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Overview

We will begin by considering the functional relevance of emotions and their possible evolutionary origins. We will look at how emotions can be measured, and examine category-based and dimensional accounts of emotion. We will review three major theories of emotions (the James–Lange, Cannon–Bard, and Schachter theories). We will then look at one technique for measuring ‘internal’ emotional (affective) states in animals.

Emotion: definitions and functions

Everybody knows what emotions are... yet they can be difficult to define in a manner that allows experimental study. As a central theme we shall consider under the umbrella of ‘emotion’ all processes in humans and other animals that involve the assessment of *value*.

Obvious functions

Simple emotions such as fear are driven by motivationally-significant stimuli and events; ‘emotional’ behaviour can be highly adaptive for an animal. Fear of heights makes you less likely to be near (and therefore fall off) cliffs; fear of snakes and spiders makes you less likely to be bitten and poisoned by them (however unlikely that is in today’s urban environment). Other emotions, especially those in the social domain, are more complex to understand.

Emotions: rationally irrational?

Schelling (1960), Frank (1988) and others view emotions as important because they are *involuntary* and *difficult to fake*; they advertise our inner states. In some situations, they are like a Doomsday machine (see Pinker, 1997, chapter 6, for light reading on this). The idea is that if your behaviour is controlled by rational mechanisms, you might change your mind, and people can bargain with you. If your behaviour isn’t rational, you may do better. Imagine it’s 1962, and you’re President of the USA. The Soviet Union has just dropped an atomic bomb on New York, but the premier responsible has just been assassinated, so you know they will not attack again. Your nation’s policy is to retaliate with a nuclear strike. But at this moment, you have nothing to gain by killing the citizens of Moscow, so you might pause. The problem is that by the time you’re at this point, your freedom of choice may cause you not to retaliate (because it isn’t particularly to your advantage at this time), but your opponent’s knowledge that you might think and behave this way is what prompted the attack in the first place. What you needed is a deterrent that everyone would *believe* — for example, automatic retaliation that you could not prevent. This is taken to its extreme in Stanley Kubrick’s famous film *Dr Strangelove (Or, How I Learned to Stop Worrying and Love the Bomb)*.

Or take the game of chicken (featured in *Rebel Without a Cause*), in which two drivers face off and drive directly at each other at high speed. Since it is rational to swerve away at the last moment before crashing, a good way to win is to enter the car conspicuously drunk and throw your steering wheel out of the window as you set off, letting your opponent know that you have no ability to stop. (If your opponent does the same thing, of course, then you’re in trouble.)

A similar argument may be applied to emotions. They may be threats: if a man is known to fly into uncontrollable rages (and, critically, is known *not to be faking it*), people will think twice before upsetting him — even if the rage is not helpful at the moment it comes. They may be promises: in choosing a sexual partner, you may be more secure if they display *emotional* responses to you (with signals that are hard to fake, like dilated pupils and flushed skin) because that means that their commitment is not under rational control — they can’t help it — so they’re less likely to leave if

a (rationally) better proposition than you comes along. (It's no coincidence that polygraphs — lie detectors — are based on measuring hard-to-fake emotional responses such as skin conductance.)

Evolution of emotions

Examples like these indicate that emotions may have benefits to the possessor, but also to other people; however, there are potential evolutionary mechanisms for both (see e.g. Trivers, 1985; Ridley, 1993; Pinker, 1997).

In fact, the first person to study the evolution of emotions was Darwin (1872). He noted that the same emotional responses (such as facial expressions) tended to accompany particular emotions, across human races and cultures. He compared human emotional behaviour with similar behaviours in other species. Darwin suggested that 'emotional expression' evolves from similar behaviours that signal what an animal is likely to do next. If such behaviours benefit the animal, they may evolve as a communication device and become to some extent independent of the original behaviour that they predicted.

