THE EFFECTS OF ANXIOUS AROUSAL ON FEAR, FEAR REDUCTION, AND THE RETURN OF FEAR

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ABSTRACT

The purpose of this investigation was to examine the applicability of several habituation models to fear processes with special reference to the effects of anxious arousal on fear, fear reduction, and the return of fear. The effects of anxious arousal on self-efficacy expectations were also explored.

Seventy-six female undergraduate students who reported a fear of snakes and met a minimum criterion of fear on a Behavioral Approach Test participated in the study. Subjects viewed a videotaped fear reduction program under either control or anxious arousal conditions. Fear and self-efficacy expectations were assessed repeatedly during the first session. During a follow-up session one month later, subjects were re-exposed to the feared stimulus under either control or anxious arousal conditions.

Although anxious arousal did not affect fear levels within-session, experiencing anxious arousal during fear reduction impeded reduction of subjective fear and, paradoxically, resulted in less heart rate response upon exposure to the feared stimulus following fear reduction. Return of subjective fear was experienced by all of the subjects except those who experienced fear reduction while in an anxious state and follow-up assessment in a calm state. These subjects experienced a substantial decrement in self-reported fear at follow-up. There was a failure to find a relationship between anxious arousal and self-efficacy.

The results were interpreted in terms of several habituation models. It was concluded that the results are better understood in terms of emotional processing models of fear.

Novel findings include evidence that: anxious arousal during fear modification impedes the return of fear, and that assessment in a calm state, following fear reduction while in an anxious state, blocks the return of fear. These findings are theoretically and clinically important.

The implications of the results to self-efficacy theory were discussed.

The clinical implications of the findings were also explored with special reference to relapse.

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OVERVIEW

Exposure-based techniques of fear reduction are efficacious in reducing fear but there is still no acceptable explanation of their efficacy. A notable attempt is the habituation model of fear (e.g., Lader & Mathews, 1968; Lader & Wing, 1964, 1966). The habituation model of fear, as well as theory and research from several other sources, have suggested that heightened arousal during exposure may impede fear reduction and facilitate the return of fear.

The purpose of the present study was to examine the applicability of several habituation models to fear reduction with special reference to the effects of anxious arousal on fear reduction and the return of fear. The results of this study are relevent for the understanding of habituation processes, fear reduction, and the return of fear. Ultimately, theoretical and clinical benefits may be derived from this research.

The literature review begins with a discussion of processes postulated to underlie fear reduction. Common to these postulated processes is an emphasis on the role of repeated exposure to the feared stimuli. Given the similarity between fear reduction and habituation, it is suggested that an examination of the theories and data of habituation will aid in the understanding of fear reduction. Consequently, an overview of habituation is presented. Research that has a bearing on the effects of arousal level on habituation to neutral and feared stimuli is discussed. The relationship

between anxious arousal and self-efficacy is then explored. Finally, a statement of the problem and a series of predictions based on the theories and data are made.

MECHANISMS OF FEAR REDUCTION

A number of therapeutic procedures are efficacious in reducing fear and anxiety. The most common of these procedures include systematic desensitization, graduated exposure, modeling procedures, and exposure and response prevention (e.g., Emmelkamp, 1982a, 1982b; Masters & Rimm, 1987; Mavissakalian & Barlow, 1981; Rimm & Masters, 1979). Although it is impossible to make unqualified statements regarding the relative efficacy of the different therapeutic procedures, it can be stated that they all show a degree of success in reducing fear and anxiety (for example, see reviews by Barlow & Beck, 1984; Emmelkamp, 1982a, 1982b; Linden, 1981; Marks, 1975, 1978; Mathews, 1978; Rachman & Wilson, 1980). These methods vary procedurally on such variables as whether the fear inducing stimulus is presented in an in vivo or imaginal manner, the length of each exposure duration, and the degree of arousal induced in the individual during exposure.

Although these treatments differ, they all include repeated exposure to the feared stimulus. This has led a number of researchers to the conclusion that the critical element determining fear reduction is repeated exposure to the feared stimulus (e.g., Barlow, 1988; Barlow & Beck, 1984; Barlow & Mavissakalian, 1981; Foa & Kozak, 1985, 1986; Leitenberg, 1976; Marks, 1975, 1978, 1987; Wilson, 1982).

Exposure is also often a component of non-behaviorally based therapies and is recognized in clinical lore as important. For example, Sigmund Freud understood the importance of exposure as an aid to anxiety reduction:

> One can hardly ever master a phobia if one waits till the patient lets the analysis influence him to give it up ...one succeeds only when one can induce them through the influence of analysis to go about alone and to struggle with their anxiety while they make the attempt. (Freud, 1924; cited in Leitenberg, 1976, p. 400)

Although fear reduction can occur in the absence of exposure to the feared stimulus, exposure is the most effective procedure for reducing fear (Bandura, 1977; Boyd & Levis, 1983; de Silva & Rachman, 1981, 1983; Foa & Kozak, 1986; Rachman, 1990).

Exposure is a descriptive term for what occurs in treatment and does not explain the process whereby exposure is effective in reducing fear. Several explanations of fear reduction have been suggested. Earlier single-process explanations have included reciprocal inhibition (e.g., Wolpe, 1958, 1982), self-efficacy theory (e.g., Bandura, 1977), extinction (e.g., Levis & Hare, 1977), and habituation (e.g., Rachman, 1978). There is currently little agreement regarding the extent to which these explanatory mechanisms account for fear reduction (e.g., Borkovec, 1978; Levin & Gross, 1985; Rachman, 1978, 1984, 1990; Wolpe 1978). Although it is parsimonious to postulate a single explanatory mechanism of fear reduction, this may be unrealistically simplistic given the complex nature of fear (e.g., Hodgson & Rachman, 1974;

Lang, 1968, 1977b; Rachman, 1978, 1990; Rachman & Hodgson, 1974). Later theories of fear reduction have responded to this challenge and attempted to integrate findings in line with the multifactorial nature of fear and fear reduction. These theories include Lang's (e.g., 1977a, 1979, 1985) bio-informational model of fear, Rachman's (1980) theory of emotional processing, and Foa and Kozak's (1985, 1986) emotional processing model of fear reduction. Each of these models will be summarized.

Lang (1977a, 1979, 1985) suggested that information about fear is propositionally coded in the form of associative networks in memory. In the associative network of memory:

> An event is represented in memory by a cluster of descriptive propositions. These are recorded in memory by establishing new associative connections among instances of the concepts used in describing the event... The contents of consciousness are the sensations, concepts, and propositions whose current activation level exceeds some threshold. Activation presumably spreads from one concept to another, or from one proposition to another, by associative linkages between them. (Bower, 1981, p. 134)

The associative network defining a specific fear includes information about properties of the stimulus (e.g., "Spiders are hairy"), response to the stimulus (e.g., "heart pounds", "run"), and meaning information that elaborates on the stimulus and response information (e.g., "Spiders are dangerous"). The degree to which the elements comprising the network defining the feared stimulus are activated determines the likelihood that a fear response will occur. Lang hypothesizes that relative to non-fearful individuals, the associative networks defining feared stimuli in fearful

individuals have a higher degree of associative strength, and activation of fewer elements of the feared stimulus are necessary to cause a fear response. Lang suggests that alteration in the network defining a feared stimulus results in behavior change. In this view, exposure is effective to the extent that it accesses the cognitive-affective network that defines the stimulus. This is congruent with the finding that greater fear reduction occurs when there is greater physiological arousal in response to initial stimulus exposures, which presumably reflects accessing of the network representing the feared stimulus (Dyckman & Cowen, 1978; Glenn & Hughes, 1978; Lang, Melamed, & Hart, 1970; Levin, Cook, & Lang, 1982; Marshall, 1988; Stern & Marks, 1973; Vermilyea, Boice, & Barlow, 1984). Lang does not focus attention on the specific mechanisms of behavior change. Fear reduction is said to occur as a result of a weakening of the associations between the stimulus, meaning, and response elements of fear, and the development and elaboration of other non-fearful associations.

Lang's (1977a) analysis of fear imagery was the impetus for Rachman's (1980) theory of emotional processing. Rachman noted that the origins of emotional processing can be traced to Freud's well known case of Anna O. Freud argued that because of circumstances Anna O. was forced to suppress emotion over the illness and death of her father. This emotional suppression was supposedly responsible for the wide range of neurotic symptoms experienced by Anna O. Emotional

processing refers to, "a process whereby emotional disturbances are absorbed and decline to the extent that other experiences can proceed without disruption" (Rachman, 1980, p. 51). The core concept of emotional processing derives from findings indicating that fear reduction is enhanced by the experience of emotion during exposure to the feared stimulus (e.g., Lang, Melamed, & Hart, 1970). Rachman suggested that reductions in fear will not be maintained if emotional processing is not complete. Direct indications of unsatisfactory emotional processing include undue persistence of fear, unprovoked return of fear, and the incubation of fear. A large number of possible factors that may facilitate or disrupt emotional processing were suggested by Rachman. Notable among the state factors that may influence emotional processing are high arousal and dysphoria, which may hinder emotional processing; and relaxation, which may facilitate emotional processing.

Foa and Kozak (1985, 1986) extended Rachman's notion of emotional processing and more fully integrated Lang's (e.g., 1985) bio-informational theory into their model of emotional processing. Their definition of emotional processing differs from Rachman's (1980) in that they suggest that the term refers to the incorporation of any new information into the fear structure regardless of its effects on fear. Central to their explanation of fear reduction are two processes: fear reduction within-session and between-session. They suggested that within-session fear reduction is the result of

habituation of autonomic responsiveness. Within-session fear reduction is postulated to have two causes: dissociation of the fear response from the stimulus and the incorporation of more adaptive responses; and a decrease in the probability and valence of perceived threat associated with the feared stimulus. The second habituation process, between-session habituation, is dependent on durable changes in cognition. Specifically, the meaning ascribed to the feared stimuli must change in order for between-session habituation to occur. These changes are accomplished through the process of within-session habituation. The most common beliefs that are necessary to be modified include the belief that the fear response will last indefinitely, and the fear of fear (e.g., Clark, 1986). Foa (1979), and Foa and Kozak suggest that between-session habituation of fear is dependent on within-session habituation; however, the occurrence of within-session habituation does not ensure between-session habituation. In other words, in their model within-session habituation is a necessary, but not sufficient, condition of long term habituation. Failures in emotional processing can occur either as the result of a failure of the stimulus exposure to sufficiently activate the fear structure underlying the feared stimulus, or because of a failure of the stimulus exposure to allow information that disconfirms the threat.

The importance attached to repeated exposures to the feared stimulus by all of the above theories of fear reduction suggests that habituation, which is defined as a decrease in

responding as the result of repeated stimulus presentation, may be a viable explanation of the fear reduction process. There are consistent data indicating that the process of exposure-based fear reduction occurs in a manner akin to habituation in the subjective report and autonomic components of the fear response (e.g., Connolly, 1979; Foa, 1979; Foa & Chambless, 1978; Klorman, 1974: Lande, 1982; Lang, Melamed, & Hart, 1970; Parkinson & Rachman, 1980). The first attempt to consider fear reduction as an habituation process was made by Lader and Wing (1964, 1966) and later elaborated by Lader and Mathews (1968). Although the similarity between the fear reduction process and habituation has not gone unnoticed by later researchers in the area of fear reduction, with very few exceptions (e.g., Rachman, 1978, 1990; Watts, 1979) they have not considered the mechanisms hypothesized to underlie habituation that have been developed in other areas of psychology. Given that the roots of behavioral approaches to fear reduction are derived from experimental psychology, it seems appropriate to consider the data and theories of habituation. It is likely that an examination of this research and theory will allow an integration of habituation theory into the area of fear reduction. This should result in an increased understanding of the fear reduction process

HABITUATION

Habituation can be defined most simply and generally as a decrease in responding as the result of repeated stimulus presentations (e.g., Harris, 1943; Thompson, Berry, Rinaldi, &

Berger, 1979; Thorpe, 1963). Response decrements due to extremely rapid stimulation (which may result in sensory adaptation or motor fatigue), trauma (Harris, 1943; Hinde, 1970; Thompson & Spencer, 1966), or drugs (Thompson et al., 1979) are excluded from the definition of habituation. Habituation has been recorded at all phylogenetic levels ranging from single-celled organisms to humans, and across responses ranging widely in function and complexity. The vast majority of research has examined habituation in nonhuman animals. Habituation research with humans has been almost limited to studies of physiological response to auditory tones. The ubiquitousness of the habituation phenomenon does not denote that its underlying mechanisms are similar across species or responses (Mackintosh, 1987; Petrinovich, 1984; Thompson, Groves, Teyler, & Roemer, 1973).

Habituation can be considered as a form of conditioning (Kimmel, 1973; Stephenson & Siddle, 1983) and may provide further understanding of more complex varieties of learning (Groves & Thompson, 1970; Stephenson & Siddle, 1983). Although most studies have examined reflex behavior, the term habituation can also correctly be used to refer to decreased responding of conditioned responses (Thompson & Spencer, 1966; Thorpe, 1963).

Although studies of habituation typically present the subject with stimuli in the form of a series of discrete trials, uninterrupted stimulus presentations can also be used to study habituation (Hinde, 1970).

Historically, response decrement due to habituation has been considered as a phenomenon that spontaneously recovers in a very short period of time following stimulus termination (i.e., within minutes). Although most studies neglect the assessment of long term habituation, even phylogenetically simple animals can exhibit significant response decrement over much longer periods. For example, Carew, Pinsker, and Kandel (1972) reported significant maintenance of habituation of the siphon withdrawal reflex in Aplysia (a bivalve mollusk) over a period of three weeks. Leaton and Jordan (1978) found evidence of continued habituation of EEG activity in rats in response to tones 32 days following habituation.

Habituation and extinction are similar processes to the extent that they are both characterized by a gradual decrease in response followed by a degree of spontaneous recovery (Kling & Stevenson, 1970). Also, both occur as a result of experience and can be relatively stable and context-specific (Peeke & Petrinovich, 1984). Beyond this, however, it is very difficult to compare these two processes as they are procedurally different. Extinction refers to response decrement in learned responses as the result of repeated presentations of a conditioned stimulus without its unconditioned stimulus (in classical conditioning), or repeated occurrences of an operant response that are not followed by positive or negative reinforcement (in instrumental conditioning). Habituation training involves repeated presentations of the same stimulus and is a simpler

procedure than extinction. Although habituation is typically studied with unconditioned stimuli, learned responses are not explicitly excluded from the definition of habituation. There is not presently sufficient evidence to unambiguously establish the extent to which extinction and habituation reflect the same process. The finding that extinction may be more permanent than habituation is usually recognized to be, suggests that the two processes may be fundamentally different.

A number of parametric properties of habituation have been summarized in reviews of this literature (e.g., Carew, 1984; Hinde, 1970; O'Gorman, 1977, 1983; Graham, 1973; Siddle, Stephenson, & Spinks, 1983; Stephenson & Siddle, 1983; Thompson & Spencer, 1966). Although there is a certain degree of variance in the specific parameters of habituation across responses and species, for the most part these properties exhibit a large degree of generality. Several theories have been developed to explain habituation. Two theories of habituation that have stimulated a considerable amount of research attention, and that are often contrasted with each other, are the dual process theory developed by Thompson and his colleagues (e.g., Groves & Thompson, 1970; Thompson et al., 1979; Thompson et al, 1973; Thompson & Spencer, 1966), and a class of theories that have as their common explanatory element the development of a cortical model of the stimulus (e.g., Sokolov, 1963; Sokolov, 1968; Sokolov & Vinograda,

1975; Wagner, 1976; Whitlow, 1975; Whitlow & Wagner, 1984). These theories will be discussed in the following sections.

DUAL PROCESS THEORY OF HABITUATION

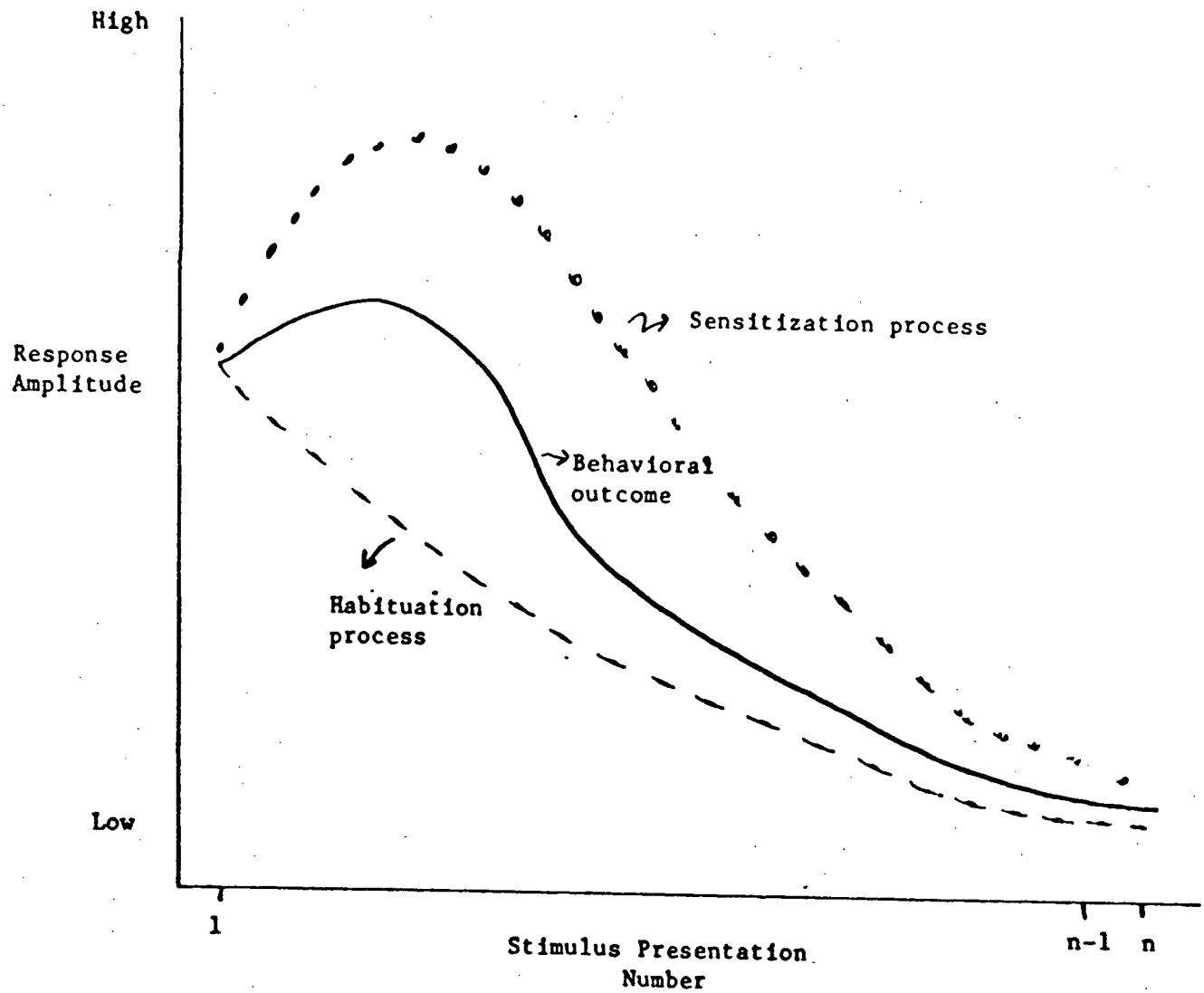
Dual process theory (Groves & Thompson, 1970; Thompson et al., 1979; Thompson et al., 1973) was developed in order to explain the parametric properties of habituation summarized in Thompson and Spencer (1966). Thompson and his colleagues contended that these properties could not be explained by any single process theory of habituation. Rather, habituation was thought to be the result of two processes: the habituation process, which occurs in the stimulus-response pathways; and the sensitization process, which occurs in the "state system" and is analogous to level of arousal. Groves and Thompson (1970) stated that any stimulus that is capable of evoking a behavioral response has two properties: it elicits a response, and it affects the arousal level of the animal exposed to the stimulus. Behavioral response is the result of the interaction of these two inferred processes. Repeated stimulus presentation results in a decrease in the strength of the stimulus-response associations (termed the habituation process). This attenuated association between stimulus and response recovers over time. However, it has a degree of permanence, especially after extended stimulus presentations. Stimulus exposure also results in sensitization (i.e., an increase in arousal). This sensitization initially increases and then decreases across stimulus exposures. The degree of sensitization is a direct function of stimulus intensity.

Sensitization is relatively transient and decays spontaneously. Sensitization has a general effect on responding, and does not only influence response to stimuli being habituated.

Dual process theory argues that overt behavioral responding is the result of the interaction of the two inferred processes of habituation and sensitization. Understanding of this theory may be aided by a visual representation of these processes.

