Emotion and Motivation **NST 1B Psychology 2003**

Lecture 3 (Tuesday 11 March)

Rudolf N. Cardinal Psychological basis and neurobiology of motivation

This is the last lecture in this series — so please fill in a feedback form. Thank you. I hope you've enjoyed them.

Overview

Motivation has been studied in many ways over many years; we will look at some historically important and interesting theories and experimental results. We will then try to examine the brain's motivational systems in the light of modern psychological theories of one of the central experimental techniques used to study motivation — instrumental conditioning.

Theories of motivation

Extremes of view

To ask questions about motivation is to ask *why* animals do what they do. There have been many theories of motivation over the years! At one end of the spectrum was Maslow (1954), who argued that humans have a hierarchy of needs (physiological → safety → social → esteem → 'self-actualization', e.g. painting and composing), and must fulfil lower-level needs before addressing higher ones. It's pretty useless experimentally; it doesn't make very many testable predictions, except that nobody should starve to death for their art. Middleton Manigault, 1887–1922, did just this attempting to 'see colours not perceptible to the physical eye'. It takes all sorts.

At the other end of the spectrum was Skinner (1938), an exponent of *radical behaviourism* (see Wilcoxon, 1969). It was well known that when some events follow animals' responses (actions), they change the likelihood that the response will be repeated. Thorndike (1905) had named this the Law of Effect, saying that events that were 'satisfying' increased the probability of preceding responses, while events that caused 'discomfort' decreased this probability. How do we know what's 'satisfying'? Because it increases the probability… a circular argument? Skinner wanted to move away from this: he called events that strengthened preceding responses *positive reinforcers*, and events whose *removal* strengthened the preceding response he called *negative reinforcers*. Reinforcers are defined by their effect on behaviour, and therefore, to avoid a circular argument, behaviour cannot be said to have altered as a *consequence* of reinforcement (Skinner, 1953). Skinner treated organisms as 'black boxes', without reference to any internal processes such as motivation — but many would argue one must take account of 'hidden' variables (like hunger) to *explain* behaviour, rather than just to describe it.

Semantic note: The term *negative reinforcement* means the strengthening of a response that removes a negative reinforcer such as electric shock — either by *escape* from the shock, or by *avoidance* of the shock. *Punishment* is the presentation of a negative reinforcer, or the removal of a positive reinforcer; it reduces the probability of the preceding response, and is therefore different from negative reinforcement.

Motivational states and homeostasis

How do motivational states (hunger, thirst) enter the picture? Hull (1943) suggested that events that *reduce drive* are positively reinforcing (so food's reinforcing when you're hungry because it reduces the hunger drive). This resembles *homeostatic* theories of motivation, such as those of Cannon (1929). These theories suggest, for example, that we eat to regulate our blood sugar, or to regulate total body fat. There is considerable interest these days in the way the hormone *leptin*, produced by fat stores, acts to suppress eating via the hypothalamus (Elmquist *et al.*, 1998; 1999).

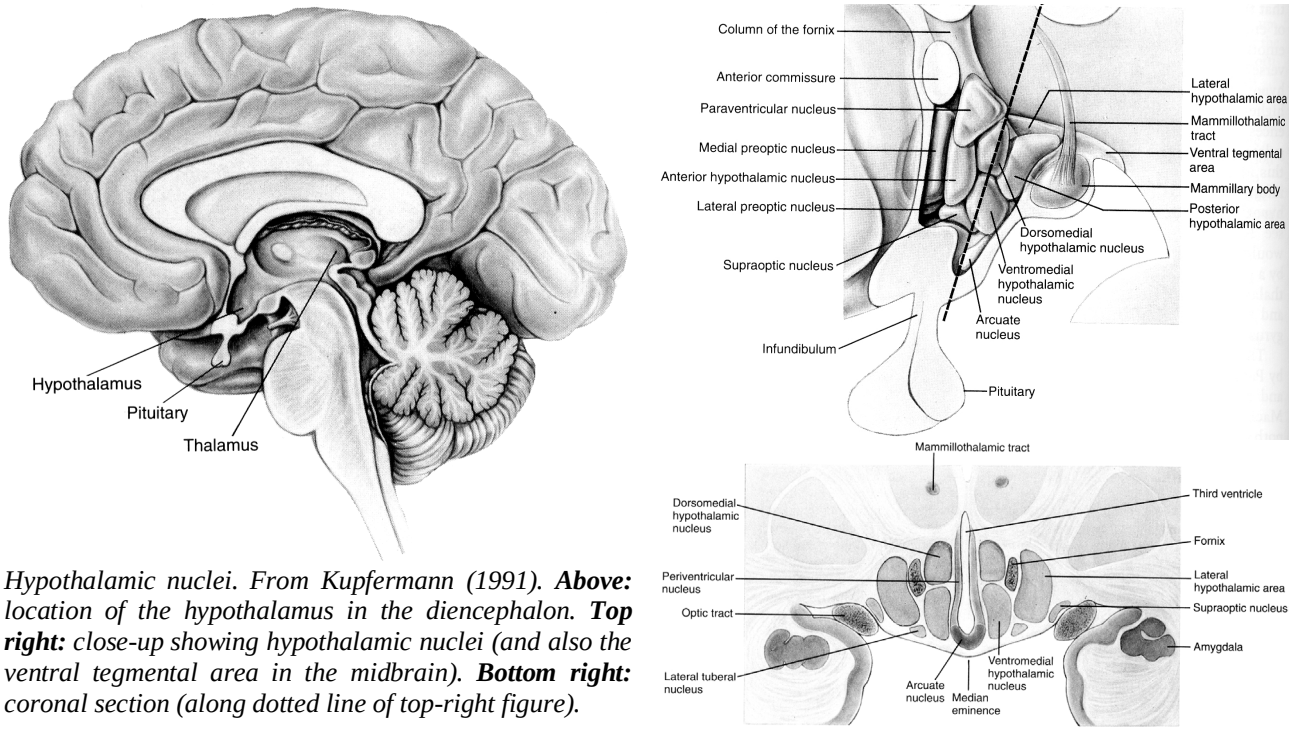
However, there are aspects of motivation that homeostatic theories don't account for well. Animals can be induced to eat or drink when they're not hungry or thirsty — their consumption doesn't just depend on their physiological needs (see Gross, 2001,