For example, rising up, facing one's enemy, and exposing one's teeth and/or claws are all necessary parts of animal combat. However, once enemies start to recognize this pattern of behaviour as signalling impending aggression, there would be a distinct advantage for any aggressor that could communicate their aggressive intent effectively enough to cause the opponent to withdraw without actually fighting. As a result, elaborate threat displays might evolve (while actual combat might decline). Darwin also noted that signals conveying opposite intent should be, and are, highly distinguishable — for example, displays of submission involve opposite movements to displays of aggression (his 'principle of antithesis'; see figure).

Measuring emotion

Emotional responses have at least *three* components:

- *subjective* (e.g. the feeling of fear)
- *behavioural* (e.g. facial expression, immobility, avoidance behaviour)
- *physiological* (e.g. autonomic responses including changes in heart rate, blood pressure, respiratory rate, pupil size, skin conductance, EEG patterns, and hormone secretion)

All can be measured in humans. Self-report techniques can be used to assess subjective feelings; observational and other measurement techniques can assess behavioural and physiological measures. Clearly, behavioural and physiological responses can be measured in animals. However, subjective experience generally cannot. Yet there are ways to infer central 'emotional' states in animals; we will mention one later.

Universal emotions? Facial expression of emotion

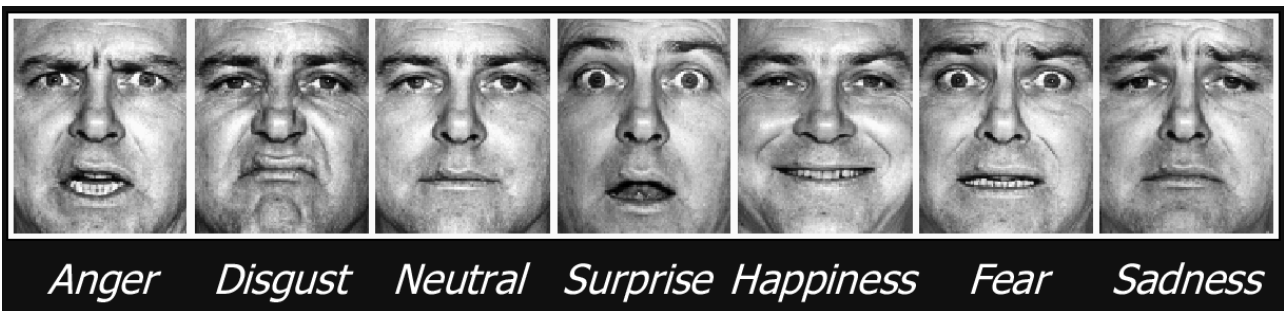
Darwin's early work on facial expression of emotions has been extended by Ekman (Ekman *et al.*, 1972; Ekman & Friesen, 1975), who identified six cross-cultural 'primary emotions' in humans — surprise, fear, anger, disgust, happiness, and sadness (see figure). Ekman views these as *universal*, and hence likely to be *innate*.

Category-based and dimensional accounts of emotion

Ekman's is a *category-based* view of emotions. An alternative is to suggest a *dimensional* account, which view different emotions as reflecting different levels of a small number of underlying dimensions. Perhaps the first dimensional system was that of Wundt (1897), who suggested that emotional experience could be described in terms of *pleasantness/unpleasantness*, *calm/excitement*, and *relaxation/tension*. More modern accounts include those of Russell (1980), based on *pleasant/unpleasant* and *aroused/not aroused*, and Rolls (1990). Rolls bases his model on animal reinforcement studies, a topic we will return to later. He attempts to use di-

mensions based on *appetitive/aversive* reinforcers (S+ or S-) — an appetitive reinforcer being something that an animal will work for, and an aversive reinforcer being something that it will work to avoid — so that presentation of a fantastic S+ causes elation, and presentation of a powerful S- causes terror. Additionally, he has a dimension related to *prediction* or *expectation*; thus, loss or omission of an expected S+ produces frustration; omission of an expected S- produces relief. It's not clear that this is a very general model of emotions.

