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The new nativism: a commentary on Gary Marcus's *The birth of the mind*

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Abstract. Gary Marcus (2004) has written a very interesting book about mental development from a nativist perspective. For the general readership at which the book is largely aimed, it will be interesting because of its many informative examples of the development of cognitive structures and because of its illuminating explanations of ways in which genes can contribute to these developmental processes. However, the book is also interesting from a theoretical point of view. Marcus tries to make nativism compatible with the central arguments that anti-nativists use to attack nativism and with many recent discoveries about genetic activity and brain development. In so doing, he reconfigures the nativist position to a considerable extent. Marcus's theory is certainly more sophisticated than any version of nativism on the market. However, in our view Marcus ends up reconfiguring the nativist position out of existence. While many of the points that Marcus makes are both interesting and correct, we see no compelling reason to classify his considered position with traditional nativism rather than anti-nativism. More generally, we that think his book points to a general moral: the opposition between nativism and anti-nativism does not help us to understand psycho-developmental issues, and should therefore be abandoned.

Gene shortage

Marcus's main objective in *The Birth of the Mind* is to argue against two popular arguments against cognitive nativism about humans: (1) the argument from gene shortage and (2) the argument from neural plasticity. Let us start with the argument from gene shortage. We now know that there are less than 100,000 genes in the human genome; the current estimate is of about 30,000. In contrast, human brains contain billions of neurons. The argument from gene shortage is that this difference in numbers means that there are far too few genes for genes to have a strong influence on brain development. Genes cannot be responsible for any detailed brain structures, but only for the general shape of the human brain. Therefore there are no innate mental structures beyond

general brain morphology. This argument can often be found in the popular press, but some eminent scientists have also endorsed it.[1]

Marcus rightly thinks that the argument from gene shortage is a bad argument. First of all, the argument is too strong. There are trillions of cells in a human body. If the argument from gene shortage showed that there are no innate mental structures, it would also show that there are no innate body structures. Second, 30,000 genes are more than enough for genes to play a central role in the development of cognitive and bodily human phenotypes.

The gene-shortage argument could work only if the mapping relation between genes and phenotypes was relatively simple, like the relation between a blueprint and the building it represents. But since the 1960s, biologists have made many discoveries that make the blueprint metaphor of the genome untenable. Gene expression has turned out very differently from the way people viewed it. We now know that even within a single organism, the relation between genes and phenotypic structures is not one-to-one but many-to-many: each gene contributes to many phenotypic structures and each phenotypic structure depends on many genes. Molecular biologists now have a well-established explanation for this phenomenon, the origin of which is to be found in a famous paper by Jacob and Monod (1961). The genome contains regulatory regions and structural regions. Structural regions serve as templates for transcription and thence for the production of new proteins and, more generally, new molecules. Regulatory regions decide whether a given structural region is transcribed or not. For example, the binding of certain molecules to a given regulatory region may constitute a mechanical barrier that prevents some nearby structural region being used as a template for transcription. Crucially, many structural regions code for molecules that themselves can bind to regulatory regions, and thereby affect whether or not *other* structural regions are transcribed. And since the molecular products of transcription are free to wander around, the products of a given structural region can influence very distant regulatory regions, on different chromosomes, say, or even in different cells. So, one pair of regulatory and associated structural region can influence the activity of other such pairs, and these pairs can influence the activity of still other pairs, and so forth. There can be branching, and there can be loops: a pair can influence the activity of a second pair that influences the activity of a third pair that influences the activity of the first pair. There are yet further complications. For example, a given stretch of structural DNA can be associated with more than one regulatory region and so give rise to different molecules in different circumstances. Moreover, what counts as a structural

[1] According to Paul Ehrlich 'Our "gene shortage" is one reason human infants and young children are so helpless. Their helplessness allows the physical and cultural environments to do the brain programming that our hereditary endowment couldn't manage. It's that environmental input that gives us the adaptability that is the hallmark of humanity. We could never have evolved as genetically controlled robots' (Ehrlich 2000). And according to Craig Venter 'The small number of genes – 30,000 instead of 140,000 – supports the notion that we are not hard-wired' (Official press release issued by the journal *Science* on the 16th of February 2001).

region or as a regulatory region may change from context to context. And the products of transcription themselves can be processed in different ways in different cellular contexts, as in alternative splicing and analogous phenomena.

