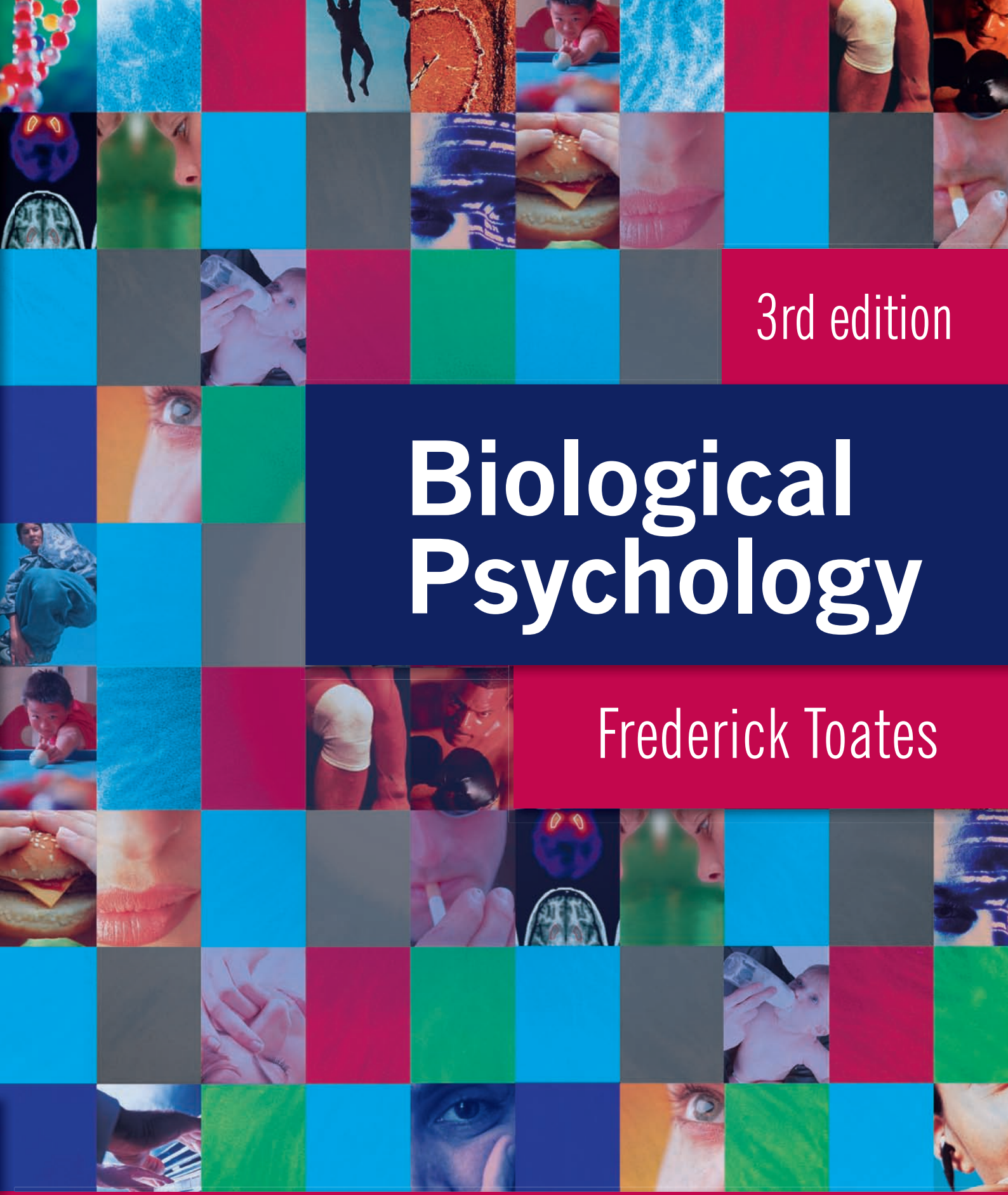

3rd edition

Biological Psychology

Frederick Toates

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Biological Psychology

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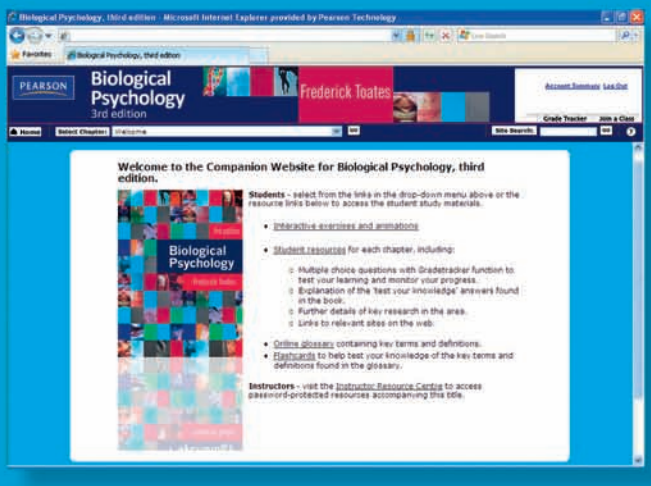

having a short lifespan, especially invertebrates, have little opportunity to learn by experience and tend to rely on closed programmes. In such animals, mating sometimes occurs only once and it is important to 'get the act together' on this occasion. In effect, instructions on what to do are inflexibly encoded on the basis of stimulus information.

An advantage of a closed programme is as an isolating mechanism, a way of eliminating mating with non-conspecifics. At best, such mating would waste time and, at worst, tie-up the reproductive process with a non-viable offspring. Mayr (1974, p. 657) summarizes it as: 'Selection should favour the evolution of a closed programme when there is a reliable relationship between a stimulus and only one correct response'.

For animals of a longer lifespan, there is often more opportunity to learn and more reliance on open programmes. An open programme is used where crucial information can be assimilated only on the basis of individual experience. Consider, for example, where an animal lives in a colony but specific parent–offspring interaction is needed (e.g. feeding the young). The programme can be closed only by the experience of the individual with its parent (Mayr, 1974). However, mate selection is still often relatively closed.

Section summary

1. Species differ in the dimension of precocial–altricial.
2. Programmes can be closed or open.

Test your knowledge

6.13 Which of the following would be described as the most precocial? (i) Pig, (ii) rat.

Answer on page 183

A personal angle

Imprinting on Konrad Lorenz

