

WEEK 7: Effects of Aversive Stimuli: Avoidance learning, conditioned fear, and human neurosis.

Avoidance Learning

Avoidance learning occurs when an instrumental response *prevents* the occurrence of aversive events. It has been studied in rats using a “shuttle box” in which animals are trained to jump over a barrier between two compartments at the onset of a signal, which precedes floor shock in the current compartment (Mowrer and Lamoreaux, 1946) and also using “free-operant conditioning” or “Sidman avoidance”, in which animals can prevent shocks which would otherwise be delivered in a Skinner box by pressing a lever (Sidman, 1953). Although classically conditioned “species-specific defence reactions” are undoubtedly a factor in many experiments, Miller (1948) demonstrated that avoidance learning can involve more than Pavlovian anticipatory defence responses. Avoidance learning of novel responses presented a problem for Thorndikean and Hullian accounts of instrumental learning, since there is no tangible external event following a response. The “two-process” (or “two-factor”) theory of avoidance learning provided a way of reconciling the phenomena with these kinds of theories by postulating an internal reinforcing event, namely the reduction of conditioned emotional reactions.

The two-process theory of avoidance learning (Mowrer, 1940; Gray, 1975; Gray and McNaughton, 1996)

In this, avoidance learning is interpreted as a product of both Pavlovian and instrumental mechanisms. The *Pavlovian* process involves the conditioning of emotional reactions (“conditioned fear”, “conditioned anxiety”) to the signal for aversive events. The *instrumental* process is used to account for the learning of novel motor responses which have the effect of alleviating this aversive internal state. The presumed “satisfying state of affairs” representing the positive outcome of responses is internal and hypothetical, but the theory is supported by results showing that avoidance learning is improved when a response produces external changes (for instance, if jumping in a shuttle-box turns off the “warning-signal”, or if lever pressing in a Skinner box allows the animal to move to a different place; Mowrer and Lamoreaux, 1946, Baron *et al.*, 1977).

Problems for the two-process theory of avoidance learning.

1. Intermittent outcomes of the warning-signal. As conditioned emotional effects are attributed to Pavlovian processes, successful avoidance appears paradoxical, since the warning signal then no longer signals aversive events. The most obvious way around this is to argue that the effective predictor of an aversive outcome is the warning signal without a response, or that avoidance responses are followed by an internal sense of relief (Kim *et al.*, 2006)

2. Persistence of responding. Nevertheless, there are data suggesting that responses learned under avoidance procedures may persist much longer than would be predicted by a standard form of the two-process theory (Solomon *et al.*, 1953). It may be that conditioned anxiety is innately more persistent than conditioned anticipations of food (see 5. above) and that anxiety persists after a limited number of conditioning trials because the experience of conditioned anxiety itself sufficiently aversive to act as a UCS.

3. Absence of measurable emotionality. A more serious problem for the straightforward two-process theory is that motor behaviour sometime persists when there are no measurable signs of physiological indicators of emotionality (Solomon *et al.*, 1953; Herrnstein, 1969; Seligman and Johnston, 1973). In these cases it is possible to appeal to automatic habits (Mackintosh, 1983), but it is probably also necessary to allow for a more cognitive (and Tolmanian) kind of instrumental avoidance learning in which the goal of the prevention of aversive outcomes operates without high levels of classically conditioned anxiety. Herrnstein (1969) and Mackintosh (1983) infer that classically conditioned emotional effects can be ignored, but this does not square with the many cases where they undoubtedly occur (e.g. Abbott *et al.* 1984; Weiss, 1968; Wolpe, 1958).

Conditioned anxiety and human neurosis

Parallels between animal reactions to aversive stimuli and human neurosis have been drawn over many decades and continue in modern forms (Rau *et al.*, 2005; Shumake *et al.*, 2005; Delgado *et al.*, 2006). Mower’s theoretical paper in 1939 had as its main reference Freud, 1936, and Rilling (2000) suggests that the classic Watson and Rayner (1920) paper was partly responsible for popularising Freud. However Wolpe’s (1958) development of the technique of systematic desensitisation, based on his own earlier experiments with cats

(Wolpe, 1952) was in his view an alternative to psychoanalytic treatment (Wolpe, 1981). Not surprisingly there are many criticisms of conditioning theories of human neurosis, and some of these come from behavioural oriented theorists (e.g. Rachman, 1977). As with Pavlovian conditioning unrelated to anxiety, there is a case that human behaviours are mediated by conscious cognitions rather than automatic responses (e.g. Lovibond, 2003) and persistent physiological responses to experienced traumas would not account for a wide range of human neurotic symptoms. It is commoner now to appeal to explicit cognitive mechanisms as both causative and curative factors (e.g. Brewin, 1989, Clark, 1986; Brewin & Holmes, 2003; Brewin, 2003, Mineka & Zinbarg, 2006). However the irrational nature of many clinical symptoms suggest that they are less directly under cognitive control than an idealized picture of human cognition would suggest. There are also some syndromes where conscious cognitions are noticeable by their absence (e.g. "Non Fearful Panic Disorder": Kushner and Beitman, 1990; Fleet *et al.*, 1998,1999; Aikens *et al.*, 1999; Bringager *et al.*, 2004; Carmin *et al.*, 2003).

