dependent on that area receiving "retinal waves" that have a particular pattern: removing this activity or replacing it with activity having a different pattern means that those areas do not form correctly. It is worth noting that if there is a strong role for endogenous activity then in no sense is Buller and Hardcastle's argument supported. Their argument depends on the idea that it is differences in the environment that make a difference in what mechanisms form, and internal activity will not vary without extreme changes in the environment because such activity is the consequence of the fundamental electrical properties of neurons and the initial wiring of the brain. Even an important role for permissive, external activity will not support their argument, because again, the only difference in this type of activity that can make a difference to the resulting structures would require extreme or rare changes to the environment. Buller and Hardcastle need *at the very least* instructive, external activity to be very important in cortical development in order to make their case that psychological mechanisms are not heritable.

Buller and Hardcastle argue that in practice, the distinction between endogenous and exogenous activity makes no difference because all endogenous activity has its source in systems shaped by exogenous activity (Buller, 2005a, p. 133). I presume (since they don't cite their sources) that they mean that the sensory systems that depend on exogenous activity form first, and are then the source of the activity that

forms the rest of the cortex. This isn't obviously the case. First, a lot of the necessary shaping activity occurs before birth or before the eyes of animals such as ferrets and kittens open. While some sensory information is probably reaching the brain prenatally (and hence some environmentally derived, instructive activity generated by this information could be involved in shaping some brain structures), this cannot be universally true: indeed, many of the studies on which the claims about the role of exogenous activity is based involve the development of the visual cortex of organisms before the eyes open, and hence before patterned visual information can reach the eyes (Crowley & Katz, 2002; Rakic, 1976).

Second, much endogenous activity occurs because neurons fire spontaneously; they don't require incoming signals to do this. Brain development seems to exploit this fact to useful purpose. Some of the sensory areas dependent on patterned activity get the "pattern" from the way that the wiring of the relevant brain areas has already been shaped before activity even begins (by processes driven by other developmental resources). The electrical activity that results comes from the spontaneous firing of neurons that is structured by the way the neurons are wired together. For example, the retinal waves described above (Shah & Crair, 2008) are generated by the structure of the retina which develops before the structures in the visual cortex are formed. However, they are not produced by "environmental information," nor is the retina from which they derive. Furthermore, even if Buller and Hardcastle were right about the source of activity, that still wouldn't mean that the activity started in the exogenously shaped areas was structuring the rest of the cortex as instructive activity; it could well be permissive (Crair, 1999). So even if the source of activity shaping an area is exogenous this doesn't amount to the area being dependent on the environment in any sense that is useful to Buller and Hardcastle.

Finally, let's address the apparent role of incoming connections in the development of fine-grained brain structures. Do experiments like the von Melchner et al. (2000) study really show that environmental activity is necessary to the formation of the features of the visual system? I think that they don't necessarily, because it isn't clear from the experiments whether changing which *connections* are coming in to an area changes what develops because it changes the *environmentally derived electrical signals* going into that area. Another possibility is that the crucial ingredient that is introduced with the new connections is a regulatory genetic signal—i.e., a set of growth factors or else an endogenous electrical signal that itself regulates growth factors in the area. The idea is that the growth factors in question are those responsible for marking out the proper location of the mechanism in question in its original area and in turn regulating the genes involved in the developmental program for forming that mechanism. If this is the case, then the formation of the mechanism in a new area when the incoming molecular signals are moved could indeed be an indicator of *high* heritability, since it appears that the genetic resources are the crucial difference makers in the case.

While this is speculation, it is speculation with some support. A study by Horng et al. (2009, which includes some of the scientists involved in the von Melchner study) have identified some sets of genes which are known to have a regulatory function and

which appear to be expressed differently in the MGN and LGN. And, indeed, rewiring retinal connections from the LGN to the MGN as in the von Melchner study changes the pattern of expression of these genes in the MGN to a pattern like that in the LGN. All developmental processes must involve gene expression, of course. But if developing an MGN rather than an LGN in the thalamus is just a matter of introducing different types of environmental signals into the two areas, then the sorts of genes switched on in these areas should be genes low in the developmental hierarchy, involved in neuron and axon growth, and so on. Differences in activity should create differences in where, when, and how much these genes are expressed in the two areas, rather than in which genes are involved.

The differences in the gene expression of the MGN and LGN, however, seem to go beyond this: for example, some of the regulatory genes involved seem to be expressed across distinct areas of the MGN and not the LGN, or only in the MGN and not the LGN or vice versa. In other words, these look like area-marking genes (there was also some new activity which the authors think may represent a plasticity mechanism). If this is the case, then the dependency of the development of the LGN on its incoming connections may well not be due to a dependency on an *environmental* signal, and the robustness of the development of the LGN mechanism in a new location may actually be an indicator that that system is highly heritable—differences in the LGN will still largely be due to differences in genes. To be sure that this is the case, of course, more needs to be understood about how these genes work.