Normally, the stimulus on which a bird imprints would be a parent but it can be another species or even Konrad Lorenz. The programme is closed by the first exposure, such that the chick will later seek the imprinted stimulus as parent, companion or mate. The programme cannot then be reversed. In one case, as a result of their early exposure to him, chicks followed Lorenz as their object of choice (Figure 6.19).

Figure 6.19 The Nobel Prize-winning Austrian zoologist Konrad Lorenz (1903–1989) being followed by a group of ducklings.
Source: Science Photo Library.

Change and plasticity in adults

Introduction

Earlier sections described changes in the brain during development. This section considers plasticity in adults: which of the changes in Figure 6.5 also occur in the brains of adults? A caution is needed. Growth of new synapses in response to increased functional demands would surely fit the category of plasticity. However, not all changes in the adult brain would be described as 'plasticity'. The death of neurons shown in Figure 6.5(h) and (i) was described as being of functional significance in the developing brain. The neuron lost was not serving a functional role and was eliminated. By contrast, any large-scale indiscriminate loss of neurons in an aging brain and associated loss of function would not be serving a functional end. It would not be described as 'plasticity'.

The notion that the cortex of the adult brain exhibits plasticity is an old one, going back to the foundations of neuroscience in the 19th century and the Spanish researcher, Santiago Ramón y Cajal (DeFelipe, 2006). Cajal proposed the 'gymnastic hypothesis': links between neurons would change and multiply as a function of mental exercise (Mora *et al.*, 2007). The young were seen as having the greatest ability to exhibit such gymnastics, an ability that declines over years.