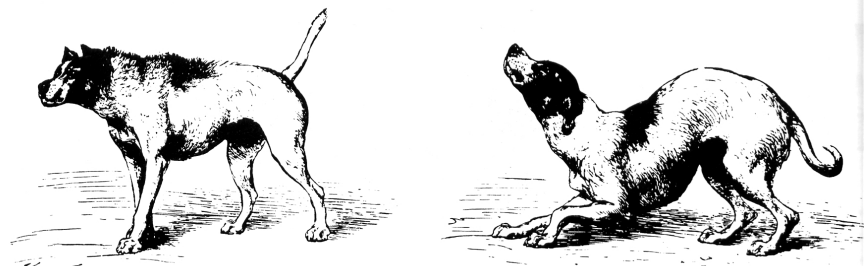
More generally, it isn't clear that two dimensions are enough to account for human emotions, particularly when the effects of brain damage need to be accounted for. Russell's model predicts that the *dimensions* are what can be affected by brain damage, and so it should be difficult or impossible for brain damage to affect the processing of some emotions without also affecting other emotions that are dimensionally related — yet there is some evidence that such dissociations can occur (see Calder *et al.*, 2001).



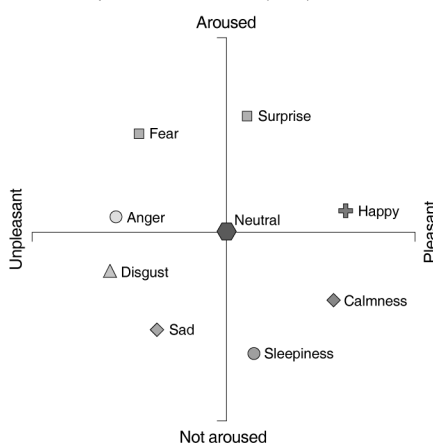
Aggression

Submission

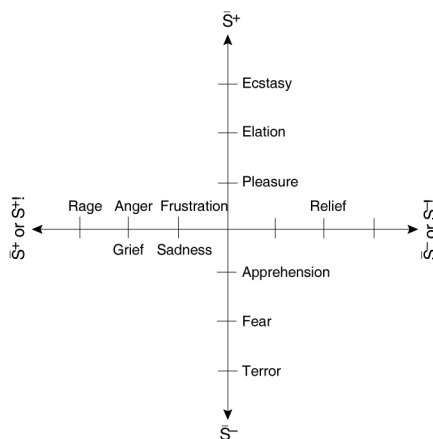
Above: Ekman's universal facial expressions. **Right:** Woodcuts from Darwin's (1872) book on emotional behaviour across species. **Below:** two-dimensional models of emotion (Russell, 1980; Rolls, 1990).



The circumplex model — Russell (1980)



Theory of emotion — Rolls (1999)



The James-Lange theory of emotion

Common sense might suggest that emotional expression results from emotional experience — that if are trekking in the jungle and see a tiger with cubs, we first feel fear (emotional experience), and this causes autonomic changes and a tendency to leave rapidly (emotional expression). James (1884) and Lange (1885 / 1967) independently suggested that the opposite might be true: that the emotional experience is a *consequence* of the bodily response, and depends on perceptual awareness (feed-

back) of that response. This theory, now known as the James–Lange theory, was initially based on anecdote and philosophical argument.

Note that James’s theory allowed that emotions could be induced by ‘visceral’ (autonomic) feedback, such as an increase in heart rate, and also by feedback from skeletal muscle activity. We will return to this below when considering facial expression of emotion. Smile — do you feel happier?

The Cannon–Bard theory of emotion

Cannon (1927) objected to the James–Lange theory on several grounds, based on the experimental evidence available at that time:

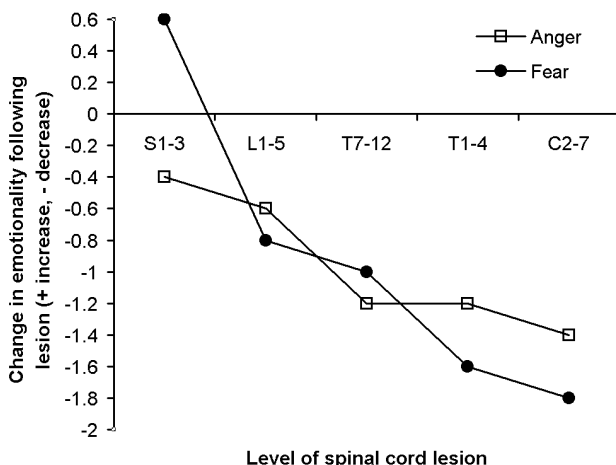
1. *total separation of the viscera from the CNS did not impair emotional behaviour observed in laboratory animals* (e.g. following sympathectomy or vagal nerve section);
2. *the same visceral changes occur in very different emotional states* (implying that they could not be the sole cause of different emotions);
3. *the viscera are relatively insensitive structures* (e.g. surgical trauma to the viscera often produces surprisingly little discomfort);
4. *visceral changes are too slow to be a source of emotional feeling*;
5. *artificial induction of the visceral changes typical of strong emotions does not actually produce emotional experience*. Marañón (1924) injected 210 subjects with adrenaline; the majority (71%) reported only physical symptoms; most of the rest reported having feelings ‘as if’ they were emotions; a very few reported actual emotions, and they recalled memories of an emotional event during the experiment.

This theory was later extended by Bard (1934). The Cannon–Bard theory essentially states that emotionally significant events *independently* cause emotional experience and physiological responses.

Many of the points made by Cannon have subsequently been disputed. Let’s look at evidence that has accumulated since then.

Subjective responses in paraplegic subjects

Hohmann (1966) found that subjective feelings of anger and fear *were* diminished in subjects who had suffered spinal cord injury, and this effect was greater with progressively higher lesions of the cord (see figure). (These lesions would affect both autonomic function and skeletal musculature, to differing degrees depending on the site.) These subjects were perfectly capable of acting *as if* they were angry, in appropriate situations — but subjectively, this anger lacked intensity and emotional colouring. This suggests that at least some part of emotional experience does depend upon the brain’s ability to interact with the body, though other aspects of emotional expression do not.



Data from Hohmann (1966) showing a decrease in ‘emotionality’ following spinal cord lesions; the higher the lesion, the more its effect. A description by one of the subjects is given below.

“It’s a sort of cold anger. Sometimes I act angry when I see some injustice. I yell and cuss and raise hell, because if you don’t do it sometimes, people will take advantage of you. But it just doesn’t have the heat to it that it used to have. It’s a mental kind of anger.”

Visceral responses to different emotions

Although emotional states induce a number of physiological changes, some of which may not differentiate between different emotional states, some studies have found that different emotions induce different ‘profiles’ of physiological response.

For example, Ax (1953) measured 14 different physiological variables while inducing fear or anger in subjects in the laboratory (by insulting them or delivering electric shocks, respectively). Of these, 7 differentiated between fear and anger (e.g. diastolic blood pressure increased more in anger than fear; muscle tension increased more during fear than anger).

Visceral responses to relived emotions and facial expression of emotion

Ekman *et al.* (1983) found similar emotion-specific autonomic changes. They asked professional actors to either (1) reconstruct facial expressions of emotions, muscle by muscle — akin to the ‘technique’ or ‘external’ system of acting favoured by Delsarte and Laurence Olivier — or (2) to relive past emotional experiences, akin to ‘method acting’ as advocated by Stanislavsky and Robert de Niro. Some autonomic measures differentiated between emotions (e.g. anger and fear produced equivalent increases in heart rate, but only anger increased finger temperature). These results held for both directed facial actions and reliving emotional experience.

As an aside, it was Charles Darwin who first suggested that feedback from facial expression was an important factor in determining subjective emotional feelings:

“The free expression by outward signs of an emotion intensifies it. On the other hand, the repression, as far as this is possible, of all outward signs softens our emotions.” (Darwin, 1872).

Emotional interpretation of skeletal muscle activity?

