

known to respond to contextual stimuli, although the kinds of stimuli that act in this role have not been well established. Those that have been identified so far seem to define a spatiotemporal backdrop against which events can be set. The modulation of place cells by more than simply 'place' extends even to humans, in which hippocampal cells have also been found to be responsive to factors such as the subject's goal, or view, as well as current location [1]. The present studies suggest that the journey that a rat is currently making can define a context too, and can be used to allow association of a particular journey with contextually related events.

The distinguishing factor between the above two possibilities will be whether time turns out to play a special role in place cell encoding, or whether it is just another context cue. In fact, modulation of place fields by the rat's journey might be more complex than it appears at first, because another study, using a broadly similar alternating task on a Y-maze, has failed to find such 'trajectory encoding' [9]. This puzzling discrepancy suggests a sensitivity of the phenomenon to as-yet-unidentified factors which might turn out to provide important constraints on how we conceptualize the information encoded by place cells.

The future...

What next? It would be interesting to know, first, whether prospective and retrospective coding can co-occur not only in the same journey, but at the same moment in time. One way of doing this would be to see how place cells fire on a double-alternation version of the Wood *et al.* task (left-left-right-right) so that, for example, an about-to-turn-right run might also be distinguished by where the rat has just come from. It would also be interesting to find out how many steps in a

behavioural sequence the cells can encode before the discrimination breaks down; in other words, how fine-grained is the sequencing? Does the granularity correlate with the ability of rats to remember behavioural sequences? And finally, we need to know whether the 'space' and the 'time' influences on these cells can be dissociated. In other words, is temporally modulated place encoding an example of a broader capacity of the hippocampus to encode sequences of events, or is space in some way fundamental to the activity of 'place' cells – and, by extension, to the encoding of episodic memory?

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doi:10.1016/j.tics.2004.03.006

Book Reviews

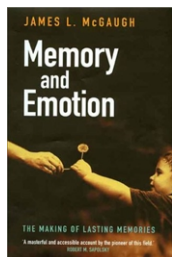
Strong memories are made of this

Memory and Emotion: The Making of Lasting Memories, by James L. McGaugh (2003) (192 pp.) Weidenfeld & Nicolson, £16.99 ISBN 0 29764 593 5, Columbia University Press, \$24.50 ISBN 0 23112 022 2

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'All memories are not created equal.' So writes James McGaugh in his charming and lucid book *Memory and Emotion*. It is more than deserving of the praise lavished upon it by Stanford neurobiologist, Robert Sapolsky, who has called the book 'A masterful and accessible account by the pioneer of this field'. What the book is really about is how experiences activate

hormone and brain processes that serve to create strong memories. Strong memories can be created by repetition, but they can also be created when emotional arousal is part of the experience. Once made strong, they can be lasting

and relatively immune to distortion. Relatively immune, perhaps, but we would stress that even lasting memories are prone to distortion – a point we will return to later.

McGaugh's field – the neurobiology of learning and memory – has a fairly recent history, much of which he recounts in the early chapters of the book. It is a history that is far shorter than that of other sciences such as biology, chemistry, physics and astronomy. It is also a history a fair bit of which McGaugh personally lived through, and thus his readers are treated to a story that comes alive through his eyes of contemporaneous experience. McGaugh's journey to becoming that pioneer and now one of the world's most pre-eminent neuroscientists began in earnest when he entered graduate school in Psychology at Berkeley in 1953. He didn't just *read about*

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the works of his significant academic ancestors, such as David Krech and Edward Tolman; he walked the halls with them and listened to their ideas take shape during weekly seminars. The science in his book is peppered with personal stories, such as the time he and fellow graduate students put on skits that made fun of the conceptual ideas of the day.

From there McGaugh would develop a long-lasting interest in the 'consolidation hypothesis'. This is the notion that things that happen after something is 'learned' can disrupt necessary continued processing of the learned material and thereby interfere with the consolidation of that material in memory. McGaugh was innovative in reasoning that if post-learning activities can sometimes disrupt consolidation and impair memory, perhaps other kinds of post-learning activities could enhance memory. Perhaps, he thought, administering stimulant drugs after learning would selectively boost the consolidation of new experiences, and thereby aid subsequent memory. He pitched this idea to his research advisors who were less than impressed: 'A bad idea...forget about it' was a rough translation of their reaction. But McGaugh persisted, and ultimately showed the memory-enhancing effects of stimulant drugs such as picrotoxin and strychnine given to non-human species after they had learned new material.