Recent evidence also points to changes in connections between neurons as the physical correlate of psychological changes, e.g. in stimulation from the environment

and learning etc. That is to say, some of the changes in Figure 6.5 also occur in adults: sprouting of dendrites and axons, particular cells die and new synapses are formed, while other synapses are lost. However, for a long time it was believed dogmatically that no new neurons are formed in the adult brain (see Gross, 2000). This assumption is false. First, we look broadly at plasticity in the adult brain. Then we consider specifically evidence for the formation of new neurons ('neurogenesis') in the adult brain and its significance.

Some examples of plasticity

Spatial cognition and brain plasticity

A study concerned the spatial abilities and brains of London taxi-drivers (Maguire *et al.*, 2000). They have to pass a formidable test on their knowledge of London streets and how best to get from A to B. From evidence on non-human species, the hippocampus has been implicated in spatial skills (Chapter 5). Could London taxi-drivers have developed particular biologically identifiable changes in the hippocampus, corresponding to the functional demands of their task? Using MRI, Maguire *et al.* found enlargement of the posterior hippocampus in taxi-drivers compared with controls.

This change might not represent plasticity but, rather, individuals with such enlargement and thereby good cognitive skills are attracted to taxi-driving. Maguire *et al.* suggest that this is not the case, since the magnitude of enlargement correlated with the length of time spent driving a taxi.

Plasticity and ageing

With age, there is a decline in the size of the human brain, which starts in the third decade (Colcombe *et al.*, 2006). It is most evident in particular regions: the temporal, parietal and frontal lobes. The decline in structure parallels a decline in cognitive ability. With an ageing population, this has profound health implications. So, what can be done?

Reasoning and problem-solving exercises and playing video games improve cognitive performance of older people (Lustig *et al.*, 2009). Neuroimaging techniques identify areas of change in structure and function associated with skill acquisition. This gives some idea where plasticity is exhibited in the adult brain. An increase in the size of a brain region and/or its increased activity suggests increased use of that region in the acquisition of a new skill. A lack of decrease relative to a control group would also draw investigators' attention. Lowering of activity in a region as a skill is acquired suggests a shift of responsibility away from the region, as when skills become more automatic and some 'higher' cognitive processing is thereby made free for other activities (Lustig *et al.*, 2009). Researchers then speculate on the nature of the changes at the level of neurons and other brain structures.

Aerobic exercise has a beneficial effect on both cognition and brain structure (Colcombe *et al.*, 2006). Work on non-humans shows that the effects of long-term exercise are mediated via increases in the levels of (i) neurotrophic factors in the brain, (ii) density of blood capillaries and (iii) the extent of dendritic connections between neurons, as well as the formation of new neurons in the hippocampus.

Colcombe *et al.* compared groups of participants aged 60–79 years: 'experimentals', who engaged in aerobic exercise for 6 months, and 'controls', who engaged in (non-aerobic) stretching exercises. The experimentals showed an increase in grey matter over the exercise period, most evidently in the frontal and temporal lobes. The frontal lobe is involved in executive functions (e.g. decision-making) and these normally show an age-related decline. In the experimentals, increases in volume were also seen in some white matter regions, e.g. the corpus callosum.

High levels of aerobic fitness help to preserve the integrity of the hippocampus (as measured by its volume) and the ability to acquire cognitive skills (Erickson *et al.*, 2009). Increased blood flow to the hippocampus appears to mediate at least some of the effects of aerobic fitness. The elevated blood flow might permit cells to survive and facilitate plasticity. Plasticity of the brain of 60-year-olds has been shown as they acquire a new skill: juggling (Boyke *et al.*, 2008). Increases in grey matter were evident in brain regions that would rather clearly be associated with the skill, e.g. the visual cortex (Figure 5.21). When practicing at the task stopped, the changes were reversed.

