Motivation

5

CHAPTER OUTLINE

Learning Objectives

By the end of this chapter you should appreciate that:

- central motivational states (such as hunger, thirst and interpersonal attraction) underlie human behaviour (feeding, drinking and sexual activity);

- these motivational states energize people to work for goals and avoid punishment;

- particular brain mechanisms are involved in linking motivation and emotion;

- there are a number of possible factors underlying obesity;

- behaviour can be described in sociobiological terms;

- links have been made between genetics, evolution and sexual reproduction.

INTRODUCTION

What motivates us to work for food when we are hungry, or water when we are thirsty? How do these motivational control systems ensure that we eat approximately the right amount of food to maintain our body weight, or drink enough to quench our thirst? And how do we explain overeating and obesity?

In this chapter, we will consider the nature of motivation and how it is controlled, focusing on the biological basis of food and fluid intake. There are two reasons for choosing these motivational behaviours:

1. There is considerable evidence about how the brain processes the relevant signals controlling food intake.
2. These are crucial survival functions. Undereating and drinking too little result in loss of body mass, impaired metabolism and – in extreme cases – starvation, dehydration and death, whereas overeating and obesity lead to significant health risks (see chapter 19). Understanding the control of food intake is therefore important.

In the last part of the chapter, we will introduce some of the biological and neural underpinnings of another type of motivated behaviour – sexual behaviour.

But first some definitions. A *reward* is something an animal will work to obtain or achieve, whereas a *punisher* is something it will work to escape or avoid. In order to exclude simple reflex-like behaviour, we use the term 'work' to refer to a voluntary behaviour (see chapter 4), also called an *operant response*. Examples are pressing a lever in a Skinner box in order to obtain a reward or avoid a punishment, putting money in a vending machine to obtain food, or

reward something for which an animal will work

punisher something an animal will work to escape or avoid

operant response (or instrumental response) an arbitrary response or behaviour performed in order to obtain a reward or escape from or avoid a punishment

removing your hand when lighting a candle to avoid singed fingers.

So motivated behaviour is when an animal (either human or non-human) performs an operant response to obtain a reward or avoid a punishment. This definition implies that learned responses are important in demonstrating motivated behaviour, and this is certainly true of two types of learning – classical conditioning and instrumental learning (see chapter 4).

Motivation also has close links with emotions, since these can be regarded as states elicited (in at least some species) by rewards and punishers (see chapter 6 and Rolls, 1999).

The material in this chapter will involve a degree of anatomical and physiological complexity, which you may find challenging. But remember that the terms used here are essentially labels created by earlier investigators. It is the principles concerned that are most important.

HUNGER AND THE CONTROL OF FOOD INTAKE

To understand how the motivation to eat (and food intake) are controlled, we first need to consider the functions of peripheral factors (i.e. factors outside the brain), such as taste, smell and gastric distension, and control signals, such as the amount of glucose in the bloodstream. Then we can examine how the brain integrates these different signals, learns about which stimuli in the environment represent food, and initiates behaviour to obtain the correct variety and amount.

PERIPHERAL FACTORS

The functions of some peripheral factors in the control of eating can be demonstrated by the sham feeding preparation shown in figure 5.2. In this preparation, the animal tastes, smells and eats

Figure 5.1

The pleasant smell and taste of food give us immediate reward, a separate process from satiety – or the feeling of fullness. The brain brings the two processes together to control the amount of food we eat.
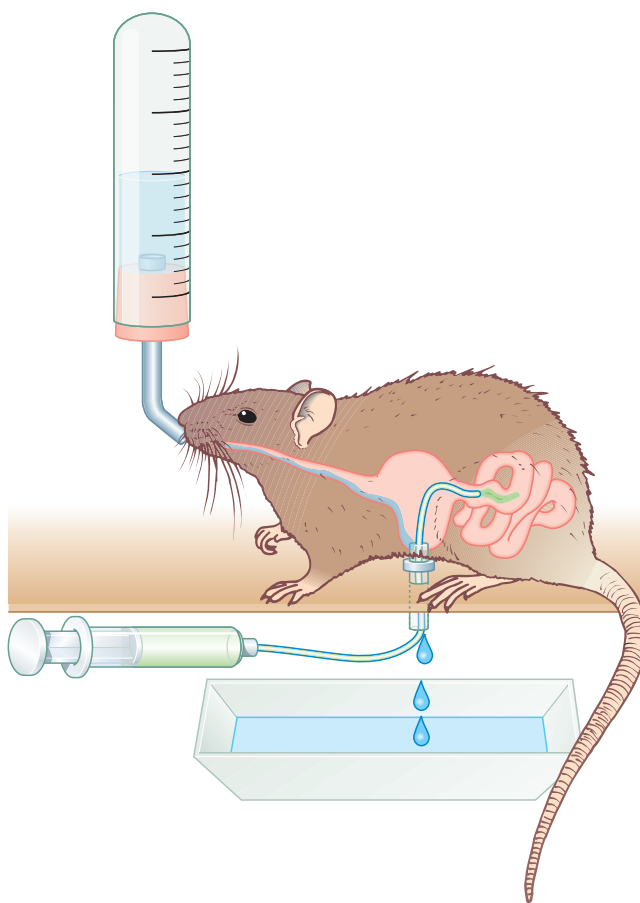

Figure 5.2

Sham feeding preparation. When food is drained from a rat's stomach it will often continue to eat for over an hour.

the food normally, but the food drains away from the stomach. This means that, although the animal consumes the food, the stomach does not become full, since the food does not enter the stomach or intestine.

Experiments have shown that rats, monkeys and humans will work for food when they are sham feeding (see Rolls, 1999), often

continuing to eat for more than an hour. This demonstrates that it is the taste and smell of food that provide the immediate reward for food-motivated behaviour. Further evidence for this is that humans are more likely to rate the taste and smell of food as being pleasant when they are hungry.

satiety reduction of appetite

oropharyngeal the oral cavity and pharynx

A second important aspect of sham feeding is that *satiety* (reduction of appetite) does not occur. From this we can conclude that taste, smell and even swallowing (i.e. *oropharyngeal* factors) do not of themselves make us feel satisfied, or satiated. Instead, satiety is produced by food accumulating in the stomach and entering the intestine.

Gastric distension is an important satiety signal, and intestinal signals also have a part to play (Gibbs et al., 1981). When an animal is allowed to eat to normal satiety and then has the food drained from its stomach, it starts eating again immediately. Moreover, small infusions of food into the duodenum (the first part of the intestine) decrease feeding, indicating satiety. Interestingly, however, animals have difficulty learning to perform a response that brings a reward of food if the food is delivered directly into the stomach, demonstrating that this form of feeding is not very rewarding in itself (see Rolls, 1999).

We can draw important conclusions about the control systems for motivated behaviour from these findings:

■ Reward and satiety are different processes.
■ Reward is produced by factors such as the taste and smell of food.
■ Satiety is produced by gastric, intestinal and other signals after the food is absorbed from the intestine.
■ Hunger and satiety signals modulate the reward value of food (i.e. the taste and smell of food are rewarding when hunger signals are present and satiety signals are not). To put this in more general psychological terms, in most behavioural situations the motivational state modulates or controls the reward or reinforcement value of sensory stimuli. So, for example, in certain species the female may apparently find the male of the species 'sexually attractive' only during certain phases of the female's reproductive cycle.
■ Since reward and satiety are produced by different bodily (i.e. peripheral) signals, one function of brain (i.e. central) processes in the control of feeding is to bring together the satiety and reward signals in such a way that satiety modulates the reward value of food.

CONTROL SIGNALS

The following descriptions of the different signals that control appetite are placed roughly in the order in which they are activated during a meal. All of these signals must be integrated by the brain.

1 Sensory-specific satiety If we eat as much of one food as we want, the pleasantness rating of its taste and smell change from very pleasant to neutral. But other foods may still taste and smell pleasant. So variety stimulates food intake.

For example, if you eat as much chicken as you want for a meal, the pleasantness rating of the taste of chicken decreases to roughly neutral. Bananas, on the other hand, may remain pleasant, so you might eat them as a second course even when the chicken has already 'filled you up', or produced satiety. This type of satiety is partly specific to the sensory qualities of the food, including its taste, smell, texture and appearance, and has therefore been named sensory-specific satiety (see Rolls, 1999).

