about 10–15% of cases of horrific trauma, such as rape or combat battle. Moreover, it is often delayed in its onset. These observations have led McGaugh and other neuroscientists to speculate that perhaps a post-trauma drug could be given to trauma victims that would block the action of stress hormones, which might attenuate (or even prevent) the development of PTSD. Early work reveals the promise in this original idea. As a result, ethical issues have already risen to public discussion. Should we be giving people pills to purposefully dampen painful, unwanted memories? Critics of this approach worry that such dimming or erasing of painful memories might disconnect people from who they really are. But others have wanted such a ‘cure’ for people who are racked by painful memories. And they have wanted the cure for hundreds of years if we draw a liberal inference from Shakespeare’s Macbeth. It is there that a doctor is urged to treat Lady Macbeth and rid her of painful memories of the past:

‘Canst thou not minister to a mind diseas’d, Pluck from the memory a rooted sorrow, Raze out the written troubles of the brain …with some sweet oblivious antidote…’

If Shakespeare could read Memory and Emotion, he’d be smiling at his own foresight.

References

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Blueprints, Swiss Army knives, and other metaphors

The Birth of the Mind: How a Tiny Number of Genes Creates the Complexities of Human Thought, by Gary Marcus, Basic Books (Perseus) 2004. $26.00 (278 pp.) ISBN 0 465 04405 0

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In 1975, a set of experiments was performed that has impacted many debates concerning the differences between humans and our closest primate relatives. The experiments were carried out by Mary-Claire King, working in the laboratory of Allan Wilson at Berkeley. The methodology involved determining the temperature at which two pieces of single-stranded DNA, one sample from a human and the other from a chimpanzee, would ‘melt’ or separate from each other. The greater the similarity between chains, the tighter the chemical fit between them and the higher the temperature required for the separation. In this case, the high temperature that was required suggested that over 98% of the human and chimpanzee genomes were identical [1].

The relevance of this finding is intuitive and immediate: if humans and chimpanzees are genetically 98% the same, how are we to reconcile the seemingly vast cognitive differences between the two species – for example, the human species’ ability to produce language, art, technology, and so forth – with the remaining mere two percent of the genome? In his new book, The Birth of the Mind, Gary Marcus quickly dispels the blueprint metaphor that leads many non-biologists astray when thinking about genes. The problem with this metaphor is that, unlike a genome, a blueprint contains a one-to-one mapping between the elements on the plan and in the finished product; an alteration of an architectural blueprint creates an equivalent amount of change in the constructed building.

This difficulty we face in understanding how subtle differences between genomes are amplified into dramatic phenotypic differences between species recurs in the form of the ‘gene shortage’ problem, which Marcus attributes to biologist Paul Ehrlich [2]. The shortage refers to the ratios between the size of the genome (in humans, by current estimates, some 30 000 genes) and the number of neurons in the brain (some 100 billion or 1011), with even higher orders of magnitude for the number of synapses between neurons, which give rise to our mental representations. An empiricist argument here might be: how could such elaborate representations be determined by such a small set of genes? The appeal of such a rhetorical device also stems from the same implicit blueprint metaphor that might lead one to expect a less dramatic ratio between the number of genes and the number of neuronal connections.

The simple answer to both of these shortages (either in terms of the absolute number of genes or the differences between genomes) is to realize that genotype–phenotype relationships are exponential. If we consider any number of developmental disorders that have been studied genetically, such as Williams Syndrome or the speech and language disorder exhibited by the KE family, it becomes immediately apparent that changes in a handful of genes, or even a single gene, can lead to exponentially larger changes in phenotype. The question then is one of how the genome ‘unpacks’ to orchestrate human development, in particular the development of the human brain. Marcus is quite up to the job of reviewing the current knowledge on
the subject, and in doing so integrates a large body of work across genetics, developmental biology, cognitive psychology, neuroscience and linguistics. As a cognitive scientist, Marcus writes particularly appropriately for that audience. Those with heavier biological training will probably find the bulk of the material familiar, but the level of integration across fields should still leave most readers with some novel insights and new questions.

First off, Marcus provides us with a more accurate (and more cogsci-friendly) alternative to the blueprint metaphor to help us imagine the genome in action: the ‘if-then’ statements of a computer program. The aptness of this metaphor can be seen in work on what are known as regulatory proteins – proteins that control the degree of expression of other genes and the proteins they encode, many of which are themselves also regulatory proteins. Such protein if-then statements are what allow a researcher to ramp up expression of a single master control gene in, for example, the antennae of a fruit fly and modulate the activity of hundreds of other genes to result in an extra eye [3]. Similar embedded cascades of gene expression underlie the differences between a normally developing child and one with a genetic developmental disorder, and even the differences between closely related species like humans and chimps, whose genomes differ by seemingly insignificant proportions.

For this reader, the more engaging sections of the book are the instances where Marcus takes a stand on some of the current conceptual challenges facing cognitive psychology as it tries to integrate sensibly the increasingly available genetic data into theories of human cognitive organization. I would like to examine three conceptual issues that, in particular, beg reconsideration in light of a small, data-compressed genome.

