Emotion and Motivation

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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 examine the 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 \rightarrow safety \rightarrow social \rightarrow esteem \rightarrow '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 be-haviourism* (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 that something's 'satisfying'? Because it increases the probability of preceding responses... a circular argument?

We can illustrate this potential circularity in other ways, too. If a theory suggests that behaviour is motivated by a 'drive', but suggests that the drive exists on the basis of observed behaviour, we may have a circular argument. Suppose we arrange matters so that response R produces an outcome O. Our subject performs response R frequently. We might suggest that the subject lacks O and has an O-seeking-drive, which motivates its behaviour. But in this simple situation we have added nothing by postulating the existence of this O drive, since the argument is circular (R is motivated by O-drive; we know O-drive exists because the subject performs response R). Even worse, if R has no obvious consequence but the animal performs R, we might suggest that the animal performs R because it likes performing R — a theory that has zero predictive value. Any behaviour, however peculiar, can be explained by assuming that the behaviour itself is the subject's objective. Why did I stand on my head on the table while holding a burning £50 note bill between my toes (after Friedman, 1990).

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. However, many would argue one must take account of 'hidden' variables (like hunger) to *explain* behaviour, rather than just to describe it. And not all attempts to suggest such hidden variables are circular arguments.

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.

Inferring internal states: why use concepts of drive or motivation?

Our understanding of the physical world is based on the idea that things do not simply happen spontaneously, but are caused to happen (this was Aristotle's and Newton's view — though there are other philosophies of causality, such as Hume's, and this view doesn't apply to the microscopic, quantum world). Clearly, when we consider the behaviour of animals, they do not always do the same thing in the same circumstances. Yet their behaviour is often clearly not random - therefore, we seek intervening variables that contribute to (cause) behaviour. Ideas of drive and motivation emerge this way. For example, the activity of a female rat running in a wheel can vary considerably, but does so with a four-day cycle (Richter, 1927; Toates, 1986, p.4); we might observe that this cycle corresponds to the oestrus cycle, postulating some internal variable that connects the two. Some forms of behaviour are reliably elicited by environmental stimuli — if a hand or paw makes contact with a very hot surface, it will withdraw rapidly and reflexively. But some behaviours are not so reliably connected to the environment. Male stags don't always attack when confronted with other males, but they do so in the breeding season. Rats confronted with food don't always eat it. We might postulate the internal state or variable of hunger to account for this variability: the rat eats more when it is hungrier.



Left: Activity of a female rat running in a wheel (from Richter, 1927; Toates, 1986, p.4). The four-day cycle in spontaneous activity corresponds to the rat's oestrus cycle; the peaks of activity correspond to the times when the female is sexually responsive. *Right:* Thirst as an intervening variable (after Toates, 1986, p.28).

Furthermore, we might think the concept of hunger is useful because it predicts many things. Food-deprived people don't just eat more (and faster) when given access to food, but they perform better on arbitrary tasks such as word recognition (Erwin & Ferguson, 1979; Ferguson, 2000, p.7). If we allow rats to discover that an arbitrary response (such as pressing a lever) produces access to food, then we would expect a starved rat to perform more of this completely arbitrary behaviour ('work harder') than a sated rat, and this can readily be observed. A simple manipulation such as food deprivation affects a whole range of behaviours — and a motivational state (hunger) is a parsimonious way to account for this. If someone is hungry, we might predict that they will eat lots/fast, be relatively undiscriminating amongst foods, exert effort or spend money to obtain food, and eat in preference to other potential activities. Furthermore, we can manipulate this large range of behaviours in many different ways: food deprivation makes rats eat more food/work harder for food/etc., but so does insulin injection; water deprivation makes them drink more/work harder for water/tolerate water that's more adulterated/etc., but so does eating dry food, injections of hypertonic salt solution, and angiotensin II injection. Central motivational states parsimoniously account for these kinds of findings, although there are often complexities in the details (see Toates, 1986, pp.28-30).

Motivational states and homeostasis

Hull (1943) used motivational states as part of his theory of reinforcement. He 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 *homeo*- *static* 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.

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 things. 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 reinforce the performance of behaviours that it engages in with a lower probability (doesn't enjoy?). For Premack, this was a basic principle of reinforcement (not surprisingly known as *Premack's principle*). 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).

Premack's principle is a *differential response probability* theory of reinforcement (more probable behaviours reinforce less probable behaviours). However, when should you measure the 'free' probability of a behaviour? This probability may vary from situation to situation. Timberlake & Allison (1974) extended Premack's idea by using a *response deprivation* analysis: they claimed that a more restricted be-

haviour would reinforce a less restricted behaviour. Indeed, this can do better than Premack's principle. Suppose that in a 'baseline' situation, a rat spends 20% of its time running in a wheel and 10% of its time drinking. We now arrange matters so that every 4 minutes spent running earns the rat 1 minute of drinking. If the rat continues to spend 20% of its time running, it can only spend 5% of its time drinking so it's drinking less than it would do freely (drinking is a deprived response). Timberlake & Allison would predict that the rat should run more than 20% of its time, to be able to drink closer to 10% of the time — i.e. the low-probability behaviour should reinforce the high-probability behaviour, contradicting Premack's principle. Timberlake & Allison's prediction was borne out (e.g. Allison & Timberlake, 1974).