Numerous accounts of human anxiety disorders (including Post-Traumatic Stress Disorder, Panic Disorder, and phobias) have been published in the last few years, in which some role for conditioned anxiety or fear is included (e.g. Brewin, 2001; Brewin & Holmes, 2003; Bouton *et al.* 2001, Lang, Davis & Ohman, 2000; Ohman & Mineka, 2001; Mineka & Ohman, 2002; Mineka & Zinbarg, 2006).

These accounts could be regarded as extended versions of the "Two-level hypothesis" described in pages 176-170 of Lieberman (2000), in which there is a relatively automatic, largely subcortical route to fear responses involving the amygdala (Cheng *et al.*, 2003) as well as a more indirect cortical and hippocampal route, which may involve verbal memories and evaluations in the human case. (See Ohman and Mineka, 2001, page 513: *Two levels of learning in human conditioning.*)

It is one of the defining features of anxiety disorders that they are resistant to rational analysis: e.g.

"at the heart of phobia, there is a dissociation between fear and cognitive understanding that is consistent with the automaticity and encapsulation of fear characterizing the evolved module."
(Ohman and Mineka, 2001; p. 502).

However, criticism of conditioning accounts continue to come from behaviourally oriented theorists (among others) and Poulton and Menzies (2002) have argued that "evolutionarily-relevant fears", such as fear of height or fear of the dark may emerge in the absence of either direct or indirect associative learning. (But see Davey, 2002). Alternatives to conditioning explanations for anxiety disorders include theories which propose that there are marked individual differences in Anxiety Sensitivity (Beck *et al.*, 1999; Weems *et al.*, 2002; Zvolensky *et al.*, 2003) or in the emotional processing of threatening stimuli (e.g. Pflugshaupt *et al.*, 2005) or more generally in genetic sensitivity to stressful events (Leonardo & Hen, 2006).

Brewin (1989, see also 2001, 2003 and 2006) drew together both conditioning and cognitive theories and proposed that emotional stimuli are subject to both conscious and non-conscious processing, making a distinction between verbally accessible knowledge, and that which can only be recovered by exposure to situational cues. He suggests that the non-conscious system is amenable to Pavlovian conditioning and in particular can influence avoidance behaviour –

"Such a system would be able to account both for conditioning in animals and for the irrational nature of some human fears and phobias (Brewin, 1989; pp. 381-2)

"... avoidance behaviour, both in animals and humans, is often mediated by nonconscious cognitive processes that reflect the influence of prior learning about stimulus-stimulus contingencies. (Brewin, 1989; p. 391)

Brewin refers to both the conscious and the non-conscious system as types of cognition, but in the non-conscious system what he calls "situational memories" (in other words classically conditioned associations) are not accessible to or modifiable by conscious experience, while there is *in addition* the possibility of conscious reconstructions of experience that can be evaluated and changed by verbal interventions.

Brewin's approach has some similarity to the "3-systems model" of human anxiety previously used by behaviour therapists (e.g. Lang, 1968; Hugdahl, 1981), in which it is recognized that "verbal/cognitive", "emotional/physiological" and "behavioural" aspects of anxiety may have separate influences.

Foa and Kozak (1986) reviewed a wide range of therapeutic interventions designed to reduce anxiety, and argued that they contained two common factors: activation of "fear structures" in memory, since fear structures stored in memory but not accessed cannot be changed; and integration of new information, some elements of which are incompatible with the original fear structures, into them –

“It is argued that some form of exposure to feared situations is common to many psychotherapies for anxiety, and that confrontation with feared objects or situations is an effective treatment” (abstract, p.20)

In fact, although his concept of anxiety was clearly much more complex, ideas about relations between anxiety, avoidance behaviour and neurosis go back to Freud –

“... symptoms are only formed in order to avoid anxiety.... thus anxiety would be the fundamental phenomenon and main problem of neurosis.” (Freud, 1959; p.144)

“Thus we attributed two modes of origin to anxiety in later life. One was involuntary, automatic... and arose whenever a dangerous situation... had established itself. The other was produced by the ego as soon as a situation of this kind merely threatened to occur, in order to call for its avoidance.” (Freud, 1959; p. 162).

Bouton et al.’s (2001) conditioning theory of panic disorder depends on differentiating between panic and anxiety

“Any theory of PD must acknowledge the strong and growing network of evidence suggesting fundamental differences between the emotional phenomena of panic and anxiety. Panic attacks have been defined as a subjective sense of extreme fear or impending doom accompanied by a massive autonomic surge and strong fight-or-flight reaction tendencies.... Anxiety has been defined as an apprehensive anticipation of future danger, often accompanied by somatic symptoms of tension or feelings of dysphoria.