2 Gastric distension Normally gastric distension is one of the signals necessary for satiety. As we saw earlier, this is demonstrated when gastric drainage of food after a meal leads to immediate resumption of eating.

Gastric distension only builds up if the *pyloric sphincter* closes. The pyloric sphincter controls the emptying of the stomach into the next part of the gastrointestinal tract, the duodenum. The sphincter closes only when food reaches the duodenum, stimulating *chemosensors* and *osmosensors* to regulate the action of the sphincter, by both local neural circuits and hormones (see Gibbs et al., 1981).

pyloric sphincter controls the release of food from the stomach to the duodenum

chemosensors receptors for chemical signals such as glucose concentration

osmosensors receptors for osmotic signals

3 Duodenal chemosensors The duodenum contains receptors sensitive to the chemical composition of the food draining from the stomach. One set of receptors respond to glucose and can contribute to satiety via the vagus nerve, which carries signals to the brain. The vagus is known to represent the critical pathway because cutting this nerve (vagotomy) abolishes the satiating effects of glucose infusions into the duodenum. Fats infused into the duodenum can also produce satiety, but in this case the link to the brain may be hormonal rather than neural (a hormone is a blood-borne signal), since vagotomy does not abolish the satiating effect of fat infusions into the duodenum (see Greenberg, Smith & Gibbs, 1990; Mei, 1993).

4 Glucostatic hypothesis We eat in order to maintain *glucostasis* – that is, to keep our internal glucose level constant. Strictly, the crucial signal is the utilization of glucose by our body and brain, as measured by the difference between the arterial and the venous concentrations of glucose. If glucose utilization is low, indicating that the body is not able to extract much glucose from the blood stream, we feel hungry, whereas if utilization is high, we feel satiated. This is confirmed by the following findings:

glucostasis constancy of glucose availability (e.g. reflected in the glucose concentration in the plasma)
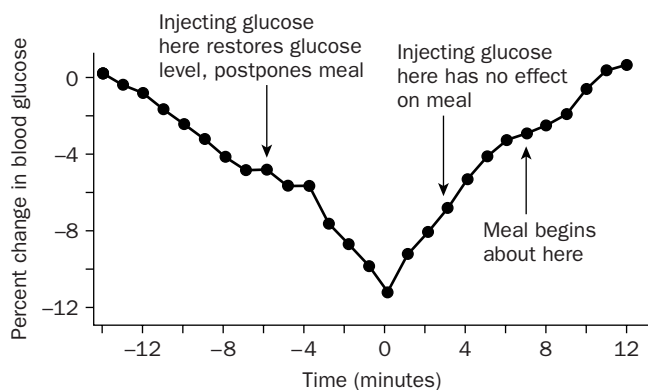
Figure 5.3

The fall in glucose concentration in the plasma typically seen in rats before a meal is initiated. Plasma glucose concentration is at its lowest at time 0, typically just before a meal starts. Source: Adapted from Campfield and Smith (1990).

- Rats show a small decrease in plasma glucose concentration just before meals, suggesting that decreased glucose concentration initiates eating (Campfield & Smith, 1990) (see figure 5.3). At the end of a meal, plasma glucose concentration rises, and so does insulin, which helps the glucose to be used by cells.
- Injections of insulin, which reduce the concentration of glucose in the plasma (by facilitating its entry to cells and storage as fat), provoke food intake.
- Infusions, or injections, of glucose and insulin (together enabling glucose to be taken up by the body's cells) can reduce feeding.
- The brain's monitoring system for glucose availability seems to be in the part of the brain called the medulla (part of the brainstem), because infusions there of a competitive inhibitor of glucose (5-thio-glucose) also provoke feeding (Levin et al., 2000).

5 Body fat regulation and the role of leptin The signals described so far help to regulate hunger from meal to meal, but they are not really adequate for the long-term regulation of body weight and, in particular, body fat. So the search has been on for scientists to identify another signal that might regulate appetite, based on, for example, the amount of fat in the body. Recent research has uncovered a hormone, leptin (also called OB protein), which performs this function (see Campfield et al., 1995).

6 Conditioned appetite and satiety If we eat food containing lots of energy (e.g. rich in fat) for a few days, we gradually eat less of it. If we eat food with little energy, we gradually, over days, ingest more of it. This regulation involves learning to associate the sight, taste, smell and texture of the food with the energy that is released from it in the hours after it is eaten.

This form of learning has been demonstrated by Booth (1985). Two groups of participants ate different flavoured sandwiches – one flavour being high energy sandwiches and the other being

low energy. On the critical test day, the participants chose to eat few of the sandwiches that tasted like the high energy ones eaten previously, but far more of the sandwiches that had the flavour of the previously consumed low energy sandwiches. And yet, on the test day, all the sandwiches consumed in fact had medium energy content. This suggests that the level of consumption of the medium energy sandwiches on the test day was strongly influenced by the energy content of the sandwiches that had been eaten previously.

HOW THE BRAIN CONTROLS EATING

Since the early twentieth century, we have known that damage to the base of the brain can influence food intake and body weight. One critical region is the ventromedial hypothalamus.

Bilateral lesions of this area (i.e. two-sided, damaging both the left and right) in animals leads to hyperphagia (overeating) and obesity (see Rolls, 1999). By contrast, Anand and Brobeck (1951) discovered that bilateral lesions (that is, damage) of the lateral hypothalamus can lead to a reduction in feeding and body weight. Evidence of this type led, in the 1950s and 1960s, to the view that food intake is controlled by two interacting 'centres' – a feeding centre in the lateral hypothalamus and a satiety centre in the ventromedial hypothalamus (see figure 5.4).

But problems arose with this dual centre hypothesis. Lesions of the ventromedial hypothalamus were found to act indirectly by increasing the secretion of insulin by the pancreas, which in turn reduces plasma glucose concentration, resulting in feeding. This has been demonstrated by cutting the vagus nerve, which
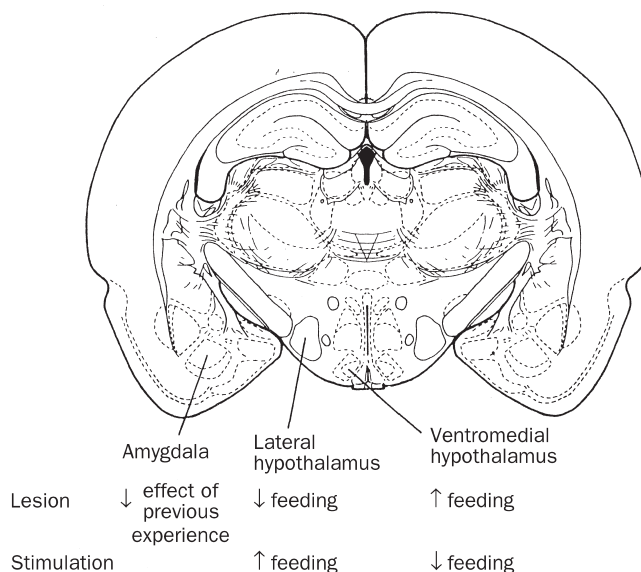

	Amygdala	Lateral hypothalamus	Ventromedial hypothalamus
Lesion	↓ effect of previous experience	↓ feeding	↑ feeding
Stimulation		↑ feeding	↓ feeding

Figure 5.4

Effects of lesions and stimulation of the lateral and ventromedial hypothalamus on eating. A coronal (transverse or vertical) section through the rat brain is shown.

Research close-up 1

Long-term regulation of body weight and fat

Research issue

Different satiety signals help to regulate hunger from meal to meal. But most of these signals are not really adequate for the long-term regulation of body weight and, in particular, body fat. So how do we regulate body weight and fat over the long term?

Recent research has uncovered a hormone, leptin (also called OB protein), which performs this function. Leptin is found in humans as well as in laboratory mammals such as mice.

Design and procedure

A variety of research methodologies have been used to address this issue, focusing on the evaluation of laboratory animals (mice), which have been especially bred to manifest a specific genetic profile.

Leptin is the hormone encoded (i.e. produced) by the mouse OB (obesity) gene. This gene comes in two forms: OB (the dominant form of the gene) and ob (the recessive form). It can be manipulated in different strains of mouse and the outcomes recorded in the laboratory. Further experimental manipulations include the administration of leptin to mice of different genetic strains.