The term 'gene' is nowadays normally used to refer to a regulatory region and some nearby structural region whose transcription depends directly on that regulatory region. This is not the only way to use 'gene,' but it is the standard one. And it is the one Marcus adopts. Marcus also uses the standard terminology of 'genetic cascades' for the multi-branched chains of chemical reactions resulting from the influence of given genes on other genes. Given this picture of genetic activity, it is easy to see that the gene-shortage argument is wrong. In principle, any phenotypic structure can be seen as the result of one or more genetic cascades. And given the complexities of gene interactions, far more possible cascades can be generated by 30,000 genes than there are neurons or even cells in a human body.

The gene-shortage argument is wrong. So far, so good. But what is this supposed to tell us about the truth or falsity of nativism? There seem to be two options. The first option is that the new understanding of how genes work shows that nativists are right and anti-nativists are wrong. The second option is that the new understanding of genetic activity shows that the debate between nativists and anti-nativists is a futile debate, a debate framed in the wrong terms. By presenting himself as a nativist, Marcus chooses the first option. However, it is not clear to us why he goes this way. Presumably, his implicit idea is that the new understanding of genetic activity can help us to see that many phenotypic traits are in some sense 'innate'. But how exactly does this work? Marcus shows that old-fashioned genetic determinism is false. The genome is not a blueprint in which every part corresponds to a different part of the phenotype. So he cannot be understanding innateness in terms of this old picture of genetic activity. Perhaps his idea is rather that a phenotypic structure is innate *if it is the product of one or more genetic cascades*, and that many phenotypic structures are innate in this sense. Some of the language and metaphors used by Marcus suggests that this is in fact his conception of innateness, For example, he talks about genes 'controlling' and 'supervising' the construction of phenotypic structures (148, 163, 168) and in the subtitle of the book he talks about genes that 'create the complexities of human thought'.

However, this way of understanding the nativist position is clearly flawed. Moreover, it is incompatible with many other claims Marcus makes. In some sections of his book, Marcus emphasizes how genetic activity can be involved in learning processes. On a traditional picture, the genes build the basic architecture of the mind – including innate learning mechanisms – and then learning improves upon this basic architecture without interference from the genes. This picture is wrong. Genetic activity and the electrical activity associated with learning can be intertwined – notwithstanding the fact that the electrical activity of learning occurs on a much faster time scale than genetic activity. Marcus describes recent work on synaptic strengthening which shows how the electrical activity of learning triggers genetic cascades that result in the

production of molecules that influence the learning process. Here learning turns out to be nothing but electrically generated gene activity. If both learned and unlearned traits can be products of complex genetic cascades, then such provenance cannot be the hallmark of innateness, since everybody agrees that learned traits should not be classified as innate. So the new understanding of gene expression does not give us – at least not by itself – a new way to make sense of the notion of innateness.

The fact that learning can influence genetic activity is only a particular case of a much more general phenomenon: genetic activity is often influenced by non-genetic factors. Which genes are expressed in a given cell – and to what extent they are expressed – depends on the cellular context. Cellular context depends in part on non-genetic factors. Non-genetic factors – such as ingested or inhaled substances, solar light, temperature, gravitational force, social interactions – switch on or off genes and influence the way gene products (mRNA transcripts and proteins) are processed and used. Genetic cascades do not occur in a vacuum. The very same genetic structures give rise to different cascades in different environments. In fact, it can be argued that the phrase 'genetic cascade' is misleading. Organisms and their traits are generated by *cascades of gene–environment interactions*.[2] This applies not only to mental structures but to all phenotypes. All this makes it unlikely that the attempt to use the new understanding of genetic activity to reformulate the nativist position is successful. We shall go back to this issue in the final section, after having discussed a different way to reformulate the nativist position.