The present article provides an integrative theory of the etiology of PD that uses contemporary learning theory as its base..... we distinguish between two aversive motivational states, anxiety and panic.... we expect that a major effect of early experience with panic is the conditioning of anxiety to cues that are associated with the episode.... As is widely recognized, the classical conditioning of anxiety also makes it possible for new operant behaviours to be reinforced when they escape or reduce it.” (Bouton et al., 2001; p.7)

Apart from direct accounts of human neurosis, there continues to be behavioural and psychobiological research on fear conditioning in human subjects in more general contexts such as the study of emotional learning (e.g. Cheng et al., 2003; Morris and Dolan, 2004; Richards and Blanchette, 2004; Kosson et al., 2006; de Rosnay et al., 2006).

Sample Essay

How is “conditioned fear” relevant to accounts of human neurosis.

Main Sources

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Further Reading (Alternatives)

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Little Albert weighed 21 lbs and was 9 months old when the tests started. He was the son of a wet nurse and very stable, having almost never cried until the experiments.

At 9 months a 4 ft x ¾ in steel bar was used to generate loud noise – needed 3 bangs before a response. But on the 3rd bang Albert “broke into a sudden crying fit”.

At 11 months and 3 days a white rat was given to Albert and the steel bar banged just behind his head as soon as he touched it). One week later there were 6 more rat + noise pairings, after which Albert rapidly crawled away when shown the rat without further loud noises.

5 days later, when shown the rat, Albert fell over and crawled away.

Then, while Albert happily played with blocks, various other stimuli were presented.

There were ‘negative reactions’ to the rats, a sealskin coat, a stuffed rabbit and dog, a Santa Claus mask (white) but no crying for Watson's hair or just cotton wool

Miller (1948)

25 rats were tested in a one-way shuttle box. One compartment was white, the other black, and there was a door between them.

Procedures

1. Rats were first allowed to explore the box with the door open. *Result: there was no preference for one compartment over the other.*
2. There were then 10 conditioning trials in which they were placed in the white compartment and shocks were delivered which they could escape by running through the open door. *Result: all animals ran to the black compartment.*
3. Next there were 5 trials in which Ss were placed in the white compartment without shock with the door open. *Result: all animals ran to the black compartment.*
4. Now there were 16 trials in which Ss were left in the white compartment (with no further shocks). The door was closed but could be opened if a wheel above it was turned slightly. *Result: 13 of the 25 rats opened the door by accident during the first few trials and from then on became quicker at opening the door by turning the wheel.*
5. Finally there were 10 more trials in which the 13 animals which had learned to turn the wheel were put in the white compartment with the door closed. The wheel no longer opened the door, but pushing a bar projecting into the white compartment next to the door would open it. *Result: 12/13 Ss stopped turning the wheel and learned to push the bar to open the door. (The other rat turned the wheel 530 rotations).*

Conclusion

Rats learn to perform new instrumental responses in order to reduce fear.

Solomon, Kamin & Wynne (1953)

Ordinary extinction of shuttle-box avoidance responses

Signal was light out in current compartment plus raising of a gate above the hurdle, 10 sec before shock. 10 trials per day, 3 min apart. Training was until 10 consecutive avoidance responses, then 20 days with no shock given.

Results: i. 0/13 dogs stopped responding; ii. dogs developed ritualized behaviour; iii. speed of jumping increased; iv. signs of emotionality decreased. (Excretion, vocalization, resistance to being put in the box)

Extinction with a glass barrier to prevent jumping

The barrier was present only during the middle 4 trials each day, to enforce “reality testing”. Normal extinction was given before the glass barrier was introduced

Results: i. 7/9 dogs did not extinguish in 10 days with the barrier; ii. dogs showed emotionality when the barrier was introduced.

Extinction with shock given for jumping

13 dogs received shock for 3 sec after jumping (the gate was lowered to stop retracing).

Results: 10/13 dogs did not extinguish in 10 days (100 trials); ii. the 3 dogs that extinguished showed no spontaneous recovered i.e. did not start again after stopping jumping.

Extinction with the glass barrier and shock for jumping

16 dogs tested which had failed to extinguish in the previous procedures. The barrier was present only during the middle 4 trials each day. 3 sec of shock was given if the dogs jumped on the first 3 or the last 3 of the 10 trials given each day.

Results: i. 14/16 dogs extinguished (10 consecutive trials without jumping) within 7 days; ii. incl. all 6 which had had just the glass barrier before iii. 2 did not extinguish in 10 days.

Sidman (1953)

Journal of Comparative and Physiological Psychology, 46, 253-261.

Rats were tested in a Skinner box. If no response was made, brief shocks were given at fixed intervals e.g. every 5 seconds. This was termed the ‘S-S’ interval. If the rats pressed a lever, this delayed the next shock for a standard period e.g. 10 seconds. This interval is termed the ‘R-S’ interval.

There was no external signal for the shocks.

Individual animals were tested with a variety of settings for the two intervals.