Laird (1974) attempted to test James’ view that emotions could follow from patterns of skeletal muscle activity. He falsely informed a group of subjects that they were participating in an experiment to measure activity in facial muscles; they were kitted out with fake electrodes attached to their faces. Laird then got them to make a range of facial movements, muscle by muscle; they were unaware of the nature of their expressions, but the patterns they made included smiles and frowns. While this was happening, they viewed cartoon slides; regardless of content, they rated as funnier the slides they’d seen while ‘smiling’. They also described themselves as happier whilst ‘smiling’, angrier when ‘frowning’, and so on. However, note that autonomic changes can accompany simulated emotional expressions (Ekman *et al.*, 1983, see above), so this experiment does not distinguish the role of skeletal muscle feedback from autonomic feedback.

Schachter’s cognitive labelling theory

Schachter (1964) held that Cannon was wrong in considering emotional experience to be independent of bodily changes, that James was right to consider that physiological changes precede the experience of emotion, but that James was wrong to consider the bodily changes to be solely responsible for emotional feelings. Schachter’s *cognitive labelling theory* suggested that physiological arousal is necessary for emotional experience, but that the nature of this arousal is immaterial — what matters is how we interpret that arousal. This theory is therefore also known as the *two-factor* theory of emotion.

Labelling of autonomic arousal

The classic demonstration of this theory was by Schachter & Singer (1962). They injected subjects with a ‘new vitamin’ to ‘test its effect on vision’. This injection was in fact adrenaline (known as epinephrine in the USA). The groups varied as follows:

1. *Epinephrine informed.* These subjects were injected with adrenaline (though they thought it was this 'new vitamin'), and informed of its side effects — tremor, palpitations, flushing, etc.
2. *Epinephrine ignorant.* These subjects were injected with adrenaline, but told that the injection was mild and had no side effects. (Therefore, the subject had no external explanation of the effects of the adrenaline.)
3. *Epinephrine misinformed.* These subjects were injected with adrenaline, but inaccurately told that it would produce numb feet, itching, and headache.
4. *Placebo.* These subjects were injected with saline, and told that it would have no side effects.

Before receiving their 'vision test', subjects waited in a room with another 'participant', who was a stooge. This stooge either acted euphorically, or angrily. The subjects' emotional experience was then assessed in two ways: (1) by self-report scales (e.g. 'How good or happy would you say you feel at present? 0 = I don't feel at all happy... 4 = I feel extremely happy'), and (2) by observers' ratings through a one-way mirror of the degree to which they joined in the stooge's behaviour (e.g. initiating activity with the stooge or agreeing with him).

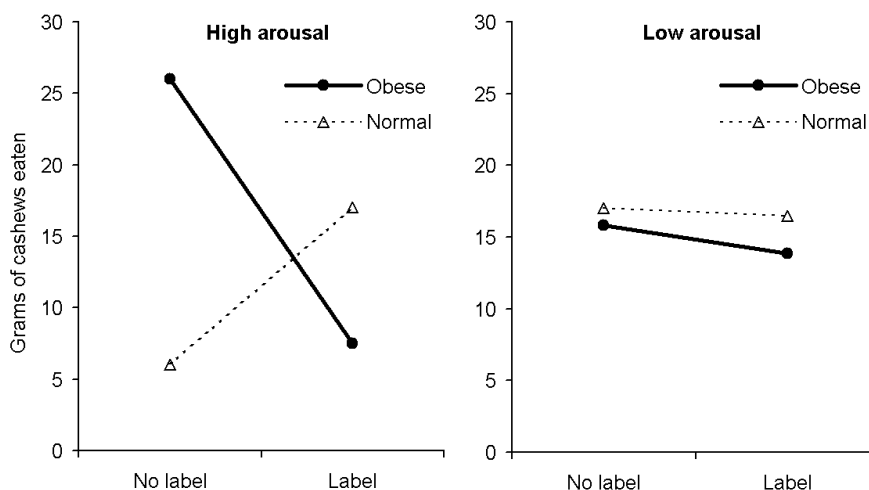
Self-report results (higher scores indicate greater euphoria; lower scores indicate anger); \uparrow and \downarrow indicate significant differences from the other groups.	Group	Condition	
		Euphoric stooge	Angry stooge
	Epi informed	0.98 –	1.91 –
	Epi ignorant	1.78 \uparrow	1.39 \downarrow
	Epi misinformed	1.90 \uparrow	data lost
	Placebo	1.61 –	1.63 –