Neural plasticity

Let us turn to Marcus's response to the anti-nativist argument from neural plasticity.[3] According to this argument, the extreme flexibility exhibited by the brain during its development rules out any innate cognitive structures.[4] One source of evidence for this argument is experiments involving brain-tissue transplants. In one experiment, neurons were taken from the somatosensory cortex of newborn rats and transplanted into their visual cortex. The transplant occurred before the transplanted neurons had acquired all the features of 'mature' somatosensory neurons. In their new location, the transplanted cells acquired all the features of normal visual neurons (O'Leary and Stanfield 1989). Some anti-nativists take this to show that learning is involved in the process that generates 'mature' visual neurons: in their view, the transplanted

[2] Cf. Bateson and Martin (1999); Gottlieb (1992, 1997, 2003); Meaney (2003); West-Eberhard (2003); Lewontin (2000); Gilbert (2001, 2003a, 2003b); Oyama et al. (2001) van Der Weele (1999); Moore (2001); Mameli (forthcoming).

[3] Cf. Marcus (2001).

[4] Cf. Elman et al. (1996); Quartz and Sejnowski (1997, 2002); Quartz (1999); Johnson (1997).

neurons became visual neurons simply because they received the same sensory inputs that visual neurons usually receive.

Marcus points out that cell transplants have been performed on other cells apart from neurons. If you transplant an eye cell into the stomach early enough in development, it will become a stomach cell. Yet we do not infer from this that the eye cell has learned to be a stomach cell, and that therefore stomachs are not innate. Of course this does not mean that learning is not crucial to cell specialization in the visual cortex, as opposed to the stomach, but it does show that this is not to be taken for granted. Given the cascade picture, which genes are 'switched on' in a cell depends in part on what molecules the surrounding cells are producing. So transformations from one kind of cell to another can always be due to contextual cues that may have nothing to do with learning. The transplanting evidence itself leaves it open whether the cues that trigger the 'presumptive' somatosensory neurons to become visual neurons involve learning or not.

At this point, advocates of the plasticity argument are likely to invoke Hubel's and Wiesel's studies of the development of ocular dominance columns in kittens (Hubel and Wiesel 1962; Wiesel and Hubel 1963; Hubel 1988). These authors used eye patches to deprive kittens of visual experience in one eye. In kittens with the patch, the ocular dominance columns disappeared and most of the relevant brain tissue became devoted to receiving and processing inputs from the open eye. Plasticity anti-nativists take this to show that visual experience is *necessary* for the normal development of the ocular dominance columns. But Marcus points out that, in kittens that had patches on *both* eyes, the columns developed normally in the absence of any visual stimulus. According to Marcus, this shows that visual experience is not *normally* involved in the development of the columns. The plasticity anti-nativists can reply that even if the two-patch experiment shows that visual experience is not *necessary* for the development of the columns, it does not show that visual experience is not *normally involved* in the development of the columns.

In order to make sense of experiments like these, Marcus distinguishes between 'hardwiring', 'prewiring', and 'rewiring'. A neural structure is 'hardwired' if it is 'read-only', that is, if it gets fixed independently of learning and cannot thereafter be altered. It is 'prewired' if there is a default setting that gets inscribed prior to learning, but which can later be 'rewired' by subsequent sensory experience. According to Marcus, hardwiring is not the right way to think about psychological development. He agrees with the plasticity anti-nativists that developmental studies show how learning and experience can have huge effects on cognitive structures, at least in some cases. At most, some brain structures are prewired independently of learning and experience. But this does not rule out the possibility that they might later be rewired.

By this stage some readers might be wondering exactly where Marcus differs from the plasticity anti-nativists. On the standard nativist picture, genes generate the 'hardware' of the brain, and learning and experience add 'software' to the already existing hardwire. As in a computer, the software does not change

the hardware. But the complexities of genetic expression and the plasticity of brain development lead Marcus to reject this 'hardwiring' nativism in favour of 'prewiring' nativism. However, many plasticity anti-nativists seem to draw just the same conclusions as Marcus does. They too insist that the developmental data show that the hardware-software metaphor is the wrong way of looking at brain development (Quartz and Sejnowski 2002). And they agree that at least some brain structures develop independently of learning, that is, they agree that some brain structures are prewired. After all, as Quine famously pointed out, everyone must concede that, for learning to occur, some brain structures must develop before any learning has taken place. So, is there a real debate here or are the two sides talking past each other?