Results: Rate of responding varied systematically with the interval settings. The maximum rate of response for a given S-S interval occurred when the R-S interval was just slightly longer than the S-S interval, and higher rates of response were found with shorter S-S intervals.

Herrnstein and Hineline (1966) and Herrnstein (1969)

Again rats could reduce the frequency of brief shocks in a Skinner box by pressing a lever. However shocks occurred **at random**, whether or not the lever was pressed. For instance, rats learned to consistently press the lever if shocks occurred **on average** every 6 sec without lever presses, but **on average** 20 sec after a lever press. There was no external signal for the shocks. Since there could be no internal timing signal either, the authors argue that the two-factor theory of avoidance learning cannot account for the results. Instead, Herrnstein (1969) argues that a reduction in the frequency of shocks is sufficient to reinforce the learned response, and there is no need to appeal to conditioned emotional effects.

TWO PROCESS THEORY OF AVOIDANCE LEARNING

(Gray, 1975; Walker, 1987)

=

TWO-FACTOR THEORY OF AVOIDANCE LEARNING

(Mackintosh, 1983; Walker 1984)

First Process or Factor

Involuntary emotional associative shifts, including physiological states and evaluative responses, which are insensitive to goal achievement.

= **Classical conditioning** e.g. anxiety induction or "Conditioned fear"

Second Process or Factor

Behavioural and cognitive responses initiated and/or reinforced by anticipated or actual achievement of emotional goals.

= **Instrumental learning** e.g. of novel motor responses which to reduce conditioned fear or anxiety.

(see Walker, 1987, pp. 218-225
or Lieberman, 2000, "The Avoidance Paradox" pp. 430-441)

Chorpita, BF and Barlow, DH (1998) The development of anxiety: the role of control in the early environment. *Psychological Bulletin*, Vol. 124, No.1, Pp.3-21.

"Current developments in cognitive and emotion theory suggest that anxiety plays a rather central role in negative emotions." [1st sentence of the abstract]

"..conditioning models.. have become increasingly relevant to traditional developmental notions of attachment..., inhibition.., and coping and resilience." [p. 3]

Examples of the conditioning models they discuss are the two below. They are in the same volume, which is housed on the periodicals shelves at Birkbeck main library, under N for Nebraska.

Gray, J.A. and McNaughton, N. (1996) The neuropsychology of anxiety: A reprise. In D.A. Hope (Ed.), *Nebraska Symposium on Motivation: Perspectives on anxiety, panic, and fear*. (Vol 43, pp. 61-134). Lincoln: University of Nebraska Press.

Mineka, S and Zinbarg, R. (1996) Conditioning and ethological models of anxiety disorders. In D.A. Hope (Ed.), *Nebraska Symposium on Motivation: Perspectives on anxiety, panic, and fear*. (Vol 43, pp. 135-210). Lincoln: University of Nebraska Press.

Rachman, S. (1977) The conditioning theory of fear-acquisition a critical examination.
Behaviour Research and Therapy, 15, 375-87.

a. many patients cannot recall crucial traumatic experiences;

b. others have lots of traumas but can deal with them -
cognitive interpretations and attributions certainly come in.

1. *Individual differences* and Eysenck's personality theory - high conditionability and sensitivity to aversive outcomes may predispose individuals to neurosis.

2. *Prepared stimuli* spiders, snakes and frogs, the dark - social disapproval, fear of failure, all these things appear to be innately associated with neurosis. This is no longer a problem for learning theory explanations.

3. *Unimportance of actual experience* - versus unconscious fears based on insecurities of arising from complex inter-personal dynamics

NB Rachman stresses lack of self-referrals for counselling about neurosis during the blitz in London and Liverpool: physical traumas did not cause neurosis.

BUT

Post-traumatic Stress Disorder (PTSD)

Gersons, B.P.R and Carlier, I.V.E (1992) "Post-traumatic stress disorder: the history of a recent concept." *British Journal of Psychiatry* 161, 742-8

Characteristic symptoms include:

i. Re-living aspects of the trauma

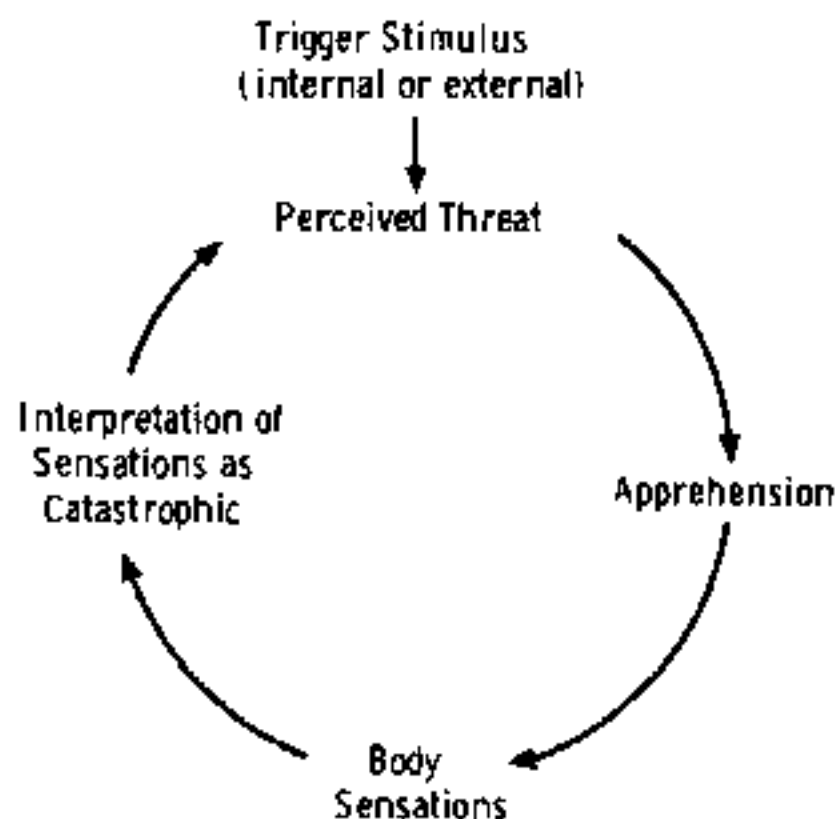
ii **Avoidance** of anything likely to remind the individual of the trauma

iii Heightened irritability including hyper-arousal, sleep disturbance and quick temper.

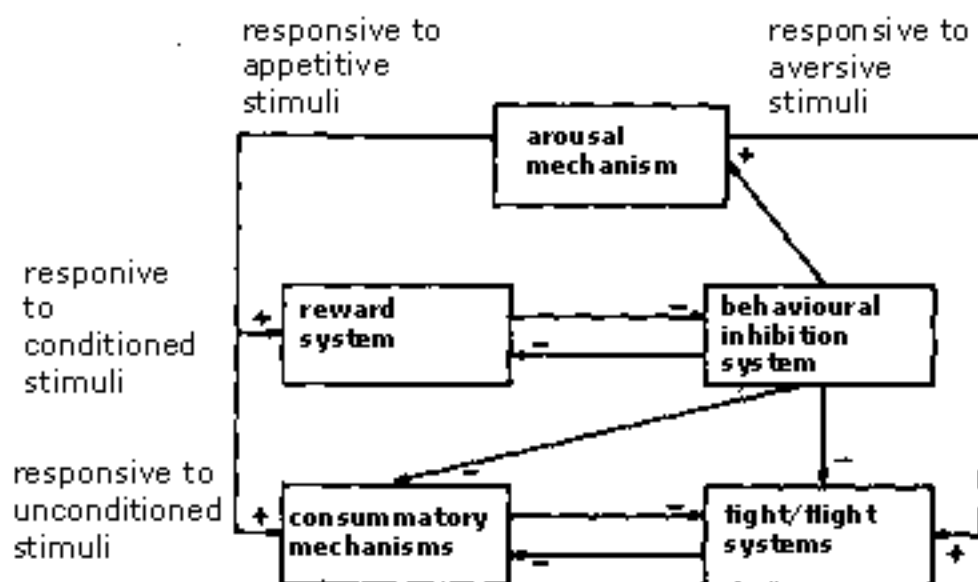
Table 2. Bodily sensations and cognitions checklist items

Cognitions		Bodily sensations	
(C1)	I am going to pass out	(S1)	Shortness of breath
(C2)	I am going to act foolishly	(S2)	Choking or smothering sensations
(C3)	I am going to have a heart attack	(S3)	Palpitations or accelerated heart rate
(C4)	I am going to suffocate	(S4)	Chest pain or discomfort
(C5)	I am going to lose control of myself	(S5)	Sweating
(C6)	I am going to scream	(S6)	Faintness
(C7)	I am going to panic	(S7)	Dizziness, light-headedness or unsteady feelings
(C8)	I am going to choke to death	(S8)	Nausea or abdominal distress
(C9)	I am going to be paralyzed by fear	(S9)	Depersonalization or derealization
(C10)	I am going to run out of air to breathe	(S10)	Numbness or tingling sensations
		(S11)	Flushes (hot flashes) or chills
		(S12)	Trembling or shaking
		(S13)	Fear of dying
		(S14)	Fear of going crazy or doing something uncontrolled

After Clark (1986) 'A cognitive approach to panic'



After Gray and McNaughton (1996)



The inhibitory (-) and excitatory (+) interactions between the various components in Gray and McNaughton's "Conceptual Nervous System". The reward system and consummatory mechanisms respond to appetitive stimuli; the behavioural inhibition and fight or flight systems respond to aversive stimuli. The middle level response to conditioned stimuli and the bottom level to unconditioned stimuli.

The '3-Systems' Model of Human Anxiety

(Lang, 1968; Foa and Kosak, 1986; Kushner and Beitman, 1990)

There are 3 components of Anxiety

1. **Physiological/emotional** (somatic symptoms)
2. **Behavioural** (actions)
3. **Verbal-cognitive** (subjective report)

Data based on the three components correlate only to a limited degree (the concept of synchrony and desynchrony between the components, with desynchrony being common in neurosis).