As shown in Figure 1, behavioral outcome is the result of the interaction of the decremental habituation process and the sensitization process, which is initially incremental and later decremental (i.e., there is habituation of the sensitization process in response to repetitive stimulus exposures). A major impetus in the development of the dual process theory was the phenomenon of dishabituation. Dishabituation refers to the consistent finding that an habituated response will recover following exposure to a novel, or otherwise arousing, stimulus. Thompson et al. (1979) summarize three findings based on the dishabituation paradigm: both habituated and non-habituated responses increase in magnitude following exposure to a novel stimulus; exposure to a dishabituating stimulus may increase responsiveness of an habituated response above baseline level; and the dishabituated response spontaneously decays to its previously habituated level in the absence of further habituation training. Thompson et al. (1979) argue that these results

Figure 1
Habituation and sensitization processes



cannot be explained with a unitary explanatory process, but are congruent with dual process theory. Within their model, the term "dishabituation" is a misnomer. Instances of response recovery in the dishabituation paradigm are the result of the influence of the superimposed process of sensitization/arousal on the habituation process.

The dual process model is a common-elements model. Generalization of habituation is regarded as a function of the amount of correspondence in the elements defining the stimuli. This degree of correspondence is paralleled in the central nervous system in terms of the extent of overlap in the specific interneurons in the stimulus-response pathway excited during exposure.

CORTICAL MODEL THEORIES OF HABITUATION

These models were developed by Sokolov (1963, 1969; Sokolov & Vinograda, 1975), and Wagner and Whitlow (Wagner, 1976; Whitlow, 1975; Whitlow & Wagner, 1984).

Sokolov's model focuses on habituation of the orienting response. The orienting response is a response to novelty or environmental change (Sokolov, 1963). It is a non-specific response to stimuli of low to moderate intensity, and its purpose is to maximize the organism's ability to perceive and react to environmental change (Sokolov, 1963). Components of the orienting response include increased receptor sensitivity, modification of skeletal muscles responsible for directing the receptors, alterations in general skeletal musculature, increased electroencephalographic arousal, and autonomic

changes (e.g., increased galvanic skin response) (Lynn, 1966). During initial exposures to the stimulus, the orienting response is of a generalized nature. After repeated exposures, however, the orienting response becomes progressively more localized and specific to the stimulus. Sokolov argues that repeated exposures to a specific stimulus results in the development of a cortical model of the stimulus. This cortical model includes stimulus parameters such as stimulus duration, intensity, and time interval between stimulus exposures. With repeated exposures, the cortical model becomes increasingly stronger and well-defined. Each stimulus presentation results in a comparison of the stimulus with the "expected" neural model. A mismatch between the stimulus and the cortical model results in cortical arousal and, as a result, excitation of the reticular formation, resulting in an orienting reaction. Given a match between the stimulus and the cortical model, the cortex does not send excitatory impulses to the reticular formation and it inhibits other impulses from the collateral afferents descending to the reticular formation. Thus, in Sokolov's model of habituation, habituation of the orienting response is the result of inhibition of the reticular formation by the cortex as the result of congruence between the stimulus and the continually developing cortical model of the stimulus. The degree of habituation is directly proportional to the degree of similarity between stimulus properties and properties of the "expected" cortical model. Sokolov's earlier writings were vague with respect to the

manner in which the "expected" cortical model was differentiated from the myriad other cortical models. He later postulated the existence of two memory systems: operative memory, "which stores the system of hypotheses that are being verified in the experiment" (Sokolov, 1969, p. 698), and a long term memory store to explain how the expected cortical model was isolated from the other cortical models.

Like Sokolov's model of habituation, the model developed by Whitlow and Wagner (Wagner, 1976; Whitlow, 1975; Whitlow & Wagner, 1984) also postulates the development of a cortical model as central to the explanation of habituation. However, unlike Sokolov's model, it is a general model of habituation, and places greater emphasis on memory processes. The theory uses an associative network model of memory (e.g., Bower, 1981; Gilligan & Bower, 1984; Lang, 1977a, 1979). In the following discussion, the elements defining the stimulus representation in memory (corresponding to Sokolov's concept of cortical model) will be referred to as a memory node.

The latest version of this model (Whitlow & Wagner, 1984) postulates three levels of memory: inactivity, corresponding to the more common term "long term memory"; and two states of activity or short term memory - primary and secondary. When a stimulus is perceived by the organism, a representation of the stimulus is stored in the primary-active memory state. This representation then decays into the secondary-active memory state and finally, into the inactive memory state. Both active memory states are of limited capacity and, as this capacity is

approached, more recent or salient information displaces the earlier information from the primary-active memory state to the secondary-active memory state to the inactive memory state.

Activation of the memory node representing a stimulus to the primary-active memory state only occurs to the extent that the node is in the inactive memory state at the time of exposure. If the memory node representing a stimulus is in the primary-active memory state at the time of stimulus exposure, this exposure will not have any further effect on the organism as the representation is redundant. If the memory node representing the stimulus is in the secondary-active memory state at the time of stimulus exposure, the stimulus is ineffective in activating the memory node to the primary-active memory state as memory nodes cannot be recalled from the secondary-active to the primary active memory state.

The principle that a memory node cannot be recalled from the secondary-active to the primary-active memory state is the central feature of habituation in this model. Habituation occurs when a greater than zero number of elements defining the node representing the stimulus are in the secondary-active memory state at the time of stimulus exposure. Elements that are in the secondary-active memory state cannot be recalled to the primary-active memory state. This results in a weakened representation of the stimulus being recalled to the primary-active memory state. As a result of the weakened stimulus representation, the associated response is also weakened.

Whitlow and Wagner contend that there are two ways in which stimuli can be represented in the secondary-active memory state at the time of stimulus exposure, thus causing response habituation:

1. Self-generated priming - which occurs as a result of a recent presentation (which has not yet decayed from secondary-active memory) of the same stimulus.

2. Retrieval-generated priming - which occurs as a result of previously learned associations between the stimulus and the environmental context in which it occurred. After repeated associations between the context and stimulus, the context by itself primes the memory node representing the stimulus to secondary-active memory.

In this model, the dishabituation phenomenon is thought to be the result of the interpolated stimulus displacing the representation of the stimulus from the secondary-active to the inactive memory state. As a result of this displacement to the inactive memory state, at the next stimulus exposure the memory node representing the stimulus will be available for recall from inactive memory to primary-active memory and, as a result, there will be recovery of the associated response.

Although many studies over the last 25 years have examined the parameters of habituation, few conclusions can be drawn regarding the ability of the theories to explain the results. Several researchers (Groves & Thompson, 1970; Mackintosh, 1987; Stephenson & Siddle, 1983; Thompson et al., 1979) have summarized this research in the context of these

theories of habituation. In lieu of an expanded discussion of these findings, pertinent comments and conclusions made by these reviewers will be presented.

In their comparison of dual process theory and Sokolov's theory of habituation, Groves and Thompson (1970) and Thompson et al. (1979) suggested that although the neural mechanisms postulated to underlie habituation differed, the theories were fundamentally similar. They argued that many of the apparent theoretical differences were simply the result of differences in the language used to describe the theories. Although Groves and Thompson are correct in stating that many of the apparent differences between these theories may be semantically based, it is incorrect to assume that they are fundamentally similar theories. For example, central to dual process theory is the notion that dishabituation is a transient phenomenon that results from sensitization. Conversely, Sokolov (as well as Whitlow and Wagner) view dishabituation as the result of a disruption of habituation. It may be difficult to reconcile these views.

Stephenson and Siddle (1983) undertook a thorough review of the habituation literature with the goal of evaluating four theories of habituation, including the three theories discussed, and concluded that statements regarding the relative efficacy of these theories were precluded for several reasons. Firstly, they noted that there is a dearth of unambiguous and reliable data with respect to differential predictions made by the theories. Secondly, the authors noted

a lack of precision in the theories, especially in the cortical model theories. Finally, the authors noted that because of the semantic differences of the theories it is difficult to determine the extent to which they encompass common processes and explanations of habituation. Stephenson and Siddle (1983) were not optimistic that a synthesis of these theories was currently possible:

> Given the present state of the theories discussed and the inadequacy of the data available, it remains to be seen whether they can be integrated into a single coherent account of habituation, or indeed, whether such a general theory is appropriate. (p. 230)

Mackintosh (1987) reviewed previous data that had been gathered in support of Whitlow and Wagner's (e.g., 1984) theory of habituation and as evidence against dual process theory (e.g., Groves & Thompson, 1970). He argued that these results are open to alternative explanations. Specifically, these findings can be explained equally well, and more parsimoniously, within the context of dual process theory. On the basis of his review and re-analysis of these findings, Mackintosh (1987) concluded that:

> no data or arguments have been advanced which should persuade us to accept this apparently more complex theory, either in the particular form proposed by Wagner (1976, 1978) or in a more general form. In the absence of compelling data or argument, we should either reject the theory, or acknowledge that it is only a re-description, in more fanciful language, of the simple S-R theory. (p. 95)

In summary, current consensus is that the available data do not differentially support any of the previously described theories. However, given the clarity and parsimony of dual

process relative to the other two theories, it seems most appropriate at this time to consider habituation within the framework of this model.

EFFECTS OF AROUSAL ON HABITUATION

Theories differ in the emphasis given to the effects of arousal on response habituation. Dual process theory (e.g., Groves & Thompson, 1970) stresses the importance of arousal, which is said to be central to the explanation of habituation. In this conceptualization of habituation, arousal during exposure results in an increase in responsiveness. However, because the arousal masks the inferred habituation process, any differences in the degree of habituation between individuals who are more and less aroused will not be evident except at dissimilar levels of arousal at the time of assessment. In other words, arousal at the time of assessment should influence response, with greater arousal resulting in greater responsiveness.

Although Lader and his colleagues (Lader & Mathews, 1968; Lader & Wing, 1964, 1966) do not have a fully developed theory regarding habituation, they discuss habituation in the context of treatment of anxiety disorders. They also postulate a central role to arousal in the process of habituation. In their model, which postulates a single process of habituation, higher levels of arousal, above some critical criterion level, result in a disruption of the habituation process. In this model, heightened arousal results in an actual impairment of

habituation that will be evident on later assessment regardless of the level of arousal at that time.

The two cortical model theories of habituation (e.g., Sokolov, 1963; Whitlow & Wagner, 1984) do not accord any significant role to arousal. However, predictions can be gleaned from their discussions of the theories. Although Sokolov does not discuss the role of arousal on habituation, he does argue that low levels of cortical arousal result in recovery of an habituated response. This occurs because lowered cortical arousal results in an impairment of the cortical mechanisms responsible for stimulus-model comparisons. As the cortical model of the stimulus is not well developed with lowered arousal levels, the cortex exerts less depression of the reticular activating system which is responsible for the orienting response. In an analogous manner, it can be argued on the basis of this model that higher levels of cortical arousal would allow a better model of the stimulus to be developed in the cortex, resulting in a corresponding inhibition of response to the stimulus.

On the basis of Whitlow and Wagner's (1984) model of habituation, it would be predicted that to the extent that arousal results in a re-direction of attention away from the stimulus and toward the environment or bodily symptoms of arousal, arousal may disrupt habituation (Wagner, 1981; Whitlow & Wagner, 1984). Furthermore, according to this model, to the extent that arousal functions as a cue to prime the stimulus into short term memory, greater maintenance of

habituation would be expected with similar levels of arousal at habituation and assessment. This focus on contextual cues is similar to the focus of theory and research that have examined the effects of affect on memory, (e.g., Blaney, 1986; Bower, 1981, 1987; Clark & Teasdale, 1982; Clark, Milberg, & Ross, 1983; Singer & Salovey, 1988; Teasdale, 1983; Teasdale & Fogarty, 1979). The major differences are that Whitlow and Wagner (1984) focus on contextual, as opposed to emotional cues, and focus on habituation, as opposed to memory. Differences in the type of cue utilized - contextual versus affective - may prove to be of little importance. For example, Eich (1989) has suggested that the influence of contextual cues on memory may be the result of differences in the affective valence associated with the different contexts. The role of affect on memory will be briefly considered.

Research on the effects of affect on memory has been conducted within the context of an associative network model of memory (e.g., Bower, 1981). The phenomenon of mood dependent retrieval "implies that what one remembers during a given mood is determined in part by what one learned (or focused on) when previously in that mood; the affective valence of the material is irrelevant" (Blaney, 1986, p. 229). This is thought to occur because re-experiencing the emotion will result in a spread of activation in memory from the elements representing the emotion to the previously learned information. This increases the probability that this information will be recalled. This effect has been well

documented; however, a number of researchers (Blaney, 1986; Bower, 1987; Eich, 1980, 1989; Overton, 1985) have noted that there have also been a number of failures to document this effect. These inconsistent findings can be explained in large part by the presence or absence of other retrieval cues in the environment (Eich, 1980; Fiedler & Stroehm, 1986). Mood dependent recall effects are much more apparent when the individual is unable to rely on alternate retrieval cues. In a related vein, Eich (1989) has suggested that memory for internally generated information is much more sensitive to changes in mood than memory for externally generated information. Within the context of habituation of fear, predictions derived from theory on mood-dependent recall would be analogous to those based on Whitlow and Wagner (1984). Specifically, it would be predicted that there would be greater memory of the previous fear reduction session given that the individual experienced congruent states of arousal during fear reduction and at follow-up. However, consideration of the phenomenon of mood congruence suggests that this prediction may not be entirely supported. The mood congruency effect assumes that information is better learned and recalled when the affective valence of the information to be learned/recalled matches that of the individual's current affect (Bower, 1981). Mood congruency effects have been documented in a number of studies (e.g., see review by Blaney, 1986).

As can be seen from the above discussion several differential predictions regarding the role of arousal on habituation can be made. A number of studies have examined the effects of arousal on habituation of the orienting response in humans. These studies will be reviewed in the following section.

Four different paradigms have been used to differentiate more and less aroused subjects:

1. Comparison of normal subjects who differ with respect to their levels of self-reported trait anxiety or neuroticism. The rationale underlying this methodology is that individuals who report higher levels of trait anxiety and/or neuroticism are more highly aroused than less anxious and/or less neurotic individuals. Although this assumption may often be correct it is not invariably the case that higher levels of trait anxiety and/or neuroticism reflect over-arousal (Eysenck, 1977; Lader, 1975, 1979).

2. Comparison of normal subjects with subjects diagnosed as clinically anxious. Studies utilizing this approach are based on the same assumption as in 1 above.

3. Comparison of normal subjects who differ with respect to their resting physiological level.

4. Experimentally inducing differential levels of arousal.

<u>Comparison of normal subjects who differ with respect to their levels of self-reported trait anxiety or neuroticism</u>

At least eight studies have compared normal subjects who differed with respect to their scores on self-report measures of anxiety or neuroticism. The results of these studies have been inconsistent. While several studies have found significantly greater habituation of physiological response in less anxious individuals (Coles, Gale, & Klein, 1971; Jackson & Berry, 1967; McGuiness, 1973; Sadler, Mefferd, & Houck, 1971), other studies have not found any effect using similar measures of physiological arousal (Chattophyay, Cooke, Toone, & Lader, 1980; Katkin & McCubbbin, 1969; Koepke & Pribram, 1966; Neary & Zuckerman, 1976). The effect of individual differences in trait anxiety or neuroticism among normal individuals is not very robust. For two major reasons this is not surprising. Firstly, given that subjects were selected from a relatively homogeneous population of university undergraduate students (Chattopadhyay et al., (1980) is the possible exception), there was not likely a large difference between the more and less anxious subjects. Secondly, and most importantly, subjects' anxiety level was assessed using measures that purport to measure stable trait characteristics. Given that arousal or anxiety level is principally a function of current environmental and interpersonal stressors (e.g., Spielberger, 1972), the consistency of the experimental situation across subjects would likely overset any differences in trait anxiety. Assessment of current anxiety or arousal levels would likely prove more appropriate.

Comparison of normal subjects with subjects who are

diagnosed as clinically anxious

Several studies conducted by Lader and his colleagues (Chattophyay et al., 1980; Lader, 1967; Lader & Wing 1964) and Raskin (1975) have compared the habituation rates of normal subjects and patients with anxiety disorders. The earlier studies by Lader (Lader, 1967; Lader & Wing, 1974) and Raskin (1975) are consistent in reporting that anxious patients are more physiologically aroused (as assessed by resting skin conductance level and the number of spontaneous fluctuations) than normal, nonanxious subjects. The anxious patients' galvanic skin response amplitude habituated more slowly than the normal subjects'. Although the amplitudes of the responses to the initial tones were not consistently different (Lader, 1967, Lader & Wing, 1964; Raskin, 1975), the normal subjects' galvanic skin resistance response during the later trials was significantly smaller than the anxious patients' (Lader and Wing, 1964), and a significantly greater proportion of normal subjects were defined as "habituators" based on their rate of response decrement across trials (Lader & Wing, 1967; Raskin, 1975). Lader (1967) compared the habituation rates of patients with different anxiety disorders. He found that patients with specific phobias had a significantly lower resting level of anxiety, and a faster rate of habituation to neutral stimuli than subjects experiencing more pervasive and generalized varieties of anxiety (e.g., agoraphobia and generalized anxiety disorder). This is not surprising given that the more generally anxious patients were more likely experiencing

anxiety during the experimental session than the patients with specific phobias who were not likely being exposed to fear-evoking stimuli at the time of assessment.

Hart (1974) and Chattopadhyay et al. (1980) also compared the responses of patients to a series of auditory tones. Both studies failed to replicate most of the findings of Lader (1967), Lader & Wing (1964), and Raskin (1975). For example, Hart (1974) found that although anxious patients exhibited a greater number of spontaneous skin fluctuations than normal subjects, their resting heart rate and skin conductance level were not different. The two groups were not significantly different with respect to galvanic skin response habituation rate or the number of subjects who are were defined as "habituators". The only significant difference between the two groups concerned the habituation of initial heart rate deceleration in response to lower intensity tones. Normal subjects displayed significant habituation of the heart rate decelerative response. Anxious subjects, however, initially responded with a lesser decelerative response that did not habituate across trials.

Given the vagueness of the diagnostic criteria used to define the patient groups, it is not possible to determine the extent to which differences in population characteristics may be responsible for differences in the obtained results. Beyond this, however, there are several other methodological differences that may explain the results (Sartory, 1983). For example, whereas Lader (1967; Lader & Wing, 1964) and Raskin

(1975) used the same stimuli across trials, Hart (1974) used stimuli of three different intensities.

Although further research is necessary to clarify the parameters of this effect, it can be tentatively concluded that subjects defined as anxiety disordered may be impaired relative to normal subjects with respect to their rate of habituation to neutral stimuli. The inconsistencies of these results stress the necessity of consistency and completeness in defining subject samples and method variables.

Comparison of subjects who differ with respect to their resting physiological level

Several studies have compared the responses of individuals differing in their resting level of physiological arousal. Physiological arousal was defined in terms of the number of spontaneous skin fluctuations (Deitz, 1982; Katkin & McCubbin, 1969; Koepke & Pribram 1966) or resting skin conductance level (Thayer & Silber, 1971). The results of the studies are consistent in finding that although more highly aroused subjects do not differ from low aroused subjects in terms of initial amplitude of the galvanic skin response, they habituate in a significantly greater number of trials (Deitz, 1982; Koepke & Pribram 1966) and to a significantly lower amplitude (Katkin & McCubbin, 1969; Koepke & Pribram, 1966).

Experimentally inducing differential levels of arousal

The most commonly used paradigm for examining the effects of arousal level on habituation has modified arousal level with various experimental manipulations. Procedures for

manipulating arousal have included sleep deprivation (Bohlin, 1973), changes in body position (Goldwater, 1987; Goldwater & Lewis, 1978), exposure to loud sounds (Epstein, 1971), engagement in motor responses or in mental/attention tasks (Hulstijn, 1978), threat of examination (Kimmel & Bevill, 1985), or threat of shock (Bohlin, 1976; Carrol & Pokora, 1976; Chattopadhayay et al., 1980; Gatchel & Gaas, 1976; Gatchel, Gaas, King & McKinney, 1977; Watts, 1975).

With the exception of Bohlin (1973), who attempted to manipulate arousal through sleep deprivation, these studies have been successful in manipulating resting physiological arousal level. Furthermore, the threat of shock has also been shown to increase the level of self-reported stress (Briush & Hendrix, 1980; Briush & Schwartz, 1980; Carroll & Podora, 1976; Chattopadhyay et al., 1980; Watts, 1975). Because the results of Bohlin (1973) suggested that the arousal manipulation was not effective in altering arousal level, the results of this study will not be included in the following discussion. Examination of the remaining studies allows one to make several tentative comments. Although many of these studies have found that arousal has significant effects on habituation, the pattern of results varies both between studies and within the same study when considering different assessment measures. For example, with respect to the habituation of the magnitude of the skin conductance response, Bohlin (1976) found that heightened arousal resulted in a slower rate of habituation, but to the same level as with

lower arousal levels. On the other hand, Carrol and Pokora (1976) found that aroused and non-aroused subjects responded similarly on initial trials, but that on later trials the aroused subjects' responses were of a greater magnitude than the non-aroused subjects'. Gatchel and Gaas (1976) found that aroused subjects initially responded with higher magnitude skin conductance responses than non-aroused subjects, and habituated to a similar level but at a slower rate, and Goldwater (1987) and Hulstijn (1978) found that arousal level had no effect on habituation of the magnitude of the skin conductance response.