Pain

Repetitive stimulation with a pain-inducing stimulus leads to an expansion of grey matter in brain regions underlying pain processing, e.g. parts of the somatosensory cortex (Teutsch *et al.*, 2008) (Chapter 5). Cessation of stimulation is followed by a return to normal levels of grey matter. By contrast, chronic pain appears to cause a reduction in size of a number of brain regions functionally related to pain processing (May, 2008; Rodriguez-Raecke *et al.*, 2009). Some are involved in counteracting pain (Chapter 14), which might have clinical implications. Whether the changes represent death of neurons, shrinkage of neuron size or reduced connections within the cortex remains open to investigation. Where surgical intervention was able to cure the pain, there was a return of the size of the brain regions to normal (Rodriguez-Raecke *et al.*, 2009).

Cognitive behaviour therapy

People with chronic fatigue syndrome (CFS) show reductions in grey matter volume (GMV) in regions of the prefrontal cortex, detected by MRI (de Lange *et al.*, 2008). Could the reduced brain volume be a predisposing risk for the disorder or be a consequence of it? One way of addressing this is to see whether the reductions can be reversed with therapeutic interventions, such as cognitive behaviour therapy (CBT). CBT consisted of guiding the patient in a gradual increase in activity, while challenging unhelpful cognitions.

Figure 6.20 shows the baseline conditions, where a lower GMV is evident in CFS. Also evident in both CFS (triangles) and controls (squares) is the decline in GMV with age. Health, physical activity and cognitive speed were improved by CBT. Figure 6.21 shows the change in GMV after CBT, as well as a break-down as a function of age. The change in GMV as a result of CBT is evident in younger rather than older patients.

It is interesting to compare the decline in volume of regions of prefrontal cortex with CFS and traumatic damage to these regions. The effect of the latter is often one of apathy. This has similarities to the problems faced in CFS and it might be more than coincidental that the success of CBT correlated with the recovery of grey matter volume (de Lange *et al.*, 2008).

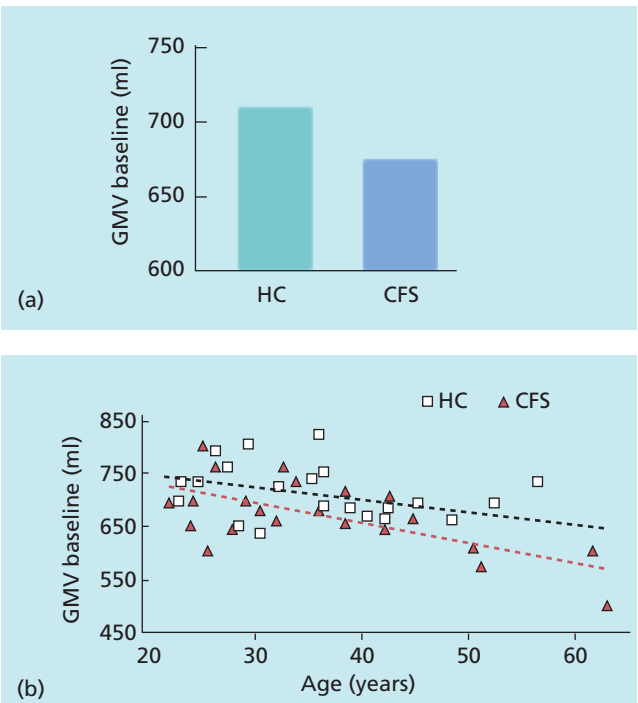

Figure 6.20 Grey matter volume (GMV) of people with CFS, compared to healthy controls (HC) at the start of the experiment (before CBT). (a) Comparison of the two samples; (b) GMV as a function of age.

Source: de Lange *et al.* (2008, Fig. 1, p. 2175).