Foa and Kozak (1986) reviewed a wide range of therapeutic interventions designed to reduce anxiety, and argued that they contained two common factors: activation of "fear structures" in memory, since fear structures stored in memory but not accessed cannot be changed; and integration of new information, some elements of which are incompatible with the original fear structures, into them –

"It is argued that some form of exposure to feared situations is common to many psychotherapies for anxiety, and that confrontation with feared objects or situations is an effective treatment (abstract, p.20)

i.e. Therapy involves the extinction of conditioned anxiety

In fact, although his concept of anxiety was clearly much more complex, ideas about relations between anxiety, avoidance behaviour and neurosis go back to Freud –

".... symptoms are only formed in order to avoid anxiety.... thus anxiety would be the fundamental phenomenon and main problem of neurosis." (Freud, 1959; p.144)

"Thus we attributed two modes of origin to anxiety in later life. One was involuntary, automatic... and arose whenever a dangerous situation... had established itself. The other was produced by the ego as soon as a situation of this kind merely threatened to occur, in order to call for its avoidance." (Freud, 1959; p. 162).

Kushner, M.G. and Beitman, B.D. (1990) Panic attacks without fear: an overview. *Behaviour Research and Therapy*, 28, 469-479.

'Non-fearful panic disorder' (NFPD) is a condition that meets DSM-III-R criteria for panic disorder but **lacks a report of subjective fear or anxiety**. .. The authors describe a wide range of overlapping phenomena includes 'somatically expressed panic', 'non-cognitive panic'... and 'masked anxiety'. The review shows that such conditions account for from 20% to 40% of the panic disorder found in various **medical** populations and that this group resembles conventional panic disorder in statistical comparisons of age, gender and other demographic variables.

NFPD patients have attacks of intense 'discomfort' without 'fear'. They have the same sort of physical symptoms as conventional panic disorder patients (e.g. heart pounding, chest pain, faintness, trembling) but little anticipatory anxiety or subjective fears such as fear of dying or losing control.

E.g. the authors analysed data for 49 cardiology patients who cardiologists identified as having no evidence of coronary artery disease. They had less inter-attack anxiety than panic patients and were less likely to be rated as agoraphobic.

There are many case histories of people which negative physical tests, who deny psychological problems but whose physical symptoms can be relieved by anti-depressant or anti-anxiety drugs.

e.g. a surgeon who had lost a patient and his partner a few months before he started having attacks before difficult operations, but denied need psychiatric help.

Conclusion. Cognitions are **not** an invariable factor in panic attacks, and the '3-systems' or 'triple-response' view of anxiety is consistent with the syndrome.

Ost, L.-G. and Hugdahl, K. (1985) Acquisition of blood and dental phobias and anxiety in clinical patients. *Behaviour Research and Therapy*, 23, 27-34.

Studied 73 patients with phobias for the sight of blood or dental experiences.

61% of patients attributed their phobia to specific conditioning experiences.

18% cited vicarious experience.

7% gave other information

15% gave no attribution.

In previous studies these authors had found that reports of conditioning experiences occurred in:

48% of animal phobias

58% of social phobias

69% of claustrophobias

81% of agoraphobias

Brewin, C.R. (1989) Cognitive change processes in psychotherapy. *Psychological Review*, 96, 379-394. This reviews **Cognitive-behavioural therapy** methods and theory, noting that most accounts of cognitive therapy are silent on the issue of conscious vs nonconscious cognition.

Brewin's model describes **conscious and non-conscious processing of emotional stimuli**. It distinguishes between **verbally accessible knowledge**, and that which can only be **recovered by exposure to situational cues**. (Abstract)

The model uses "**Two cognitive systems**"

One is for conscious experience and is under the control of the individual.

The other is for **information processing that is not verbally accessible and is not under the control of the individual**.

Brewin, C. R. (2001). A cognitive neuroscience account of posttraumatic stress disorder and its treatment. *Behaviour Research and Therapy*, 39(4), 373-393.

Recent research in the areas of animal conditioning, the neural systems underlying emotion and memory, and the effect of fear on these systems is reviewed. This evidence points to an important distinction between hippocampally-dependent and non-hippocampally-dependent forms of memory that are differentially affected by extreme stress. The cognitive science perspective is related to a recent model of posttraumatic stress disorder, dual representation theory, that also posits **separate memory systems** underlying vivid reexperiencing versus ordinary autobiographical memories of trauma. This view is compared with other accounts in the literature of traumatic memory processes in PTSD, and the contrasting implications for therapy are discussed. (C) 2001 Elsevier Science Ltd. All rights reserved.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, 108(1), 4-32.