5.2 The Nature of the Instantiation of Psychological Mechanisms in Cortical Mechanisms

Suppose, however, for the sake of argument, that Buller and Hardcastle are right that exogenous, instructive activity is important in the formation of the cortex. This still doesn't answer the question of whether the activity dependence of the formation of *cortical* mechanisms means that the *psychological* features of the psychological mechanisms they instantiate vary with differences in that instructive activity. The upshot of activity dependence will depend greatly on the way those mechanisms are instantiated in the cortex. I want now to describe four possible psychological interpretations of the contribution of externally generated neural activity to cortical development: it might be interpreted as *tuning* of the connections necessary to form psychological mechanisms; as *parameter setting* of psychological mechanisms already in place; as mechanisms already in place *learning representations*; or as *wholesale construction* of psychological mechanisms.

Let's look at *tuning* first. On the tuning view, the role of neural activity is to correct and strengthen connections that have already been put in place by other developmental resources. On this view, the rough overall *pattern* of these connections develops robustly across individuals and varies most with genes and little with the environment in populations—i.e., getting psychological mechanisms in place with the right computational features may require that there be a connection between some point A and point B, and this connection needs to be heritable. However, what could vary between individuals with the same genes on this view is the exact pattern of lower-level

connections that gets A and B connected: perhaps because there are differences in the precise details of the cellular and embryonic environment during development. Alternatively, activity might be involved in the *initial* building of strong connections between cells, but where this process is so constrained by the other mechanisms involved that while the resulting fine-grained pathways might vary between individuals with the same genes, those individuals reliably end up with the same overall pattern. In both these cases, the overall pattern of connections, which give the system its psychological properties, would be heritable, even if the lower-level features are not.

The second possible way to interpret the role of neural activity (especially of the type that involves exuberant branching and pruning of connections) is as *parameter setting*. Parameter setting is derived from a view about how certain proposed psychological mechanisms which drive learning processes are supposed to work—most importantly, the grammar of language. Languages themselves are clearly learned, but one popular view in cognitive science is that the mechanisms responsible for language learning greatly constrain the search process by limiting the learner to a range of possible options for the key parameters of the grammars of languages—experience then permits him or her to "decide" between these parameters. So one possibility is that the initial structure of the cortex before the arrival of activity involves what are effectively many different possible mechanism pathways or representations—the possible parameters—and neural activity "prunes away" most of these pathways or representations to leave the appropriate one in place. In this case, it is the pre-pruned state—the capacity to be "set" in a variety of different ways—that would be heritable, since differences in this mechanism would largely be due to differences in genes. The variable outcomes of the "parameter setting" process (i.e., Swahili or Japanese grammar in the case of a language mechanism) would have low heritability, however, since in this case it is differences in the environment that make most of the difference in what develops. This is the view of most Evolutionary Psychologists with regard to language: it is the capacity to learn the grammar of languages that is an adaptation, rather than the capacity to employ Swahili or Japanese grammar. Evolutionary Psychologists think that some or even many mechanisms may turn out to be of this type, so there will be no problem for them if this is what the role of neural activity comes to.

The third option for interpreting the psychological role of neural activity is that it involves mechanisms *learning or acquiring representations*: in this case, the neural activity counts as a representation of the environment. The preexisting cortical mechanisms receiving that representation and being modified count as psychological mechanisms engaging in *learning* of those representations: for example, a mechanism that permits us to make moral judgments acquiring representations of moral norms by learning the appropriate norms from the environment. In this case, the preexisting moral judgment mechanism would be heritable, since again, differences in which learning mechanisms individuals have could be largely accounted for in terms of differences in their genes; the post-learning state (i.e., the particular norms acquired) would not be heritable, however. It's worth pointing out that pretty much all the Evolutionary Psychologists think that at least some human mental representations are learned. Consequently, *some* cases where

the activity dependence of a mechanism counts as that mechanism learning representations is completely compatible with their view.

The final possibility is the one that Buller and Hardcastle want us to accept: that the contribution of neural activity represents *wholesale construction* of most psychological mechanisms from the cortex. In this case, the changes in neural structures induced by neural activity counts as the formation of new mechanisms in the cortex, not just the acquisition of representations or the setting of parameters. The idea is that when neural activity varies, different mechanisms are formed. This means that differences in the environment across a population result in differences in those mechanisms; hence, the resulting mechanisms would not be heritable. Uncontroversial examples of "constructed" mechanisms might include mechanisms that make chess playing or bike riding possible and rapid.