Results and implications

- The possession of the OB gene appears to regulate whether or not obesity occurs in the mouse. More specifically, genetically obese mice that are *double recessive* for the obesity gene (i.e. ob ob mice), and thereby lack the OB gene, produce no leptin.
- Leptin administration decreases food intake in wild type (lean) mice (who have OBOB or OBob genes, so that they produce leptin) but also in obob mice. This finding shows that obob mice do have receptors sensitive to leptin, but they do not produce it spontaneously, because they lack the OB gene.
- The satiety effect of leptin can be produced by injections into the brain.
- However, leptin does not produce satiety (i.e. decrease food intake) in another type of genetically obese mouse (db db mice, which are double recessive for the diabetes gene). These mice may be obese because they lack the leptin receptor, or mechanisms associated with it, so that even when leptin is administered artificially it cannot produce satiety in these mice.
- Leptin fluctuates over 24 hours, but not in relation to individual meals. It might therefore be appropriate for the long-term regulation of appetite.

> **double recessive** the two copies of a gene in an animal are both recessive (i.e. non-dominant), as opposed to one copy being dominant (in which case the phenotype, or body characteristic, will be that of the dominant gene)

These and other related pieces of experimental evidence lead to the hypothesis that leptin represents one of the important signals in humans that controls how much food is eaten.

Campfield, L.A., Smith, F.J., Guisez, Y., Devos, R., & Burn, P., 1995, 'Recombinant mouse OB protein: Evidence for a peripheral signal linking adiposity and central neural networks', *Science*, 269, 546–9.

disconnects the brain from the pancreas, preventing ventromedial hypothalamic lesions from causing hypoglycaemia, and therefore preventing the consequent overeating.

So the ventromedial nucleus of the hypothalamus is now thought of as a region that can influence the secretion of insulin and, indirectly, affect body weight, but not as a satiety centre per se.

On the other hand, the hypothesis that damage to the lateral hypothalamus produces a lasting decrease in food intake and body weight has been corroborated by injecting focal neurotoxins (agents that kill brain cells in a very specific manner, such as ibotenic acid), into rats. These damage the local cell bodies of neurons but not the nerve fibres passing nearby. Rats with lateral hypothalamus lesions also fail to respond to experimental interventions that normally cause eating by reducing the availability of glucose (Clark et al., 1991).

A matter of taste

How are taste signals (which provide one of the most significant rewards for eating) processed through different stages in our brains, to produce (among other effects) activation of the lateral hypothalamic neurons described above (see Rolls, 1996, 1997, 1999)?

Research close-up 2

What happens in the brain when we see or taste food?

Research issue

What information relevant to food and feeding is mediated by the lateral hypothalamus? This question can be addressed by recording cellular activity while presenting food (sight and/or taste) to a laboratory animal.

Design and procedure

In these experiments, laboratory animals (typically monkeys) are anaesthetized and an electrode is surgically implanted into the lateral hypothalamus. This enables researchers to record cell response before and after the presentation of food.

It is also possible to electrically stimulate cells in the lateral hypothalamus, in a manner analogous to natural stimulation (such as might occur, for example, after the presentation of food), using surgically implanted electrodes.

Results and implications

- Some neurons in the lateral hypothalamus respond only to the sight of food (11.8 per cent), some respond to the taste of food (4.3 per cent), and some of these (2.5 per cent) respond to both the sight and taste of food (Rolls, Burton & Mora, 1980).
- The neurons only respond to the sight or taste of food if the monkey is hungry. This suggests that these lateral hypothalamic neurons represent information that is closely related to activity in the autonomic nervous systems (see chapter 3) because autonomic responses to food and eating behaviour only occur if hunger is present.
- If a lateral hypothalamic neuron has ceased to respond to a food on which the monkey has been fed to satiety, then the neuron may still respond to a different food (Rolls et al., 1986). This is reflected in the monkey's rejection of the food on which he has been fed to satiety, and acceptance of other foods.
- Hypothalamic neurons can learn to respond to the sight of a previously neutral stimulus – a container – from which the monkey has been fed orally. The neurons stop responding to the stimulus if it is no longer associated with food (Mora, Rolls & Burton, 1976; Wilson & Rolls, 1990). This type of learning underlies some forms of classical conditioning (see, for example, the classical studies of Pavlov reported in chapter 4).

Direct evidence of this type is essential for understanding how the brain works in representing rewards (see Rolls & Treves, 1998). Other evidence suggests that animals will work to obtain activation of these lateral hypothalamic neurons. It therefore seems likely that it is the stimulation of these brain regions by the consumption of food in the natural state that makes food psychologically rewarding (see Rolls, 1999).

Rolls, E.T., 1999, *The Brain and Emotion*, Oxford: Oxford University Press.

Some of the brain connections and pathways in the macaque monkey described in this chapter are shown in figure 5.5. The monkey is used to illustrate these pathways because neuronal activity in non-human primates is considered to be especially relevant to understanding brain function and its disorders in humans.

During the first few stages of taste processing (from the *rostral* part of the nucleus of the solitary tract, through the thalamus, to the primary taste cortex), representations of sweet, salty, sour, bitter and protein tastes are developed (protein represents a fifth taste, also referred to as 'umami'). The reward value or pleasantness of taste is not involved in the processing of the signal as yet, because the primary responses of these neurons are not influenced by whether the monkey is hungry or satiated. The organization of these first few stages of

> **rostral** towards the head or front end of an animal, as opposed to caudal (towards the tail)

processing therefore allows the primate to identify tastes independently of whether or not it is hungry.

In contrast, in the secondary cortical taste area (the *orbitofrontal cortex*), the responses of taste neurons to a food with which the monkey is fed to satiety decrease to zero (Rolls et al., 1989, 1990). In other words, there is modulation or regulation of taste responses in this taste-processing region of the brain. This modulation is also sensory-specific (see, for example, figure 5.6). So if the monkey had recently eaten a large number of bananas, then there would be a decreased response of neurons in this region of the orbitofrontal cortex to the taste of banana, but a lesser decrease in response to the taste of an orange or melon. This decreased responding in the
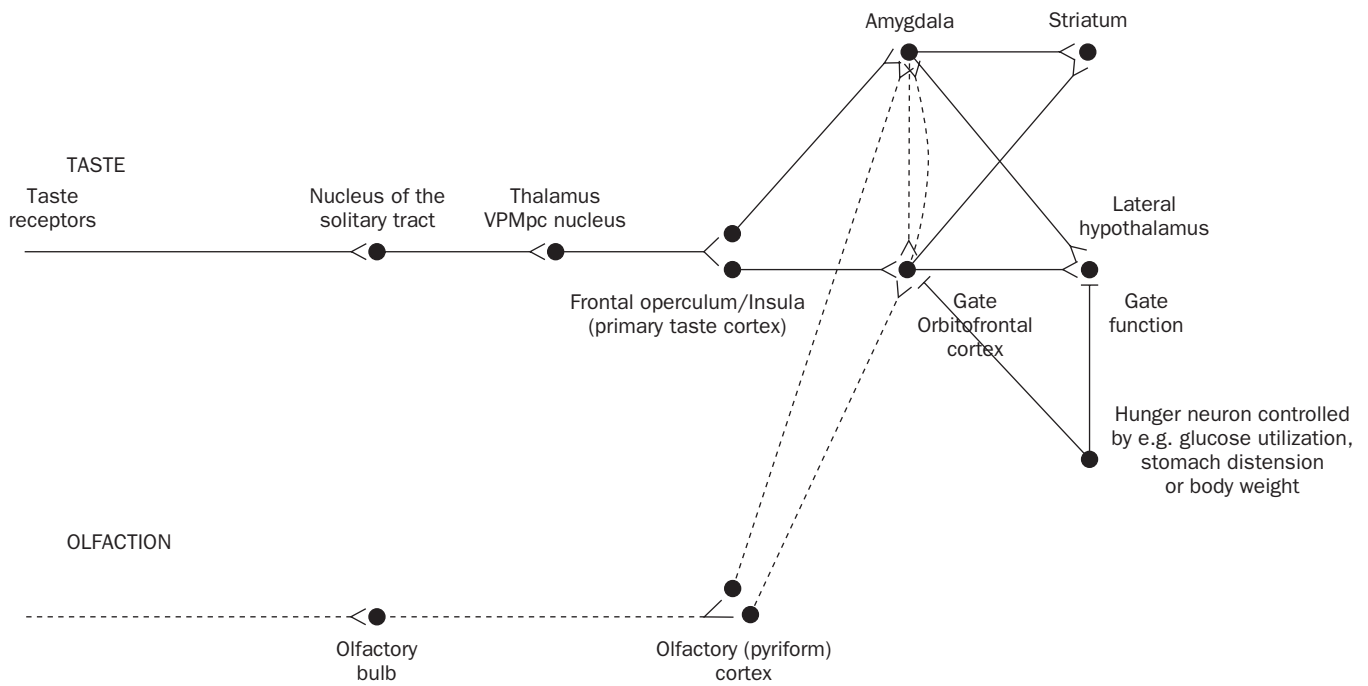
> **orbitofrontal cortex** above the orbits of the eyes, part of the prefrontal cortex, which is the part of the frontal lobes in front of the motor cortex and the premotor cortex