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. (We'll see how the ideas of behavioural economics can explain this in the next lecture.)

We've been talking about natural reinforcement, but there are also reinforcers that are *really* odd. Drugs of abuse are one example. Rats will work for and selfadminister 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.

So far, we've been looking at motivated behaviour in very global terms. But it turns out that many psychological processes contribute to motivated behaviour. Let's take a step backwards and examine those processes individually.

'Bottom up': psychological processes contributing to instrumental behaviour

There are many psychological mechanisms for action. Last time we talked about *Pavlovian (classical) conditioning.* Also, we mustn't forget that many forms of behaviour are *unlearned.* These include simple spinal and brainstem reflexes, which influence skeletal musculature (respiratory movements, postural reflexes, pain withdrawal reflexes, etc.) and autonomic function (such as the regulation of heart rate and arteriolar smooth muscle tone to maintain arterial blood pressure). Swallowing is a more complicated example of unlearned behaviour: it involves the activation of at least ten different muscles in a precise order (Doty & Bosma, 1956). Innate behavioural patterns can also be very complex. For example, the female greylag goose exhibits an innate, species-specific and highly stereotyped behaviour (a 'fixed action pattern') in which it rolls eggs — or any vaguely similar object — into its nest. It will continue the movement even if the egg is lost or removed by an experimenter (Lorenz, 1939; Tinbergen, 1948).

However, when we choose to measure motivation we are often interested in behaviours that are directed at obtaining particular goals, not just behaviours that animals perform once those goals are at hand. The difference can be phrased in several ways: *appetitive* versus *consummatory* is one popular way. In fact, it's been clear for some time that consummatory behaviour (e.g. eating, drinking, copulating — directly related to using behavioural 'goals') — is separable from appetitive behaviour (directed to obtaining these goals in the first place). Much of this evidence comes from neurobiological studies (for those doing NST 1B Neurobiology, see B.J. Everitt's lectures). 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 — consummatory behaviour is impaired. However, these rats can still learn to press a lever to obtain hot or cool air, and can regulate their temperature this way — appetitive behaviour is intact (Carlisle, 1969). In fact, the two can be doubly dissociated: lesions of the medial preoptic area of the hypothalamus prevent male rats from copulating (impaired 'consummatory' response) but do not prevent them from working to obtain a female (normal 'appetitive' response). In contrast, lesions of the basolateral amygdala have the opposite effect (Everitt & Stacey, 1987; Everitt *et al.*, 1989). So how is goal-directed behaviour organized psychologically?

Goal-directed behaviour: instrumental (action-outcome) contingencies

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.

In Pavlovian (or 'classical') 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 (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. First off, instrumental conditioning is demonstrable different from Pavlovian conditioning. Grindley (1932) showed that instrumental conditioning is not explicable in terms of Pavlovian conditioning; he trained guinea pigs to turn their heads left when a buzzer sounded in order to receive access to carrot delivered in front of them. When he reversed the instrumental contingency (head-turning-carrot relationship) by requiring that they turn right instead to get carrot, they did turn the other way, even though the Pavlovian contingency (buzzer-carrot relationship) stayed the same. This demonstrates that their behaviour was controlled by the instrumental contingency. Furthermore, Pavlovian conditioning is not explicable in terms of instrumental conditioning. For example, Sheffield (1965) trained dogs that a tone predicted food (Pavlovian conditioning: tone \rightarrow food) except that if the dog salivated, it lost the food (instrumental contingency: salivating \rightarrow no food). The subjects continued to salivate. So some behaviours, such as head-turning in guinea pigs (Grindley, 1932), and lever-pressing in rats (Bolles et al., 1980), can be controlled by instrumental contingencies; others can't, such as salivation (Sheffield, 1965) and walking around (Hershberger, 1986).

What does happen during instrumental conditioning? 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 *goaldirected* if it depends on the twin representations of (1) the instrumental *contingency*



Goal-directed actions (based on declarative representations) versus stimulus-response habits (based on procedural representations). After Dickinson (1980). We and rats have both. How can we tell the difference? One way is to train the subject to perform the action (press the lever), then poison the food. If the subject refrains from pressing the lever with no further training, it must be using declarative information that includes a representation of the food. Habits have no representation of the outcome (food), so they cannot immediately respond to changes in the value of the outcome. 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.



Adams & Dickinson (1981) trained rats to press a lever for food A. They also gave them food B for free. Next, they poisoned either food A (group P for Paired poison) or food B (group U for Unpaired poison). Then they tested the rats' responding on the lever for food A in extinction (no food was actually delivered). If rats' lever-pressing is goal-directed (if they know that pressing the lever produced food A), and they represent the value of the goal, and that value was altered by poisoning and eating poisoned food, then group P should respond less than group U. And they did.