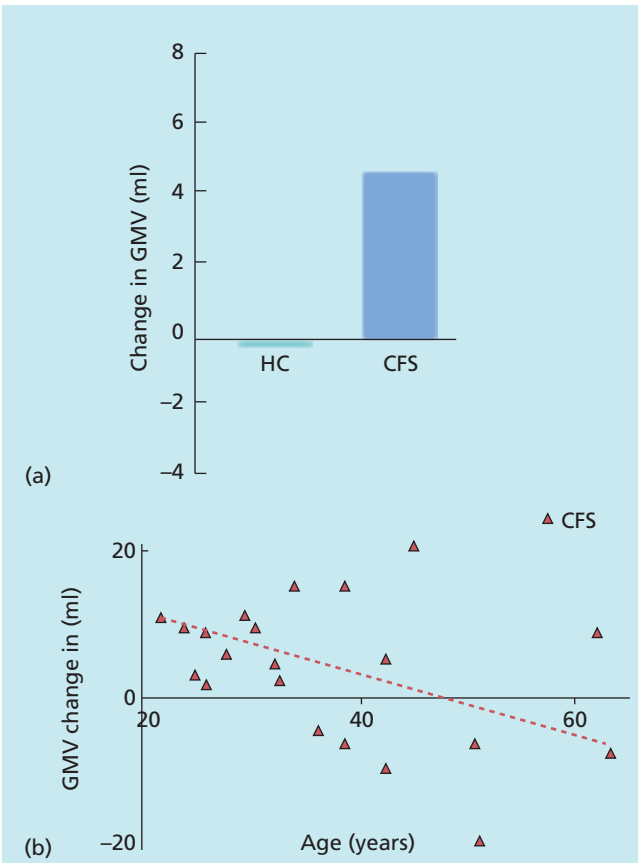

Figure 6.21 Change in GMV. (a) CFS patients compared with healthy controls (HC). (b) Change in GMV as a function of the age.

Source: de Lange *et al.* (2008, Fig. 2, p. 2176).

Stroke

A trigger for reorganization within the adult brain is loss of tissue as in stroke. A stroke occurs when neurons in part of the brain are denied their supply of blood, as in blockage of a blood vessel or rupture of its wall. Neuronal death follows. When the stroke is in motor regions, there will be disruption to movement, e.g. of an arm. However, there is usually at least some recovery from stroke and lost abilities return albeit not to their former extent. Consider where neurons in the motor cortex of the brain are lost (Dancause *et al.*, 2005). The functional connections that other neurons make to the motor neurons are also lost, which can be the trigger for sprouting until other target neurons are found.

So, for some time there has been an acceptance of the existence of plasticity in the adult brain, in the form of changed connections between an existing population of neurons. This leads to the controversial issue of whether an additional contribution to brain plasticity arises from the birth of new neurons (Gross, 2000), described next.

The issue of neurogenesis

Background

Old dogmas die hard and this was the case with the dogma that there is no neurogenesis in the adult brain. It is not hard to appreciate reasons for the dogma. Early neuroanatomists had reasoned (Gross, 2009, p. 327): 'Because the elaborate architecture of the brain remained constant in appearance, the idea that neurons were continually added to it was, understandably, inconceivable'.

A personal angle

A prophet before his time

In the 1960s, Joseph Altman employed what is termed a 'cell birth marker', a substance known as ^{3}H-thymidine (Altman, 1962). This was injected into the brains of adult rats and it labelled cells at the time of their division (Figure 6.5(b)). Altman then followed the fate of the 'daughter cells' that also carry the label (Figure 6.5(c)–(e)). Cells carrying the label were subsequently found in various brain regions (e.g. cortex), implying that their birth was in the adult animal. Despite publication in the prestigious journal *Science*, researchers ignored or dismissed the result (Gross, 2009). Why? The techniques available did not permit the newborn cells to be unambiguously classified as neurons. Researchers puzzled – how could the adult brain repeat the developmental feat of migration of cells from their site of origin to their final location, as in Figure 6.5(c)–(e) (see Nottebohm, 2002). Altman was relatively unknown and the result did not fit the dogma of the times (Gross, 2009). Now such cells have been shown to have the form and characteristics of neurons and Altman takes his well-deserved place in the scientific literature.