chapter 9). In humans, social and stimulus-based control of eating and drinking is very prominent. Do animals have a latent drive to take cocaine? To stimulate parts of their own brain electrically? Do humans? This seems to push the 'drive' concept too far — to examine these forms of motivation we need to look deeper at the processes that govern instrumental behaviour.

The hypothalamus; consummatory and appetitive behaviour

The hypothalamus and motivation

It is clear that different cell groups within the hypothalamus are involved in a large number of behaviourally significant activities. We saw last time that it coordinated 'rage'. Similarly eating can be suppressed by electrolytic lesions of the lateral hypothalamus (LH; Anand & Brobeck, 1951), and enhanced by lesions of the ventromedial hypothalamus (VMH; Hetherington & Ranson, 1939). Stellar (1954) suggested that the LH and VMH were 'hunger' and 'satiety' centres, respectively.



Hypothalamic nuclei. From Kupfermann (1991). ***Above:*** *location of the hypothalamus in the diencephalon.* ***Top right:*** *close-up showing hypothalamic nuclei (and also the ventral tegmental area in the midbrain).* ***Bottom right:*** *coronal section (along dotted line of top-right figure).*

However, this 'drive centre' hypothesis doesn't hold that well. Firstly, these electrolytic lesions affected *lots* of types of behaviour. Secondly, electrolytic lesion destroy a whole set of fibres passing *through* the hypothalamus; excitotoxic lesions, which destroy just local neurons, have less of an effect. (We will see later that these fibres of passage, inadvertently destroyed, included important axons of the *medial forebrain bundle*, connecting brainstem neurotransmitter systems to the forebrain; damage to this bundle replicates many of the effects of electrolytic LH lesions.) Thirdly, animals *recovered* to some extent from many of the effects of these lesions (Teitelbaum & Stellar, 1954). Fourthly, there are effects of these lesions that this hypothesis doesn't explain well — such as the finding that if rats have their body weight lowered *before* LH lesions, they can eat *more* for a while, suggesting that the lesion lowers a 'set point' for body weight (Powley & Keesey, 1970), and the observation that VMH lesions also affect food preference and metabolism (see Powley, 1977). Fifthly, we can ask whether studies examining food consumption measure *motivation* at all, in the sense of animals being likely to work for something.

Measuring motivation: distinguishing appetitive from consummatory behaviour

In fact, it is quite clear that *consummatory* behaviour (e.g. eating, drinking, copulating — directly related to using behavioural 'goals') — is neurally separable from *appetitive* behaviour (directed to obtaining these goals in the first place). Of these, appetitive behaviour is more obviously related to 'motivation' as we might com-

monly conceive it, but the hypothalamus is clearly involved more in consummatory behaviour. For example, lesions of the preoptic area of the hypothalamus prevent rats from shivering, eating more, building nests, or running around when it gets cold. However, these rats can still learn to press a lever to obtain hot or cool air, and can regulate their temperature this way (Carlisle, 1969).

A double dissociation between 'appetitive' (instrumental) and 'consummatory' behaviour has been shown for sexual behaviour: lesions of the medial preoptic area of the hypothalamus prevent male rats from copulating ('consummatory' response) but do not prevent them from working to obtain a female ('appetitive' response). In contrast, basolateral amygdala (BLA) lesions have the opposite effect (Everitt & Stacey, 1987; Everitt *et al.*, 1989).