Summary: 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	Results of	Devalued group	Change occurring in de-
-	(L = lever)	comparison	(LiCl = lithium chloride)	valued group
Training	$L \rightarrow food$		$L \rightarrow food$	
Devaluation	food		$food \rightarrow LiCl$	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.

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). So the way responding depends on motivational state is *learned*.

Summary: 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. You have to *learn* what goals are appropriate for your motivational state.

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 poisoned with lithium chloride, 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).

The same incentive learning process may operate when it comes to drugs of abuse. Consider opiates, such as heroin. Opiates produce euphoria, and brain opiate systems may be involved in the 'hedonic' process we've discussed (Berridge, 2000). But even heroin's value can vary, and incentive learning seems to operate. With drawal from opiates may create a 'new' motivational state that animals can perceive. When you're in withdrawal, the hedonic impact of opiates may be enhanced, and this in turn teaches animals that it is worth working more for opiates — that opiates have a higher instrumental incentive value — when they're in a state of opiate withdrawal (Hutcheson *et al.*, 2001). The hedonic impact of a reinforcer may be a 'com-

mon currency' for determining the value of widely varying reinforcers (e.g. Cabanac, 1992).

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).

Summary: actions can become habitual.

This may also have relevance for addiction — it's possible that some drugs of abuse induce habitual responding faster than natural reinforcers. For example, Dickinson *et al.* (2002) found that alcohol-seeking was more habitual than food-seeking in rats. Alcohol is aversive to rats and humans when they first experience it, though they get used to it and the aversive reactions wane with experience (Kiefer & Dopp, 1989). So a good way to get rats — and people — to drink alcohol is to mix it with sugar at first (Samson, 1986). Witness the rise of alcopops.

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-toinstrumental transfer (PIT) (Estes, 1948; Lovibond, 1983).

Summary: 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



Habits (Adams, 1982). Left: Rats were trained to press a lever for food, and allowed to do so 100 times. Then they ate some of the food and were poisoned. Subsequently, in an extinction trial (one in which no food was given), they pressed the lever much less than unpoisoned rats (group 100-P < group 100-U). This indicates that their behaviour was goal-directed; they no longer valued the goal. However, another group of rats were trained for 500 lever presses. When they were poisoned, they carried on pressing the lever (group $500-P \ge group 500-U$) — their responding was habitual. **Right:** the poisoning worked in all groups. When the food is delivered again, responding drops in the 500-P group and the 100-P group relative to the two unpoisoned groups.



Pavlovian-instrumental transfer (redrawn from Estes, 1948). A tone is paired with food. The rat is then trained to press a lever for food. Finally, the rat is allowed to press in extinction (no food is delivered) and the experimenter switches the tone on and off. The Pavlovian CS enhances lever-pressing; this is termed Pavlovian-instrumental transfer. Group C is a control group (no tone); groups A and B hear the tone at different times (• tone, o no tone). Estes had previously shown that tones that were not paired with food had no effect on lever-pressing.

so much any more. (And supermarkets attempt to place advertising and other product-associated cues as close as possible to the point of purchase; this may reflect the same principle of cue-induced motivation.)

PIT depends directly on motivational state in a way that goal-directed action does not. Although we've seen that you have to *learn* that the instrumental incentive value of food depends on your state of hunger, through incentive learning, PIT depends on motivational state without the need for incentive learning. For example, fluid-associated cues have a bigger impact when you're thirsty, even if you've never drunk that fluid when thirsty before; salt-associated cues promote responding when you're salt-deprived (Dickinson, 1986; Dickinson & Dawson, 1987a; 1987b).

A real-world application: it's a popular belief that people buy more in supermarkets if they do their weekly shop whilst hungry. Why should this be so, and is it true? There are at least two reasons to think it would be so. First, the food has a higher instrumental incentive value when they're hungry (assuming they've eaten those foods when hungry before, i.e. had an opportunity for incentive learning). Second, food-associated cues tend to promote responding for food more when subjects are hungry. And indeed, it does appear to be true (e.g. Dodd *et al.*, 1977; Mela *et al.*, 1996).

Summary

Motivated behaviour is complex. Reinforcement must be defined carefully to avoid circular arguments. Motivational states are internal 'hidden' variables that help to explain behaviour. 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 'consummatory' response patterns, such as the process of eating, to use it.



Routes to action in the rat (modified from Cardinal et al., 2002). *Goal-directed* lever pressing depends on the actionoutcome (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 $(S^D s)$. With time, actions can become habits (direct connections between environmental stimuli and responses). Finally, Pavlovian CSs that signal a motivationally relevant outcome can enhance responding (PIT).

All references cited in the handout

I'm not suggesting that you read these! They are here as pointers to the original literature; so if you are for some reason keen to read more, or if you disagree with something I've claimed, you can check for yourself.

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