Figure 5.5

Schematic diagram showing some of the gustatory (taste) and olfactory pathways involved in processing sensory stimuli involved in the control of food intake. Areas of processing where hunger affects the neuronal responses to the sight, smell or taste of food are indicated by the gating or modulatory function of hunger.
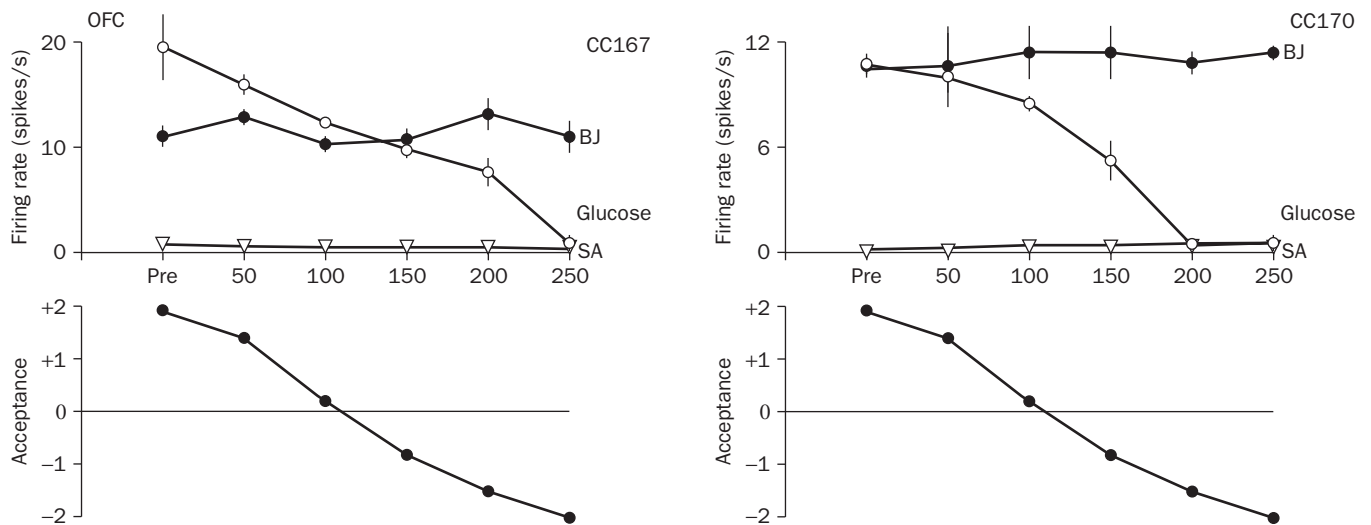
Figure 5.6

The effect of feeding to satiety with glucose solution on the responses of a neuron in the secondary taste cortex to the taste of glucose and of blackcurrant juice (*BJ*). The spontaneous firing rate is also indicated (*SA*). Below the neuronal response data for each experiment, the behavioural measure of the acceptance or rejection of the solution on a scale from +2 to −2 (see text) is shown. The solution used to feed to satiety was 20 per cent glucose. The monkey was fed 50 ml of the solution at each stage of the experiment as indicated along the abscissa (*x*-axis) until he was satiated, as shown by whether he accepted or rejected the solution.

Pre – the firing rate of the neuron before the satiety experiment started; *OFC* – orbitofrontal cortex; *CC167* and *CC170* – two different neurons. Source: Adapted from Rolls, Sienkiewicz and Yaxley (1989).

orbitofrontal cortex neurons would be associated with a reduced likelihood for the monkey to eat any more bananas (and, to a lesser degree, any more orange or melon) until the satiety had reduced.

So as satiety develops, neuronal activity in the secondary taste cortex appears to make food less acceptable and less pleasant – the monkey stops wanting to eat bananas. In addition, electrical stimulation in this area produces reward, which also decreases in value as satiety increases (Mora et al., 1979). It is possible that outputs from the orbitofrontal cortex subsequently influence behaviour via the connections of this region to the hypothalamus, where it may activate the feeding-related neurons described earlier.

TASTE + SMELL = FLAVOUR

Flavour refers to a combination of taste and smell. The connections of the taste and olfactory (smell) pathways in primates (see figure 5.5) suggest that the necessary convergence may also occur in the orbitofrontal cortex. Consistent with this, Rolls and Baylis (1994) showed that some neurons in the orbitofrontal cortex (10 per cent of those recorded) respond to both taste and olfactory inputs (see figure 5.7). Some of these neurons respond equally well to, for example, both a glucose taste and a fruit odour. Interestingly, others also respond to a visual stimulus representing, say, sweet fruit juice.

olfactory pathways smell pathways through the brain

This convergence of visual, taste and olfactory inputs produced by food could provide the neural mechanism by which the colour of food influences what we taste. For example, experimental participants reported that a red solution containing sucrose may have the flavour of a fruit juice such as strawberry, even when there was no strawberry flavour present; the same solution coloured green might subjectively taste of lime.

There is also another olfactory area in the orbitofrontal cortex (see figure 5.5). Some of these olfactory neurons respond to food only when the monkey is hungry, and so seem to represent the pleasantness or reward value of the smell of food. These neurons therefore function in a similar manner with respect to smell as the secondary taste neurons function with respect to taste.

The orbitofrontal cortex also contains neurons that respond to the texture of fat in the mouth. Some of these fat-responsive neurons also respond to taste and smell inputs, and thus provide another type of convergence that is part of the representation of the flavour of food. A good example of a food that is well represented by these neurons is chocolate, which has fat texture, sweet taste and chocolate smell components.

The orbitofrontal cortex

Neurons that respond to the sight of food do so by learning to associate a visual stimulus with its taste. Because the taste is a reinforcer, this process is called stimulus-reinforcement association learning. Damage to the orbitofrontal cortex impairs this type of learning by, for example, altering food preferences. We know this because monkeys with such damage select and eat substances they would normally reject, including meat and non-food objects (Baylis & Gaffan, 1991; Butter, McDonald & Snyder, 1969).

The functioning of this brain region could have critical implications for survival. In an evolutionary context, without this function of the orbitofrontal cortex, other animals might have consumed large quantities of poisonous foodstuffs and failed to learn which colours and smells signify nutritious foods.

The orbitofrontal cortex is therefore important not only in representing whether a taste is rewarding, and so whether eating should occur, but also in learning about which (visual and olfactory) stimuli are actually foods (Rolls, 1996, 1999, 2000c).