Although a number of studies report that the experimental manipulation of arousal significantly influences habituation, there are also several notable exceptions. Although there are several methodological differences that may account for these results, the most consistent difference seems to be related to the specific type of manipulation used. With the exception of Chatopadhyay et al. (1980), all of the studies that manipulated arousal using the threat of shock or examination identified one or more detrimental effects of arousal on habituation. (Unfortunately, the study by Chattopadhay et al (1980) is not sufficiently described to allow comparison with the other studies that manipulated arousal through the use of threat.) On the other hand, the effects of the other arousal manipulations are apparently less robust in their influence on habituation of physiological responding. Although unqualified statements regarding the differences between the threat of

shock or examination and other paradigms cannot be made at the present time, two possible explanations of this difference may be suggested. Firstly, perhaps threat of shock or examination exerts a more potent effect, or a different pattern of effects on physiological arousal than do the other arousal manipulations. Secondly, it is reasonable to expect that the threat of shock or examination may have other effects on cognitive or affective processing that do not occur with other arousal manipulations. For example, it seems self-evident that the threat of shock would be much more likely to result in physiological arousal and negative affect than standing (e.g., Goldwater & Lewis, 1978) or pressing a dynamometer (Hulstijn, 1978). In fact, while the arousal generated by the last two tasks would be primarily of a physiological nature, threat would produce physiological arousal because of the mediation of cognitive and affective variables. Perhaps this influence on cognitive or affective processing results in further impairments in habituation.

Ability of the habituation models to explain the influence of arousal

In summary, with the exception of studies examining self-reported trait anxiety, there is evidence that increased levels of arousal may disrupt habituation of physiological response to neutral stimuli. Unfortunately, unqualified statements regarding the parameters or specific effects of arousal cannot be made at this time. Even more importantly, with very few exceptions, these studies have been carried out

without the explicit objective of evaluating specific predictions made by the various theories of habituation discussed earlier. It is difficult to evaluate these theories given the post-hoc nature of the data currently available. Any statements regarding this issue must be made very cautiously.

The studies discussed previously suggest that heightened arousal may impede habituation. Although this finding casts doubt on the ability of Sokolov's (1963) theory to accurately explain and predict the effects of heightened arousal (assuming that the prediction that heightened arousal will facilitate habituation is congruent with Sokolov's theory), it does not differentially support the theories of Thompson (e.g., Groves & Thompson, 1970), Lader (Lader & Mathews, 1968; Lader & Wing, 1964, 1966), or Whitlow and Wagner (e.g., 1984). In order to differentiate between these theories a more specific examination of the pattern of results is necessary. The main differential prediction is with respect to the rate of habituation across repeated presentations. Dual process theory (Groves & Thompson, 1970) would predict that the rate of habituation would not be affected by arousal level. Aroused subjects should show increased responsiveness to stimuli; however, their rate of response decrement should be relatively constant across arousal levels. Because habituation rate is assumed to be a property of the stimulus and independent of arousal level, the rate of habituation should not vary given presentation of the same stimulus across conditions. On the other hand, Lader's model (Lader & Mathews, 1968; Lader &

Wing, 1964, 1966) would predict that higher levels of arousal disrupt the process of habituation, resulting in a lesser rate of response decrement with higher levels of arousal. Whitlow and Wagner's (1984) model would predict that arousal would disrupt the rate of habituation because of the resulting distraction.

Re-examination of the studies discussed previously (excluding those that examined individual differences in trait anxiety or neuroticism, or that did not determine that the arousal manipulation was effective in manipulating resting arousal level at the time of assessment) provides support for the notion that heightened arousal impedes the rate of habituation to neutral stimuli. Of the 11 studies that examined whether or not there was differential rate of change across stimulus exposures, seven studies reported that heightened arousal disrupted the rate of habituation (Bohlin, 1976; Carrol & Pokora, 1976; Goldwater & Lewis, 1978; Lader, 1967; Lader & Wing 1964; Neary & Zuckerman, 1976; Raskin, 1975), and one study (Katkin & McCubbin, 1969) reported a disruption in the rate of habituation with higher, but not lower, intensity stimuli. Of the other available studies, Goldwater (1987) and Hart (1974) failed to replicate this finding, and Gatchel and Gaas (1976) found that more highly aroused subjects had a greater rate of response decrement.

These results suggest that increased arousal may disrupt the rate of habituation. Although this conclusion seems incongruent with dual process theory, this incongruence can be

resolved with a modification of the theory. It can be argued that heightened arousal has two effects on habituation. Not only does it result in an increase in general responsiveness in the "state" system that is independent of the habituation process (e.g. Groves & Thompson, 1970), but it also may have an effect on the habituation process because it re-directs the individual's focus of attention away from the stimulus. If an individual is experiencing heightened levels of arousal, he or she may search the environment to identify the source of arousal and/or attend to physiological manifestations of arousal. As arousal becomes more salient for the individual, especially if the arousal is accompanied by negative affect, other cues in the environment will likely become more salient and less attention will be directed to the stimulus. The disruptive effects of arousal and anxiety on attention and concentration are well documented (e.g., Eysenck, 1977, 1882; Hockey, 1984; Fenz & Epstein, 1965; Fenz, 1965, cited in Lang, 1985; Wine, 1971, 1980). For example, arousal and anxiety increase the frequency of negatively valenced, repetitive, and intrusive thoughts (e.g., Horowitz, 1975; Parkinson & Rachman, 1981). Further, Rachman and Levitt (1987) found that claustrophobic subjects' report of bodily symptoms ("Flushes (hot flashes) or chills"; Nausea or abdominal distress") and/or anxiety-related cognitions ("I am going to pass out"; "I am going to lose control") related to anxiety following initial exposure to the feared stimulus significantly predicted the extent to which their fear would habituate

across trials. Specifically, report of these symptoms and/or cognitions was predictive of an absence of later habituation. Attention to irrelevant information increases cognitive processing demands and results in processing overload (Hamilton, 1980; Tobias, 1986), and may adversely affect performance at one of three points: attention to relevant environmental stimuli; encoding and processing of these stimuli; and selection of the appropriate response (Sarason, 1975). With respect to habituation, the spreading of attention will subtract "processing time" from the immediate task at hand (e.g., Eysenck, 1979, 1982) and result in lessened "functional exposure" (e.g., Borkovec & Grayson, 1980) to the stimulus. Cognitive manipulations have been demonstrated to influence habituation to neutral (e.g., Iacono & Lykken, 1983, 1985) and feared (e.g., Grayson & Borkovec, 1978; Grayson, Foa & Steketee, 1982, 1986) stimuli. As attention is drawn away from the stimulus, the stimulus-response associations determining response to the stimulus will have less opportunity to be weakened as a result of the repeated stimulus presentations.

Expansion of the dual process model to consider the influence of attentional and cognitive/affective factors is consistent with the model and may allow greater explanatory power, especially when considering habituation to more complex stimuli in humans. In fact, Thompson et al. (1979) acknowledged that although present knowledge does not permit an understanding of how cognitive factors affect functioning

on a neural level, such influences nonetheless have a significant influence on habituation.

In summary, the results of the studies discussed previously suggest that arousal disrupts the process of habituation. This finding is inconsistent with the predictions based on Sokolov's (1963) theory of habituation; however, it is not clearly supportive of either the revised version of dual process theory just described, or Whitlow and Wagner's cortical model theory. A modification of the paradigm used to assess the effects of arousal may help to clarify this issue. The results of a study that assesses the effects of arousal during the process of habituation, as well as the extent of habituation at a later point in time when all individuals are in a similar state of arousal will result in a set of differential predictions. Both the revised version of dual process theory and Whitlow and Wagner's cortical model theory would predict that heightened arousal may impair the process of habituation. However, the revised version of dual process theory would predict that individuals who are aroused during habituation would manifest a further response decrement (i.e., increased habituation) when later assessed in a state of reduced arousal. Whitlow and Wagner (1984) and Lader (Lader & Mathews, 1968; Lader & Wing, 1966) would also predict that any effects of arousal that were apparent at the time of habituation would be maintained at later assessment. However, to the extent that arousal is a salient cue to prime the stimulus into short term memory, subjects who were aroused

during habituation but not at the time of retest would be expected to show a further response increase (i.e. a decrease in habituation) at later assessment relative to subjects who were not aroused during habituation.

THE EFFECTS OF AROUSAL ON FEAR REDUCTION AND THE RETURN OF FEAR

Arousal may impede habituation to neutral stimuli and several studies have noted that arousal also affects fear and the fear reduction process.

A relationship between initial autonomic responsivity to imaginal and in vivo presentations of feared stimuli and fear reduction has been noted by several investigators (Dyckman & Cowen, 1978; Glenn & Hughes, 1978; Lang, Melamed, & Hart, 1970; Levin, Cook, & Lang, 1982; Marshall, 1988; Stern & Marks, 1973; Vermilyea et al., 1984). Specifically, responsiveness to initial presentations of the feared stimuli is predictive of greater fear reduction via exposure treatment. These results suggest that emotional processing of fear during exposure, as evidenced by an increase in autonomic arousal in response to the stimulus exposure, expedites fear reduction. This finding is central to the previously discussed models of fear developed by Lang (e.g., 1985) Rachman (1980), and Foa and Kozak (1985, 1986).

Although autonomic arousal in response to initial exposure to feared stimuli promotes fear reduction, other findings suggest that high basal level of arousal affects fear and hinders the fear reduction process. There are suggestions, based on Lang's associative network model of fear that experiencing increased arousal that is unrelated to the feared stimulus intensifies the fear response (e.g., Lang, 1988). Barlow (1988) suggested that baseline anxiety level is a

"platform" for fear and that the probability of responding fearfully upon exposure to a feared stimulus is increased given high levels of baseline anxiety. Rachman (1990) used the term "emotional spill-over" in his discussion of the relationship between general arousal, especially anxious arousal, and fear. He argued that increased anxious arousal primes the cognitive networks defining feared stimuli, thus increasing the likelihood that a fear response will occur upon exposure to a feared stimulus.

Most of the support for the suggestion that increased arousal affects fear and fear reduction is derived from research on the return of fear. The return of fear refers to "the reappearance of fear that was present earlier but had undergone a decline" (Rachman, 1979, p. 165). The return of fear should not be confused with the phenomenon of relapse which can take many forms (Rachman, 1987). The return of fear is a robust phenomenon and has been examined in circumscribed fears (Grey, Rachman, & Sartory, 1981; Grey, Sartory & Rachman, 1979; Philips, 1985; Rachman & Lopatka, 1988; Rachman, Robinson, & Lopatka, 1987; Rachman & Whittal, 1989a, 1989b; Samsom & Rachman, 1989; Sartory, Rachman, & Grey, 1982), performance based fears (Craske & Rachman, 1987), and in obsessive-compulsive disorders (Foa, 1979; Grayson, Foa, & Steketee, 1982, 1986; Likierman & Rachman, 1980). Return of fear can be assessed either between sessions of fear reduction or following the completion of treatment (Craske & Rachman,

1987). The return of fear more properly refers to partial, as opposed to complete, return of fear (Rachman, 1987).

Grey et al. (1979) examined the effects of treatment demand on the return of fear. Demand was manipulated by altering the distance that the subject was from the feared stimulus during in vivo exposure. They found that there was less subjective report of return of fear under conditions of low demand compared with increasing or high demand. Although the findings for the autonomic index of fear (heart rate) were in the same direction as those for subjective fear, they were not significant. A follow-up study (Grey et al., 1981) attempted to determine the extent to which the dissipation of arousal that resulted from high or increasing levels of demand during exposure was responsible for the earlier results. Subjects were exposed to feared stimuli under conditions of either massed or distributed practice. Contrary to predictions, there were no significant effects with respect to the return of fear for any of the three indices of fear (subjective report of fear, heart rate, or behavioral approach) assessed either half an hour or one week following exposure. Upon closer examination of the data, however, the authors did find that four subjects in the distributed practice condition who displayed very high heart rates during the initial behavioral avoidance test in spite of reports of an absence of fear, also displayed a greater return of subjective fear one week following treatment compared with the other subjects who received distributed practice. There was

little evidence of return of fear using the other two indices of fear.

A further follow-up study by Sartory et al. (1982) investigated the possibility that exposure to high intensity stimuli and imaginal rehearsal of the feared stimuli immediately following treatment would result in a return of fear relative to exposure to low intensity fear stimuli and distraction following treatment. There were no significant effects of the manipulations on the return of fear 30 minutes following treatment (i.e., following the distraction/imagination period). The intensity of exposure also did not affect the subjective report of the return of fear one week following treatment; however, low intensity exposure resulted in a greater return of fear for minimally, but not maximally, feared stimuli. Contrary to prediction, rehearsal following treatment reduced the extent of the return of fear upon exposure to the medium and high fear stimuli. The authors based their prediction regarding the effects of imaginal rehearsal on the idea that imaginally rehearsing the feared stimuli results in increasing levels of arousal that in turn results in return of fear. This contrary result is not surprising, however, if rehearsal of the feared stimuli is conceived of as imaginal exposure to the feared stimuli. This imaginal exposure could contribute further therapeutic benefit in addition to the in vivo exposure experienced immediately prior to the imaginal rehearsal period. Once again, the authors noted that there was a subset of subjects who

exhibited very high heart rates while reporting an absence of fear upon initial exposure to the feared stimuli. Although the earlier finding of a relationship between high heart rate during initial exposure and the return of fear was not replicated, it was found that initial heart rate was predictive of the return of fear in subjects who approached the feared stimuli. This effect was not found in those subjects who avoided the stimuli. The small number of subjects exhibiting high heart rate precluded statistical analysis of these results.

A study by Craske and Rachman (1987) attempted to more systematically examine the role of arousal in the return of fear. In this study a population of musicians with performance anxiety were treated and assessed. In addition to examining the effects of initial autonomic arousal, as indexed by heart rate, the authors sought to examine the effects of initial level of perceived skill on the return of fear. Among their findings were that elevated heart rate was predictive of greater subjective report of the return of fear at follow-up, and greater report of anxious thoughts at all assessment occasions. Post hoc analyses indicated that subjects who exhibited a return of fear at follow-up had a significantly greater initial heart rate, lower initial perceived skill, more anxious thoughts, and performed less often during the follow-up period than subjects who did not show a return of fear. A multiple regression analysis which examined the ability of initial heart rate, initial perceived skill, actual

skill level, number of anxious thoughts, and number of follow-up performances to predict the amount of return of fear following treatment was conducted. None of these variables contributed significantly to the regression equation beyond the variance explained by initial heart rate. The combined effect of all the remaining variables also failed to add significantly to the ability of initial heart rate to predict the return of fear.

Taken together these four studies suggest that autonomic arousal prior to fear reduction is predictive of the return of fear. There are several problems with these data that must temper the conclusion. Most importantly, in examining the effects of arousal on the return of fear all studies used groups that were selected on the basis of pre-existing differences in arousal level. As a result, it is not possible to determine the extent to which other subject variables correlated with the level of autonomic arousal prior to exposure may explain the results. The most obvious alternative explanation involves autonomic arousal level at follow-up. An examination of arousal level at follow-up in the Grey et al. (1981) Sartory et al. (1982) and Craske and Rachman (1987) studies does not allow rejection of the possibility that arousal level at follow-up determines the amount of return of fear. (Note: The study by Grey et al. (1979) did not directly examine the effects of autonomic arousal on the return of fear).

There are thus at least two possible explanations for the previously obtained results. Firstly, perhaps autonomic arousal upon initial exposure to the feared stimuli (and presumably throughout exposure) impedes the process of habituation, as evidenced over the longer term but not at the end of the session. As discussed in the previous section on the effects of arousal on habituation, arousal during exposure to neutral stimuli may hinder habituation. A major difference between those findings and the findings regarding the effects of arousal on the return of fear concerns the time interval over which the deficit in habituation becomes apparent. The research on the effects of arousal on habituation to neutral stimuli indicates that arousal has a detrimental effect on habituation during the exposure interval. It is presently not known what effects arousal has on the long term habituation to neutral stimuli as there has not been an assessment of these effects. In contrast to this, research on the return of fear suggests that arousal during fear reduction may result in a return of fear even when it fails to disrupt within-session habituation (Grey et al., 1981).

It may be that there is more than one process governing habituation to feared stimuli, and that heightened arousal disrupts mechanisms governing long term (i.e., between-session) but not short term (i.e., within-session) habituation. A number of researchers have attempted to garner support for a two-process (within-session and between-session) explanation of habituation. For example, Groves and Lynch

(1972) suggested that within-session habituation is mediated by the reticular formation and between session habituation, "involves elaboration by forebrain structures, most particularly frontal (or other association) cortex and hippocampus, but probably other forebrain structures as well" (p. 237). Likewise, Whitlow and Wagner (e.g., 1984) discuss the differential importance of short term and long term memory processes, and self-generated and retrieval-generated priming, in within- and between-session habituation. Foa (1979) and Foa and Kozak (1985, 1986) postulated the existence of two partially dependent habituation processes that account for within- and between-session reductions in fear.

Although postulating two processes of habituation may facilitate explanation of the finding that individuals who show return of fear do not differ from those who do not show a return of fear with respect to within-session fear reduction, it is worthwhile to consider the arguments of Mackintosh (1987) in his insightful analysis of habituation. He reviewed data that have been used as evidence in support of the existence of separate short term and long term habituation processes and concluded that although a two process explanation is congruent with the data, there is a more parsimonious explanation. Specifically, he argued that concepts of incomplete retention across the follow-up interval and differences in generalization decrements across conditions could equally well account for the results. In a similar manner, when considering the habituation of fear responses, it

is plausible that within- and between-session habituation represent a single process, and that disruptions in the habituation of fear become apparent only in the long term because arousal disrupts long term retention of the habituated response.

The second major explanation for the effect of autonomic arousal on the return of fear is that the return of fear is the result of heightened arousal at the time of retest. The habituation literature has documented examples of the effects of interpolation of a novel or otherwise arousing stimulus on habituation to a neutral stimulus. Specifically, results of several studies indicate that exposure to a novel stimulus results in a transitory increased level of response to the original stimulus (e.g., Groves & Thompson, 1970; Magliero, Gatchel & Lojewski, 1981; McCubbin & Katkin, 1971,; Rust, 1976; Thompson & Spender, 1966). It is not entirely clear, however, that this increased level of responsiveness is entirely due to increases in arousal (Edwards & Siddle, 1976; Rust, 1976). Central to the dual process theory of habituation (e.g., Groves & Thompson, 1970) is the prediction that increased arousal will result in a generalized increase in responding. There are also clinical accounts of a return of fear during, or following, treatment as the result of the individual experiencing increased levels of stress (e.g. Bilsbury & Morley, 1979; Rachman, 1987). Possibly increased arousal at the follow-up assessment, independent of arousal

level at the beginning of treatment, results in a dishabituation of the fear response.

Rachman and Whittal (1989a) examined the effects of increased anxious arousal at follow-up on the return of fear. They found that increased anxious arousal did not result in increased return of fear. However, this finding is limited by the fact that the anxious arousal manipulation that they used did not significantly increase heart rate compared to their control condition.

In summary, although the previous studies that noted the effects of arousal on the return of fear focused attention on the role of autonomic arousal at the time of pretest, it is equally plausible that the return of fear is augmented by arousal at the time of follow-up assessment. Unfortunately, currently available studies do not allow an evaluation of the two possibilities.

In order to clarify the role that autonomic arousal has on fear reduction and the return of fear, it is necessary to conduct further research that more systematically controls and manipulates arousal level.

SELF-EFFICACY AND AROUSAL

Bandura (1977) formulated the theory of self-efficacy as an explanation of behavior change. Self-efficacy expectations refer to beliefs of an individual that he or she can successfully behave so as to achieve a certain goal or outcome. Bandura argues that reductions in fear (and more generally, changes in behavior) (Bandura, 1977, 1982, 1986)

are mediated by changes in self-efficacy. Self-efficacy expectations affect "people's choice of activities and behavioral settings, how much effort they expend, and how long they will persist in the face of obstacles and aversive experiences. The stronger the perceived self-efficacy, the more active the coping efforts" (Bandura & Adams, 1977, p. 287-288). Self-efficacy expectations are based on four sources of information. These sources of information include performance accomplishments, vicarious experience, verbal persuasion, and level of emotional arousal (Bandura, 1977). Although previous research has demonstrated that self-efficacy expectations predict fear behavior (e.g. Bandura & Adams, 1977; Bandura, Adams, & Beyer, 1977; Bandura, Adams, Hardy, & Howells, 1980; Bandura, Reese, & Adams, 1982; Bandura, Taylor, Williams, Mefford, & Barchas, 1985; Craske & Craig, 1984; Craske & Rachman, 1987; Kendrick, Craig, Lawson, & Davidson, 1982; Williams & Watson, 1985), its status as the primary mediator of behavior has not been established (Borkovec, 1978; Craske & Rachman, 1987; Eastman & Marzillier, 1984; Feltz, 1982; Wolpe, 1978).