Development of ideas of motivation and reinforcement

What is reinforcing? Natural reinforcement, drugs of abuse, ICSS

There are many natural reinforcers. Rats, for example, will work for food if hungry, water if thirsty, salt if salt-deprived, sex, warmth/cold if they are too cold/warm… but they'll also work for less obvious reinforcement. For example, rats will work for the opportunity to run in a wheel. Premack (1963) found that behaviours that a rat has a high probability of engaging in spontaneously (enjoys?) will reinforcer the performance of behaviours that it engages in with a lower probability (doesn't enjoy?) — for Premack, this was a basic principle of reinforcement. Thus, if the rat normally drinks more than it runs, you can reinforce running if drinking is made *contingent* upon running (i.e. it'll run more if you, the experimenter, arrange such that the rat has to run in order to drink). If it normally runs more than it drinks, however (perhaps when it's not thirsty), then you can reinforce drinking with running (i.e. it'll drink in order to be allowed to run).

If this weren't complex enough, the same thing can be both a positive and a negative reinforcer. Hundt & Premack (1953) used apparatus in which pressing a bar switched on a motorized running wheel, so that the rat inside was forced to run; licking a drinking spout then caused the wheel to stop. They found that the rats increased their rate of bar-pressing (positive reinforcement) *and* licking (negative reinforcement)… so running was positively reinforcing when the rats weren't running, and negatively reinforcing when they were running. Fickle creatures.

Then there are reinforcers that are *really* odd. Drugs of abuse are one example. Rats will work for and self-administer nearly all drugs that humans abuse — including heroin, cocaine, and nicotine. Finally, there's one of the most powerful reinforcers of all — intracranial self-stimulation (ICSS). Olds & Milner (1954) found that rats would perform an arbitrary response (such as pressing a lever) to deliver electrical stimulation to certain areas of their brain, including the septum and lateral hypothalamus. It was the power of this reinforcer that was so striking: one rat, for example, made >2000 responses per hour for 24 consecutive hours; and rats would also cross electrified floors to reach a lever that would deliver intracranial self-stimulation (ICSS). Animals will deliver ICSS to a variety of sites; conversely, stimulation of other sites is negatively reinforcing. ICSS was a clear challenge to simple forms of 'homeostatic' or 'drive' theories of motivation — there's no obvious deprivation state for ICSS.

The dopamine hypothesis of reward

One neurotransmitter has perhaps attracted more attention than every other in the study of reinforcement: dopamine (DA). There are four main DA systems:

- the *tuberoinfundibular* system, in the hypothalamus (this regulates the pituitary hormone prolactin, involved in the control of lactation);
- the *nigrostriatal* system projecting from the substantia nigra pars compacta (SNc) in the midbrain to the dorsal striatum, part of the basal ganglia (this regulates the initiation of movement and goes wrong in Parkinson's disease);

- the *mesocortical* system projecting from the ventral tegmental area (VTA) near the SNc in the midbrain to the prefrontal cortex;
- the *mesolimbic* system, projecting from the VTA to the *nucleus accumbens* (Acb) in the ventral striatum, another part of the basal ganglia.

Wise (1982) was probably the first to suggest that mesolimbic DA mediated *pleasure*. His hypothesis was based around the suggestion that DA-blocking drugs prevent the pleasure of rewards, so it was termed the 'anhedonia hypothesis' — alternatively, that the effects of DA were 'hedonic' (pleasurable). The theory was criticized from the outset but became very popular; it still features prominently in the news ('the brain's pleasure chemical', and all that).

All natural reinforcers that have been studied increase mesolimbic DA. Pretty much all drugs of abuse do, too — cocaine and amphetamine do so directly, while heroin, nicotine, ethanol, and cannabis increase mesolimbic DA indirectly via their own receptors. Many of the 'hot spots' for ICSS run along the medial forebrain bundle (MFB), which is a fibre tract that includes the axons of mesolimbic DA neurons. ICSS of the lateral hypothalamus stimulates the MFB and therefore stimulates DA release in the Acb, for example. Partially blocking DA receptors makes rats deliver more ICSS, as if to 'overcome' the blockade; if the blockade is extensive enough, they eventually cease responding, as if responding no longer had an effect (see Wise, 1994). All this suggested that animals repeat actions that increase mesolimbic DA — Wise initially suggested that this was because mesolimbic DA meant pleasure, though he has since retracted that view (Wise, 1994).

However, Berridge & Robinson (1998) found that profound DA depletion doesn't affect rats' ability to express or even to *learn* 'hedonic' responses to foods. Furthermore, there is extensive evidence that DA systems respond to *conditioned stimuli* predicting reinforcement as much as they respond to actual (primary) reinforcement — or even more (see Berridge & Robinson, 1998)! Both these suggest that DA is not a 'pleasure' chemical. However, DA is firmly implicated in 'appetitive' behaviour (Robbins & Everitt, 1992) — we will look at one explanation in a moment.

Psychological processes contributing to instrumental behaviour

Let's move on to modern theories of instrumental behaviour (e.g. Dickinson, 1994). They're a bit complex, because instrumental behaviour is complex. Even an apparently simple thing like lever-pressing in rats is controlled by *many* processes.