Because of its reward-decoding function, and because emotions can be understood as states produced by rewards and
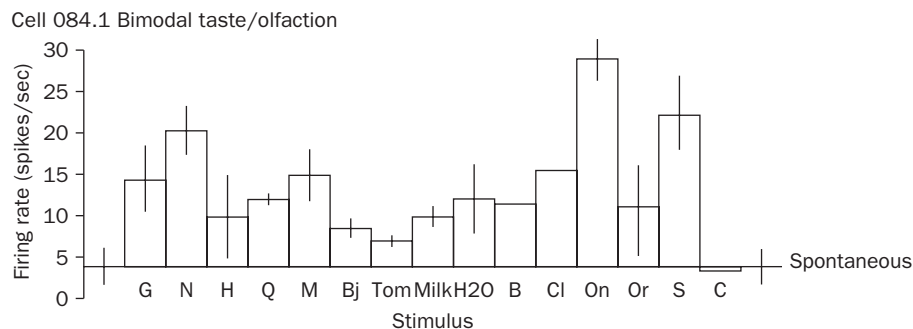

Cell 084.1 Bimodal taste/olfaction

Figure 5.7

The responses of a bimodal neuron recorded in the caudolateral orbitofrontal cortex. The neuron responded best to the tastes of NaCl and monosodium glutamate and to the odours of onion and salmon.

G – 1M glucose; *N* – 0.1M NaCl; *H* – 0.01M HCl; *Q* – 0.001M Quinine HCl; *M* – 0.1M monosodium glutamate; *Bj* – 20 per cent blackcurrant juice; *Tom* – tomato juice; *B* – banana odour; *Cl* – clove oil odour; *On* – onion odour; *Or* – orange odour; *S* – salmon odour; *C* – control no-odour presentation. The mean responses ± se (standard error of the mean) are shown. Source: Rolls & Baylis (1994).

in exchange for electrical stimulation of the amygdala. For example, they might be prepared to press a lever for a long period of time to receive amygdalar stimulation (via an electrode which has been implanted in their brain), implying that this stimulation is significantly rewarding. In addition, single neurons in the monkey amygdala have been shown to respond to taste, olfactory and visual stimuli (Rolls, 2000a).

But although the amygdala is similar in many ways to the orbitofrontal cortex, there is a difference in the speed of learning. When the pairing of two different visual stimuli with two different tastes (e.g. sweet and salt) is reversed, orbitofrontal cortex neurons can reverse the visual stimulus to which they respond in as little as one trial. In other words, neurons in the orbitofrontal cortex that previously 'fired' in response to a sweet taste can start responding to a salty taste, and neurons that previously 'fired' in response to a salty taste can start responding to a sweet taste, very quickly (see Rolls, 1996, 2000c). Neurons in the amygdala, on the other hand, are much slower to reverse their responses (Rolls, 2000a).

To explain this in an evolutionary context, reptiles, birds and all mammals possess an amygdala, but only primates show marked orbitofrontal cortex development (along with other parts of the frontal lobe). So the orbitofrontal cortex may be performing some of the functions of the amygdala but doing it better, or in a more 'advanced' way, since as a cortical region it is better adapted for learning, especially rapid learning and relearning or reversal (Rolls, 1996, 1999, 2000c).

The striatum and other parts of the basal ganglia

We have seen that the orbitofrontal cortex and amygdala are involved in decoding the stimuli that provide the rewards for feeding, and in connecting these signals to hunger / satiety signals. How do these brain regions further connect to behavioural systems?

One path is via the hypothalamus, which is involved in autonomic responses during feeding (such as the need for increased blood flow to the gut, to facilitate the assimilation of food into the body), and also in the rewarding aspects of food. Another route is via the striatum (one part of the basal ganglia, requiring dopamine to function – see chapter 3) and then on through the rest of the basal ganglia (see figure 5.5). This route is important as a behavioural output / feeding system, because disruption of striatal function results in aphagia (lack of eating) and adipsia (lack of drinking) in the context of a general akinesia (lack of voluntary movement) (Rolls, 1999; Rolls & Treves, 1998).

Neurons in the ventral striatum also respond to visual stimuli of emotional or motivational significance (i.e. associated with rewards or punishments; Williams et al., 1993), and to types of reward other than food, including drugs such as amphetamine (Everitt, 1997; Everitt & Robbins, 1992).

OBESITY – POSSIBLE FACTORS

With all of these brain functions promoting food regulation, why, then, is there such a high incidence of obesity in the world today?

Figure 5.8

There are neurons in the orbitofrontal cortex that respond to the texture of chocolate. Add its distinctive flavour (taste + smell) and you have an appealing combination.

punishers, the orbitofrontal cortex plays a very important role in emotion (see Rolls, 1999).

The amygdala

Many of the amygdala's connections are similar to those of the orbitofrontal cortex, and indeed it has many connections to the orbitofrontal cortex itself (see figure 5.5).

Bilateral damage to the temporal lobes of primates, including the amygdala, leads to the Kluver–Bucy syndrome, in which, for example, monkeys place non-food as well as food items in their mouths and fail to avoid noxious stimuli (Aggleton & Passingham, 1982; Baylis & Gaffan, 1991; Jones & Mishkin, 1972; Kluver & Bucy, 1939; Murray et al., 1996). Rats with lesions in the basolateral amygdala display similar altered food selections.

Given the neural connectivity between the orbitofrontal and amygdalar regions, we might relate these phenomena to the finding that lesions of the orbitofrontal region lead to a failure to correct inappropriate feeding responses.

Further evidence linking the amygdala to reinforcement mechanisms is illustrated when monkeys perform physical work

Many different factors can contribute to obesity, and there is only rarely a single cause (see Garrow, 1988). Occasionally, hormonal disturbances, such as hyperinsulinemia (that is, substantially elevated levels of insulin in the bloodstream), can produce overeating and obesity. Otherwise, there are a number of possible contributory factors:

- It is possible that the appetite of some obese people is more strongly stimulated by external factors such as the sight and smell of food (Schachter, 1971). The palatability of food is now much greater than it was in our evolutionary past, leading to an imbalance between the reward from *orosensory* control signals and the gastrointestinal and post-absorptive satiety signals controlling the reward value of sensory input. In other words, the rewards from the smell, taste and texture of food are far greater than the satiety signals can control.

  > **orosensory** the sensory systems concerned with the oral cavity, including taste, smell and the texture of what is in the mouth

- Animals evolved to ingest a variety of foods, and therefore nutrients. So satiety is partly specific to a food just eaten, while appetite remains for foods with a different flavour. Overeating may therefore be partially explained by the tremendous variety of modern foods, encouraging us to eat more by moving from one food to another.
- Modern humans take less exercise than our ancestors due to our more sedentary lifestyles, so unless regular exercise is proactively built into our daily lives, we may be inclined to gain weight.
- Human meal times tend to be fixed. Animals normally regulate their food intake by adjusting the inter-meal interval. A long interval occurs after a high energy meal, and a short interval after a low energy meal. Quite simple control mechanisms, such as slower gastric emptying (and therefore a feeling of fullness for a long time after an energy rich meal) may contribute to this. But the fixed meal times often preferred by humans deter this control mechanism from operating normally. Obese people tend to eat high energy meals and then eat again at the next scheduled mealtime, even though gastric emptying is not yet complete.
- Obese people often eat relatively late in the day, when large energy intake must be converted into fat and is less easily burned off by exercise and heat loss. Regulation of heat loss is one way that animals compensate for excessive energy intake. They do this by activating brown fat metabolism, which burns fat to produce heat. Although brown fat is barely present in humans, there is nevertheless a mechanism that, when activated by the sympathetic nervous system, enables metabolism to be increased or reduced in humans, depending on energy intake (see Garrow, 1988; Trayhurn, 1986).
- Obesity may be related to higher stress levels in contemporary society. Stress can regulate the sympathetic nervous system to increase energy expenditure, but at the same time it can also lead to overeating. Rats mildly stressed (e.g. with a paperclip on their tail) show overeating and obesity.

Figure 5.9

Obesity has many possible contributing factors and is rarely the result of a single cause.

THIRST AND THE CONTROL OF DRINKING

But what of water intake, and drinking? The human body can survive without food for very much longer than it can survive without water – how does our physiological make-up help direct this vital function?

Body water is contained within two main compartments, one inside the cells (intracellular) and the other outside (extracellular). Intracellular water accounts for approximately 40 per cent of total body weight; and extracellular water for about 20 per cent, divided between blood plasma (5 per cent) and interstitial fluid (15 per cent) (see figure 5.10).

When we are deprived of water, both the cellular and extracellular fluid compartments are significantly depleted. The depletion of the intracellular compartment is shown in figure 5.11 as cellular dehydration, and the depletion of the extracellular compartment is known as hypovolaemia (meaning that the volume of the extracellular compartment has decreased).