Bandura (1977, 1982, 1986) maintains that there is an inverse relationship between arousal level and expectations of self-efficacy. He argues that heightened arousal indicates to the individual that he or she is currently vulnerable to stress and anxiety resulting in a decrease in expectations of self-efficacy. Bandura (1982) states that arousal level affects behavior indirectly because of its effect on

expectations of self-efficacy: "It is not arousal per se but self-evaluative rumination that is detrimental to performance" (Bandura, 1986, p. 442). Specifically, Bandura would predict that heightened arousal would lower self-efficacy which would, in turn, increase fear. Although there do not appear to be any studies that directly address this issue, a finding reported by Craske and Rachman (1987) suggests that arousal level and level of perceived competence (a measure similar to self-efficacy) are not related (r.=-.10, n=63). Although this finding suggests that arousal level may not influence self-efficacy expectations, it can be criticized for not adequately addressing this issue. Firstly, Craske and Rachman (1987) assessed perceived competence as opposed to self-efficacy. It is not entirely clear how these two measures are related to each other. Secondly, the correlation obtained by Craske and Rachman (1987) was based on single measures of pre-existing levels of perceived competence and arousal. Feltz (1982) and Williams and Watson (1985) also found that pre-existing levels of arousal and self-efficacy were unrelated. Conversely, Williams, Dooseman, and Kleifield (1984) and Williams, Turner, and Peer (1985) found that self-efficacy and self-reported levels of performance anxiety were significantly related in an inverse fashion. All of these studies have examined pre-existing levels of arousal and self-efficacy; it is unknown what effects changes in arousal level have on expectations of self-efficacy.

There is little information on the effect of arousal on self-efficacy expectations. Research in this area would allow a further clarification of the role of arousal on self-efficacy and behavior change.

STATEMENT OF PURPOSE

The main purpose of the present study was to investigate the effects of anxious arousal[1] during fear reduction training and at follow-up assessment on fear reduction and the return of fear. Justification for this study comes from several sources. Firstly, predictions regarding the role of anxious arousal on fear reduction and the return of fear can be made based on hypotheses derived from the major theories of habituation. Secondly, previous literature in the area of the return of fear has suggested that heightened levels of physiological arousal has detrimental effects on the maintenance of fear reduction. At present, however, specific parameters of anxious arousal on fear reduction and the return of fear are unknown.

A second purpose of the present study was to examine the effects of anxious arousal on self-efficacy expectations. Although specific predictions can be derived from self-efficacy theory, they have been largely untested.

The results of this study will be useful in several ways: firstly, they will allow a greater understanding of the

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1 There is debate regarding the use of the term arousal (Anderson, 1990; Neiss, 1988, 1990). It has been argued that the term is meaningless without reference to the psychological context in which the arousal occurs (Neiss, 1988, 1990). Arousal produced through different means likely results in different effects. For example, arousal that accompanies fear is not entirely the same as arousal resulting from physical exertion or sexual excitement. In the present study, use of the term 'anxious arousal' allows greater definitional specificity than the term 'arousal'. Anxious arousal encompasses physiological arousal as well as the cognitive effects associated with increased anxious arousal such as worry, apprehension, and distraction (e.g., Barlow, 1988; Sarason, 1984, 1985).

ability of the major theories of habituation to explain fear reduction. Secondly, they will allow greater insight regarding the effects of anxious arousal during fear reduction and at the time of follow-up on fear reduction and the return of fear. Thirdly, the results of the study will allow greater insight into the effects of anxious arousal on self-efficacy. Ultimately, both theoretical and clinical benefit may be derived from these findings.

A number of hypotheses and specific predictions regarding the effects of anxious arousal on fear reduction and the return of fear can be made based on the revised version of the dual process theory and cortical theory (e.g. Whitlow & Wagner, 1984). Within the context of the present study, these two theories lead to several identical predictions as well as several differential predictions. Results congruent with predictions that are identical across theories are useful as they allow a more systematic understanding of, and alternative explanations of, the effects of anxious arousal on fear reduction. Differential predictions are most useful, however, as they not only allow a more systematic understanding of the phenomena under study, but they also allow statements to be made regarding the relative explanatory ability of each theory with respect to the effects of anxious arousal on fear reduction.

Hypotheses and predictions based on self-efficacy theory (e.g., Bandura. 1977, 1982, 1986) will also be presented.

Hypotheses

Several hypotheses regarding the influence of anxious arousal on fear reduction were based on the revised version of dual process theory (henceforth to be referred to as dual process theory). It was hypothesized, based on dual process theory, that anxious arousal has two major effects on habituation: firstly, the experience of an increase in anxious arousal results in an increase in the sensitization process. Behaviorally, this is manifested as a relatively transient increase in responsiveness (in the case of fear, increased fear). In other words, as the anxious arousal is allowed to dissipate, the inflated fear response also dissipates. Secondly, the experience of heightened levels of anxious arousal during habituation training (in the present study, during fear reduction) impedes the habituation process. Behaviorally, this impediment to habituation is manifested by a decrease in the amount of fear reduction during exposure to the feared stimulus. This disruption is the result of re-direction of the individual's attention away from the feared stimulus. Rather than focussing on the feared stimulus, the individual tends to attend to other cues in the environment and/or bodily sensations of anxious arousal, and/or may be distracted by negatively valenced intrusive thoughts. Unlike the postulated effects of anxious arousal on the sensitization process, these effects are hypothesized to be permanent, representing an impairment in learning.

Several hypotheses regarding the influence of anxious arousal on fear reduction were based on the cortical theory of habituation (e.g., Whitlow & Wagner, 1984). Firstly, it was hypothesized that anxious arousal during habituation (in the present case, during fear reduction) results in a direction of attention away from the feared stimulus, resulting in decreased habituation. In the present study, this is represented behaviorally as impaired fear reduction. This effect represents an impairment in learning and is permanent. Secondly, it was hypothesized that long term habituation (represented behaviorally as maintenance of fear reduction) is determined by the extent of similarity in anxious arousal level during fear reduction and at retest. Specifically, experiencing similar levels of anxious arousal during fear reduction and at retest results in greater maintenance of the fear reduction than results given incongruent levels of anxious arousal during fear reduction and at retest. This was hypothesized to occur because the individual's anxious arousal state serves as a cue that is used to access memories of the stimulus from long term memory. The extent to which the stimulus is primed in memory prior to exposure to the (previously) feared stimulus is determined in part by the individual's anxious arousal state. The extent to which the stimulus is primed into short term memory determines the extent of maintenance of the habituated response.

It was hypothesized based on self-efficacy theory (e.g., Bandura, 1977, 1982, 1986) that self-efficacy level is

directly influenced by anxious arousal level. Specifically, as anxious arousal level increases, self-efficacy decreases. When anxious arousal level is allowed to return to normal, self-efficacy shows a corresponding increase.

Overview of Method and Design

Subjects were female undergraduate students who reported a fear of snakes. Upon the subject's arrival at the laboratory during the first session, the intensity of her fear was assessed using a standardized Behavioral Approach Test (BAT). The dependent measures assessing fear included self-reported fear (subjective units of distress) and heart rate response. Subjects who exhibited a minimum criterion level of self-reported fear were asked to participate in the rest of the study. Subjects were randomly assigned to one of two groups: anxiously aroused (i.e., received a series of randomly timed shocks) vs. control (did not receive any shocks). Once anxious arousal level was manipulated, the subject's fear level was again assessed using the BAT. Strength of self-efficacy was also assessed. The subject then viewed a videotaped fear reduction program under control or anxious arousal conditions. Following this, the subject again participated in a BAT and measures of fear and self-efficacy were taken. Subjects in the anxious arousal group then had their anxious arousal reduced (i.e., through termination of shock/shock threat). A fourth BAT then occurred. Subjects returned for a follow-up session one month later. Half of the subjects in each of the above groups were randomly assigned to conditions of either anxious

arousal or control conditions during follow-up. A final BAT was used to assess fear levels. Self-efficacy was also assessed.

In order to assess predictions regarding the effects of arousal level during fear reduction on fear and self-efficacy, the study used a two by four factorial design with repeated measures on the second factor. The design consisted of anxious arousal level during fear reduction (Anxiously aroused vs. Control) by time of assessment (Prior to the anxious arousal manipulation (Time 1) vs. Immediately after the anxious arousal manipulation (Time 2) vs. Immediately after fear reduction (Time 3) vs. Five minutes following the termination of fear reduction - Anxious arousal equalization (Time 4)) factorial design with repeated measures on the second factor. In order to evaluate the interactive effects of anxious arousal level during fear reduction and at follow-up, subjects in each of the above two groups were randomly assigned at follow-up to either the anxious arousal or the control condition.

Each of the three dependent variables (heart rate response (i.e., maximal heart rate during the BAT, covarying out resting heart rate immediately prior to the BAT), self-reported fear, and self-efficacy) was assessed on each occasion.

Predictions

Predictions were made for the two dependent variables assessing fear (i.e., heart rate response and self-reported

fear) based on hypotheses derived from each of the two major theories of habituation. Although the following predictions were made in absolute terms, it was not implied that proponents of either theory would not allow for the influence of other factors on fear and the fear reduction process. Given the complex nature of fear and the fact that these theories have been based on the study of organisms, stimuli, and responses that are less complex than those currently under study, it is unlikely that any one theory would be able to completely explain fear behavior. Rather, the previously discussed theories are more appropriately considered as "mini models" (Mineka, 1985) that guide research and allow further insight into, but not a complete understanding of, the phenomena under study. The predictions were as follows:

1. Effects of anxious arousal on pre-exposure levels of fear.

(Fear at time 2).

Dual process theory

The subjects in the anxiously aroused group would experience significantly greater heart rate responsiveness (i.e., maximal heart rate during the BAT, covarying out resting heart rate immediately prior to the BAT) and self-reported fear relative to the subjects in the control group.

The reason for the anxiously aroused subjects' greater response relative to the subjects in the control group is because of their increased level of anxious arousal which,

according to dual process theory, would increase responsiveness.

Cortical theory (e.g., Whitlow & Wagner, 1984)

Although cortical theory does not make any specific predictions regarding the effects of anxious arousal on habituation, it was argued on the basis of this theory that the subjects in the anxiously aroused group would be re-exposed to the feared stimulus at time 2 under a different level of anxious arousal compared with time 1. As a result, the feared stimulus would be less likely to be cued in memory prior to the BAT. As a result of this, these subjects would show increased heart rate response and self-reported fear relative to subjects in the control group.

Both dual process theory and cortical theory made identical predictions regarding the effects of anxious arousal on pre-exposure levels of fear.

2. Effects of anxious arousal during fear reduction on fear level immediately following fear reduction.

(Fear at time 3).

Dual process theory

The subjects in the anxious arousal group would experience significantly greater residual fear immediately following fear reduction on the two measures of fear (heart rate response and self-reported fear) than the subjects in the control group.

The reason for this prediction was because anxious arousal results in an increase in general responsiveness and

distracts attention away from the feared stimulus during exposure. This decreased functional exposure results in decreased fear reduction.

Cortical theory

The subjects in the anxious arousal group would experience significantly greater fear on the two measures of fear (heart rate response and self-reported fear) than the subjects in the control group.

The reason for this prediction was because increased anxious arousal distracts attention away from the feared stimulus during exposure. This decreased functional exposure results in decreased fear reduction.

Dual process theory and cortical theory made identical predictions regarding the effects of anxious arousal on fear levels assessed immediately following fear reduction.

3. Effects of removal of anxious arousal on fear level. (Change between time 3 and time 4).

Dual process theory

Upon re-assessment under condition of non-anxious arousal (i.e., when not under the threat of shock), the subjects who experienced fear reduction under conditions of anxious arousal would show a decrease in heart rate response and self-reported fear relative to the control subjects.

The reason for this prediction was that as the previously aroused subjects' arousal level decreased, their level of general responsiveness as indexed by heart rate response and self-reported fear would also decrease.

Cortical theory

Upon re-assessment under conditions of non-anxious arousal (i.e., when not under the threat of shock), the subjects who experienced fear reduction under conditions of anxious arousal would show increased heart rate response and self-reported fear relative to the subjects in the control group who experienced fear reduction under conditions of non-anxious arousal. This prediction differs from that based on dual process theory.

The reason for this prediction was that anxious arousal functions as a memory cue and subjects in the control group would experience a greater degree of congruence in anxious arousal level between exposure and the second post-test than subjects in the anxious arousal group.

4. Effects of anxious arousal during fear reduction and at follow-up on the return of fear.

(Change between time 4 and time 5).

Dual process theory

The subjects who were anxiously aroused at follow-up would show a significantly greater increase in fear scores between exposure and follow-up relative to the subjects who were not aroused at follow-up.

The reason for this prediction was that as arousal level increased, the level of general responsiveness as indexed by heart rate response and self-reported fear would show a corresponding increase.

Cortical theory

The subjects who experienced congruent states of anxious arousal during fear reduction and at follow-up (i.e., either anxiously aroused on both occasions or non-anxiously aroused on both occasions) would evidence significantly less return of fear than subjects who experienced incongruent states of anxious arousal on the two occasions.

The reason for this prediction was that anxious arousal functions as a memory cue and the subjects who experienced congruent states of arousal during fear reduction and at follow-up would show significantly less responsiveness.

Refer to Tables 1 and 2 for tabular presentations of the predictions regarding the effects of anxious arousal on fear.

Predictions regarding self-efficacy were made based on Bandura's (e.g., 1977, 1982, 1986) model of self-efficacy.

1. Effect of anxious arousal on self-efficacy prior to fear reduction.

(Self-efficacy at Time 2).

The subjects in the anxious arousal group would report significantly less self-efficacy relative to the subjects in the control group. The reason for this decreased level of self-efficacy relative to the subjects in the control group was because of the anxiously aroused subjects' increased level of anxious arousal which, according to self-efficacy theory, was a source of information indicating to the individual that she was currently vulnerable to stress which may impede coping abilities.

Table 1

Predictions regarding the effects of anxious arousal prior to and during fear reduction on fear

	Predictions derived from Dual Process Theory	Predictions derived from Cortical Theory
Effects of anxious arousal on pre-exposure levels of fear	HRR: A>C SUDS: A>C	HRR: A>C SUDS: A>C
Effects of anxious arousal during fear reduction on fear levels immediately following fear reduction.	HRR: A>C SUDS: A>C	HRR: A>C SUDS: A>C
Effects of removal of anxious arousal on change in fear levels (i.e., Fear level at Time 4 minus fear level at Time 3)	HRR: A<C SUDS: A<C	HRR: A>C SUDS: A>C

Note: HRR=Heart rate response. SUDS=Subjective units of distress.

A=Anxious arousal prior to/during fear reduction. C=Control group.

Table 2

Predictions regarding the effects of anxious arousal during fear reduction and at follow-up on the return of fear

	Predictions derived from Dual Process Theory	Predictions derived from Cortical Theory
Fear response at Time 5 minus fear response at Time 4	HRR: (CA+AA)>(CC+AC) (and cortical theory predicts that (CA+AA)=(CC+AC))	HRR: (CA+AC)>(CC+AA) (and dual process theory predicts that (CA+AC)=(CC+AC)
	SUDS: (CA+AA)>(CC+AC) (and cortical theory predicts that (CA+AA)=(CC+AC)	SUDS: (CA+AC)>(CC+AA) (and dual process theory predicts that (CA+AC)=(CC+AA)

Note:

CC=Control group - session 1, Control group - session 2.

CA=Control group - session 1, Anxious arousal group - session 2.

AC=Anxious arousal group - session 1, Control group - session 2.

AA=Anxious arousal group - session 1, Anxious arousal group - session 2.

2. Effect of anxious arousal during fear reduction on self-efficacy following fear reduction.

(Self-efficacy at time 3).

The subjects in the anxious arousal group would report decreased self-efficacy relative to the subjects in the control group.

The reason for this prediction was two-fold: firstly, the subjects in the anxious arousal group would continue to experience heightened levels of anxious arousal despite exposure to the feared stimulus. This continued anxious arousal may result in a disruption in development of self-efficacy expectations. Secondly, experiencing anxious arousal would result in decreased functional exposure to the feared stimulus which would allow less opportunity to increase self-efficacy expectations.

3. Effect of removal of anxious arousal on self-efficacy.

(Change between Time 3 and Time 4).

The subjects who were aroused during fear reduction would experience a significant increase in self-efficacy relative to the subjects in the control group.

The reason for this change in self-efficacy relative to subjects in the control group was because the reduction of anxious arousal constituted removal of a source of information that promotes decreased levels of self-efficacy.

4. Effect of anxious arousal at follow-up on self-efficacy.

(Change between Time 4 and Time 5).

The subjects who were anxiously aroused at follow-up would show a significantly larger decrease in self-efficacy between exposure and follow-up than subjects in the control group.

The reason for this change in self-efficacy relative to the subjects in the control group was because the increased anxious arousal constituted addition of a source of information that promotes decreased levels of self-efficacy. Refer to Table 3 for a tabular presentation of the predictions regarding the effects of anxious arousal on self-efficacy.

Table 3

Predictions regarding the effects of anxious arousal on self-efficacy expectations

Effects of anxious arousal prior to fear reduction on SEE	A<C
Effects of anxious arousal during fear reduction on SEE immediately following fear reduction	A<C
Effects of removal of anxious arousal on changes in SEE (i.e., SEE at Time 4 minus SEE at Time 3)	A>C
Effects of anxious arousal at follow-up on changes in SEE (i.e., SEE at Time 5 minus SEE at Time 4)	A<C

Note: SEE=Self-efficacy expectation. C=Control.

A=Anxious Arousal.

METHOD

SCREENING

Undergraduate students in introductory psychology were administered an abbreviated version of the Fear Survey Schedule (Wolpe & Lang, 1964) (see Appendix A) in order to determine their appropriateness for inclusion in the present study. Female students who indicated that they were "extremely fearful of" or "terrified of" snakes were then contacted via telephone and asked to participate in the study. It was decided to use only female students in the current study for two main reasons: firstly, the incidence of animal phobias is much more frequent in women than men (Bourdon, Boyd, Rae, Burns, Thompson & Locke, 1988; Hersen, 1973; Marks, 1969, cited in Sturgis & Scott, 1984); secondly, it has been observed that men exhibit greater discordance between self-report and other indices of fear (e.g., behavioral avoidance, physiological arousal) (Hersen, 1973; Lopatka, 1987). If the subject agreed to participate, the experimenter arranged a convenient appointment time.

MEASURES

Self-report measures: Several self-report measures were used in this study. The state version of the State-Trait Anxiety Inventory (STAI) (Speilberger, Gorsuch, & Lushene, 1970) consists of 20 statements that focus upon qualities of anxiety, tension, worry, and apprehension. Subjects indicate the extent to which each of the statements describes their current state. Test-retest reliabilities for the state version

of this scale range from .16 to .54 across time periods ranging from 1 hour to 20 days. Internal consistency as measured by the K-R 20 ranges from .83 to .92 (Katkin, 1978; Mason-Dreger, 1978). The validity of the scale has been has been empirically validated in a number of studies (e.g., Katkin, 1978; Kendall, 1976).

A mood scale, consisting of six 100 mm. visual analog scales was administered to each subject on a number of occasions. Each scale represents a different emotion (i.e., anxiety, sadness, agitation, happiness, relaxation, and apprehension) (see Appendix B). Subjects were to indicate the extent to which they were currently experiencing each emotion. This mood scale is similar to scales used in previous studies of fear and has been found to be sensitive to experimental manipulations of mood (e.g., Rachman & Whittal, 1989a; Samsom & Rachman, 1989; Sutherland, Newman & Rachman, 1982).

Subjective units of distress (SUDS) upon exposure to the snake during the Behavioral Approach Test (BAT) were assessed by asking the subject to verbally report how much fear they experienced at the point of closest exposure to the snake, with zero indicating no fear and 100 indicating terrifying fear. SUDS are very commonly used in research on fear and are a very sensitive measure of changes in fear (Agras & Jacob, 1981).

Strength of self-efficacy regarding the ability to touch the snake was assessed by having the subject indicate on a 100 point visual analog scale the extent to which she was

confident that she could approach a live but harmless snake (see Appendix C). The specific behavior that the subject was asked to evaluate corresponded to the closest approach point to the snake that the subject engaged in during the initial BAT.

Heart rate: Heart rate was recorded with a Sanyo heart rate monitor model number HRM-700E. This monitor consists of a photoplethysmograph that is attached to the earlobe. It has a digital display that is accurate to within 3 %. During sesion 1, heart rate during exposure to the modeling program was sampled for five second intervals every one minute. The maximum heart rate during each five second interval was recorded, resulting in 14 samples of heart rate data. During session 2, the maximum heart rate during each five second interval was sampled at one minute intervals for six minutes beginning immediately after the subject was told that she would be exposed to the series of tones (in the control group) or shocks (in the anxious arousal group), resulting in six samples of heart rate data. Heart rate immediately prior to the BAT was defined as the maximum heart rate in the five second interval occurring between 25 and 30 seconds following completion of the self-report measures. Heart rate response during the BAT was defined as the maximum heart rate during the five second interval immediately following the closest approach point to the snake. Heart rate was chosen as a measure of autonomic arousal as it is reliable, more highly correlated with other measures of fear than other autonomic

measures (Agras & Jacob, 1981; Bellack & Lombardo, 1984; Craske, 1982; Hodgson & Rachman, 1974; Hugdahl, 1989; Lang et al., 1970), and is the autonomic index used in previous research on the return of fear.