Following Altman's study, a number of pieces of evidence emerged pointing to the existence of adult neurogenesis (Gross, 2000).

Song learning in birds

Nottebohm (2002) studied song learning in birds, identifying its neural basis. Two nuclei (collections of neurons) attracted interest, since they showed enormous fluctuations in size over time, e.g. with season and following injection of testosterone. At first, Nottebohm felt that the increase in size of a nucleus following administration of testosterone might be due to just increased growth of dendrites or to the formation of new synapses, as in Figure 6.5(h). Later he asked whether the production of new neurons also contributes to the enlargement. A labelling technique revealed the addition of thousands of new neurons daily and these neurons were assimilated into functioning circuits.

Nottebohm also studied a species of bird, the Black-capped chickadee, which stores ('caches') hundreds of items of food in different locations, an awe-inspiring feat of spatial cognition. This behaviour occurs as the days shorten and the temperature falls, a time at which incorporation of new neurons into the hippocampus is at its maximum. The new neurons only survive for a few weeks, which coincides with the length of time between caching an item of food and its retrieval.

Rodents

Refined techniques confirm the existence of neurogenesis in the adult rat hippocampus (Nilsson *et al.*, 1999) and in the olfactory bulb (discussed by Gross, 2009). Furthermore, the rate of neurogenesis in the hippocampus increases as the environment is made more complex and, in parallel with this, learning ability also increases. This suggests that adult neurogenesis is not simply some functionless vestige, a 'left-over' from early evolution or development. An enlarged hippocampus could have functional significance in facilitating the animal's coping with environmental complexity (Gross, 2000).

In mice, increased locomotor activity is associated with increased sprouting of new blood vessels and thereby increased blood flow to regions of the hippocampus where neurogenesis occurs (Pereira *et al.*, 2007). Regional cerebral blood volume correlates with neurogenesis.

Primates

In adult macaque monkeys, newborn cells appear to migrate from their birth-place near to the lateral ventricle, through white matter to various regions of the cortex (Gould *et al.*, 1999). The regions, such as temporal cortex, have a role in learning and memory. In humans, neurogenesis occurs within regions of the hippocampus (Eriksson *et al.*, 1998). Aerobic exercise increases the sprouting of new blood vessels in these regions, a possible measure of neurogenesis (Pereira *et al.*, 2007).

Broad reflections

Could adult neurogenesis have evolved as a means of repair of the brain, e.g. to counter natural 'wear-and-tear'? Nottebohm rejects this suggestion on the grounds that it appears to be selective to particular brain regions, which are not obviously the most vulnerable to damage.

Rather, he suggests (p. 746) that: 'it evolved to keep circuits functionally young, able to master skills in the way that young brains do …'.

In an argument compatible with this, others (Balu and Lucki, 2009; Gross, 2000) suggest that adult neurogenesis might be important for learning (Chapter 11), drawing upon several sources of evidence:

1 The phenomenon is most evident in brain structures most closely involved with learning and memory, e.g. the hippocampus.
2 Factors that decrease neurogenesis, such as stress, also impair learning on tasks that involve the hippocampus.
3 Presenting learning tasks that involve the hippocampus improves the survival of newborn neurons.
4 Factors that increase neurogenesis in the hippocampus (e.g. increasing environmental complexity) enhance learning.
5 Neurons formed when the animal is adult appear to be more plastic than older neurons, which could make them particularly suitable as a basis of learning. For example, they show a high capacity to sprout extensions.

There could be important health implications of adult neurogenesis. There are indications that the efficacy of anti-depressive interventions (e.g. medication, physical exercise) could lie, in part, in increasing neurogenesis in the hippocampus (Balu and Lucki, 2009).