CELLULAR DEHYDRATION

When our bodies lose too much water, or we eat foods rich in salt, we feel thirsty, apparently because of cellular dehydration, leading to cell shrinkage. For instance, if concentrated sodium chloride solution is administered, this leads to withdrawal of water from the cells of the body by *osmosis*, and results in drinking.

Cellular dehydration is sensed centrally in the brain, rather than peripherally in the body. For instance, low doses of hypertonic sodium chloride (or sucrose) solution infused into the carotid arteries, which supply the brain, cause dogs to drink water, but similar infusions administered into peripheral regions of the body, which don't directly supply the brain, have no effect (Wood et al., 1977).

Everyday Psychology

Obesity: a disease of affluence?

Obesity is an extremely common disorder in our society. The prevalence of overweight or obese people in many Western countries is over 50 per cent and increasing. One estimate suggests that the average adult in developed countries has been adding one gram per day to body weight over the last decade. This has significant health consequences, and can cost these countries around 3 to 5 per cent of their total health budgets.

How can societies tackle the obesity problem? Major recent research investment has been made in seeking to understand the physiology, psychology and genetics of obesity. And many public education programmes publicize the value of healthy eating and exercise, although the effect of these programmes has been fairly minimal.

The National Health and Medical Research Council (NH&MRC) in Australia (a country that suffers from the same 'obesity epidemic' as other Western countries) commissioned a report, Acting on Australia's Weight, published in 1997. It suggested that the driving forces behind the increasing prevalence of obesity in recent decades are likely to be found in environmental changes inherent in modernizing societies. The report argued that a new model is needed to tackle the obesity epidemic, shifting the emphasis away from metabolic defects and genetic mutations.

A key concept within this framework is that obesity reflects a 'normal physiology within a pathological environment'. It is accepted that some people are genetically predisposed to obesity, but that the major role in most cases is played by psychosocial factors. To tackle this issue requires an environmental or lifestyle change.

Other relevant factors cited in the report are quantity and type of energy (food) intake and level of physical activity. The report argues that 'different combinations of genetic effect, food intake and energy output, modulated by psychosocial factors, interact in different populations, ethnic groups and families to produce overweight and obesity – no single or simple cause has been isolated'.

In industrialized countries, if we reflect on our everyday lives it becomes apparent that energy-dense foods are heavily promoted and readily available commercially. Also, labour-saving – and therefore physical activity reducing – devices are common both at home and in the workplace. The report argues that encouraging individuals to control their weight and providing them with information about how to do this is unlikely to be effective unless steps are taken to modify the environmental influences that underpin weight problems. For example, a programme that promotes low-fat foods is likely to meet with limited success unless there are enough low-fat products in supermarkets that are clearly labelled, placed near to full-fat alternatives and realistically priced.

Similarly, a strategy that encourages physical activity as part of a daily routine is unlikely to succeed if shops and workplaces are not within easy walking distance of people's homes, or if there are limited opportunities for physical activity outdoors.

The Australian NH&MRC document therefore proposes that public health strategies should be developed to promote environmental changes supporting healthy weight-control behaviours, including:

- encouraging schools to provide programmes that emphasize healthy dietary and physical activity behaviour in children; and
- increasing opportunities for people to be physically active in the community (for example, through better urban planning and the provision of exercise facilities at or near the workplace).

The report concludes that a major challenge for future research will be to identify precisely which environmental variables influence people's eating and exercise behaviour most effectively.

Commonwealth of Australia, 1997, Acting on Australia's Weight: A Strategic Plan for the Prevention of Overweight and Obesity: Summary Report.

The part of the brain that senses cellular dehydration appears to be near or in a region extending from the preoptic area through the hypothalamus.

EXTRACELLULAR THIRST STIMULI

Although the amount of fluid in the extracellular fluid (ECF) compartment is less than that in the cells, it is vital that the ECF be conserved to avoid debilitating changes in the volume and pressure of fluid in the blood vessels. The effects of extracellular fluid loss can include fainting, caused by insufficient blood reaching the brain. The behavioural response of drinking in response to hypovolaemia, a disorder consisting of a decrease in the volume of blood circulation, ensures that plasma volume does not fall to dangerously low levels.

There are a number of ways that ECF volume can be depleted experimentally, including haemorrhage, lowering sodium content in the diet, and excessive sweating, urine production or salivation. Two main thirst-inducing systems are activated by
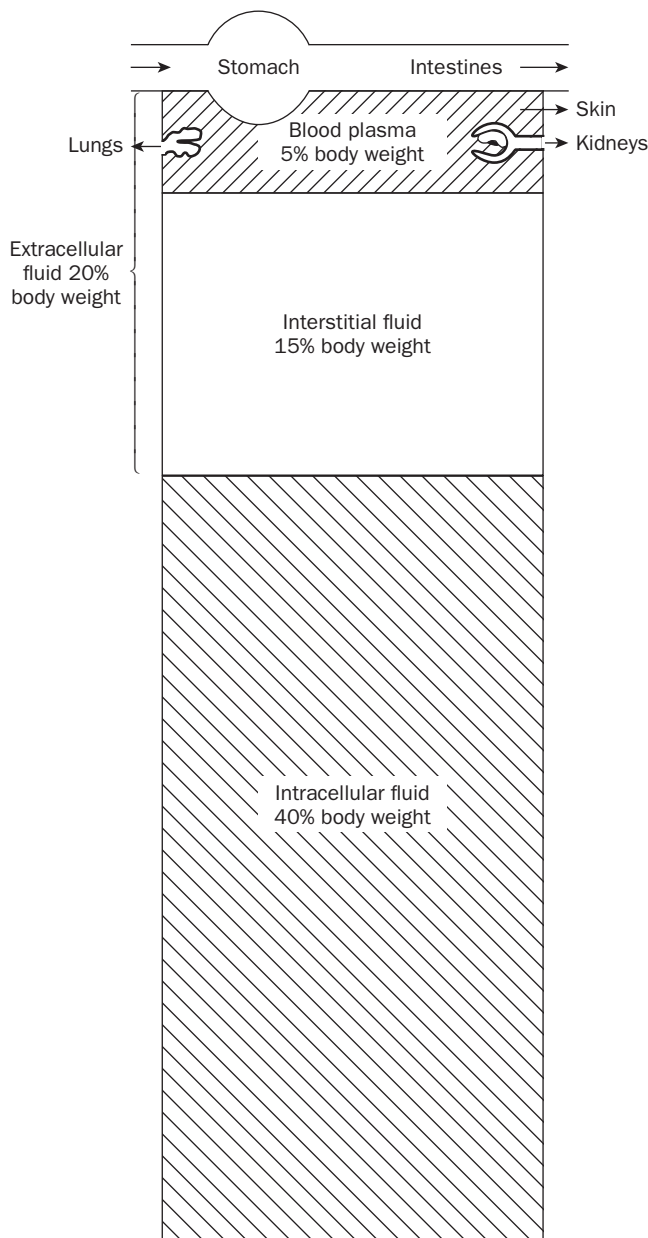
Figure 5.10

Body water compartments. Arrows represent fluid movement. Source: Adapted from Rolls and Rolls (1982).
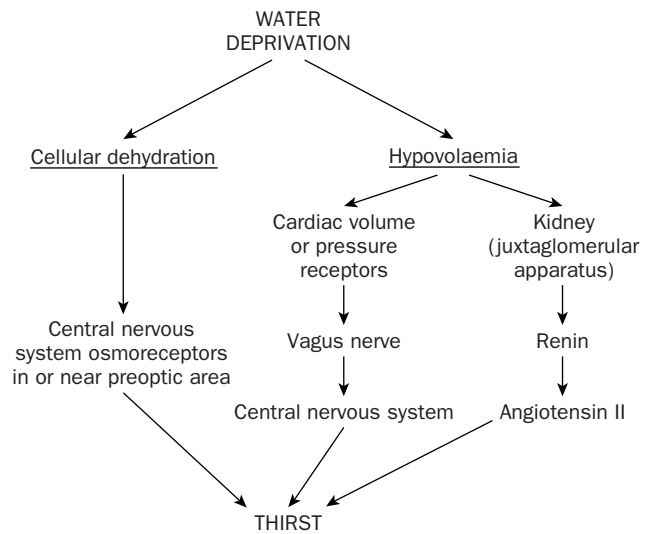

Figure 5.11

A summary of the factors that may lead to drinking after water deprivation. Source: Adapted from Rolls and Rolls (1982).

are located in the venous circulation around the heart, since the compliance (i.e. the ability to change diameter) of these vessels is high, making them responsive to changes in blood volume. Information from these receptors is probably carried to the central nervous system via the vagosympathetic nerves, from where information is relayed to the brain and drinking behaviour can be regulated.