If the contribution of neural activity to cortical development is to constitute wholesale construction, then most of this activity is going to have to be instructive, externally generated activity. What's more, if Buller and Hardcastle are to make their case against the Evolutionary Psychologists, then external, instructive activity is going to have to be necessary for the formation of *many* or *most* cortical mechanisms (even Evolutionary Psychologists would accept that mechanisms for bike riding or chess playing are constructed). However, as I have already discussed, this is just not true. Even in cases (such as Shah & Crair, 2008) where mechanisms do require instructive activity, it isn't obvious that they can vary without extreme experimental manipulations (as Shah and Crair had to do) which would be rare or impossible in the natural environment. Even if variations in such instructive activity did cause variations in some cortical structure, that variation might still count as "tuning" at the psychological level. Finally, "sometimes" does not cut it—Buller and Hardcastle need to show that *many* or *most* mechanisms require instructive environmental activity in order to make their case. Consequently, there seems no reason to accept Buller and Hardcastle's argument that the role of brain activity in the development of most or many "central" psychological mechanisms means they have low heritability.

6. Some General Concerns about the Studies to Which Buller and Hardcastle Appeal

One final and general point I want to make about most of the studies to which Buller and Hardcastle appeal is that they are (almost) all based on sensory motor model systems. Model systems are important for understanding the brain and brain development—the nature, development, and even computational features of the visual system and other sensory systems are the best understood. However, model systems do thereby have significant limitations on what they can tell us about the development of the rest of the brain, in particular the "central" psychological mechanisms about which Buller and Hardcastle are concerned. First, even supposing that Buller and Hardcastle had managed to establish their conclusions, the best their evidence could show is that *sensory motor* mechanisms were not heritable adaptations.

This is a peculiar result, since Buller and Hardcastle themselves agree with Fodor's view that the "sensory input" mechanisms of the mind are probably modular and "genetically specified" (Buller & Hardcastle, 2000, p. 314), along with many other philosophers and cognitive scientists. Part of the reason may be that Buller and Hardcastle take most of these transducer mechanisms to be those instantiated in *subcortical* mechanisms (e.g., Buller, 2005a, p. 142). However, much of the human cerebral cortex is also given over to basic sensory processing.

Second, there are plausibly some significant reasons to expect differences between the development of the sensory motor cortices and that of the "central" cognitive systems which are the focus of Buller and Hardcastle's argument. Quartz (1999) suggests, for example, that sensory and motor systems need instructive environmental input in order to calibrate themselves and keep up with the development of the body and senses over time. The brain systems cannot "predict in advance" what the body will be like in great detail (beyond a very approximate idea of size and what bits it will have!) so the representations those mechanisms employ of the body and the senses need to be flexible in response to the changing form of the sensory input. The same is not obviously true of the systems elsewhere in the cortex.

7. Conclusion

In this paper, then, I have shown that the evidence Buller and Hardcastle present does not give us a reason to expect that most central psychological mechanisms will not be heritable. To make their argument, Buller and Hardcastle needed to show that most variation in psychological mechanisms was likely to be due to variation in the environment, rather than variation in genes. Buller and Hardcastle's strategy was to show both that there would have been insufficient genetic variation to account for the variation in psychological mechanisms that would have had to be present in the human EEA if those mechanisms were to evolve by natural selection, and to show that the contribution of environmentally driven activity to the plasticity and development of psychological mechanisms suggests that any variation in those mechanisms would have to have been due to variations in the environment. My argument has been that Buller and Hardcastle are wrong about the possible range of genetic variation. They also misunderstand the nature of neural activity and the way it contributes to the development and plasticity of *cortical* mechanisms; even if they were right, this does not mean that environmentally derived neural activity is important to forming the psychological features of *psychological* mechanisms. Consequently, they do not show that psychological mechanisms have low heritability.

Notes

[1] Please note that in Buller's book *Adapting minds* (Buller, 2005a), the chapter in which he addresses these arguments is labeled as co-authored by Hardcastle, so I will also describe arguments in this chapter as "Buller and Hardcastle's" arguments.

[2] Interestingly, as Sterelny (2007) suggests, broad sense heritability is not needed for natural selection to act so long as *environments themselves* are transmitted reliably across generations (e.g., by powerful niche construction). Technically, the Evolutionary Psychologists could avoid Buller and Hardcastle's argument if there was good reason to think most psychological mechanisms were transmitted across generations in this way; however, I won't rely on this idea at all in this paper.

[3] Some adaptations are, of course, designed to vary in response to standard changes in the developmental environment (they are facultative or plastic adaptations); the kinds of variation Buller and Hardcastle have in mind here go beyond variation that is part of the adaptation.

[4] Technically, a proper demonstration of the degree of heritability of human psychological mechanisms would require a statistical analysis of how much variation in such mechanisms is due to genetic and environmental variation in human populations. Buller and Hardcastle aren't claiming to have done this, and doing so would be extraordinarily difficult. Instead, they take their evidence to tell against the high heritability of human psychological mechanisms in the weaker sense described here.

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