As shown by the discussion about the experiments on the development of ocular dominance columns, there remain substantial differences between Marcus and the plasticity anti-nativists. They may agree that, in the context of brain development, the contrast between maturation and learning is not a matter of hardware versus software: everything is in principle 'rewirable'. But this leaves room for real disagreement on the importance of those developmental processes that generate brain structures without any learning. Marcus thinks that many important brain structures (including many cortical structures) are not due to learning. In contrast, the plasticity anti-nativists believe that only a very small number of brain structures (and cortical structures in particular) develop independently of learning. Moreover, Marcus thinks that a lot of non-learned brain structures remain intact in the mature brain. In contrast, plasticity anti-nativists deny this: in their view, almost everything gets rewired during the developmental process and, more generally, the life of the organism. That is, Marcus and the plasticity anti-nativists disagree about the extent to which learning and experience affect the development of the brain. Consistently with this, Marcus can allow that experience driven rewiring will occur in abnormal environments (Hubel's and Wiesel's one-patch kittens), yet hold that in normal cases the relevant structures are intact prewirings which owe nothing to experience (this is what he thinks about ordinary kittens). The plasticity anti-nativists, by contrast, think that even in normal cases (ordinary kittens) the relevant brain structures develop only with the help of experiential input (Elman et al. 1996; Quartz and Sejnowski 2002).

Both Marcus and the plasticity anti-nativists present evolutionary selectional arguments in support of their view. According to Marcus, it makes adaptive sense to have psycho-developmental strategies that do not depend on learning because these strategies make brain development more reliable and because learning is costly. According to the plasticity anti-nativists, it makes adaptive sense to use experience because learning is reliable in stable environments and because learning makes brain development more sensitive – and thereby more adaptable – to local conditions (Quartz and Sejnowski 2002). In our view, both these arguments appeal to untested intuitions. There will be cases and cases. We need detailed models that will help us assess the costs and benefits of the

two developmental strategies for different brain structures and for different environmental contexts.[5]

Neo-nativism and learning

In the previous two sections, we saw that, in his attempt to redefine and strengthen the nativist position, Marcus makes two moves. The first move is to abandon old-fashioned genetic determinism in favour of the new way of conceiving genetic activity in terms of cascades. The second move is to abandon the hardwiring view of psychological development in favour of the prewiring view of psychological development. We also saw that the first move, while entirely appropriate for anyone who has kept up to date with the advances in molecular and developmental biology, does not provide any comfort to those who want to argue that the nativism/anti-nativism debate is a fruitful approach to the study of biological systems. On the contrary, the new theories of genetic activity seem to give support to the view the nativism/anti-nativism dispute is wrong-headed and futile. In contrast, the debate about the importance for brain development of psycho-developmental strategies that do not depend on learning seems to be a substantive debate.

This suggests that one good way of understanding the nativism/anti-nativism debate in the context of current scientific knowledge is as a debate about prewiring. Old-fashioned nativists believed in genetic determination and hardwiring. In contrast, *neo-nativists* believe that there is a lot of prewiring and that prewired structures often (in normal developmental circumstances) remain intact. And their opponents, the *neo-anti nativists*, believe that there is little prewiring and that almost all prewired structures get rewired (even in normal developmental circumstances). When framed in this way, being a neo-nativist becomes a matter of degree: the more one believes that prewiring is important, the more neo-nativist one is. Moreover, one may be a neo-nativist concerning some brain structures (e.g. sub-cortical structures) but not others (e.g. cortical structures).

This way of redefining the nativism/anti-nativism debate depends on the notion of prewiring, which in effect serves as a successor for the concept of innateness. But when exactly does a trait count as innate/prewired? One obvious thought is that a trait is prewired if and only if *learning does not contribute to its development*. However, there are problems with this definition. For a start, let us suppose that there is a brain area A1 that implements a particular cognitive structure. It turns out that the development of A1 depends in part on receiving some structural support from a nearby brain area A2, which develops earlier than Al. Moreover, the development of A2 is heavily affected by learning. So, without learning, A2 does not grow and this causes A1 to lack the structural support it needs to develop properly. The provision of structural support is the only contribution that learning makes to the development of A1. Given that

[5] Bateson (2004a); West-Eberhard (2003); Sterelny (2003); Laland et al. (1996).

learning plays such an indirect role in the development of A1, do we want to say that A1 is not prewired? Marcus's discussion suggests that he does not. He would count A1 as prewired. This strikes us as the right thing to say. But then more needs to be said about what makes something prewired/innate, since A1 does not develop independently of learning. One way to get around this problem is to distinguish between mere causes and explanatory causes. Learning contributes *causally* to the development of A1 by providing structural support. But the internal features and complexity of A1 are not *explained* by learning and experience. Given this, the suggested definition of prewiring might be refined as follows: a trait is prewired if and only if learning is irrelevant to *explaining the internal complexity of the trait*.