Goal-directed behaviour

In Pavlovian conditioning (Pavlov, 1927), as we discussed last time, an experimenter arranges a contingency between two stimuli in the world, presenting those stimuli independent of an animal's behaviour. In 'instrumental conditioning' (or 'operant' conditioning), the experimenter arranges a contingency between an animal's behaviour and a reinforcing outcome (Thorndike, 1911). No assumptions are made about the nature of learning — as we've seen, what an animal does in fact learn has been a matter of debate for decades. Instrumental conditioning is not explicable in terms of Pavlovian conditioning (Grindley, 1932); nor is the opposite true (Sheffield, 1965).

Early theorists took the position that the delivery of reward strengthened a direct associative connection between environmental stimuli and a particular response (Thorndike, 1911; Grindley, 1932; Guthrie, 1935; Hull, 1943). Such 'habit' learning would represent *procedural* knowledge (Dickinson, 1980), as the structure of the representation directly reflects the use to which the knowledge will be put in controlling the animal's behaviour. It would also be inflexible, because subsequent changes in the value of the reward would be unable to affect responding.

However, it has been shown that rats form more sophisticated and flexible representations in instrumental conditioning tasks. Behaviour may be said to be *goal-directed* if it depends on the twin representations of (1) the instrumental *contingency* between an action and a particular outcome, and (2) a representation of the outcome

as a *goal* (Tolman, 1932; Dickinson & Balleine, 1994). Simply put, a goal-directed organism presses a lever for food because it knows that lever-pressing produces food *and* that it wants the food. Rats can be goal-directed. When rats press levers, they know what the lever produces (Bolles *et al.*, 1980) and they know that they want the food (Adams & Dickinson, 1981). They can also use *discriminative stimuli* in the environment to tell them when lever-pressing will produce food, and when it won't — in the same way that humans can learn not to press the button on a Coke machine if it's unplugged (Colwill & Rescorla, 1990; Rescorla, 1990a; 1990b).

Goal-directed behaviour — incentive value

Saying that rats 'know that they want the food' is the same as saying 'the food has high *incentive value* for the rat'. Adams & Dickinson (1981) showed this by training rats to press a lever for food, and then giving the rats the same food followed by lithium chloride, to induce nausea and consequently an aversion to that food. The rats were then returned to the chamber with the levers, in an *extinction* session — no food was actually delivered. So they're previously pressed a lever only for nice food; now they were being asked to press that lever again. They never got a chance to press the lever and actually obtain 'nasty' (aversive) food, so they couldn't learn some sort of direct connection between lever-pressing and 'nastiness'. Yet they did press the lever less — indicating that their internal representation of the *value* of the food had been decreased by the poisoning. It makes sense for the rat.

Key point 1: rats know what they're doing when they press levers, just like us.

Incentive learning — the trickiest bit to understand in this lecture

What's much more surprising is that this only happens if the rats get a chance to *eat* the poisoned food after the poisoning event. This is really quite extraordinary. Consider the following experiment (Balleine & Dickinson, 1991):

Stage	Control group (L = lever)	Results of comparison	Devalued group (LiCl = lithium chloride)	Change occurring in devalued group
Training	L → food		L → food	
Devaluation	food		food → LiCl	Hedonic change
Test 1	L	=	L	
Re-exposure	food	>	food	Incentive learning
Test 2	L	>	L	

Both groups are trained to press a lever for food. The 'devalued' group then eat the food, and are poisoned. The control group aren't poisoned. If you then immediately test their lever-pressing, it's the *same* in the two groups. And yet the poisoned rats have certainly learned something: they'll eat less of the food than the control rats. And once they've actually eaten it, then they'll press less for it. This result implies that rats have *two value-processing systems.* One system responds as soon as the food is poisoned, and causes them to eat less of the food next time. It's quite likely that this reflects the *hedonic value* of the food (Garcia, 1989) — how much they *like* the food. The other value, the one governing their lever-pressing — the *instrumental incentive value*, or how much they *want* the food — doesn't change straight away. Only when the rats actually eat the food, experiencing its new unpleasantness, is the value governing lever-pressing updated.

To restate this hypothesis: the devaluation procedure modifies the neural system responsible for hedonic experience, so that it will react with disgust rather than pleasure when the devalued foodstuff is next experienced. In the meantime, the more 'cognitive' incentive value remains high, so the animal still works for the devalued food. The next time the food is consumed, direct experience of the food leads to the disgust reaction being evoked, which re-writes the neural representation of incentive value and leads the animal to work less for the food in the future.