The first of these issues concerns the distinction between the developmental process and the mental representations that result from that process, particularly with regard to the issue of domain-specificity. Marcus touches on this point in a discussion that contrasts the logic of association and dissociation following brain lesions (which are disruptions of previously developed representations) and the logic of association and dissociation in developmental disorders (which are disruptions of the developmental process itself). Marcus suggests that although the failure to find neuropsychological dissociations might provide evidence against domain-specific representations, the failure to find developmental dissociations probably does not. This is because the loss of a gene affects a multitude of genetic cascades in disparate areas of the brain, and can result in the disruption of developmental processes that in normal development would have culminated in domain-specific representations. It seems here that Marcus is envisioning a brain that develops domain-specific representations (e.g. for language), but he breaks from more hard-core nativist accounts by allowing the genetic constraints involved not to be themselves domain-specific. If this is true, Marcus may be close to meeting the ‘anti-modularists’ – whom he often pits himself against – halfway; Annette Karmiloff-Smith, along with her colleagues Jeffrey Elman and Elizabeth Bates, has long advocated a view of development in which the domain-specificity of developmental processes and that of their products, mental representations, are dissociated in a very similar manner [4,5].

The second issue where there seems to be some convergence concerns the realization that the expression of a gene is not likely to be specific to one representation, one cortical region, or even the nervous system. Together with Simon Fisher, Marcus nicely illustrated this in a recent and much needed review of the FOXP2 gene, the aberrant gene in the speech-disordered KE family [6]. This issue is relevant because on first impression one might expect that more modular views of brain organization would require some degree of genetic specificity. Marcus points to a 1998 article by Karmiloff-Smith [7] that he feels is an example of genetic naïveté of this kind. I think that Karmiloff-Smith’s article is worth a second read, because it is not only more sophisticated than Marcus gives credit for, but also reflects a view of brain development that is actually strikingly similar to Marcus’s own. Both authors describe ‘unique-marker’ genes as implausible. Both authors also describe a developmental process in which genetic constraints largely establish different cortical regions with, in Marcus’s words, the ‘subcomponents for computation, not complete systems for single-handedly solving complex cognitive tasks’ (p. 133, italics original). Thus, the two are in agreement to a surprising extent over the way in which the genome contributes to the establishment of mental representations, and both even allow some of these end representations to be specific to different cognitive domains. The real tension in this debate, it seems, stems from the ‘Swiss-Army-knife’ view of the brain, and what this metaphor implies, not only about human development but also human evolution.

This brings us to the third issue, which concerns the implications of small numbers of genes and comparative genetic differences for evolutionary psychology. The real problem of the ‘gene-shortage’ is not that it undermines the truism that brain development proceeds under genetic guidance, but it does however call into question the Swiss-Army-knife view of the mind that currently dominates evolutionary psychology [8]. According to this view, the brain does not merely end up with dozens of different domain-specific representations, but is believed to have been designed for acquiring those representations by dozens of selection pressures acting on specific, adaptive cognitive abilities. The small number of genes separating humans from the last common ancestor is highly suggestive of a scenario long advocated by Stephen Jay Gould and Richard Lewontin [9], which proposes that many of humanity’s unique cognitive abilities (or the capacity to develop them culturally) were not directly selected, but were instead consequences of other selection pressures. Given the strong data compression found in the genome, natural selection of a master control gene in response to one trait or behaviour might result in an ‘unintended’ reorganization of multiple other systems. Marcus himself hints at such a scenario with regard to the evolution of language: ‘But since…a single change can induce a new cascade or block an old one, phenotypic changes need not be gradual. A single mutation (or duplication) can have a large effect on the phenotype… if language arose by a
novel combination of existing elements – such as neural structures for memory, the automatization of repeated actions, and social cognition – it is possible that it could have developed relatively quickly.’ (p. 140). Marcus’s primary point in this passage concerned the pace of language evolution, but that scenario certainly also begs questions about what physical traits or behaviours drove it. Despite Marcus’s sophistication in critiquing the ‘gene for’ concept, my main disappointment with The Birth of the Mind was that Marcus was not so cautious in his use of the Swiss-Army-knife view of the brain, with its very explicit ‘evolved for’ connotation. Perhaps the Swiss Army knife is like the blueprint when it comes to human development and evolution: a metaphor in need of replacement.

References

Corrigendum
Corrigendum: Shared representations between self and other: a social cognitive neuroscience view

In the article by J. Decety and J.A. Somerville, published in the December 2003 issue of TICS, we wish to correct the following with respect to two of the illustrations.

In Box 2 on p. 529, Figure I is not adapted from reference [39] as stated, but is from an unpublished study that contrasted ‘social emotion’ conditions with neutral conditions. The appropriate figure to illustrate subjective perspective-taking can be found in reference [38].

Also, in Figure 1 on p. 530, two activations, one in the lateral view and one in mid-sagittal, marked as yellow hexagons in the frontal cortex (anterior) as being ‘imagining action’ from reference [38], were not in fact found in that study. An activation at this location in medial prefrontal cortex was found in reference [39] in a conceptual perspective-taking task. Reference [38] found an activation in the frontopolar cortex (Brodmann area 10).

We apologise to readers for this incorrect information.

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