FEAR REDUCTION PROGRAM

A modeling sequence to be presented to the subjects via videotape was developed for use in the present study. The program, which is approximately 15 minutes in duration, depicts two individuals who were initially fearful of snakes undergoing fear reduction through the use of graduated participant modeling. All four models in the program (two clients and two therapists) are female and appear to be in their late teens or early twenties. Three of the four models are caucasian, and the fourth is oriental. The program depicts both coping (the clients) and mastery (the therapists) models. Throughout the modeling sequence there is provision of information regarding snakes and how to handle them, and the client is given frequent feedback and positive reinforcement about her performance. The outcome of both therapist-client interactions is clear: the client is gradually able to more competently, and less fearfully, handle the snake. In the final moments, both clients are able to handle the snake without assistance while reporting very low levels of fear. These factors (i.e., use of multiple models, similarity between the models and the subjects, use of coping models, provision of information, feedback, and positive reinforcement) serve to maximize the effects of the modeling

procedure (e.g., Kazdin, 1974, Kazdin, 1975, Meichenbaum, 1972). Although videotape modeling is a less effective method of fear reduction than participant modeling (e.g., Bandura, 1969), it is nevertheless a robust fear reduction procedure. Furthermore, it was a more appropriate procedure than participant modeling for use in the present study as it allows complete standardization between subjects with respect to amount and type of exposure to the feared stimulus. This degree of standardization cannot be achieved with participant modeling procedures. The use of participant modeling procedures would likely have resulted in systematic group differences regarding the parameters of exposure to the snake.

FEAR REDUCTION SESSION

Upon arrival at the laboratory, the subject was escorted to the testing room by the experimenter, a man in his late twenties, and seated in a reclining chair. She remained in this chair for the duration of the experiment. The heart rate monitor was then shown to the subject and attached to her right ear lobe. The experimenter then explained to the subject how to complete the mood scale and the state version of the STAI. The subject was then given the consent form (Appendix D) to read and sign. Any additional questions of the subject were answered at this time.

The research assistant, who was blind to the hypotheses of the study and the condition of the subject, entered the room and the experimenter was seated by the shock equipment which was behind a screen to the right of the subject. After a

five minute adaptation period, the subject completed the mood scale and the STAI, and her heart rate was recorded.

The subject was then asked to participate in a BAT in order to further assess her level of fear. The research assistant uncovered the container housing the snake which was 18 feet from the subject. The subject was given the following instructions:

> Inside the container is a live harmless garter snake. Can you see it from where you are sitting? In a moment I am going to ask you to report the peak amount of fear that you are experiencing using a scale from 0 to 100, with "0" being no fear and "100" being terrifying fear. I will move the container toward you and when I reach where you are sitting, I would like you to pick up the snake for five seconds while continuing to look at it. I will tell you when the five seconds are up. If you are unable to pick up the snake please let me know when the snake is as close to you as you can possibly tolerate. It is important to do so as quickly as possible. Do you have any questions? I will now move the snake toward you. (Experimenter slowly moves the container toward the subject) Now I would like you to report the peak amount of fear you are experiencing using the 0 to 100 scale.

The subject's heart rate and level of self-reported fear were recorded at the closest approach point to the snake. The closest approach point was held constant for each subject for subsequent BATs so as to allow comparison across BATs.

Subjects who were able to touch the snake while reporting a fear level of less than 70 were excluded from the study. Use of a minimum cutting score of 70 is consistent with other similar studies of fear and the return of fear (e.g., Samsom & Rachman, 1988).

After the BAT, the research assistant left the room and the subject was randomly assigned to one of two conditions:

1. Anxious arousal condition - An electrode was attached to the subject's forearm. It was held in place by a tensor bandage wrapped around the subject's arm. The subject was given the following instructions:

> You will now receive a series of brief 0.5 second shocks through this electrode. The shocks are completely harmless and will cause absolutely no lasting pain or damage. Each succeeding shock will be slightly more intense than the previous one. I would like you to tell me when you first experience the shock. You will continue to receive shocks until you are unable to tolerate any further shocks. At any time during the sequence of shocks, simply saying "Stop" will end the series of shocks. Please do your best to tolerate as intense a shock as possible. Do you have any questions?

The subject was then given a series of 0.5 second shocks at 10 second intervals in order to establish the subject's pain sensitivity range - pain threshold to tolerance. Pain threshold was defined as the level of shock first discerned by the subject and pain tolerance was the level at which the subject was not prepared to accept any further increases in shock intensity. Following this procedure, the subject was given the following instructions:

> During the rest of the experiment, until you are told otherwise, you will receive a number of shocks. The shocks will be of various intensities and the timing of the shocks will be randomly determined. You cannot do anything to change the intensity or timing of the shocks. Do you have any questions?

2. Control condition - An electrode, different in size and shape from that used for subjects in the arousal condition, was attached to the subject's forearm. It was held in place by a tensor bandage. The subject was given the following instructions:

> The purpose of this electrode is to measure your level of physiological responding. In order to calibrate the equipment, I need to assess your physiological response to a series of standardized tones. You will now hear a series of brief tones. I would like you to do nothing but simply listen to the tones. Do you have any questions?

The subject was then exposed to a series of 11 brief tones that were presented at 10 second intervals. This number is equivalent to the mean number of shocks needed to establish the pain sensitivity range of subjects in the anxious arousal group during pilot testing and served to equate the amount of time that subjects in the two groups were in the laboratory prior to viewing the modeling videotape. Following this procedure the subject was given the following instructions:

> You are in the control condition and will not at any time be exposed to shock. There is absolutely no possibility that you will receive a shock. Throughout the procedure please do your best to sit quietly. Do you have any questions?

At this point any questions of the subject were answered and, if the subject agreed, the experiment continued. At this point, the subject was asked to not discuss with the research assistant any aspects of the study. The research assistant then re-entered the room. She asked the subject to complete the STAI, the mood scale, and the self-efficacy scale, and recorded the subject's heart rate. A second BAT was then conducted. The research assistant left the testing room and the experimenter gave the subject the following instructions:

> I would now like you to watch a short videotaped program. You will see two different individuals who, like yourself, were fearful of snakes. On the program they will be taught skills that helped them to become more skillful in handling snakes. Ultimately, they both became relatively fearless of

harmless snakes like the one in the program. Please pay attention to the program.

Subjects in the anxious arousal condition were then told, "Please remember that you will continue to receive one or more shocks until you are told otherwise and disconnected from the shock equipment." Subjects in the control condition were told, "Please remember that you absolutely will not receive any shocks throughout the procedure."

Subjects then viewed the modeling program. Subjects in the anxious arousal group received a series of 10 shocks during the program.[2] The 15 minute program was divided into 10 intervals of 1.5 minutes. Within each interval one shock was delivered to the subject. The intensity of the shocks varied from .50 to .95 of the interval from pain threshold to pain tolerance. Heart rate was sampled for five second intervals every one minute throughout the program. Following the program the research assistant re-entered the room, asked the subject to complete the three scales, recorded her heart rate, and conducted a third BAT. The research assistant then left the testing room. The electrode was removed from the subject's forearm. Subjects in the arousal condition were told, "You will no longer be exposed to any further threat of shock." Subjects in the control condition were told, "Your level of physiological responsiveness will no longer be assessed."

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2 Pilot testing was initially conducted using a shock threat manipulation. The subjects in the shock threat group were connected to an electrode and informed that they would receive a painful shock on one or more occasions during the session. However, they did not actually receive any shocks. It was found that this manipulation did not reliably increase either self-reported anxious arousal or heart rate and, consequently, actual shock was used in order to generate anxious arousal.

Subjects were asked to sit quietly for five minutes and were given a magazine article to read. The purpose of this interval was to allow subjects in the anxious arousal and control conditions to develop similar levels of anxious arousal.

Following the five minute anxious arousal normalization period, the research assistant asked the subject to complete the three scales completed previously, recorded her heart rate, and conducted a fourth BAT. The subject was then thanked for her cooperation and a second appointment, four weeks later, was scheduled.

FOLLOW-UP SESSION

The follow up session took place in the same testing room as in the first session. The subject was asked by the research assistant to complete the mood scale and the STAI and, after a five minute adaptation period, her heart rate was recorded. Half of the subjects in each of the two conditions of session 1 were randomly assigned to the anxious arousal group and the remaining subjects were assigned to the control group. The shock sensitivity range of subjects in the anxious arousal group was established in an identical fashion to that used in session 1. Subjects in the control group were exposed to a series of tones using the same procedure as in session 1.

Following the establishment of the shock sensitivity range, subjects in the anxious arousal group were given the following instructions:

> During the rest of the session, until you are told otherwise, you will receive a number of shocks. The shocks will be of varying intensities and the timing of the shocks will be randomly determined. You

> cannot do anything to change the intensity or timing of the shocks. Do you have any questions?

After exposure to the series of tones, subjects in the control condition were given the following instructions:

> You are in the control condition and will not at any time be exposed to shock. There is absolutely no possibility that you will receive a shock. Please do your best to sit quietly. Do you have any questions?

Any questions of the subject were answered. Subjects in the arousal condition then experienced a series of 6 shocks over a six minute interval. One shock was received within each one minute interval. The intensity of the shocks varied between .50 and .95 of the subject's pain sensitivity range. Subjects in the control group were asked to sit quietly for six minutes. Immediately following the six minute interval, the research assistant re-entered the room. The subject completed the mood scale, the self-efficacy scale, and the STAI, and her heart rate was recorded. The subject then participated in a BAT using identical instructions to those in the previous BATs.

The subject was then debriefed, paid a stipend of ten dollars, and thanked for her cooperation.

RESULTS

A total of 91 subjects attended the initial session. Of these subjects, 1 subject refused to participate because her fear level was too great, and 14 subjects were judged unsuitable because their initial fear levels were not sufficiently high. Thus, all of the analyses to be presented regarding session 1 are based on the results of 76 subjects, 38 in the control group and 38 in the anxious arousal group. Of these subjects, 1 subject, who was assigned to the anxious arousal group during both sessions, did not attend the follow-up session. Thus, all analyses involving the follow-up session are based on the results of the remaining 75 subjects. The results section consists of three subsections. First, data regarding the effects of the experimental manipulation on anxious arousal will be presented. This will be followed by the principal set of analyses, those concerning the effects of anxious arousal on fear. The final subsection presents the results regarding the effects of anxious arousal on self-efficacy.

EVALUATION OF THE EXPERIMENTAL MANIPULATION

Self-reported anxiety (anxiety subscale on the Mood Scale (see Appendix B) and the STAI and heart rate immediately prior to each Behavioral Approach Test (BAT) (i.e., resting heart rate) were evaluated on four occasions during session 1 and on two occasions during session 2. On each occasion, responses of subjects in the anxious arousal group were compared with subjects in the control group. It was hypothesized that

subjects who were in the anxious arousal group during session 1 would exhibit significantly greater anxious arousal as assessed by the three variables immediately following the anxious arousal manipulation (Time 2) and immediately following the fear reduction procedure (Time 3). It was hypothesized that they would exhibit similar levels of anxious arousal to subjects in the control group prior to the anxious arousal manipulation (Time 1) and following anxious arousal equalization (Time 4). It was also hypothesized that subjects who were in the anxious arousal group during session 2 would exhibit greater anxious arousal following the anxious arousal manipulation (Time 6), but not prior to this manipulation (Time 5). The results of the analyses for each dependent variable on each occasion will now be considered.

Session 1:

1. Anxious arousal level prior to the anxious arousal manipulation

(Anxious arousal level at time 1):

The means and standard deviations for the three dependent variables assessing anxious arousal at time 1 are shown in Table 4. A univariate t-test was conducted on each of the three variables assessing anxious arousal level. The two groups did not differ on any of these three variables (anxiety subscale of the Mood Scale: t (74) = 0.01, p > .90; STAI: t (74) = 1.41, p >.15; heart rate: t (74) = -0.18, p > .80.).

Analyses of anxious arousal level at times 2, 3 and 4:

Table 4

Responses of subjects on the anxious arousal measures at time 1

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Anxiety subscale[1]	42.5	19.6	42.4	20.8
STAI[1]	45.1	8.4	42.2	9.5
Heart rate[2]	76.5	11.5	77.0	12.3

[1] Greater values indicate greater self-reported anxiety.

[2] Measured in beats per minute.

univariate t-tests that compared the responses of subjects in the two groups on each assessment occasion. Heart rate immediately prior to each BAT was evaluated with a one-way analysis of covariance, with resting heart rate at time 1 (prior to the anxious arousal manipulation) as the covariate. In order to control Type I error rate, the critical level of significance for the three analyses was calculated using the Bonferroni inequality. Each univariate t-test was evaluated at the .05/3 = .017 probability level.[3]

2. Anxious arousal level immediately following the anxious arousal manipulation

(Anxious arousal level at time 2):

Refer to Table 5 for a presentation of mean scores for the two groups on the three measures of anxious arousal at time 2. The subjects who were in the anxious arousal group reported significantly greater anxious arousal on the anxiety subscale of the Mood Scale (t (74) = -3.18, p < .005) and on the STAI (t (74) = -3.73, p < .0005) and experienced significantly greater heart rate (F (1, 73) = 11.62, p < .0005) compared with subjects in the control group.

3. Anxious arousal level following the fear reduction procedure

(Anxious arousal level at time 3):

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3 Initially, the two self-report measures of anxiety were analysed using analysis of covariance procedures. The results of these analyses indicated that there was no significant linear relationship between the covariate and the dependent variable for three of the analyses. Departures from linearity reduce the efficiency of analysis of covariance and result in biased estimates of the treatment means (Kirk, 1982; Winer, 1971). Consequently, it was more appropriate to analyse these data with analysis of variance procedures.

Table 5

Responses of subjects on the anxious arousal measures at time 2

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Anxiety subscale[1]	31.5	23.7	48.8	23.6
STAI[1]	40.3	9.0	48.8	10.6
Heart rate[2]	72.0	8.8	77.5	12.1
Heart rate$_{adj}$	72.2		77.3	

[1] Greater values indicate greater self-reported anxiety.

[2] Measured in beats per minute.

Refer to Table 6 for a presentation of the mean scores for the two groups on the three variables assessing anxious arousal at time 3. There was no significant difference between the two groups regarding their responses on the anxiety subscale of the Mood Scale (t (74) = -2.26, p = .03); however, the subjects in the anxious arousal group reported significantly more anxious arousal on the STAI (t (74) = -4.53, p < .0001) and experienced significantly higher heart rate (F (1, 73) = 23.97, p < .001) than did subjects in the control group.

As a further check on the experimental manipulation, heart rate was sampled on 14 occasions during the fear reduction procedure. These data were subjected to a two (Group: Control vs. Anxious arousal) by 14 (Time) analysis of variance, with repeated measures on the second factor. It was found that there was a significant main effect of group (F (1, 74) = 13.38, p < .001). The subjects in the anxious arousal group had a significiantly greater heart rate (M = 79.8) than the subjects in the control group (M = 71.9). In evaluating the effect of time and the interaction of time and group, the degrees of freedom were adjusted using the procedure recommended by Kirk (1982). This procedure controls for violations of the sphericity assumption and involves adjusting the degrees of freedom from (k-1) and (n-1)(k-1) (where k is the number of treatments and n is the number of subjects) to (k-1) and (k-1)(n-1) resulting in .4817 (13) = 6.26 and .4817 (13)(75) = 469.7 degrees of freedom. Using

Table 6

Responses of subjects on the anxious arousal measures at time 3

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Anxiety subscale[1]	29.5	23.3	41.7	23.6
STAI[1]	37.4	9.7	49.5	13.2
Heart rate[2]	72.3	7.4	78.7	9.5
Heart rate$_{adj}$	72.4		78.6	

[1] Greater values indicate greater self-reported anxiety.

[2] Measured in beats per minute.

this adjustment, it was found that there was a significant effect of time (F (6.26, 469.7) = 3.68, p < .01). As this effect is not pertinent, simple main effects were not calculated. The interaction of group and time was not significant (F (6.26, 469.7) = 1.96, p > .05).

4. Anxious arousal level following anxious arousal equalization

(Anxious arousal level at time 4):

Refer to Table 7 for a presentation of means for the two groups on the three variables assessing anxious arousal at time 4. The subjects in the two groups did not differ with respect to their self-reported anxious arousal level on either the anxiety subscale of the Mood Scale (t (74) = 0.81, p > .40), the STAI (t (74) = -2.20, p = .03) or with respect to their resting heart rate (F (1, 73) = 4.75, p = .03).

Session 2:

5. Anxious arousal level prior to the arousal manipulation

(Anxious arousal level at time 5):

Refer to Table 8 for a presentation of the group means on the three variables assessing anxious arousal at time 5. Self-reported anxious arousal and heart rate were evaluated with three univariate analyses that compared subjects who were in the control and anxious arousal groups during session 2. The subjects in the two groups did not differ with respect to these three variables (anxiety subscale of the Mood Scale: t (73) = -0.33, p > .70; STAI: t (73) = 0.49, p > .60; heart rate t (73) = 1.33, p > .15).

Table 7

Responses of subjects on the anxious arousal measures at time 4

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Anxiety subscale[1]	16.6	22.6	20.2	15.3
STAI[1]	33.6	8.9	38.4	9.8
Heart rate[2]	71.9	8.9	75.2	10.4
Heart rate$_{adj}$	72.1		75.1	

[1] Greater values indicate greater self-reported anxiety.

[2] Measured in beats per minute.

Table 8

Responses of subjects on the anxious arousal measures at time 5

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Anxiety subscale[1]	36.3	25.7	38.1	24.1
STAI[1]	40.5	10.8	39.3	10.2
Heart rate[2]	75.7	11.1	72.6	8.8

[1] Greater values indicate greater self-reported anxiety.

[2] Measured in beats per minute.

6. Anxious arousal level following the anxious arousal manipulation

(Anxious arousal level at time 6):

Refer to Table 9 for a presentation of the mean scores on the three measures of anxious arousal at time 6. Each of the two self-report measures of anxious arousal was evaluated using a univariate t-test that compared the responses of subjects in the control and anxious arousal groups. The subjects in the anxious arousal group reported significantly more anxious arousal as assessed by the anxiety subscale of the Mood Scale (t (73) = -5.14, p < .0001) and the STAI (t (73) = -5.51, p < .0001). Heart rate immediately prior to the BAT was analyzed with a one-way analysis of covariance, with heart rate at time 5 as the covariate. The subjects in the two groups did not differ significantly with respect to their heart rate prior to the BAT (F (1, 72) = 0.15, p > .60). Because there was heterogeneity of the regression slopes (F (1, 71) = 7.6, p < .01), the adjusted means are not presented.

Heart rate was also sampled on six occasions during the interval prior to the last BAT, while subjects in the anxious arousal group were exposed to the random shock contingency. These data were evaluated with a 2 (Group: Control vs Anxious arousal) by 6 (Time) analysis of variance, with repeated measures on the second factor. There was a significant main effect of group, (F (1, 73) = 5.12, p < .05). The subjects in the anxious arousal group had significantly greater heart rate

Table 9

Responses of subjects on the anxious arousal measures at time 6

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Anxiety subscale[1]	22.9	21.4	50.1	24.3
STAI[1]	35.4	9.7	48.8	11.3
Heart rate[2]	73.8	10.7	72.1	7.1

[1] Greater values indicate greater self-reported anxiety.

[2] Measured in beats per minute.

($\underline{M}$ = 75.9) than the subjects in the control group ($\underline{M}$ = 71.6). In evaluating the effect of time and the interaction of time and group the degrees of freedom were adjusted using the epsilon adjustment procedure that was described earlier, resulting in .7700(5) = 3.8 and .7700(5)(74) = 284.9 degrees of freedom. Neither the effect of time ($\underline{F}$ (3.8, 284.9) = 1.23, $\underline{p}$ > .25) nor the interaction of group and time ($\underline{F}$ (3.8, 284.9) = 1.69, $\underline{p}$ > .15) were significant.

In summary, with several minor inconsistencies, the data indicate that the arousal manipulation increased both subjective and physiological indices of anxious arousal.

ANALYSES OF THE EFFECTS OF ANXIOUS AROUSAL ON FEAR

Analyses concerning each set of predictions regarding fear levels will be presented in this section. In all of the analyses, self-reported levels of fear (SUDS scores) were evaluated using either univariate t-tests or univariate analysis of variance procedures. Heart rate responses were analyzed using analyses of covariance. Heart rate immediately prior to each BAT (i.e., resting heart rate) was covaried out in these analyses. In order to control for Type I error rate, the critical level of significance for each univariate test was calculated using the Bonferroni inequality (i.e., .05/3 = .017).[4]

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2 On each assessment occasion, three dependent variables pertinent to fear (SUDS score, heart rate response, self-efficacy) were evaluated. The results for the third dependent variable, self-efficacy, will be presented in the next section.

1. Fear levels prior to the anxious arousal manipulation (Fear at time 1).

The level of self-reported fear of subjects in the control and anxious arousal groups at time 1 were compared using a univariate t-test. The subjects in the two groups were not significantly different with respect to their self-reported levels of fear (t (74) = 0.65, p > .50). Subjects in the control group reported a mean SUDS score of 80.9 (SD = 8.2) and subjects in the anxious arousal group reported a mean SUDS score of 79.8 (SD = 8.1).