Section summary

1 Some of the changes that characterize early development can occur to a limited extent in adults.
2 Adult experience influences plasticity of neural connections and in some cases neurogenesis.

Test your knowledge

6.14 At a cellular level what could contribute to an enlarged hippocampus as a result of experience?

Answer on page 183

Bringing things together

The development of behaviour reflects developing neural processes and, in turn, development of neural processes depends on behaviour. In turn, the environment is influenced by the behaviour of the developing animal.

Given the complexity of interactions that determine development, you might wonder whether the role of any factor can ever be understood. Rather than despair, complexity can be the stimulus for experimentation and theory. For example, a role of a hormone can be established but to do so can involve considering both the nervous system and the dynamics of interaction between two animals. Given such tortuously complex dynamic interactions underlying development, you might also have cause to wonder how a viable animal ever emerges. Even more so, how is sufficient consistency of form among conspecifics maintained that they are able to recognize each other as potential sexual partners and produce offspring? Awe seems an appropriate reaction, as is, in more down-to-earth terms, a consideration of the stabilizing effects of environmental consistencies.

Moving on from development to consider adult systems, the kinds of change in the nervous system that underlie development (e.g. synaptic restructuring) can also be seen in some cases in adult nervous systems.

We asked, how do we distinguish between development and learning? With newer findings, the distinction becomes even more blurred (Elman *et al.*, 1996). The plasticity of the adult nervous system contributes to the blur since we can no longer assume an absolute distinction between early (development) and later changes (learning). The old distinctions between experience-independent (development or maturation) and experience-dependent (learning) are now suspect. Elman *et al.* suggest that we might risk calling the early series of changes consisting of cell division, migration, etc., 'maturation' as distinct from learning. Life seemed simpler before but there is no going back now, so we have to live with complexity and try to better understand it.

See the video coverage for this chapter to appreciate where a study of brain development informs the psychology of development.

Summary of Chapter 6

1. Development depends upon several layers of interacting factors.
2. Development of the nervous system consists of the net production of an increasing number of cells, accompanied by an increasing degree of complexity of these cells and their interconnections.
3. The changes in connections between cells associated with development have some similarities with more limited changes ('plasticity') in the adult system. In each case, changes depend upon what role the system of connected cells plays.
4. Hormones exert both organizational effects and activational effects on the nervous system and thereby behaviour. Sex hormones exemplify these roles.
5. The emergence of cognitive and social skills is associated with identifiable features of the development of the nervous system.
6. Atypical development can be contrasted with typical development and links made between developmental outcome, genes and environment.
7. Differences in functional demands posed on different species can be linked to differences in the development of their nervous systems.
8. Limited plasticity is evident in adult brains.

Further reading

For biological aspects, see Gazzaniga *et al*. (2008). For links between brain development and cognition, see Goswami (2008). For poverty, malnutrition and brain development, see Lipina and Colombo (2009). For the dynamics of interaction, see McCartney and Phillips (2008).

Answers

Explanations for answers to 'test your knowledge' questions can be found on the website www.pearsoned.co.uk/toates

6.1 (ii) Phenotype
6.2 (ii) The bottom
6.3 Myelination
6.4 Zygote (or fertilized egg cell); genotypes; phenotypes
6.5 (i) An increase in the number of postsynaptic receptors; (iii) an increase in the number of presynaptic vesicles
6.6 (iii) Cholinergic
6.7 (ii) Contralateral
6.8 Axon
6.9 Membrane
6.10 (i) Organizational; (ii) activational
6.11 (ii) Axon terminals
6.12 The larger the number of receptors, the higher the inhibitory feedback and the lower the excitatory input to the adrenal gland.
6.13 (i) Pig
6.14 (i) Formation (or enlargement) of new synapses or dendrites; (ii) neurogenesis.

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