Several theories of the development of panic disorder (PD) with or without agoraphobia have emerged in the last 2 decades. Early theories that proposed a role for classical conditioning were criticized on several grounds. However, each criticism can be met and rejected when one considers current perspectives on conditioning and associative learning. The authors propose that **PD develops because exposure to panic attacks causes the conditioning of anxiety (and sometimes panic) to exteroceptive and interoceptive cues**. This process is reflected in a variety of cognitive and behavioral phenomena but fundamentally involves emotional learning that is best accounted for by conditioning principles. Anxiety, an anticipatory emotional state that functions to prepare the individual for the next panic, is different from panic, an emotional state designed to deal with a traumatic event that is already in progress. However, the presence of conditioned anxiety potentiates the next panic, which begins the individual's spiral into PD. Several biological and psychological factors create vulnerabilities by influencing the individual's susceptibility to conditioning. The relationship between the present view and other views is discussed. (PsycINFO Database Record (c) 2000 APA, all rights reserved)

Ohman, A., & Mineka, S. (2001). Fears, **phobias**, and preparedness: Toward an evolved module of **fear and fear learning**. *Psychological Review*, 108(3), 483-522.

An evolved module for fear elicitation and fear learning with 4 characteristics is proposed. (a) The fear module is preferentially activated in aversive contexts by stimuli that are fear relevant in an evolutionary perspective. (b) Its activation to such stimuli is automatic. (c) It is relatively impenetrable to cognitive control. (d) It originates in a dedicated neural circuitry, **centered on the amygdala**.

Evidence supporting these propositions is reviewed from conditioning studies, both in humans and in monkeys; illusory correlation studies; studies using unreportable stimuli; and studies from animal neuroscience. The fear module is assumed to mediate an emotional level of fear learning that is relatively independent and dissociable from cognitive learning of stimulus relationships.

Carmin, C. N., Wiegartz, P. S., Hoff, J. A., & Kondos, G. T. (2003). Cardiac anxiety in patients self-referred for electron beam tomography. *Journal of Behavioral Medicine*, 26(1), 67-80.

Studies have repeatedly shown that as many as 43% of patients undergoing coronary angiograms have no evidence of coronary heart disease (CHD). Fear of cardiac-related sensations has been posited as one explanation for complaints of chest pain in patients without CHD. The purpose of this study is to examine variables associated with cardiac anxiety in a sample of individuals self-referred for noninvasive coronary calcium screening. Nearly one quarter of the subjects screened experienced chest pain in the absence of coronary artery calcium (CAC). Individuals without evidence of CAC were more likely to report higher levels of heart-focused attention, even when subjects with any risk factors for CHD were excluded from the analyses. Men were more likely to have evidence of coronary calcium, although a greater proportion of women reported chest pain. Women generally endorsed higher levels of cardioprotective behavior, heart-focused attention, and fear of heart-related sensations. Findings are discussed in relation to treatment of cardiac anxiety and the prevention of unnecessary medical procedures.

Morris, J. S., & Dolan, R. J. (2004). Dissociable amygdala and orbitofrontal responses during reversal fear conditioning. *Neuroimage*, 22(1), 372-380.

The neural mechanisms underlying the persistence and plasticity of human emotional learning are unknown. Here we describe dissociable neural responses in amygdala and orbitofrontal cortex during acquisition and reversal of discriminatory fear conditioning. During acquisition, increased responses in bilateral amygdala were elicited by a face stimulus (A = CS+) predictive of an aversive noise compared to another nonpredictive face (B = CS-). With subsequent reversal of the conditioning contingency, face B (new CS+) elicited enhanced responses in right orbitofrontal cortex, while face A (old CS+) continued to evoke increased responses in right ventral amygdala. Thus, while orbitofrontal cortex exhibited rapid reversal of acquired fear responses, ventral amygdala showed a persistent, nonreversing "memory" for previous fear-related stimulus associations. (C) 2004 Elsevier Inc. All rights reserved.

Mineka, S., & Ohman, A. (2002). Phobias and preparedness: The selective, automatic, and encapsulated nature of fear. *Biological Psychiatry*, 52(10), 927-937.

We describe evidence for an evolved module for fear elicitation and fear learning with four primary characteristics. First, it is preferentially activated by stimuli related to survival threats in evolutionary history. Thus, fear-relevant stimuli lead to superior conditioning of aversive associations compared with fear-irrelevant stimuli. Second, the module is automatically activated by fear-relevant stimuli, meaning that fear activation occurs before conscious cognitive analysis of the stimulus can occur. Third, the fear module is relatively impenetrable to conscious cognitive control, and fear conditioning with fear-relevant stimuli can occur even with subliminal conditioned stimuli. Fourth, the amygdala seems to be the central brain area dedicated to the fear module. Finally, we propose that there are two levels of fear conditioning, with an emotional level that is relatively independent of the cognitive contingency level, each mediated by different brain areas. (C) 2002 Society of Biological Psychiatry.

Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders - It's not what you thought it was. *American Psychologist*, 61(1), 10-26.