Heart rate response was analyzed with a one-way analysis of covariance, with heart rate immediately prior to the BAT as the covariate.[5] The two groups did not differ with respect to their heart rate response at time 1 (F (1, 73) = 0.73, p > .30). Group means for heart rate immediately prior to the BAT, heart rate response and adjusted heart rate response at time 2 are presented in Table 10.

2. Effects of anxious arousal on pre-exposure fear level (Fear at time 2).

The levels of self-reported fear in the anxious arousal and the control groups at time 2 were compared using a univariate t-test. The subjects in the two groups were not significantly different with respect to this dependent variable (t (74) = -1.15, p > .25). Subjects in the control

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5 It may seem that analysis of covariance procedures will remove that effect of the treatment. This is not the case, however, as the independent variable of interest is not the anxious arousal manipulation. Rather, the analysis of interest examines the effects of exposure to the feared stimulus given the different levels of the independent variable.

Table 10

Mean initial heart rate, heart rate response, and heart rate response adjusted for initial heart rate at time 1

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Initial heart rate[1]	76.5	11.5	77.0	12.3
Heart rate response[1]	100.6	13.2	98.7	12.7
Adjusted heart rate response[1]	100.7		98.6	

[1] Measured in beats per minute.

group reported a mean SUDS score of 57.0 (*SD* = 18.7) and subjects in the anxious arousal group reported a mean SUDS score of 61.9 (*SD* = 18.4).

Heart rate response was analyzed with a one-way analysis of covariance, with heart rate immediately prior to the BAT as the covariate. The two groups did not differ with respect to their heart rate response at time 2 (F (1, 73) = 3.07, p > .05). Group means for heart rate immediately prior to the BAT, heart rate response and adjusted heart rate response at time 2 are presented in Table 11.

2. Effects of anxious arousal during fear reduction on fear level immediately following fear reduction

(Fear at time 3)

The SUDS scores reported by subjects in the two groups at time 3 were compared using a univariate t-test. The subjects in the anxious arousal group reported significantly greater fear (M = 41.3, *SD* = 22.0) than did the subjects in the control group (M = 28.0, *SD* = 20.4) (t (74) = -2.73, p < .01). Heart rate response was evaluated with a one-way analysis of covariance, with heart rate immediately prior to the BAT as the covariate. There was a significant group difference (F (1, 73) = 6.03, p < .017). The subjects in the control group experienced significantly greater heart rate response upon exposure to the snake than did the subjects in the anxious arousal group. Refer to Table 12 for a presentation of mean scores for each group.

Table 11

Mean initial heart rate, heart rate response, and heart rate response adjusted for initial heart rate at time 2

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Initial heart rate[1]	72.0	8.8	77.5	12.1
Heart rate response[1]	91.3	13.4	91.8	13.2
Adjusted heart rate response[1]	93.6		89.5	

[1] Measured in beats per minute.

Table 12

Mean initial heart rate, heart rate response, and heart rate response adjusted for initial heart rate at time 3

	Group			
Variable	Control		Arousal	
	M	SD	M	SD
Initial heart rate[1]	72.3	7.4	78.7	9.5
Heart rate response[1]	84.1	12.8	85.8	13.8
Adjusted heart rate response[1]	87.8		82.2	

[1] Measured in beats per minute.

3. Effects of the removal of heightened anxious arousal on fear level

(Change in fear between time 3 and time 4):

A two (Group: Control versus Anxious arousal) by two (Time: Following fear reduction [Time 3] versus Following anxious arousal equalization [Time 4]) univariate analysis of variance with repeated measures on the second factor was conducted with the SUDS score data. There was a significant main effect of group (F (1, 74) = 9.16, $p < .005$). The subjects in the anxious arousal group (M = 35.0, SD = 23.7) reported significantly greater fear than did subjects in the control group (M = 21.3, SD = 19.2). There was also a significant main effect for time (F (1, 74) = 96.45, $p <$ .0001). The subjects reported significantly less fear following anxious arousal equalization (Time 4) (M = 21.7, SD = 21.3) than they did following fear reduction (Time 3) (M = 34.7, SD = 22.1). The interaction effect was not significant (F (1, 74) = 0.13, $p > .70$).

Heart rate response was analyzed with a two (Group: Control versus Anxious arousal) by two (Time: Following fear reduction [Time 3] versus Following anxious arousal equalization [Time 4]) analysis of covariance with repeated measures on the second factor. Heart rate immediately prior to each BAT functioned as the covariate in the analysis. There was a significant main effect for time ($\underline{F}$ (1, 73) = 12.39, $\underline{p} < .001$). The subjects' mean adjusted heart rate reponse was significantly less during the fourth BAT compared with the third BAT. Neither the main effect of group ($\underline{F}$ (1,

73) = 4.41, $p > .017$) nor the interaction of group and time (F (1, 73) = 0.14, $p > .70$) were significant. The means for heart rate immediately prior to each BAT, heart rate response during each BAT, and adjusted heart rate response are presented in Table 13.

5. Effects of anxious arousal during fear reduction and at follow-up on the return of fear.

(Change in fear between time 4 and time 6):

The following predictions were evaluated with three two-way univariate analyses of variance and two two-way univariate analyses of covariance. In order to control the problem of escalating Type I error rate among the univariate analyses, the overall error rate was set at .15 (the sum of the .05 error rates for the two main effects and the interaction of the main effects) (Kirk, 1982). Using the Bonferroni inequality, each of the 15 effects (two main effects and an interaction effect in each of three analyses of variance and two analyses of covariance) as well as any subsequent simple effects (Winer, 1971) were evaluated at the .15/15 = .01 significance level.

In order to evaluate the prediction made by dual process theory (i.e., that subjects who are aroused at follow-up will show a significantly greater increase in fear between exposure and follow-up relative to subjects who are not aroused at follow-up), a two (Anxious arousal level during the follow-up assessment: Control versus Anxious arousal) by two (Time: Time 4 versus Time 6) univariate analysis of variance with repeated

Table 13

Mean initial heart rate, heart rate response and heart rate response adjusted for initial heart rate at time 3 and time 4

		Group			
		Control		Arousal	
Time	Variable	M	SD	M	SD
Time 3	Initial heart rate[1]	72.3	7.4	78.7	9.5
	Heart rate response[1]	84.1	12.8	85.8	13.8
	Adjusted heart rate response[1]	86.1		82.1	
Time 4	Initial heart rate[1]	71.9	8.9	75.2	10.3
	Heart rate response[1]	80.6	12.3	82.3	13.4
	Adjusted heart rate response[1]	83.0		81.7	

[1] Measured in beats per minute.

measures on the second factor was conducted using the SUDS scores. A graphic representation of the group means at time 4 and time 6 is presented in Figure 2. This analysis indicated that there was not a significant main effect of group (F (1, 73) = 0.22, p > .60), but there was a significant main effect of time (F (1, 73) = 19.16, p < .0001). The subjects reported significantly greater fear at follow-up (M = 31.1, SD = 26.5) than they did at the end of the first session (M = 21.7, SD = 21.3). The interaction of group and time was not statistically significant at the .01 level (F (1, 73) = 6.15, p = .015). The effect of anxious arousal level at follow-up on heart rate response was evaluated using a two (Anxious arousal level during the follow-up assessment: Control versus Anxious arousal) by two (Time: Time 4 versus Time 6) analysis of covariance with repeated measures on the second factor. Heart rate prior to each BAT was used as a covariate in this analysis. The main effect of group (F (1, 72) = 4.15, p > .01), time (F (1, 72) = 2.00, p > .15), and the interaction of group and time (F (1, 72) = 0.59, p > .40) were all nonsignificant. Refer to Table 14 for a presentation of the mean scores.

In order to evaluate the prediction made by cortical theory, namely that subjects who are in congruent states of anxious arousal during fear reduction and at follow-up will evidence significantly less return of fear than will subjects who experience incongruent states of anxious arousal on the two occasions, a two (Group: Congruent states of anxious arousal versus Incongruent states of anxious arousal) by two

Figure 2
Mean SUDS scores at time 4 and time 6 for subjects in the control group and anxious arousal group at follow-up

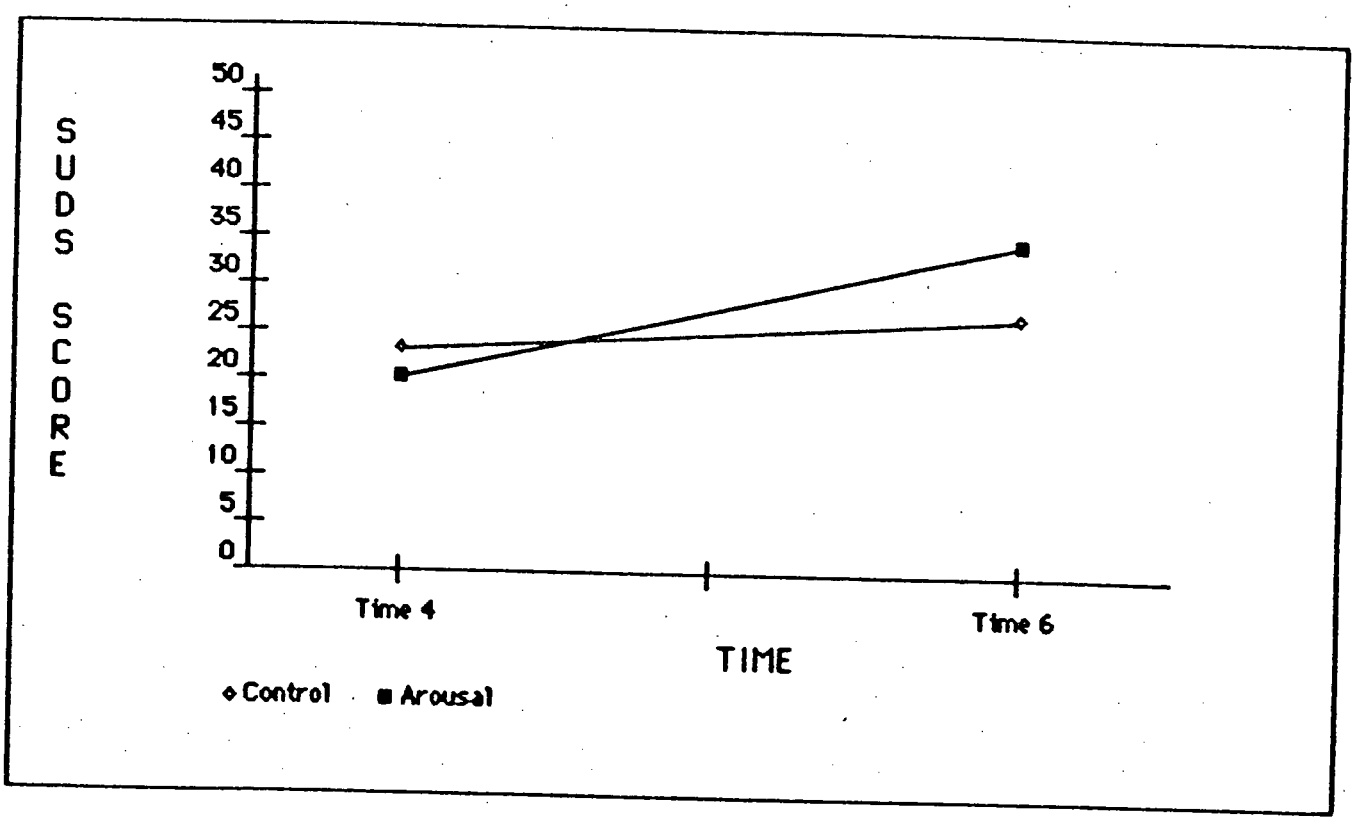


Table 14

Mean initial heart rate, heart rate response and heart rate response adjusted for initial heart rate at time 4 and time 6 for subjects who were in the control and anxious arousal groups at follow-up

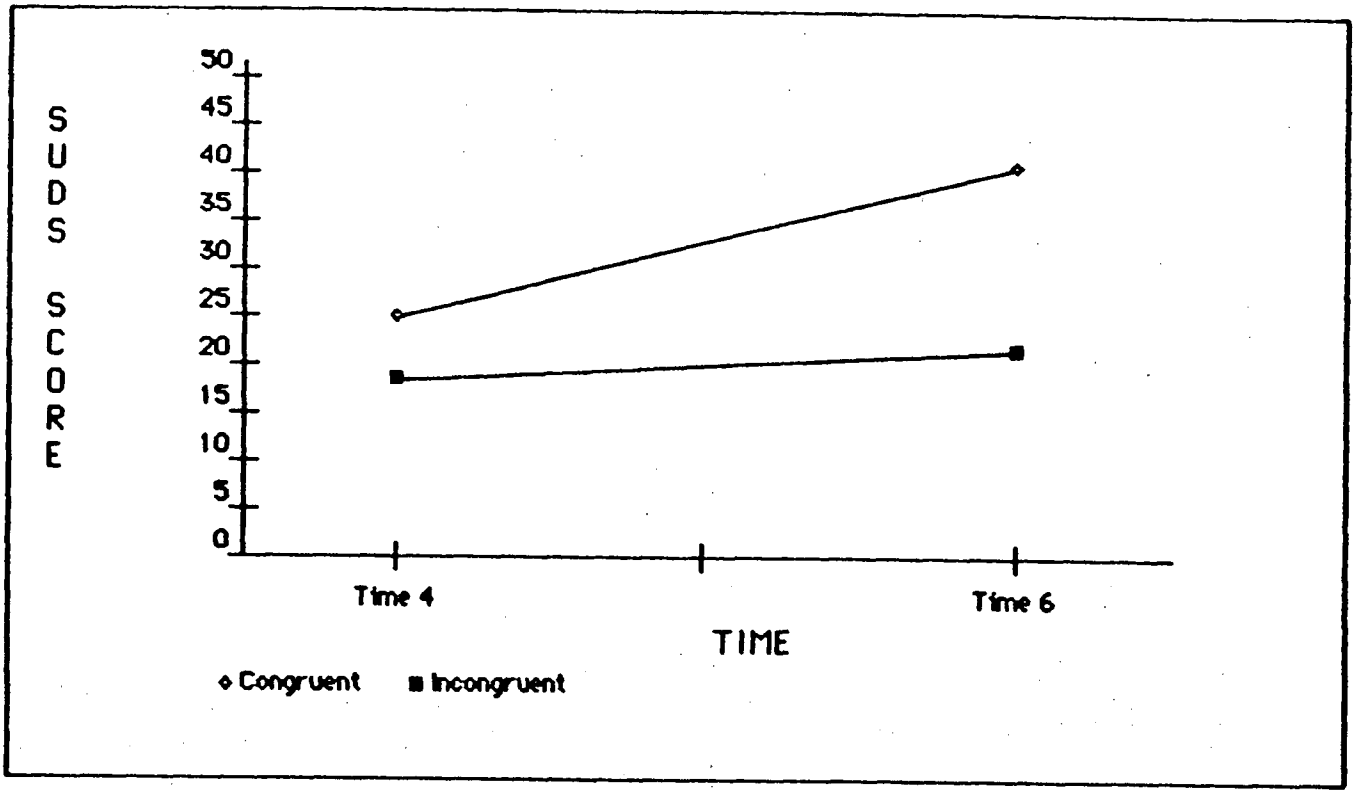
		Group			
		Control		Arousal	
Time	Variable	M	SD	M	SD
Time 4	Initial heart rate[1]	73.8	10.5	73.6	9.0
	Heart rate response[1]	80.5	13.0	82.6	12.9
	Adjusted heart rate[1] response	80.1		82.3	
Time 6	Initial heart rate[1]	73.8	10.7	72.1	7.1
	Heart rate response[1]	81.3	11.7	83.9	12.9
	Adjusted heart rate[1] response	80.8		85.1	

[1] Measured in beats per minute.

(Time: Time 4 versus Time 6) analysis of variance with repeated measures on the second factor was conducted on the SUDS scores. The main effect of group was not significant (F (1, 73) = 6.48, p = .013), however, there was a significant main effect of time (F (1, 73) = 19.96, p < .0001). This effect was qualified by a significant interaction af group and time (F (1, 73) = 9.20, p < .01). Subsequent simple main effects analyses indicated that subjects who experienced congruent states of anxious arousal during the first session and at follow-up did not differ from subjects who experienced incongruent states of anxious arousal with respect to their reported fear at time 4 (F (1, 98.9) = 1.07, p > .25); however, subjects who experienced congruent states of anxious arousal reported significantly greater fear at time 6 than subjects who experienced incongruent states of anxious arousal on the two occasions (F (1, 98.9) = 11.42, p < .005). Although subjects who experienced incongruent states of anxious arousal on the two occasions did not differ with respect to their reported fear at time 4 compared with time 6 (F (1, 74) = 1.04, p > .25) subjects who experienced congruent states of anxious arousal did report significantly greater fear at time 6 compared with time 4 (F (1, 72) = 27.79, p < .001). Refer to Figure 3 for a graphic representation of the group means at time 4 and time 6. In order to examine these findings further, these data were subsequently analysed with an analysis of variance on the residual gain SUDS scores at time 6 which compared the responses of subjects in each of the four groups:

Figure 3

Mean SUDS scores at time 4 and time 6 for subjects in congruent and incongruent states of anxious arousal during session 1 and session 2



control group-session 1, control group-session 2 (CC); control group-session 1, anxious arousal group-session 2 (CA); anxious arousal group-session 1, control group-session 2 (AC); anxious arousal group-session 1, anxious arousal group-session 2 (AA). Refer to Appendix E for a description of this analysis. The results indicated that the groups differed with respect to their residual gain scores (F (3, 71) = 6.65, p < .001). The data were further analysed using Newman-Keuls multiple comparison procedures. The results of this analysis indicated that subjects in group AC exhibited significantly less return of fear than predicted compared with subjects in group AA (p < .001) or group CC (p < .01). Subjects in group AC exhibited less return of fear than predicted compared with subjects in group CA, however, this difference was not significant (p = .015). None of the other differences were significant (all ps > .10). Refer to Table 15 for a presentation of SUDS scores at time 4, time 6, and the residual gain SUDS scores for subjects in the four groups.

The four groups were also compared with respect to the proportion of subjects in each group who exhibited return of fear (defined as an increase in self-reported fear of at least 10 SUDS units between time 4 and time 6). The results of a chi square test indicated that the four groups differed significantly with respect to the proportion of subjects who experienced return of fear (χ^2 (3, N = 75) = 14.72, p < .01). Refer to Table 16 for a tabular presentation of the percentage of subjects in each of the four groups who experienced return of fear. Follow-up multiple comparison

Table 15

SUDS scores at time 4, time 6, and residual gain SUDS scores for subjects in the four groups

	Group							
	AC		AA		CC		CA	
	M	SD	M	SD	M	SD	M	SD
SUDS score-Time 4	27.5	20.2	31.1	28.0	18.9	15.6	10.0	14.1
SUDS score-Time 6	21.9	16.5	49.2	29.6	32.6	24.8	21.6	25.8
Residual gain score	-14.1		10.0		4.0		0.7	

Table 16

Proportion of subjects in each of the four groups who showed return of fear

	Group							
	AC		AA		CC		CA	
	%	(n)	%	(n)	%	(n)	%	(n)
Return of fear	11	(2)	67	(12)	63	(12)	47	(9)
No return of fear	89	(17)	33	(6)	37	(7)	53	(10)

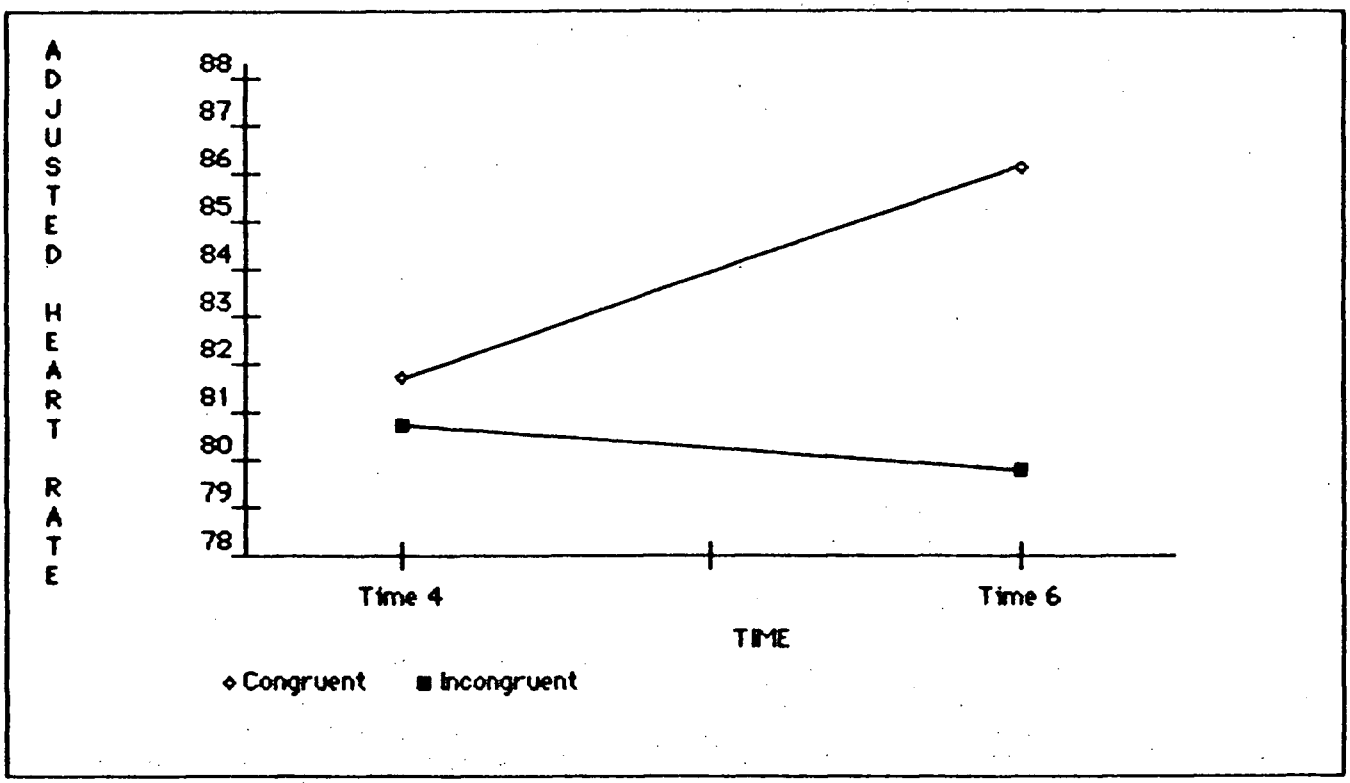
tests on the proportions indicated that the percentage of subjects who exhibited return of fear was significantly smaller in group AC compared with group AA ($p < .001$) or group CC ($p < .001$) The proportion of subjects in group AC who exhibited return of fear was smaller than the proportion of subjects in group CA who exhibited return of fear, however, this difference was not significant ($p = .03$). All other comparisons were nonsignificant (all ps $> .30$).