CONTROL OF NORMAL DRINKING

In drinking caused by, for example, water deprivation, both the cellular and extracellular thirst systems are activated. Experiments show that, in many species, it is the depletion of the cellular, rather than the extracellular, thirst system that accounts for the greater part of the drinking, typically around 75 per cent (see Rolls & Rolls, 1982; Rolls, 1999).

It is important to note that we continue to drink fluids every day, even when our bodies aren't deprived of water. The changes in this type of thirst signal are smaller, partly because drinking has become conditioned to events such as eating foods that deplete body fluids, and also because humans have a wide range of palatable drinks, which stimulate the desire to drink even when we are not thirsty. (See earlier explanation of sensory-specific satiety, which means that we can drink much more if we are offered a variety of different drinks than if we were presented with only orange juice.)

SEXUAL BEHAVIOUR

Just as we need to eat to keep ourselves alive, working to obtain rewards such as food, so we need to have sex and reproduce in

hypovolaemia. One is the renin–angiotensin system mediated by the kidneys. When reductions in blood pressure or volume are sensed by the kidneys, the enzyme renin is released, leading to the production of the hormone angiotensin II which stimulates copious drinking

A second thirst-inducing system activated by hypovolaemia is implemented by receptors in the heart. For example, reducing the blood flow to the heart in dogs markedly increases water intake (Ramsay et al., 1975). It is still not clear precisely where such cardiac receptors are located. But it seems likely that they

order to keep our genes alive. In this part of the chapter, we will look at the following two questions:

How can a socio-biological approach (that is, an approach which seeks to reconcile our biological heritage as a species with our highly social organization) help us to understand the different mating and child-rearing practices of particular animal species? How does the human brain control sexual behaviour?

SOCIOBIOLOGY AND SEXUAL BEHAVIOUR

Sperm warfare

Monogamous primates (those with a single mate) living in scattered family units, such as some baboons, have small testes. Polygamous primates (those with many mates) living in groups, such as chimpanzees, have large testes and copulate frequently. This may be related to what sociobiologists call 'sperm warfare'. In order to pass his genes on to the next generation, a male in a polygamous society needs to increase his probability of fertilizing a female. The best way to do this is to copulate often and ejaculate a large quantity of sperm, increasing the chances that his sperm will reach the egg and fertilize it. So, in polygamous groups, the argument is that males have large testes to produce large numbers of sperm.

In monogamous societies, with less competition between sperm, the assumption is that the male just picks a good partner and produces only enough sperm to fertilize an egg without the need to compete with others' sperm. He also stays with his partner to bring up the offspring in which he has a genetic investment, and to guard them (Ridley, 1993).

What about humans? Despite widespread cultural pressure in favour of monogamy or restricted polygamy, humans are intermediate in testis size (and penis size) – bigger than might be expected for a monogamous species. But remember that although humans usually do pair, and are apparently monogamous, we also live in groups, or colonies. Perhaps we can find clues about human sexuality from other animals that are paired but also live in colonies.

A problem with this type of comparison, though, is that for most primates (and indeed most mammals) it is the female who makes the main parental investment – not only by producing the egg and carrying the foetus, but also by feeding the baby until it becomes independent. In these species, the male apparently does not have to invest behaviourally in his offspring for them to have a reasonable chance of surviving. So the typical pattern in mammals is for the female to be 'choosy' in order to obtain a healthy male, and for the males to compete for females.

But, because of its large size, the human brain is not fully developed at birth, so infants need to be looked after, fed, protected and helped for a considerable period while their brain develops and they reach independence. So in humans there is an advantage to paternal investment in helping to bring up the children, because the paternal resources (e.g. food, shelter and protection) can increase the chances of the father's genes surviving into the next generation to reproduce again. In humans, this therefore favours more complete pair bonding between the parents.

Couples in colonies

It is perhaps more useful to compare humans with birds that live in colonies, in which the male and female pair up and both invest in bringing up the offspring – taking turns, for example, to bring back food to the nest. Interestingly, tests on swallows using DNA techniques for determining paternity have revealed that approximately one third of a pair's young are not sired by the male of the pair (Ridley, 1993). So the female is sometimes mating with other males – what we might call committing adultery! These males will probably not be chosen at random: she may choose an 'attractive' male by responding to indicators of health, strength and fitness.

One such indicator in birds is the gaudy tail of the male peacock. It has been suggested that, given that the tail handicaps movement, any male that can survive with such a large tail must be very healthy or fit. Another theory is that a female would choose a male with an attractive tail so that her sons would be attractive too and also chosen by females. (This is an example of the intentional stance, since clearly the peahen is incapable of any real propositional thought; but it has also been criticized as representing a somewhat circular line of argument.) Choosing a male with an attractive tail may also benefit female offspring, so the argument goes, because of the implied health/fitness of the fathering peacock.

In a social system such as the swallows', the 'wife' needs a reliable 'husband' to help provide resources for 'their' offspring. A nest must be built, the eggs must be incubated, and the hungry young must be well fed to help them become fit offspring ('fit' here means capable of 'successfully passing on genes into the next generation'; see Dawkins, 1986). The male must in some sense 'believe' that the offspring are his – and, for the system to be stable, some of them must actually belong to him. But the female also benefits by obtaining genes that will produce offspring of optimal fitness – and she does this by sometimes 'cheating' on her

Figure 5.12

The gaudy tail of the male peacock is one indicator of attractiveness in birds. Given that the tail handicaps movement, any male that can survive with such a large tail must be very healthy or fit, which may explain why peahens have evolved to choose males with large tails.

'husband'. To ensure that the male does not find out and therefore leave her and stop caring for her young, she 'deceives' him by 'committing adultery' secretly, perhaps hiding behind a bush to mate with her 'lover'. So the 'wife' maximizes care for her children by 'exploiting' her 'husband', and maximizes her genetic potential by finding a 'lover' with better genes that are subsequently likely to make her offspring more attractive to future potential mates.

The implication is that genes may influence our motivational behaviour in ways that increase their subsequent success (see Rolls, 1999, ch. 10).

Are humans like swallows?

Again, how might this relate to human behaviour? Though it is not clear how important they are, there is some evidence to suggest that such factors could play some part in human sexual behaviour.

One potentially relevant piece of evidence in humans concerns the relatively large testis and penis size of men. The general argument in sociobiology is that a large penis could be adaptive in sperm competition, by ensuring that the sperm are placed as close as possible to where they have a good chance of reaching an egg, and so displacing other sperm, thereby winning the 'fertilization race'.

A second line of evidence is that studies in humans of paternity using modern DNA tests suggest that husbands are not the biological fathers to about 14 per cent of children (Baker & Bellis, 1995; see Ridley, 1993). So it is possible that the following factors have shaped human sexual behaviour in evolution:

- women might choose a partner likely to provide reliability, stability, provision of a home, and help with bringing up her children;

- women might also be attracted to men who are perhaps successful and powerful, increasing the likelihood of producing genetically fit children, especially sons who can themselves potentially have many children;
- men might engage in (and be selected for) behaviours such as guarding the partner from the attentions of other men, to increase the likelihood that the children in which he invests are his; and
- men might be attracted to other women for their childbearing potential, especially younger women.

Much of the research on the sociobiological background of human sexual behaviour is quite new and speculative, and many of the hypotheses have still to be fully tested and accepted. But this research does have interesting implications for understanding some of the factors that may influence human behaviour (see Baker, 1996; Baker & Bellis, 1995; Buss, 1999; Ridley, 1993).

HOW THE BRAIN CONTROLS SEXUAL BEHAVIOUR

We can be pretty sure that, in males, the preoptic area (see figure 5.13) is involved in the control of sexual behaviour (see Carlson, 1998; Rolls, 1999) because:

1. lesions of this region permanently abolish male sexual behaviour;
2. electrical stimulation of this area can elicit copulatory activity;
3. neuronal and metabolic activity is induced in this area during copulation; and
4. small implants of the male hormone testosterone into this area restore sexual behaviour in castrated rats.