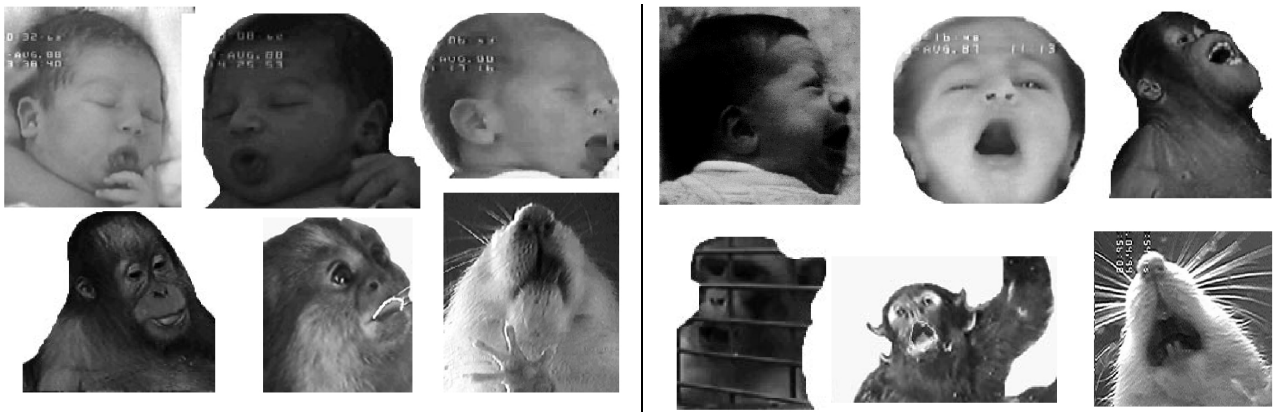
Incidentally, the same process controls how animals work when they're hungry or sated. Hungry rats will work for a nice food, and they'll carry on working for it even if they're sated. Only when they've actually *eaten* the food while sated, thereby

learning that the food is 'worth less' when they're sated, will they stop working. From this moment on, they'll work hard for it when they're hungry, but not when they're sated (Balleine, 1992).

Key point 2: just because rats work for something and know what they're working for, they may not like it when they get it. Next time, they know better.

Measuring hedonic value directly: taste reactivity patterns?

If we're going to suggest that animals might work for things (high incentive value) that they don't like (low hedonic value), we need to be able to measure 'liking' independently of a tendency to work. We can simply ask humans whether they like things or not (e.g. Baeyens *et al.*, 1990). We can't ask rats. However, there may be behavioural responses that directly reflect 'liking' or 'disliking'. Steiner (1973) found that newborn humans show characteristic facial expressions that distinguish pleasant tastes (e.g. sweet) from unpleasant ones (e.g. bitter). Grill & Norgren (1978) showed that rats exhibit similar responses. In fact, they are more than simple responses to tastes; they can be *learned* as well. For example, sweet tastes initially evoke 'appetitive' reactions; if a rat is given this taste, and shortly afterwards is given LiCl, it will subsequently show *aversive* reactions to the same taste (see Berridge, 2000). Dubious as it might sound (Wise, 1994), taste reactivity patterns are probably the best way of measuring 'liking' in rats.



Taste reactivity patterns, suggested to be an index of hedonic experience. ***Left:*** *tongue protrusion to sweet substances.* ***Right:*** *gaping to bitter substances. Figures from Berridge (2000).*

Habits

Nearly done. If rats spend ages pressing a lever for food, that response can become *habitual* — the behaviour is no longer goal-directed, but is controlled by a simple stimulus–response (S–R) association. At this point, if you poison the food, even if you let them eat the food afterwards, then their lever-pressing continues. They don't eat the food, but they carry on pressing the lever (Adams, 1982).

Key point 3: rats' actions can become habitual, just like ours.

Pavlovian to instrumental transfer

Last bit. Pavlovian conditioned stimuli (CSs) can modulate instrumental performance (Dickinson, 1994; Dickinson & Balleine, 1994). For example, if a rat's busy pressing a lever for food, and you present a CS that predicts the arrival of food, the rat will increase the rate of its lever-pressing. This is termed Pavlovian-to-instrumental transfer (PIT) (Estes, 1948; Lovibond, 1983).