A different problem with the style of defining prewired/innate is that learning is a theoretically controversial notion. Learning is variously regarded as hypothesis testing, parameter setting, a kind of symbolic manipulation, conditioning (classical or operant), synaptic pruning, any change in synaptic strengths due to perceptual processing, etc. The relations between these views are far from clear. But in many cases a trait counts as learned on one of these accounts and as not learned on another. For example, Quartz and Sejnowski (1997) argue convincingly that many neural structures count as not learned on the view that learning is hypothesis testing and at the same time count as learned on the view that learning is a change in neural structure due to perceptual processing.[6] Which notion of learning should one adopt in defining prewiring? What kind of process must be irrelevant to explain the complexity of a mental structure in order for that mental structure to be prewired?

Marcus's use of the wiring metaphor suggests to us that, for the purposes of defining prewiring, learning should be understood in a relatively liberal way as *any change in the way neurons are connected to each other that results from the impact on neural networks of electrical impulses generated by the interaction between the external environment and the sensory apparatuses*. The phrase 'change in neural networks' is intended to include cases in which new connections between neurons are *generated*, cases in which existing connections are *modified* (as in synaptic strengthening), cases in which existing connections are *eliminated* (as in synaptic pruning), cases in which some neurons (rather than just connections) are *destroyed* (as in neuronal death), and cases in which new neurons are *added* to the network (as described by Quartz and Sejnowski 1997). This definition has many advantages. The developmental changes caused by the impact of electrical impulses on brain structure seem to constitute a theoretically important class of changes, with properties that differ in important respects from developmental changes due to other causes. Due to the speed with which electrical impulses propagate and to the possibility of targeting these impulses in very precise ways, these changes provide the brain with dynamic properties that no other bodily organ possesses. Moreover, the definition is liberal enough to be acceptable, at least as an operational definition, by

[6] Cf. also Quartz (1999).

people who disagree about what the 'essence' of learning is, such as classicists who believe learning is symbol manipulation and connectionists who deny this.

This proposal about what counts as learning is liberal but not too liberal. It is important to emphasize that this view does not count all developmentally significant interactions between organism and external environment as learning. The term 'experience' is often used ambiguously. In some cases, the term refers to any kind of interaction between organism and environment ('due to the intervention of the experimenters, the *E. coli* experienced an environment with a high concentration of lactose and a low concentration of glucose'). In other cases, the term refers to a special kind of interaction between organism and environment, namely, those where: (1) the organism has a nervous system and a sensory apparatus, (2) the environment impacts on the nervous system of the organism by impacting on the sensory apparatus, (3) the environment impacts on the sensory apparatus of the organism in such a way that electrical impulses are 'passed' from peripheral neurons to more internal neurons. Only this restricted class of interactions falls under our definition of learning. We believe that this definition captures the way Marcus talks about learning in this book.[7] Moreover, we believe that this definition is theoretically motivated, since the special features of neurons and of their electrical activity mean that interactions belonging to the restricted class have many important features in common, whereas the larger class includes interactions due to all sorts of mechanisms and so has far fewer important features in common.

Neo-nativism and genes

With these clarifications about learning in hand, let us now go back to the idea that a mental trait is innate/prewired if and only if learning is irrelevant for explaining the internal complexity of the trait. This proposal faces an obvious objection. Consider someone who becomes sociopathic as a result of an accident that causes brain damage to its prefrontal cortex.[8] In this case, learning is irrelevant to explaining the internal complexity of the newly acquired mental trait. So, on the suggested definition, acquired sociopathy should count as innate/prewired. But intuitively acquired sociopathy is definitely not innate.

A natural response is to add a second clause to the definition of innateness. This clause says that a trait is innate only if it results from normal development. Acquired sociopathy results from accident or disease and, as such, is not the result of normal development. Hence, it should not count as innate/prewired despite the fact that learning does not explain its internal complexity.