But we don't need to have external stimulants applied to our weak memories in order to enhance consolidation. A bit of emotional arousal will do the trick. That's why McGaugh can remember that he was at a scientific meeting in Portland, Oregon on November 22, 1963 when he heard the news that President Kennedy had been shot. That's why one of us remembers she had just flown from Los Angeles to New York, and was in a car traveling to Yale for the Harvard vs. Yale football game when she learned the news. (The other of us doesn't remember at all, as he wasn't born yet!) But for those of us who were over the age of eight at the time of Kennedy's assassination, the memories are there, part of our life story, and trotted out on many anniversaries of that tragic day. Something about that emotion and rehearsal makes these memories different. In McGaugh's words: 'Although most memories fade over time, traumatic memories may last a lifetime' (p. 124).

Or do they? As has been amply demonstrated, 'flashbulb' memories of highly emotional and personally meaningful events can seem special. Certainly they seem stronger. But in some other ways they behave like ordinary memories: they are prone to error and distortion [1]. In *Memory and Emotion*, McGaugh does a fine job of reviewing the evidence in support of flashbulb memories. He seamlessly interweaves the cognitive and social psychological work on this topic with the neurobiology underlying enduring memories. Through clear and concise discussion, he explains the roles of the hippocampus, amygdala and caudate nucleus, in addition to the stress hormones epinephrine, norepinephrine and cortisol in the formation and consolidation of enduring memories.

There seems to be compelling neurobiological evidence supporting the notion of highly durable memories. And this is the point that McGaugh wants his readers to

remember – some memories are stronger than others. By 'stronger', we can read 'lasting' (the subtitle of the book, after all, is *The Making of Lasting Memories*). For McGaugh, the more emotionally arousing the original event, the more lasting the memory. But even he has to concede that 'like memories of ordinary events, they [flashbulb memories] are neither completely accurate nor, in all cases, permanently strong' (p. 91). However, for McGaugh these imperfections arise mostly out of eventual forgetting, and the strength rather than the changeability of memory has been his emphasis.

Our own experimental research on the malleability of human memory leads us to a somewhat different emphasis regarding emotional memories. Although hundreds and perhaps thousands of experiments have been conducted on the malleability of memory more generally [2], relatively few have involved deliberate attempts to distort genuine traumatic memories. Despite the ethical difficulties of conducting such research, a few key studies have been done, most of them too new to be mentioned in *Memory and Emotion*. One study demonstrated the malleability of memory for a serious life-and-death situation. High-school students had attended an important football game at which a player on the field went into cardiac arrest. Paramedics tried to resuscitate the player and appeared to have failed. The audience reactions ranged from complete silence, to sobbing, to screaming, unaware then that the player would ultimately be revived at the hospital. Six years later, many of these students were interviewed, and their recollections contained numerous errors. Moreover, when exposed to misleading information about this traumatic event, many individuals incorporated the misinformation into their recollections. For example, more than 25% of the subjects were persuaded that they had seen blood on the player's jersey after receiving a false suggestion to this effect (J.J. Abhold, unpublished).

In more recent work, Russian subjects were led to believe falsely that they had seen a wounded animal in the aftermath of a terrorist attack on two Moscow apartment buildings that had been bombed several years earlier [3]. In yet another study, active duty military personnel underwent a rather traumatic survival-school training ordeal. A relatively short time later many of them made errors when they tried to remember the individual who was primarily responsible for their torment (Morgan, C.A. *et al.*, pers. commun.).

Thus, even traumatic events are susceptible to contamination. Put another way, whereas McGaugh suggests that strong memories might succumb to eventual forgetting, we believe that even 'lasting' or strong memories are susceptible to contamination by post-event misinformation. Moreover, it is not necessary to wait for the original memories to fade to find that strong memories are vulnerable to distortion.

Research on strong memories figure in McGaugh's thinking in another important way, one that has the potential for an enormously important practical application. Some traumas are so horrible that they lead some individuals to develop Post-Traumatic Stress Disorder (PTSD), a condition that is often accompanied by horribly intrusive memories of the trauma. PTSD seems to occur in

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.

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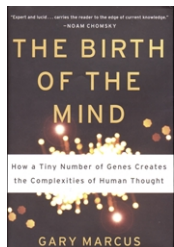
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doi:10.1016/j.tics.2004.03.005

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 10^{11}), 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

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