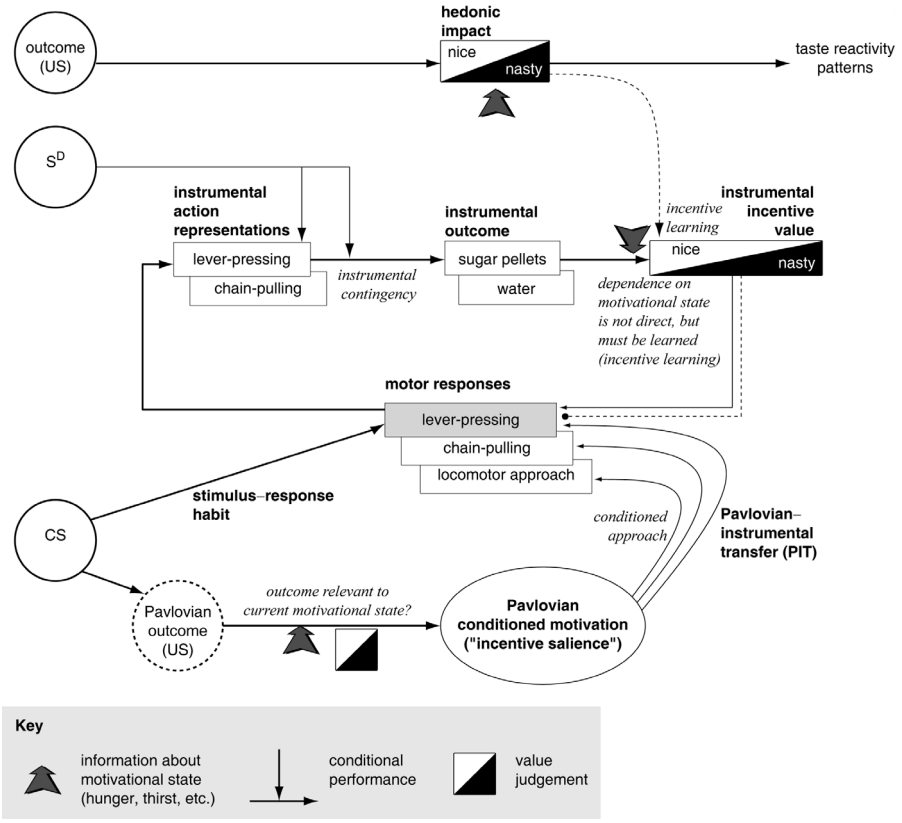
Key point 4: Pavlovian CSs that we have no control over can influence instrumental actions.

Is this important? Yes. For example, it may be an important contributor to drug abuse. Drug-associated cues (e.g. syringes, needles, the place where you shoot up, your friend the drug dealer) can induce *craving* in addicts, and cause them to *relapse*

(Tiffany & Drobes, 1990; Gawin, 1991; O'Brien *et al.*, 1998). Robinson & Berridge (1993) suggested that PIT — which they confusingly termed 'wanting' — might become stronger over time as a *consequence* of drug-taking, and might explain the phenomenon of addicts who continue to take drugs even though they don't like them so much any more.

Summary

Motivated action, exemplified by lever-pressing in rats, is a complex business! If we understand what's going on in the rat's mind, we might be better equipped to understand what's going on in its brain.

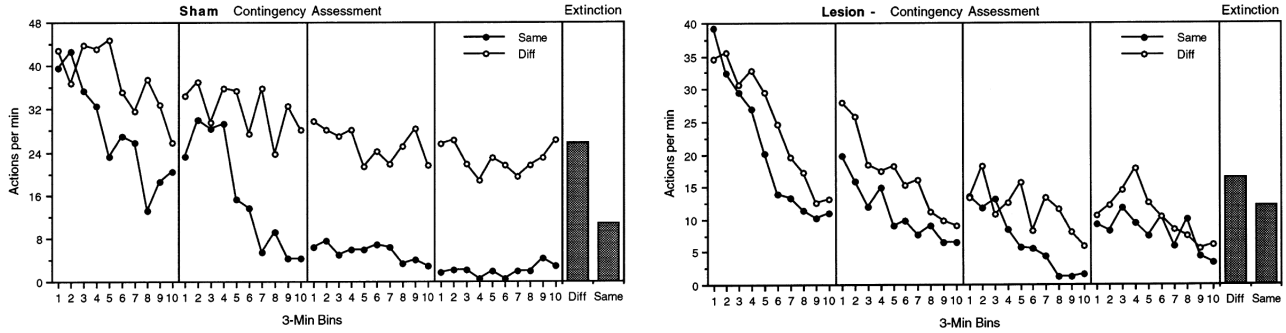


Routes to action in the rat (modified from Cardinal et al., 2002). Goal-directed lever pressing depends on the action–outcome (instrumental) contingency ('lever causes food') and the instrumental incentive value ('food is nice'). The rat needs to learn that food has value in a given motivational state via direct hedonic experience as it eats the food (incentive learning). The instrumental contingencies currently in force can be signalled by discriminative stimuli (S^Ds). With time, actions can become habits (direct connections between environmental stimuli and responses connections). Finally, Pavlovian CSs that signal a motivationally relevant outcome can enhance responding (PIT).