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[7] See, for example, the discussion at the end of chapter 6. Marcus says that learning requires electric activity, but in those cases in which the electric activity originates endogenously (rather than from the interaction between the sensory apparatus and the external environment) the electric activity does not count as learning.

[8] Cf. Damasio (1994).

The same clause would yield the right non-innate classification for many other abnormally acquired mental structures.

The proposal is then to reformulate the nativism/anti-nativism dispute in terms of the following definition: a mental T is innate/prewired if and only if (1) *learning is irrelevant for explaining the internal complexity of T and* (2) *T results from normal development*.[9] But this second requirement is fiat less simple than it looks. For a start, note that sociopathy as a general *type* is not always due to abnormal development: some people 'mature naturally' into psychopaths, without the aid of any traumatic events, and presumably one would want to count their sociopathy as indeed innate/prewired. The natural move here is to say that cognitive structure C is innate/prewired in individual X if and only if (1) *learning doesn't explain C's complexity and* (2) *C results from normal development in all individuals with the same genome as* X. But problems remain. Presumably 'normal development' is what happens in a 'normal environment'. However, 'normal environment' cannot just mean *currently statistically normal*, otherwise some C – a high degree of euphoria, say – could *become* innate/prewired just because a drug that produces C – Prozac – became culturally universal. Perhaps the way to deal with this would be to equate 'normal environment' with the 'environment of evolutionary adaptation', that is, to specify that C is only innate/prewired if C would develop in the environment in which C was selected as an evolutionary adaptation. But then what do we do about Cs which are not biological adaptations, and so do not have an 'EEA', but which should count as innate, such as some psychological traits due to genetic disorders?

Another possibility is to appeal to the role of genes in development. Thus we might try adopting the following definition: trait T is innate/prewired if and only if (1) *learning is irrelevant for explaining the internal complexity of T and* (2) *T is genetically specified*. Brain damage due to an accident clearly is not in any sense genetically specified, so acquired sociopathy does not count as innate/prewired on this new definition. Marcus, like most biologists, is happy to talk about genes specifying phenotypic structures, and he seems to view this as relevant to nativist controversies. However, note that this definition removes any need for the first clause about the explanatory irrelevance of learning. Presumably it will be agreed on all sides that a trait is not genetically specified if learning is needed to explain its internal complexity. So genetic specification is sufficient for the irrelevance of learning, with the result that the conjunction of clauses (1) and (2) is equivalent to (2) alone. The proposed definition collapses into the claim that a trait is innate/prewired if and only if it is genetically specified.[10]

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[9] This reformulation is similar to that proposed by Samuels (1998, 2002, 2004). Cf. also Cowie (1999) and Khalidi (2002).

[10] It is interesting to note that both Chomsky (1993) and Fodor (1981, 2001) believe that the notion of innateness that is useful in cognitive science will eventually be reduced to some notion of genetic specification, but they never consider the problems that this reduction may pose.

A first problem with this definition is that it is incredibly difficult to make sense of the notion of genetic specification. Genetic specification is not genetic determination. As said in section 2, both genes and environments are necessary for the development of all phenotypic traits. Because of this, many biologists nowadays think of genetic specification in terms of genes carrying *information* about phenotypic outcomes. Both genes and environments are needed, but – in the cases of innate traits – only genes carry the information required for the development of the phenotype. Innate phenotypes are genetically encoded. But the idea that genes encode phenotypes (rather than just mRNA products and proteins) is deeply problematic. Developmental systems theorists have launched a strong attack against the idea that genetic causes are distinguished from environmental causes by bearing some kind of informational content: any good sense that can be made of the notion of content in this context would seem to allow for contentful environmental causes as much as genetic ones.[11]

A definition of genetic specification that does not appeal to the notion of genetic information is thereby required. One possibility is to say that a trait is genetically specified if and only if *its internal complexity is explained by genetic factors rather than environmental factors*. Note that the requirement is that the trait's internal complexity must be *explained* (rather than merely caused) by genetic factors. As pointed out above, nothing is *caused* entirely by genes without environmental help. Still, many biologists think that genes have a privileged *explanatory* role. In fact, it is probably this that they have in mind with talk of 'genetic information': such talk is most usefully viewed as a metaphor that allows biologists to convey the message that genes and not environmental factors are what matters when it comes to explaining the complexity of 'innate' traits. It is unlikely that this proposal will work. If the view presented in section 2 is correct, all phenotypes are the result of cascades of gene–environment interactions. Hence, all satisfactory explanations of phenotypic development will have to mention both genetic factors and non-genetic factors.