The effects of congruence of anxious arousal between sessions on heart rate response was evaluated using a two (Group: Congruent versus Incongruent) by two (Time: Time 4 versus Time 6) analysis of covariance with repeated measures on the second factor. Heart rate prior to each BAT was used as a covariate in this analysis. Neither the main effect of time (F (1, 72) = 2.23, $p > .10$), the main effect of group, (F (1, 72) = 5.61, $p = .02$), nor the interaction of group by time (F (1, 72) = 4.41, $p = .04$) were significant using the corrected alpha level. Refer to Figure 4 for a graphic representation of the group means at time 4 and time 6.

In order to examine these findings further, these data were subsequently analysed with an analysis of variance on the residual gain heart rate responses at time 6. This analysis compared the responses of subjects in the four groups (CC vs. CA vs. AC vs. AA). Heart rate response at time 6 (adjusted for heart rate immediately prior to the BAT) was regressed on the adjusted heart rate response at time 4. The score that would be predicted at time 6 from the adjusted heart rate response at time 4 was then computed. The residual adjusted heart rate

Figure 4

Mean adjusted heart rate responses at time 4 and time 6 for subjects in congruent and incongruent states of anxious arousal during session 1 and session 2



response at time 6 was computed as the difference between the adjusted heart rate response at time 6 and the predicted adjusted heart rate response at time 6. The results indicated that the residual heart rate responses of subjects in the four groups were not significantly different (F (3, 71) = 2.75, p = .05). The residual gain scores were as follows: AC: M_{res} = -5.40; CA: M_{res} = .74; CC: M_{res} = 2.41; AA: M_{res} = 3.41.

ANALYSIS OF THE EFFECTS OF ANXIOUS AROUSAL ON SELF-EFFICACY

1. Effect of anxious arousal on self-efficacy prior to fear reduction

(Self-efficacy at time 2):

The levels of reported self-efficacy at time 2 of subjects in the control and anxious arousal groups were compared using a t-test. The subjects in the two groups did not differ (t (64.4) = 0.51, p > .60). Subjects in the control group reported a mean level of self-efficacy of 66.2 (SD = 20.3) compared with subjects in the anxious arousal group who reported a mean level of self-efficacy of 63.2 (SD = 30.4).

2. Effect of anxious arousal during fear reduction on self-efficacy following fear reduction

(Self-efficacy at time 3):

The levels of reported self-efficacy at time 3 of subjects in the two groups were compared with a t-test. The subjects in the two groups did not differ (t (74) = 0.22, p > .80). Subjects in the control group reported a mean level of self-efficacy of 79.8 (SD = 22.3) compared with subjects in

the anxious arousal group who reported a mean level of self-efficacy of 78.7 (SD = 20.5).

3. Effect of the removal of heightened anxious arousal on self-efficacy

(Change in self-efficacy between time 3 and time 4):

Self-efficacy scores were analysed using a two (Group: Control vs. Anxious arousal) by two (Time: Following fear reduction [Time 3] vs. Following anxious arousal equalization [Time 4]) univariate analysis of variance with repeated measures on the second factor. There was a significant main effect of time ($F(1, 74) = 13.59$, $p < .0005$). Subjects reported significantly greater self-efficacy at time 4 ($M = 88.0$, $SD = 18.3$) compared with time 3 ($M = 79.2$, $SD = 21.3$). Neither the univariate main effect of group ($F(1, 74) = 0.87$, $p > .35$) nor the interaction of group and time ($F(1, 74) = 1.11$, $p > .25$) were significant. 4. Effect of anxious arousal at follow-up on self-efficacy

(Change in self-efficacy between time 4 and time 6):

The results of a two (Anxious arousal level during the follow-up assessment: Control vs. Anxious arousal) by two (Time: Time 4 vs Time 6) univariate analysis of variance with repeated measures on the second factor indicated that there was not a significant main effect of group ($F(1, 73) = 4.27$, $p > .01$). There was a significant main effect of time ($F(1, 73) = 7.87$, $p < .01$). Subjects reported significantly less self-efficacy at follow-up ($M = 80.8$, $SD = 18.2$) compared with the end of the first session ($M = 87.9$, $SD = 18.4$). The interaction of group and time was not significant ($F(1, 73)$

= 0.90, $p > .30$). Refer to Table 17 for a presentation of mean self-efficacy scores of subjects in the two groups at the end of the first session and at follow-up.

Table 17

Mean self-efficacy scores at time 4 and time 6 of subjects in the control and anxious arousal groups at follow-up

	Group			
Time	Control at follow-up		Arousal at follow-up	
	M[1]	SD	M[1]	SD
Time 4[1]	90.0	11.0	85.3	23.7
Time 6[1]	85.3	13.7	76.1	21.0

[1] Greater values indicate greater reported self-efficacy.

DISCUSSION

The effectiveness of the experimental manipulation on measures of anxious arousal is discussed first in this chapter. This is followed by discussions regarding the effect of anxious arousal on fear, fear reduction, and the return of fear. The impact of anxious arousal on self-efficacy expectations is considered next. This is followed by a discussion of the theoretical and clinical implications of the findings. The chapter concludes with a summary of the findings and suggested directions for future research.

EVALUATION OF THE EXPERIMENTAL MANIPULATION

Several predictions were made regarding the effects of the experimental manipulation on the subjects' level of anxious arousal. As predicted, the subjects in the two groups did not differ on any of the three measures of anxious arousal prior to the anxious arousal manipulation during the first session. Also as predicted, the anxious arousal manipulation effectively increased self-reported levels of anxious arousal and heart rate prior to the fear reduction procedure and immediately following the procedure. Subjects who were in the anxious arousal group also experienced increased heart rate during the fear reduction procedure relative to the subjects in the control group. The one exception to these findings was with respect to a nonsignificant difference between the two groups on the anxiety subscale of the Mood Scale following fear reduction. However, although the subjects in the two groups did not

differ on this measure, the difference was in the expected direction and approached significance ($p = .03$).

The anxious arousal manipulation was more robust than expected in the sense that residual anxious arousal effects were still apparent following the anxious arousal equalization period. Although the subjects in the two groups were not significantly different on the three measures of anxious arousal at this time, their responses on the STAI and their resting heart rate indicated a tendency for the subjects who were previously anxiously aroused to continue to be anxiously aroused relative to the subjects in the control group. This reduces the likelihood of identifying differences that are dependent on changes in anxious arousal level.

As predicted, subjects who were in the control and anxious arousal groups during session 2 did not differ on the three measures of anxious arousal prior to the anxious arousal manipulation. As in session 1, implementation of the anxious arousal manipulation during the second session resulted in subjective reports of increased anxious arousal for the subjects in the anxious arousal group compared with the subjects in the control group. The subjects in the anxious arousal group also experienced greater heart rate during the six minute interval prior to the BAT, while they were exposed to the random shock contingency, than did the subjects in the control group; however, the two groups did not differ with respect to heart rate immediately prior to

the BAT. It is not entirely clear why there is this inconsistency, however, two possible explanations are apparent. Firstly, heart rate was sampled on six occasions during the six minute interval compared with one sample of heart rate which was taken immediately prior to the BAT, resulting in the prior data set being more reliable. Secondly, during the six minute interval, subjects were actually exposed to the random shocks; the subjects were not told that the random shock contingency was no longer in effect immediately prior to the BAT but it may be that their previously elevated heart rate had decreased as a result of not receiving any shocks during the previous several minutes.

In summary, although there are several minor inconsistencies, the data indicate that the anxious arousal manipulation successfully increased both subjective and physiological indices of anxious arousal. This is consistent with prior research that has found threat of shock to be an efficacious method of increasing anxious arousal (e.g., Bohlin, 1976; Briush & Schwartz, 1980; Carrol & Pokora, 1976; Watts, 1975).

EFFECTS OF ANXIOUS AROUSAL ON FEAR

Results pertinent to the effects of anxious arousal on fear, fear reduction, and the return of fear will be discussed in the following three subsections.

Effects of anxious arousal on within-session changes in fear

It was predicted based on dual process theory (e.g., Groves & Thompson, 1970; Thompson et al., 1979; Thompson et al., 1973; Thompson & Spencer, 1966) that changes in anxious arousal level would influence fear levels. Specifically, it was predicted that fear levels would increase given increases in anxious arousal, and decrease given decreases in anxious arousal. It was found that neither increases in anxious arousal prior to fear reduction nor decreases in anxious arousal following fear reduction significantly influenced either self-reported levels of fear or heart rate response upon exposure to the feared stimulus.[5] Thus, it can be concluded that there is no support for the prediction based on dual process theory that level of anxious arousal influences fear within-session. This result is problematic for dual process theory as the prediction that increased sensitization (or, to use the terminology adopted in the present study "anxious arousal") will result in increased general responsiveness is a fundamental aspect of this theory. However, the relationship between anxious arousal level and responsiveness has been documented in several

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5 Because the two groups were not completely equivalent with respect to their arousal levels following the anxious arousal equalization period, the evaluation of the effect of reductions in anxious arousal is not as cogent a test of this prediction as would be desired. However, given that there is not any evidence of a trend to support this prediction (both ps for self reported fear and heart rate response are greater than .70) it is unlikely that this result would differ substantially given greater equivalence between the two groups following arousal equalization.

studies (e.g., Edwards & Siddle, 1976; Groves & Thompson, 1970; McKubbin & Katkin, 1971; Rust, 1976; Thompson & Siddle, 1966) and it appears that the effect is a relatively robust one. Consequently, the theory is not seriously disputed by this finding. There are two possible explanations to account for the inability to replicate this finding in the current study. Firstly, it may be that the experimental manipulation was ineffective in changing anxious arousal level. This possibility is unlikely, however, as the present study used a manipulation that seems more potent than other manipulations (e.g., low intensity tone, light stimulus) that have found an effect. Furthermore, the manipulation had the intended effect with respect to both self-reported anxious arousal level and heart rate.

The second possibility concerns the type of stimulus and responses studied. Prior researchers have used habituating stimuli (e.g., auditory tones, single shock pulses) and responses (e.g., skin conductance response, hind limb flexion reflex, startle reflex) that have differed radically from those used in the current study, and the clearest results have been obtained from nonhuman subjects. The stimuli typically used in habituation studies differ from the stimulus used in the current study in at least two fundamental respects. First of all, the snake stimulus is a more complex stimulus than the stimuli used in other studies and secondly, it has, initially at least, a negative

affective valence (i.e., subjects are fearful of the stimulus). As well as the differences in stimuli, there are differences in the responses that were assessed. Self-reported fear is a more complex response than that typically assessed. An individual's self-reported fear level and the magnitude of his or her heart rate response depends on a number of attentional and cognitive operations and evaluations. Although there was a high degree of experimental control for a study of this type, the current study does not allow the degree of experimental precision that has been achieved in previous habituation studies. It would be impossible to achieve the degree of control that can be achieved in other studies that have used different stimuli, responses, and experimental preparations. Although the results of the current study do not invalidate dual process theory, they do question its generalizability to human fear responses to exposure to a feared stimulus.

Cortical theory (e.g., Wagner, 1976; Whitlow, 1975; Whitlow & Wagner, 1984) does not accord a significant role to the effects of anxious arousal per se on fear. However, it was predicted based on this theory that increases in anxious arousal prior to fear reduction would result in the feared stimulus being less likely to be cued in memory prior to the BAT, which would result in an increase in fear relative to subjects in the control group who were not exposed to the feared stimulus under conditions of anxious arousal. It should be noted that although the rationale for

this prediction differs from that of dual process theory, the prediction is the same. It was found that exposure to the anxious arousal inducing stimulus prior to fear reduction did not affect the levels of fear as assessed either through self-report or heart rate response. It was also predicted based on cortical theory that the removal of anxious arousal which was present during fear reduction would result in an increase in fear relative to subjects who experienced fear reduction under conditions of normal arousal. The reason for this prediction was that anxious arousal functions as a memory cue to prime the feared stimulus into short term memory. The subjects who were previously anxiously aroused experienced a greater degree of incongruence in anxious arousal level between fear reduction and later assessment than did subjects in the control group and thus the feared stimulus will be less primed in memory upon exposure to the actual stimulus. This prediction differs from that based on dual process theory. It was found that the degree of congruence in anxious arousal level did not have any effect on either measure of fear. Thus, this prediction is not supported. This failure to find an effect of anxious arousal is problematic for cortical theory but does not necessarily invalidate the theory. It is possible that other contextual cues were used to prime the stimulus into short term memory. All aspects of the experimental situation, other than anxious arousal level state, were held constant. If these other contextual factors were the salient

ones for the individual, then, as was found in the current study, differences in fear response would not be expected as a result of changes in anxious arousal level. Thus, although it can be concluded from these results that anxious arousal state was not an important cue used to prime the feared stimulus into short term memory, it cannot be concluded that the priming process per se is not important in determining level of fear response upon exposure to feared stimuli. As well, the current discussion concerns short term within-session (rather than long term) influences of anxious arousal on fear.

Effects of anxious arousal on fear reduction

Both dual process theory and cortical theory predict that subjects who experienced anxious arousal during fear reduction would experience significantly greater fear following fear reduction (i.e., significantly less fear reduction) than would subjects who did not experience anxious arousal during fear reduction. Both theories make this prediction based on the fact that anxious arousal results in a distraction of attention away from the feared stimulus, resulting in less functional exposure (Borkovec & Grayson, 1980) and emotional processing (e.g., Foa & Kozak, 1985, 1986; Lang, 1977; Rachman, 1980) than would occur given lower levels of anxious arousal. Dual process theory, in addition, explains this effect as due to an increase in general responsiveness at the time of assessment as a result of the anxious arousal. However, as discussed previously,

neither decreases nor increases in anxious arousal had any impact on fear levels within-session and, consequently, any differences that resulted from anxious arousal during fear reduction are not likely due simply to increased anxious arousal causing an increase in fear.

A very interesting pattern of results emerged. The prediction made by these two theories was supported with respect to self-reported fear. Fear reduction treatment that occurred while the individual was in a state of anxious arousal resulted in less fear reduction (i.e., greater fear following exposure treatment) than did occur under conditions in which less anxious arousal was present. This finding is congruent with previous research that has found that anxious arousal during habituation training impedes habituation (e.g., Bohlin, 1976; Carrol & Pokora, 1976; Goldwater & Lewis, 1978; Lader & Wing, 1964).

The opposite pattern was found with respect to heart rate response. The subjects who experienced anxious arousal during fear reduction exhibited less heart rate response upon exposure to the feared stimulus following fear reduction. This finding is contrary to the prediction made by the dual process and cortical theories. It seems peculiar that treatment conditions that facilitated the reduction of self-reported levels of fear would inhibit the reduction of heart rate response. The subjects in both groups experienced heart rate responses that were of approximately the same magnitude ($\underline{M}$ = 84.1 for the subjects in the control group

versus 85.8 for the subjects in the anxious arousal group). The difference in response is accounted for by the significant difference in their resting heart rates: the subjects in the control group had a resting heart rate of 72.3 compared with a resting heart rate of 78.7 for subjects in the anxious arousal group. Although the responses of the subjects in the two groups were of the same magnitude, the amount of change upon exposure to the feared stimulus was greater for subjects who were in the control group. This finding is a clear example of discordance (Hodgson & Rachman, 1974; Rachman & Hodgson, 1974) between the self-report and physiological (as indexed by heart rate response) components of fear. If heart rate response is seen solely as a measure of fear, this finding seems unusual. If, instead, it is viewed as an index of attention to the stimulus and of emotional processing (Foa & Kozak, 1985, 1986; Lang, 1977, 1985; Rachman, 1980) the result seems reasonable. The subjects in the control group were in a relatively calm, nonanxious state prior to exposure to the feared stimulus. Upon exposure to the stimulus, they were able to fully attend to the stimulus and, consequently, responded with a moderate heart rate response. In contrast, the subjects in the anxious arousal group were more anxiously aroused prior to exposure to the feared stimulus (as evidenced by their greater resting heart rate and higher self-reported anxious arousal level) than were the subjects in the control group. Consequently, it is conjectured that they were likely

vigilant regarding the anxious arousal inducing stimulus, focussing on internal sensations of anxious arousal, and experiencing intrusive cognitions of a worrisome nature. As a result of these processes they did not likely attend to the feared stimulus during the BAT as fully as did the subjects in the control group. Consequently, the magnitude of their heart rate responses was not as great. It should be noted that this explanation is speculative and needs empirical investigation. This finding is remarkably similar to that which was recently reported by Borkovec and Hu (1990). They examined the effects of worry on fear induced by imaginally presented phobic scenes. They found that although worry prior to visualization resulted in greater self-reported fear in response to visualization, less heart rate response occurred compared with subjects who were not worried prior to visualization. The authors concluded that,

> to the degree that cardiovascular reaction reflects emotional processing, the present outcome suggests that worry may inhibit the processing of phobic material and thus result in a maintenance of the cognitive/affective fear structure despite repeated exposures. It does so without affecting the strong subjective fears that the person reports in response to the phobic presentations. (p. 72)

The present study differs from Borkovec and Hu's in several respects. They used imaginal stimuli as opposed to in vivo stimuli, and they induced task relevant worry rather than anxious arousal. The relationship between anxious arousal and worry is an interesting one that will be

discussed in a later section. In spite of these differences, the above conclusion seems applicable to the current study.

The above results suggest two important facts. First of all, high levels of anxious arousal impede the emotional processing of feared stimuli and fear reduction. Secondly, the amount of heart rate response upon exposure to the feared stimulus (i.e., the magnitude of change) may be an index of emotional processing and fear reduction. As will be discussed later, these results have important theoretical and clinical implications.

Effects of anxious arousal on the return of fear

The results regarding the effects of anxious arousal during fear reduction and at follow-up on the return of fear will now be discussed. It was predicted based on dual process theory that the subjects who were anxiously aroused at follow-up would show a significantly greater increase in fear between the two sessions (i.e., significantly more return of fear) than would the subjects who were not anxiously aroused at follow-up. This prediction was made based on the theoretical premise that experiencing anxious arousal results in an increase in general responsiveness as indexed by the two measures of fear. The overall interaction did not support this prediction ($p = .015$). The results of a subsequent data analysis, which will be discussed shortly, allow a more complex interpretation of these results.

Anxious arousal at follow-up did not have a significant effect on heart rate response. These results do not support

the prediction of dual process theory. The finding of significant differences with respect to self-reported fear (as will be discussed later), but not with heart rate response reflects the fact of differential response system sensitivity (Agras & Jacob, 1971) and is congruent with other studies that have found a similar discrepancy between self-reported fear and heart rate (e.g., Craske & Rachman, 1987).

It was predicted, based on cortical theory, that the subjects who experienced congruent states of anxious arousal during the two sessions would evidence significantly less return of fear than would the subjects who experienced incongruent states of arousal. This prediction is based on the theoretical premise that anxious arousal functions as a memory cue that primes the representation of the feared stimulus into short term memory. Contrary to this prediction, the subjects who experienced congruent, rather than incongruent, states of anxious arousal on the two occasions showed a significant increase in self-reported fear across the follow-up interval. The results regarding the heart rate response data were not significant but suggested a similar pattern of results: subjects who experienced congruent states of anxious arousal on the two occasions showed an increase in the magnitude of their heart rate response across the follow-up interval compared with subjects who experienced incongruent states of anxious

arousal, who did not exhibit any change across the follow-up interval.