Neural structures contributing to instrumental behaviour

Instrumental action–outcome contingency: prefrontal cortex

Balleine & Dickinson (1998) have looked directly at action–outcome contingency learning. They used a task in which rats are offered two actions (lever pressing and chain pulling). Action A produces food of type 1; action B produces food of type 2. Initially, the instrumental contingency *P*(outcome | action) – *P*(outcome | no action) is perfect (1 – 0 = 1) for both action. Then, food of type 1 starts to be delivered *for*

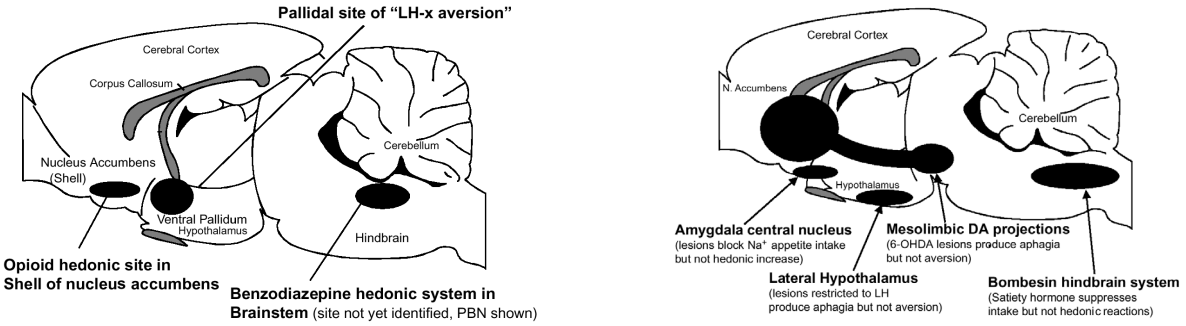


Action–outcome contingency learning (Balleine & Dickinson, 1998). ***Left:*** *normal rats are aware whether their actions increase the likelihood of food arriving ('same', rate of performing the action that produces the same food as that being delivered for free; 'diff', rate of performing the other action).* ***Right:*** *rats with prelimbic cortex lesions aren't. See text.*

free. As a result, the contingency for action A drops — the benefit of performing this action, in terms of the amount of extra food this produces, is reduced. The contingency remains the same for action B. Therefore, rats that are aware of this contingency should perform action A less than action B. Normal rats do this (see figure); rats with prelimbic cortex lesions don't. Yet in other experiments lesioned rats could distinguish food 1 from food 2, and action A from action B. This suggests that their actions are not under the control of action–outcome contingency knowledge; they don't know what their actions do, so they may be pure 'creatures of habit'. The prelimbic cortex may be equivalent to dorsolateral prefrontal cortex in primates.

Hedonic value: opioid systems in the nucleus accumbens shell, and others

We mentioned earlier that Berridge & Robinson (1998) found that profound DA depletion did not prevent rats from expressing, and indeed learning, 'hedonic' responses (taste reactivity patterns). What does? There is evidence (reviewed by Berridge, 2000) that opioid receptors injected systemically or into part of the Acb (the 'shell' region) make things taste nicer; so do benzodiazepines injected systemically or into the hindbrain. Lesions of the Acb shell and ventral pallidum affect taste reactivity, too (see figure).



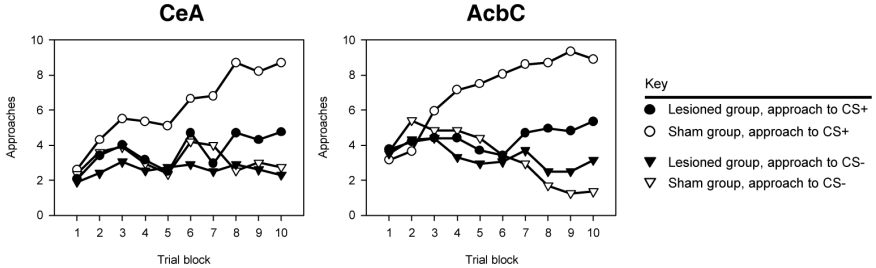
*L**eft:** sites suggested by Berridge (2000) to mediate hedonic impact, as measured by taste reactivity patterns. **Right:** sites that mediate some aspects of reinforcement, but do **not** affect hedonics (taste reactivity).*

There have also been studies that may provide information about the rat's 'other' value system controlling actions, the instrumental incentive value (see Cardinal *et al.*, 2002), but we won't discuss these today.

Pavlovian–instrumental transfer: dopamine systems and an amygdala–accumbens circuit

When it comes to the impact of Pavlovian CSs on behaviour, the amygdala, the Acb, and Acb DA all seem to play a vital role.

- *Conditioned reinforcement.* Animals will work for CSs previously paired with primary reinforcement (e.g. food) — this is termed *conditioned reinforcement* (CRf). The ability of animals to work for CRf depends upon the BLA (Burns *et al.*, 1993). Remember that we talked about the BLA last time as being important for imparting value to CSs. The BLA sends information to the Acb. Injection of amphetamine into the Acb, which releases DA and blocks its reuptake from the synapse, enhances the effect of CSs to act as conditioned reinforcers (Taylor & Robbins, 1984). This effect is blocked by DA antagonists (Cador *et al.*, 1991).
- *Pavlovian–instrumental transfer.* DA antagonists block PIT (Dickinson *et al.*, 2000), as do lesions of the 'core' region of the Acb and lesions of the CeA (Hall *et al.*, 2001). Injection of amphetamine into the Acb, which increases DA levels there, enhances PIT (Wyvell & Berridge, 2000).
- *Conditioned approach.* The tendency of animals to approach CSs that predict reward also depends on the CeA and the Acb (Parkinson *et al.*, 2000a; 2000b).