Olfactory bulb

Medial preoptic area

Ventral tegmental area/
periaqueductal gray
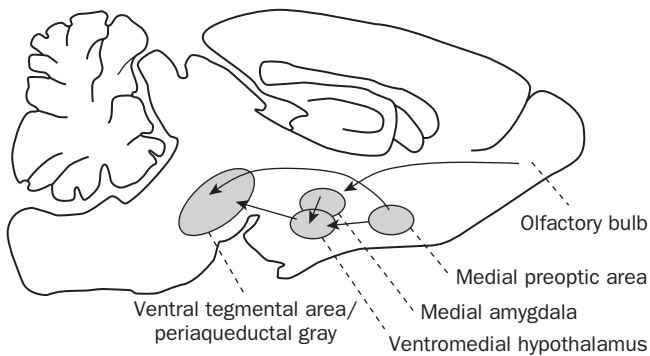
Medial amygdala

Ventromedial hypothalamus

Figure 5.13

A midline view of the rat brain showing some of the brain regions involved in the control of sexual behaviour.

In females, the preoptic area is involved in the control of reproductive cycles, and is probably directly involved in controlling sexual behaviour too.

The ventromedial nucleus of the hypothalamus (VMH) is also involved in sexual behaviour. Outputs from the VMH project to the periaqueductal gray of the midbrain, and this region is also necessary for female sexual behaviour, including lordosis (the position adopted by a female to accept a male) in rodents. This behaviour can be reinstated in ovariectomized female rats by injections of the female hormones oestradiol and progesterone into the VMH brain region.

Can the brain help us to understand sexual arousal at the sight and smell of someone to whom we are sexually attracted? By receiving inputs from the amygdala and orbitofrontal cortex, the preoptic area receives information from the inferior temporal visual cortex (including information about facial identity and expression), the superior temporal auditory association cortex, the olfactory system and the somatosensory system. It is presumably by these neural circuits that the primary rewards relevant to sexual behaviour (such as touch and perhaps smell) and the learned stimuli that act as rewards in connection with sexual behaviour (such as the sight of a partner) reach the preoptic area. And it is likely that, in the preoptic area, the reward value of these sensory stimuli is modulated by hormonal state, perhaps (in females) related to the stage of the menstrual cycle – women are more receptive to these sensory stimuli when they are at their most fertile.

The neural control of sexual behaviour may therefore be organized in a similar way to the neural controls of motivational behaviour for food. In both systems, external sensory stimuli are needed to provide the reward, and the extent to which they do this depends on the organism's internal state, mediated by plasma glucose concentration for hunger and hormonal status for sexual behaviour. For sexual behaviour, the internal signal that controls the motivational state and the reward value of appropriate sensory stimuli alters relatively slowly. It may change, for example, over four days in the rat oestrus cycle, or over weeks or even months in the case of many animals that only breed during certain seasons of the year.

Figure 5.14

We now know that the pleasantness of touch is represented in the human orbitofrontal cortex. This finding contributes to our understanding of the motivational rewards involved in sexuality.

The outputs of the preoptic area include connections to the tegmental area in the midbrain. This region contains neurons that are responsive during male sexual behaviour (Shimura & Shimokochi, 1990). But it is likely that only some outputs of the orbitofrontal cortex and amygdala that control sexual behaviour act through the preoptic area. The preoptic area route may be necessary for some aspects of sexual behaviour, such as copulation in males, but the attractive effect of sexual stimuli may survive damage to the preoptic area (see Carlson, 1998). Research findings suggest that, as for feeding, outputs of the amygdala and orbitofrontal cortex can also influence behaviour through the basal ganglia.

Much research remains to be carried out into how the amygdala, orbitofrontal cortex, preoptic area and hypothalamus represent the motivational rewards underlying sexual behaviour. For instance, it has recently been found that the pleasantness of touch is represented in the human orbitofrontal cortex (Francis et al., 1999). Findings such as these can enhance our understanding of sexuality in a wider context.

FINAL THOUGHTS

Motivational states lead animals (including humans) to work for goals (such as food, drink or sex). In this chapter, we have principally been looking at motives arising from the biological goals that we must reach in order to guarantee our own survival (eating, to ward off starvation and to promote healthy growth), and for the survival of our genes (sexual behaviour).

Part of the adaptive value of motivational states is that they specify the goal for behaviour (whether that be obtaining food, drink or sex), and then an appropriate behavioural action is co-ordinated to attain that goal. Emotional states are elicited by rewards (which we are motivated to obtain) and punishers (which we are motivated to avoid). Examples include fear produced by a noise that has been previously associated with pain, or joy produced by the sight of a long-lost loved one.

Motivations and emotions are not merely theoretical concepts, but also have considerable significance in the real world. For example, one of the central topics of this chapter is obesity, which is becoming a major problem is Western society. Why do so many people consistently eat in excess of their body's energy requirements when food supplies are plentiful? On the other side of the coin, why are some people motivated to deprive themselves of adequate nutritional input (see chapter 15 on anorexia and bulimia)? It will be very important in future to apply our developing knowledge of the many factors involved in motivated behaviours to provide help and support to those at risk of obesity and other clinical disorders in which motivational and emotional mechanisms appear to become dysfunctional.

Summary

- Motivational states are states that lead animals (including humans) to work for goals. Motivation also has close links with emotions, since these can be regarded as mental states (present in at least some species) that are elicited by rewards and punishments.
- Goals can be defined as rewards that animals will work to obtain, while punishers are events or situations that animals will escape from or avoid.
- One example of a goal is a sweet taste, which is rewarding when the motivational state of hunger is present. Hunger is signalled by decreases of glucose concentration in the bloodstream.
- The reward for eating is provided by the taste, smell and sight of food.
- Satiety is produced by (a) the sight, taste, smell and texture of food, (b) gastric distension, (c) the activation by food of duodenal chemosensors, (d) rises in glucose concentration in the blood plasma, and (e) high levels of leptin. Satiety signals modulate the reward value of the taste, smell, and sight of food to control appetite and eating.
- The orbitofrontal cortex contains the secondary taste cortex and the secondary olfactory cortex. In this brain region, neurons respond to the sight, taste and smell of food, but only if hunger is present. The orbitofrontal cortex is the first stage of processing at which the reward or hedonic aspects of food is represented. It is the crucial site in the brain for the integration of the sensory inputs activated by food (taste, smell, sight etc.) and satiety signals.
- The lateral hypothalamus has inputs from the orbitofrontal cortex and it also contains neurons that are necessary for the normal control of food intake. Once again, neurons in the lateral hypothalamus respond to the sight, taste and smell of food, but only if hunger is present. These neurons thus reflect the reward value of food, by reflecting the integration between the sensory inputs that maintain eating and satiety signals.
- The orbitofrontal cortex, and the amygdala, are involved in learning which environmental stimuli are foods (for example, in learning which visual stimuli taste good).
- Sexual behaviour has been influenced in evolution by the advantages to genes of coding for behaviours such as parental attachment, which increase the probability of survival of those genes.
- As with other motivational systems, such as hunger, genes achieve this by coding for stimuli and events that animals find rewarding. This is achieved by specifying, in parts of the brain such as the amygdala, orbitofrontal cortex, preoptic area and hypothalamus, which sensory inputs and events should be represented as rewards.

REVISION QUESTIONS

1. Is sensory-specific satiety a feature of most reward systems? How do you think that sensory-specific satiety is adaptive, i.e. would benefit the survival of the organism?
2. Discuss factors that may contribute to obesity, and possible treatments for obesity.
3. What are the functions of the orbitofrontal cortex and amygdala?
4. How plausible are sociobiological 'explanations' of behaviour?
5. Justify your response with respect to what you have learned in this chapter about the regulation of sexual behaviour.
6. Do you think that humans are intrinsically monogamous?

FURTHER READING

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A fascinating and controversial volume presenting analyses and hypotheses regarding some of the factors involved in human sexual behaviour and reproduction.

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An influential and provocative sociobiological perspective on how genes influence behaviour.

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