The authors describe how contemporary learning theory and research provide the basis for perspectives on the etiology and maintenance of anxiety disorders that capture the complexity associated with individual differences in the development and course of these disorders. These insights from modern research on learning overcome the shortcomings of earlier overly simplistic behavioral approaches, which sometimes have been justifiably criticized. The authors show how considerations of early learning histories and temperamental vulnerabilities affect the short- and long-term outcomes of experiences with stressful events. They also demonstrate how contextual variables during and following stressful learning events affect the course of anxiety disorder symptoms once they develop. This range of variables can lead to a rich and nuanced understanding of the etiology and course of anxiety disorders.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, 108(1), 4-32.

The first sentence of this paper refers to Pavlov (1927), Watson and Rayner (1920), Mowrer (1947) and Solomon Kamin and Wynne (1953), as people very interested in the relevance of their experimental work on conditioning and learning to human neurosis.

The third sentence notes that enthusiasm for this relationship waned in the 1970s in response to critics such as Rachman (1977).

In the second paragraph they announce that “The major goal of the present article is to spell out the relevance of some contemporary work on classical conditioning, and learning theory more broadly, to understanding the etiology and maintenance of anxiety disorders with an emphasis on one of the more common anxiety disorders: panic disorder (PD).

Panic disorder

Criteria include: unexpected panic attacks without any obvious cues or triggers, *plus* anxiety about having another attack or about the implications of the attack (e.g. having a heart attack, losing control, going crazy).

Many people with PD develop agoraphobia, the fear of being in places in which a panic attack might cause major difficulties. Typically agoraphobia involves fears of clusters of situations, most broadly fear of being outside alone, and also commonly fears of being on public transport, or being in crowds, or being in shops. Agoraphobia, according to these authors, is the most severe of all phobias. However not all PD sufferers have it, and among those who do, there is a continuum from mild to severe avoidance of feared situations.

Current theories of panic disorder

Cognitive theories

Associated with Clark’s (e.g. 1986) notion of “catastrophic misinterpretations” (see page 11 of the handout).

Bouton et al. suggest that problems for the purely cognitive theory include the incidence of nocturnal panic attacks during periods of non-dreaming sleep, panic attacks during the day with no reported catastrophic misinterpretations, and the phenomena of “non-fearful panic”, in which people have physical symptoms alone, without reported subjective anxiety (e.g. Kushner & Beitman, 1990 — see page 12 of the handout)

Anxiety sensitivity theory

E.g. Reiss (1991) and McNally (1996) {neither listed on the handout but see Beck et al, 1999, Liddell and Gosse, 1998; Norton et al. 1999, Schmidt et al. 1997, Van der Does et al., 2000; Watt, Stewart and Cox, 1998}

This theory assumes that some individuals have an enduring trait like tendency to be excessively frightened by the symptoms of anxiety, because they believe that anxiety and its symptoms will have long-term deleterious consequences. This differs from the cognitive theory in that the cognitive theory has people worried about immediate impending disaster, whereas anxiety-sensitivity theory has people worried about long-term side effects.

Conditioning theories

They are on the side of conditioning theories, saying that these have “ a long and distinguished tradition of helping to understand the etiology of anxiety disorders”, quoting Eysenck (1979) and Wolpe and Rowan (1988) which are both on the handout, among others.

When stimuli, events, or situations are *paired* with a panic attack, they become conditioned stimuli capable of triggering more panic and anxiety when they are encountered again.

Bouton et al. say that an important additional idea in conditioning theory is the “fear of fear” process (see Walker, 1984, page 114) and they also mentioning the conditioning of internal physiological effects (“interoceptive conditioning”) which relates to the “3-systems model” (see page 13 of the handout).

Some pertinent Clinical Evidence (p.15 of Bouton et al, 2001)

In detailed surveys, it transpires than panic attacks to not come out of the blue, even though this claim is sometimes made in retrospective recall. 80% of panics in one study were classed as predictable, and the majority of patients report that panics arise from a preceding period of milder anxiety. Bouton et al. support the position that “an underlying apprehension is a precursor to panic”, and that expectancies of panic attacks “can be thought of as developing from a conditioned fear of panic attacks”.

Vulnerabilities for the Development of PD (page 18 of Bouton et al, 2001)

Nonspecific Biological (Genetic) Factors

E.g. the heritability of “trait anxiety” or “neuroticism”.

Prior experience with control and mastery, vs prior experience with unpredictability

Vicarious and Instrumental Learning

Bouton et al. review studies suggesting that, for instance, adults are more likely to miss work as a result of illness if they have been reinforced (e.g. special toys or food) when they were ill as children. Generally, learning experiences which encourage “sick role behaviour” are correlated with vulnerability to PD, and patients with PD are also more likely than other anxious groups to report that they had observed their parents suffering from panic symptoms.

Lecturer’s comments

This is a very long and exhaustive account of some current conditioning theories of one particular anxiety disorder. There are many alternative theories, but this paper is a useful reference for showing that conditioning theories still count as one of the options for interpreting some of the phenomena relating to human anxiety disorders.