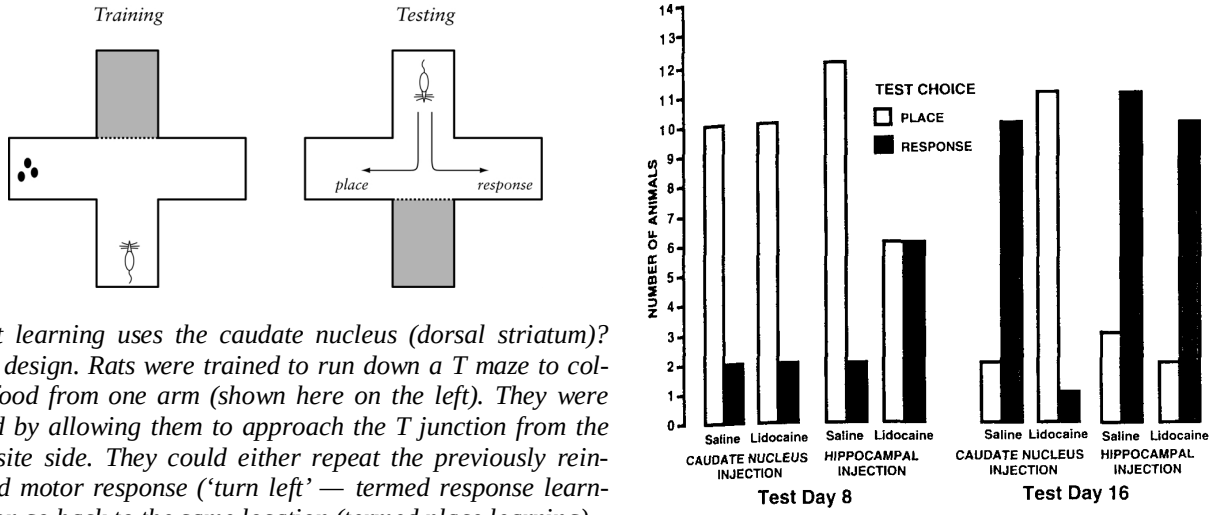


An example of an effect of a Pavlovian CS that depends on the amygdala and nucleus accumbens. Normal animals learn to approach CSs that predict food (CS+) more than they approach control stimuli (CS–). Rats with lesions of the CeA or Acb core don't (Parkinson et al., 2000a; 2000b).

These behaviours are 'appetitive' rather than consummatory: animals work for CSs, are 'energized' by them, and approach them, because this tends to bring them closer to primary reinforcement. These powerful *learned* motivational effects appear to be mediated by an amygdala–accumbens limbic circuit.

Habits: the dorsal striatum?

Perhaps the best attempt to examine the neural basis of habit learning is that by Packard & McGaugh (1996); their elegant study is illustrated below. It demonstrates that a stimulus to motor response (S–R) mapping or habit develops slowly during reinforced training, and it comes to dominate behaviour in this task; its performance depends upon the caudate (with the caveat that local anaesthetics such as lignocaine can inactivate fibres of passage as well as cell bodies). (In contrast, and not relevant to our present discussion, a hippocampus-dependent place-based memory develops rapidly and is superseded by the S–R memory under normal circumstances.)



*Habit learning uses the caudate nucleus (dorsal striatum)? **Left:** design. Rats were trained to run down a T maze to collect food from one arm (shown here on the left). They were tested by allowing them to approach the T junction from the opposite side. They could either repeat the previously reinforced motor response ('turn left' — termed response learning) or go back to the same location (termed place learning). **Right:** results (number of rats displaying each type of behaviour). If rats were tested on day 8, they exhibited place learning ('saline' groups = normal rats). This was blocked by pre-test injections of lidocaine (lignocaine), a local anaesthetic, into the dorsal hippocampus; these rats performed at chance. Intra-caudate injections had no effect. On day 16, rats exhibited response learning. This was not blocked by inactivation of the hippocampus, but it was blocked by inactivation of the caudate, which reinstated 'place responding'.*

Consummatory behaviour: the hypothalamus

We've already talked about this: once you've obtained your goal, the hypothalamus integrates 'consummatory' behaviour such as eating, copulation, and aggression.

Summary

Motivated behaviour is complex. Obtaining goals — 'appetitive' behaviour — involves the integration of cognitive knowledge about your goals with habits and the motivational impact of environmental stimuli (CSs). Once you've obtained your goal, you need to integrate complex 'consummatory' response patterns to use it. Structures within the brain's limbic system play an important role in appetitive and consummatory behaviours; we can distinguish those structures contributing to each.

All references cited in the handout

I'm not suggesting that you read all these! If you want further information, I'd suggest Robbins & Everitt (1999) — chapter 48 of *Fundamental Neuroscience*.

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