The results (above) indicate that subjects who experienced *unlabelled arousal* (those injected with adrenaline who were either ignorant or misinformed of its effects) were more likely to experience emotion, but the *quality* of that emotion could be influenced by the cognitive context of the subject — the *labelling* of that arousal. The observers' rating provided similar results.

Others (e.g. Marshall & Zimbardo, 1979) have since suggested that subjects' ability to label arousal is not all that flexible; they found that the unexplained effects of adrenaline were not interpreted as positive emotions in the presence of a euphoric stooge (i.e. they failed to replicate some aspects of the results of Schachter & Singer, 1962), suggesting that unexplained autonomic arousal is simply a bit unpleasant. We'll consider some more examples with positive emotions in a moment.

Unlabelled arousal and eating behaviour

Schachter (1968) suggested some interesting applications of his theory. He suggested that some forms of obesity arise from an inability to distinguish internal states of emotions such as anxiety from internal states of hunger, perhaps as a consequence of early experience. Slochower (1976) tested this hypothesis. She took subjects of normal weight and obese subjects; while waiting to perform some completely irrelevant experiment, they heard feedback of their own heart rate. This feedback was false (i.e. not their own), and was either at a normal rate (*low arousal*) or



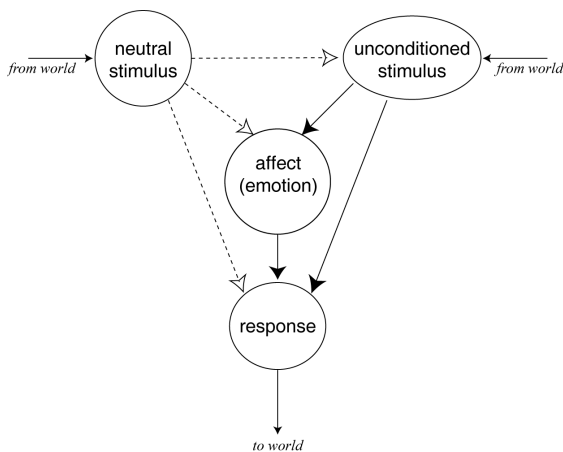
Data from Slochower (1976).

Measuring emotions in animals

One traditional way of measuring emotion in animals — and one that has greatly illuminated the neurobiological basis of emotion — is to use a Pavlovian conditioning procedure in which an initially neutral stimulus (conditioned stimulus, CS) is paired with a motivationally significant unconditioned stimulus (US). The animal's response to the CS is often then taken to reflect its emotional state, in the absence of any consequences of presenting the US. To make this concrete, imagine that our subject, a rat, experiences a tone (CS) paired with electric shock (US). If you subsequently watched the rat when it next hears the tone, you could do worse than to say that it exhibits fear. It exhibits behavioural changes (typically, it becomes immobile, or *freezes*); it exhibits physiological changes (it secretes adrenaline and ACTH, its heart rate changes, and so on). Does it also feel fear? Tricky question. To answer this, we need to look at the psychological representations formed in the brain by Pavlovian conditioning.

Pavlovian conditioning generates multiple representations of the world

Pavlovian conditioning has the potential to create multiple associative representations in the brain (see figure); experimental analysis has shown that CS–US pairings may cause the CS to enter into several such associations (reviewed by Dickinson, 1980; Mackintosh, 1983; Gewirtz & Davis, 1998). Thus, Pavlovian conditioning is *not* a unitary process.



Left: Pavlovian conditioning (CS–US pairing) can create multiple representations in the brain, including (1) stimulus–stimulus, or CS–US associations; (2) CS–affect associations; (3) stimulus–response, or CS–UR associations. Only a single response is shown. Dotted lines represent associative links. **Below:** blocking and transreinforcer blocking.