One might reply that, despite the fact that both kinds of factors must be mentioned, in some cases genetic factors play a *more important* explanatory role. But in order for this proposal to work one needs a way of deciding what counts as 'more important' and what counts as 'less important' in this context. Intuitions are unlikely to be enough, given that they are so easily swayed by considerations that have nothing to do with objective features of developmental interactions. For example, some will emphasize genetic over environmental factors simply because they have analytic tools that make it easier to identify genetic than environmental factors. Or they may emphasize genetic factors because they know that the popular press and funding agencies like gene-talk. Or they may have other political agendas. Analogous biases may of course lead others to

[11] Cf. Gray (1992, 2001); Griffiths and Gray (1994, 1997, 2001); Griffiths (2001, forthcoming); Oyama (2000a, 2000b); Godfrey-Smith (1999, 2000). For replies and discussion see Sterelny et al. (1996); Kitcher (2001); Maynard Smith (2000); Sterelny (2000, 2001, 2004); Stegmann (2004); Jablonka (2002).

downplay genes and emphasize the explanatory importance of environmental factors. In order to vindicate a comparative notion of genetic specification, we need some objective metric for measuring and comparing the explanatory importance of genetic and environmental factors. Given the complex nature of their interactions, we doubt that such a metric will ever be constructed.

There is a yet further reason for thinking that a notion of innateness/prewiring based on 'genetic specification' is not going to help to make sense of Marcus's commitment to 'nativism'. Genetic specification applies to a far wider range of biological traits than neural structures, and moreover requires that internal complexity not be explained by environmental causes of any kind, not just that it not be explained by learning. Given this, it seems unlikely that an understanding of prewiring/innateness in terms of genetic specification is going to cast any useful light on the debate between Marcus and his 'anti-nativist' opponent about brain development. That debate was specifically about the relevance of *learning* to the development of *neural structures*. There is no obvious rationale for presenting this as a special case of a far more general debate about the extent to which environmental conditions of all kinds play a role in shaping the structure of biological traits. By defining innateness/prewiring in terms of genetic specification, we are in danger of losing sight of the specifics of this debate.

Conclusions

The question of whether or not neurobiological learning influences the development of some brain structure is perfectly intelligible, without posing it as a matter of 'innate' brain structures. As we have seen, it is by no means obvious that any coherent notion of innateness can be extracted from the debate. Substantial obstacles face any attempt to define innateness either in terms of learning or in terms of genetic specification. The obvious solution is simply to ditch the notion of innateness, and stop thinking of the debate as between nativism and anti-nativism. Given that all the interesting issues can be raised without using any notion of innateness, why insist on trying to characterize the issues in this way?

The only possible motivation for hanging onto some notion of innateness would be a desire to relate commonsense views of 'nature' and 'nurture' to the new discoveries in developmental biology and psychology. This desire is bound to remain unsatisfied. There is too large a gap between the commonsense notions and current knowledge of brain development. Far from helping us to refine the innate/acquired distinction, the new developmental discoveries show that the distinction has no coherent core.[12]

In conclusion, it is interesting to note that, after having spent the whole book defending nativism and trying to formulate what we have called a 'neo-nativist' position, in the last chapter – with a sudden twist – Marcus concludes that,

[12] Cf. Bateson (1991, 2000, 2001, 2004b); Griffiths (1997, 2002).

after all, the best thing to do is to give up the distinction between nature and nurture. Other authors have recently made a similar move: they have denounced the flaws of the nature/nurture distinction while at the same time hanging onto some kind of neo-nativist position.[13] But if the nature/nurture distinction really is theoretically inadequate, and given that (as we have argued in this paper) no satisfactory reformulation can be found, we should all agree to forget about 'nativism vs. anti-nativism'. All we really need are theories (note the plural) that accommodate the complexities of the many different kinds of developmental interactions that exist. Old-fashioned nativism is dead, and neo-nativism should be dead too.

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[13] Cf. Pinker (1998, 2002, 2004); Ridley (2002); Tooby and Cosmides (1992).

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