Blocking.

A → US

AB → US

Test response to A: substantial conditioning.

Test response to B: much less conditioning than if B had been paired with the US on its own (A already predicted the US, so learning about B was blocked by A).

Transreinforcer blocking.

A → shock

AB → omission of expected food

Test response to A: substantial conditioning.

Test response to B: reduced conditioning (B was blocked by A).

Yet A did not predict food omission! All it predicted was something unpleasant. Therefore transreinforcer blocking depends on affect (in this case, unpleasantness).

Firstly, the CS may become directly associated with the *unconditioned response* (UR), a simple stimulus–response association that carries no information about the identity of the US (e.g. Kandel, 1991). In fact, a single US may elicit several responses (for example, a US such as a puff of air delivered to the eye may elicit a simple motor act such as blinking, and a ‘central’ process such as an enhancement of arousal or attention) — any of these can potentially enter into association with the CS.

Secondly, the CS can become associated with the *specific sensory properties* of the US — including its visual appearance, sound, feel, and smell. A rigorous demonstration of this kind of representation is sensory preconditioning (Brogden, 1939), in which two neutral stimuli are first associated; one stimulus is then paired with a biologically significant US, and the other stimulus can subsequently evoke a conditioned response (CR).

Thirdly, and most important for our present discussion, the CS can evoke a representation of *affect* — such as fear or the expectation of reward. This embodies the concept of an emotional ‘tone’ that is tagged to a stimulus. It is demonstrated by the

complicated phenomenon of *transreinforcer blocking*. Blocking (Kamin, 1968; 1969) is a feature of Pavlovian conditioning in which an animal does not learn about one CS in the presence of another CS that already predicts the same US (see figure). In *transreinforcer* blocking, the presence of a CS previously paired with shock can block or prevent conditioning to a CS paired with the absence of otherwise expected food reward (Dickinson & Dearing, 1979). These two reinforcers share no common properties other than their aversiveness and therefore the blocking effect must depend upon an association between the CS and affect (emotion). Affective states can therefore be independent of the specific reinforcer and response — they are pure ‘value’ states.

So rats do have central emotional states whose presence can be inferred. As yet, I know of no neurobiological studies using the transreinforcer blocking technique, so our discussion of the neural basis of emotion in animals next lecture will have to be based on simpler behavioural and physiological measures!

Summary

We have considered, speculatively, the obvious and less obvious functions of emotions in an evolutionary context; we have examined ways of classifying and measuring emotions. We have looked in some detail at the development of theories of emotion, and the interaction between physiological arousal and emotional state. We have seen that sufficiently advanced behavioural techniques can demonstrate the existence of emotional states in animals, in the sense of affective states that are independent of environmental stimuli and overt behavioural/physiological responses (though perhaps we can never know what it ‘feels like’ to experience their emotions). Next time, we will consider the neural structures that contribute to emotional processing; we will move from there to consider motivation.

Suggested reading

For full titles see the reference list below.

- Chapters in Robbins & Cooper (1988) — out of print, but we should have copies in the Experimental Psychology library.
- Chapters in Gross (2001) — simple, but very clear
- Cardinal *et al.* (2002) — some of the learning theory, and much of the neurobiology (relevant to lectures 2 and 3). Available as a PDF at www.pobox.com/users/rudolf/publications.

Footnote

There’s always someone who manages to confuse some of the following. Don’t let it be you.

- *Affect* (verb) — to influence (also to like, or to use ostentatiously, or to assume a false appearance). ‘The results of the experiment were affected by the weather.’ Common.
- *Affect* (noun) — an emotion. ‘Transreinforcer blocking has demonstrated the existence of affect in rats.’ Rare.
- *Effect* (verb) — to bring about or accomplish. ‘A police officer may use reasonable force to effect an arrest.’ Rare.
- *Effect* (noun) — a consequence. ‘Blocking is an effect discovered by Kamin.’ Common.

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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