These puzzling findings were the impetus of a further set of analyses that examined the pattern of change for all four groups of subjects: those who were in the control group during both sessions (CC), those who were in the control group during one session and in the anxious arousal group during the other session (CA or AC), and those who were in the anxious arousal group during both sessions (AA). The subjects in three of the groups (AA, CC, CA) showed an increase in self-reported fear across the follow-up interval, exhibiting return of fear, as would be expected. These three groups did not differ significantly with respect to the mean amount of return of fear that they exhibited. In contrast, the subjects who were anxiously aroused during exposure treatment, but not at follow-up (AC), showed a decrease in subjective fear across the follow-up interval. Refer back to Table 15 for a presentation of these mean scores. An analysis of the proportion of subjects in each of the four groups was congruent with this finding. Only 11 percent of subjects who were aroused during exposure treatment but not at follow-up experienced a return of fear, compared with 47 to 67 percent of the subjects in the other three groups. A similar analysis using the residual heart rate response data was not significant ($p = .05$) but showed a similar configuration as was found with the self-reported fear data (i.e., the subjects in group AC exhibited a lesser

heart rate response than expected, while the subjects in the other three groups exhibited a greater heart rate response than expected). Thus, the two earlier conclusions, namely that anxious arousal at follow-up does not influence the return of fear, and that there is greater return of fear given congruent, as compared to incongruent, states of anxious arousal, are not entirely correct and must be qualified. Increased anxious arousal at the time of follow-up does not result in increased return of fear. Rather, the flipside of this proposition seems more appropriate: follow-up assessment in a calm state following exposure while in a state of anxious arousal impedes the return of subjective fear and, in fact, results in a substantial decrement in the fear response. This finding cannot be explained simply on the basis of anxious arousal level at the time of follow-up assessment as would be predicted on the basis of dual process theory. If this were the case, in the subsequent analysis of the four groups, the two groups experiencing anxious arousal at follow-up would show greater return of fear than the subjects who were in a relatively calm state on both occasions. Yet, these three groups did not differ. On the other hand, if the amount of return of fear was partially accounted for by anxious arousal level at the time of follow-up, as would be predicted on the basis of dual process theory, and given that there was still residual anxious arousal present during the final assessment during session one, it would be predicted that group AC would show

the least amount of return of fear. This is what was found. Thus, this pattern of results offers partial support for dual process theory.

The results cannot be explained in terms of cortical theory. Congruence in anxious arousal state does not impact on the return of fear. Although the initial evaluation of congruence effects suggested an effect that was opposite to that predicted on the basis of the theory, the subsequent analysis indicated that it was a state of incongruence of a specific type, anxious arousal during fear reduction and nonanxious arousal at follow-up, that promoted further habituation of fear. Cortical theory does not make any allowances for this type of asymmetry. Thus, this theory cannot, except perhaps through some theoretical gymnastics, explain this finding.

Craske and Rachman (1987) found that elevated heart rate prior to treatment did not influence fear reduction but it did result in increased return of subjective fear. The present study found, in contrast to Craske and Rachman, that anxious arousal impeded the reduction of fear. It is unclear why the current study found an effect, but a number of methodological differences may account for this discrepancy. For example, the nature of the fear, the source of the anxious arousal, and the treatment all differed. Craske and Rachman concluded that heightened heart rate prior to treatment resulted in increased return of fear, however, the pattern of results in these two studies do not exclude the

possibility that anxious arousal level during treatment and at follow-up may interactively influence the return of fear.

Rachman and Whittal (1989a) were unable to find evidence of increased return of fear as a result of anxious arousal at follow-up. As mentioned previously, their test was not a cogent one as their manipulation did not increase heart rate. However, it may also be due in part to the fact that all of the subjects experienced fear reduction in an equivalent state of anxious arousal.

EFFECTS OF ANXIOUS AROUSAL ON SELF-EFFICACY EXPECTATIONS

Several predictions regarding the effect of anxious arousal on self-efficacy expectations were made on the basis of Bandura's (1977, 1978, 1982, 1986) self-efficacy theory. There was an absence of support for these predictions. Each prediction will be discussed in turn.

It was predicted that the subjects who were anxiously aroused during the first session would report significantly less self-efficacy relative to the subjects in the control group prior to fear reduction. This prediction was based on the theoretical premise of self-efficacy theory that increased anxious arousal is a source of information indicating to the individual that he or she is currently vulnerable to stress which may impede coping abilities. The subjects in the two groups did not differ with respect to the magnitude of their reported self-efficacy expectations.

It was also predicted that the subjects who were anxiously aroused during fear reduction, and who continued

to be anxiously aroused during the subsequent assessment, would report decreased self-efficacy relative to the subjects in the control group. The reason for this prediction was two-fold. Firstly, the experience of anxious arousal throughout fear reduction would result in decreased functional exposure and would disrupt the development of self-efficacy expectations. Secondly, to the extent that anxious arousal at the time of assessment is a source of information to the individual that he or she is vulnerable to stress, there should be a further decrease in self-efficacy. The subjects in the two groups did not differ with respect to their reported self-efficacy expectations following exposure treatment.

It was predicted that the subjects who experienced an alleviation of anxious arousal would show an increase in self-efficacy relative to the subjects in the control group. It was found that although the subjects in both groups reported an increase in self-efficacy expectations across the time interval, there was not any evidence of a differential increase.

The final prediction concerned the effect of anxious arousal at follow-up on self-efficacy. It was predicted that the subjects who were anxiously aroused at follow-up would evidence a larger decrease in self-efficacy across the follow-up interval than would the subjects in the control group. The subjects in both groups experienced a decline in the magnitude of their self-efficacy expectations across the

follow-up interval, however, the magnitude of this decline did not differ between the two groups.

In summary, the results suggest that a state of anxious arousal, which Bandura (1977, 1978, 1982, 1986) states is one of the four types of information on which self-efficacy judgements are made, did not have any influence on self-efficacy expectations. This study is the first empirical attempt to examine the role of changes in anxious arousal on self-efficacy and is consistent with previous research that did not find a relationship between pre-existing levels of anxious arousal and self-efficacy (Craske & Rachman, 1987; Feltz, 1982; Williams & Watson, 1985) but inconsistent with the two studies that did report such a relationship (Williams et al., 1984; Williams et al., 1985).

Several methodological arguments may be made regarding why there was an absence of an effect. First, perhaps anxious arousal did not have an effect on self-efficacy because the measure was not sensitive. This argument does not seem valid as there were highly significant changes in self-efficacy across assessment occasions that were in the expected direction (i.e., self-efficacy expectations increased during the first session with repeated exposures to the feared stimulus and decreased significantly across the follow-up interval). Further, as has been found in other studies (e.g., Bandura & Adams, 1977; Williams et al., 1984; Williams et al., 1985), a significant inverse relationship between self-reported fear and level of self-efficacy was

noted.[6] The subjects' self-efficacy expectations showed highly significant change as a result of experience with the feared stimulus and showed an inverse relationship with level of self-reported fear. In spite of this, their self-efficacy expectations were not influenced by generalized changes in anxious arousal level. It is interesting to note that in spite of the fact that experiencing anxious arousal during fear reduction disrupted fear reduction, it did not have any impact on self-efficacy expectations. Given the close relationship between fear and self-efficacy that has been claimed (e.g., Bandura & Adams, Bandura et al., 1977; Bandura et al., 1985; Craske & Craig, 1984; Craske & Rachman, 1987; Kendrick et al., 1982) it is surprising that this discrepancy occurred. It suggests that fear response can be influenced without a corresponding change in self-efficacy expectations.

It can be argued that the lack of relationship between anxious arousal level and self-efficacy in the present study is the result of the nature of the anxious arousal manipulation. There are several ways in which the specific nature of the anxious arousal mechanism may have prevented the detection of differences that may have been identified using alternate methods of increasing anxious arousal.

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6 These results were not reported in the results section. It was found that the correlation between level of self-reported fear and self-efficacy on each assessment occasion ranged from -.26 to -.43 with a mean correlation of -.34 (df = 74 for 3 of the comparisons and df = 73 for 1 of the comparisons, $p < .01$).

First, it may be that the amount of anxious arousal was not sufficient to cause a disruption of self-efficacy. The manipulation did successfully increase anxious arousal but perhaps a greater increase, above some minimum criterion level of anxious arousal, is necessary. Although this is an interesting possibility, it is a post hoc argument that is not a part of self-efficacy theory in its present form.

Bandura (1986) states that, "activities are often performed in situations containing varied evocative stimuli. This creates ambiguity about what caused the physiological reactions. The efficacy import of the resultant arousal on self-efficacy will, therefore, vary depending on the factors singled out and the meaning given to them" (p. 406). Perhaps anxious arousal only has an effect on self-efficacy expectations if the source of the anxious arousal can not be specifically identified, or is identified by the individual (whether correctly or incorrectly) as being caused by the feared stimulus. If the individual cannot identify the source of anxious arousal, excitation transfer (Zillman, 1983) may occur and the individual may misattribute the source of anxious arousal as being due to the feared stimulus. Individuals tend to attribute their anxious arousal to stimuli that they are focussing on and that strike them as obvious sources of anxious arousal (Zillman, 1983). In the current study, it may be that the cause of the anxious arousal was specifically identified, and identified as being external to, and independent of, the feared

stimulus. It is not possible to determine the extent to which the anxious arousal that was generated in the current study was attributed by the subjects to the feared stimulus versus the experimental manipulation. However, the attention of subjects was focussed on the feared stimulus which was, by definition, a source of anxious arousal for the subjects.

A final issue concerns the relative influence of anxious arousal compared with the other three possible sources of self-efficacy information (i.e., performance attainments, vicarious experience, and verbal persuasion). Performance attainments and vicarious experience are identified as the most potent sources of efficacy information (Bandura, 1986) and the robust effect of exposure treatments and vicarious experience in reducing fear and increasing self-efficacy are well documented (e.g., Barlow & Beck, 1984; Emmelkamp, 1982a 1982b; Linden, 1981; Rachman & Wilson, 1980). In the current study, both of these sources of efficacy information were present. It may be that they are much more salient and powerful influences on self-efficacy compared with anxious arousal state and, consequently, they override any effect that it may have on self-efficacy if this information is not available. Bandura states that the cognitive processing of self-efficacy information involves two factors: the type of information that individuals attend to and endorse in making self-efficacy judgements; and the rules and heuristics that they use in integrating all of the self-efficacy information that

is available. Furthermore, "people are much more likely to act on their self-perceptions of efficacy inferred from many sources of information rather than relying primarily on visceral cues" (Bandura, 1986, p. 444). If this other information had not been available, anxious arousal may have had an effect on self-efficacy.

In summary, the current study was unable to document an effect of anxious arousal on self-efficacy expectations. Several possible reasons for this failure were discussed. In light of the difficulty in establishing a relationship between anxious arousal and self-efficacy, it is concluded that the relationship may not be a robust one and/or that it is tempered by limiting or qualifying conditions. Several boundary conditions were hypothesized. Further clarifications of the theory, as well as accompanying research, will be necessary in order to further delimit these boundaries.

THEORETICAL IMPLICATIONS

A number of theoretical implications derive from the findings of the current study. The first issue to consider concerns the ability of the two theories of habituation, dual process theory and cortical theory, to explain these results. A second issue concerns the ability of emotional processing theories to explain these results. Finally, the implications of the findings with respect to self-efficacy will be considered.

Neither dual process theory nor cortical theory is a clear winner in this comparison. With respect to dual process theory, the following conclusions can be made. When considering short term within-session effects of anxious arousal, neither increases nor decreases in anxious arousal had an effect on either measure of fear. The role of "sensitization" on response magnitude is a hallmark of this theory, and the inability to document an effect within-session is problematic. Given that sensitization effects have been documented in other studies, the current findings do not invalidate the theory. Rather, they suggest a lack of generalizability of the theory.

The second finding concerns the effect of anxious arousal on fear reduction. Experiencing anxious arousal substantially impedes reduction of subjective fear. It should be noted that this prediction was made on the basis of the "revised version" of dual process theory that was developed earlier and that the earlier versions of dual process theory do not consider the impact of anxious arousal on the process of fear reduction. This finding offers further support for this revision of dual process theory and is congruent with prior studies that have noted the inhibitive effects of anxious arousal on the habituation process.

Unexpectedly, anxious arousal had contrary effects on heart rate response (i.e., subjects who were anxiously aroused showed less heart rate response) following fear

reduction. This effect was not predicted by dual process theory. This effect, as well as the accompanying discordance between the two measures of fear, is better understood within the context of emotional processing. This was discussed in a previous section and will be considered again later.

An examination of the effects of anxious arousal at follow-up found that it did not have an effect on fear as assessed by either measure. A subsequent analysis, however, clarified these results. Anxious arousal at follow-up does not result in increased return of fear, as would be predicted by dual process theory. Rather, exposure to the stimulus at follow-up while in a calm state given fear reduction while anxiously aroused substantially impedes the return of fear. This finding is congruent with dual process theory, however, it would also be predicted on the basis of dual process theory that subjects who experienced fear reduction in a calm state and follow-up assessment in an anxious state would experience the most return of fear. This was not found, however. The finding that fear reduction that occurs while in a state of anxious arousal followed by long-term assessment that occurs while in a calm state impedes the return of fear is similar to the phenomenon that was termed 'greater than 100 percent habituation' by Thompson et al., (1973). They predicted, and had supporting evidence, that if an animal is habituated to a high intensity stimulus and later assessed with a low intensity stimulus, greater

habituation would occur than would occur if the animal was habituated to and tested with the same low intensity stimulus.

In the current context, although the specific feared stimulus (i.e., the snake) remained the same on each assessment occasion, it could be argued that anxious arousal during the first session became associated with the fear network defining the feared stimulus. At follow-up, experiencing the snake while in a calm state resulted in a lesser activation of the network that was developed during the first session (and which now consists of elements of anxious arousal as well as the snake). This could explain the further decrement in fear at follow-up in subjects who were anxiously aroused during fear reduction but not at follow-up. The problem with this explanation is that anxious arousal state did not inflate fear of the snake during the first session. This suggests that the anxious arousal state generated by the experimental manipulation was not part of the network defining the feared stimulus. However, it may be that a time interval is necessary in order to allow this consolidation to occur. The issue remains why the reverse does not hold. In other words, if fear reduction is conducted while the individual is in a calm state and follow-up occurs in a state of anxious arousal why isn't there increased return of fear? As well as being affectively congruent with fear, the experience of anxious arousal is much more salient than the experience of a calm

state, and, consequently, more likely associated with the network defining the feared stimulus.[7]

The predictions made on the basis of cortical theory regarding the effects of within-session changes in anxious arousal were not supported by the data. Neither decreases nor increases in anxious arousal state influenced either measure of fear. As predicted by cortical theory, anxious arousal during fear reduction impeded fear reduction with respect to subjective fear. However, the reverse was found with respect to heart rate response (i.e., there was less heart rate response following fear reduction in the subjects who were anxiously aroused during fear reduction). Regarding the effects of anxious arousal on the return of fear, there is no support for this theory. An asymmetrical effect of congruence of arousal states was found (i.e., experiencing anxious arousal during fear reduction and a calm state at follow-up impedes the return of fear). This finding was not predicted by the theory and cannot easily be explained by it.

In summary, both theories of habituation received support from the finding that anxious arousal impedes the process of fear reduction with respect to subjective reports of fear, but the finding that anxious arousal during fear

7 Although the results of the multiple comparison tests were not significant, the subjects who experienced anxious arousal during both occasions did show the greatest return of fear (see Table 15). This would be expected if the network defining the feared stimulus was modified during the first session so as to include the anxious arousal that was present during the session.

reduction was accompanied by decreased heart rate response following fear reduction was not predicted by either theory. There is further partial support for dual process theory regarding the effects of anxious arousal on the return of fear. Both theories have rather limited success in predicting the effects of anxious arousal per se on fear.

The results of the current study offer support for several predictions made on the basis of emotional processing theories of fear. Rachman (1980, 1990) predicted that relaxation and calm rehearsals facilitate emotional processing and fear reduction relative to high arousal which impedes them. The current study offers clear support for this prediction. He also predicted that autonomic reactivity enhances emotional processing and that unresponsive autonomic reactions impede emotional processing. Although this study does not offer direct support for this prediction, the finding that subjects who were anxiously aroused experienced greater subjective fear following fear reduction but less heart rate response is consistent with this. If heart rate response is thought of as an index of emotional processing as compared to a measure of fear, it would be predicted that individuals who show greater reduction of subjective fear, but who still report a minimum level of subjective fear, would continue to show moderate heart rate responses to the feared stimuli. In contrast, individuals who experience minimal heart rate response to moderately fearful stimuli will show lesser habituation of

subjective fear. However, as subjective fear levels approach zero, individuals who previously exhibited heart rate responses will cease to do so. In other words, a lack of autonomic reactivity can indicate either that the feared stimulus is not being emotionally processed or that it has been successfully emotionally processed.

Rachman and Whittal (1989a) predicted that anxious arousal at follow-up increases the return of fear. The findings of the current study suggest a more complex relationship between anxious arousal state and fear. Assessment in a calm state which follows fear reduction in an anxiously aroused state results in less return of fear than occurs otherwise.

Lang (e.g., 1985) and Barlow (1988) argue that arousal, even if unrelated to the feared stimulus, can serve to intensify the fear response. In a similar fashion, Rachman (1990) predicted that through a process of emotional spill-over, in which one emotional state influences another, increases in arousal, especially anxious arousal, may result in a kindling of the fear network resulting in an inflation of fear. The findings of the current study did not find evidence of this process within-session. Over the long term, however, the results are congruent with the idea that if fear reduction occurs while in an anxious state this anxious arousal becomes associated with the network defining the feared stimulus and that later assessment in a nonanxious state will result in an inhibited activation of the network,

resulting in a lesser fear response than would otherwise occur.

Rachman (1990) differs from Lang (e.g., 1985) and Barlow (1988) in that he states that the type of arousal influences the extent to which emotional spill-over occurs. This differentiation of types of arousal seems to be a useful one. As argued by Neiss (1988, 1990), the concept of arousal is not meaningful without reference to its psychological context. It seems that anxious arousal has co-effects that do not result from forms of arousal (e.g., as induced by physical exertion) that are not negatively valenced with respect to emotion. Exercise, for example, would activate response propositions that are shared with the fear network (e.g., "heart pounds"); however, it would not likely activate meaning propositions that elaborate the relationship between the stimulus and the response elements of the network (e.g., "This is a dangerous and threatening situation.") Anxious arousal, in contrast, would be more likely to share common meaning propositions that are associated with the feared stimulus. Thus, it may be that anxious arousal may more effectively result in emotional spill-over to the network defining the feared stimulus than will other forms of arousal. Although this specific proposition remains to be evaluated, the finding that the "worry" component of anxiety has a more detrimental impact on task performance in those with performance anxiety than the physiological component of anxiety (Sarason, 1984, 1985)

suggests that meaning information may be more likely to ignite fear than will physiological arousal.

The findings of the current study do not accord any significant effect of anxious arousal on self-efficacy. In the previous section, possible reasons for this lack of relationship were discussed. It does not seem that the failure to find a significant effect was due to insensitivity of the self-efficacy measure. Although the anxious arousal manipulation was robust, it is possible that a greater increase in anxious arousal, above some minimum criterion level, was necessary in order to find an effect. Alternatively, it may be that the nature of the anxious arousal that was generated, specifically the discrete nature of its source, prevented anxious arousal effects that would have otherwise occurred. These two possibilities can not be evaluated in the current study and are not addressed by self-efficacy theory in its current form. A final issue that was discussed concerns the impact of anxious arousal information relative to other sources of information such as performance accomplishments or vicarious experience. It may be that these sources of information overrode any effects of anxious arousal.

In summary, the results of the current study suggest that the role of anxious arousal on self-efficacy needs to be reconsidered. Anxious arousal may not have a robust effect on self-efficacy, and the effect, if any, is tempered by several qualifying conditions.

CLINICAL IMPLICATIONS

The results of the current study suggest several important clinical implications. Before commenting further, however, it is important to realize that the present study was conducted with the main purpose of further understanding processes underlying fear, fear reduction, and the return of fear. Although the findings suggest several important ramifications regarding processes underlying fear, further research will be necessary in order to clarify these findings and to extrapolate them to other populations, settings, treatments, and fears. Given these caveats, several comments can be made. Firstly, the results of the present study suggest that the process of fear reduction is facilitated by exposure to the feared stimulus while the individual is in a calm state. Anxious arousal impedes the habituation of fear.

There has been discussion in the past regarding the importance of training in relaxation as a component of fear reduction treatment. Inconsistent effects of relaxation have been noted (Borkovec & O'Brian, 1976; Borkovec & Krogh-Sides, 1979; Levin & Gross, 1985; Mathews, 1971). It has been suggested that although training in progressive muscle relaxation was not a necessary component of fear reduction procedures, it may facilitate the process of fear reduction in highly fearful individuals (McGLynn, Mealiea, & Landau, 1981). The findings of the current study are congruent with this conclusion. However, it is not simply that relaxation

is important only during exposure. Rather, the second major finding, that calm exposures at follow-up given fear reduction in an anxiously aroused state results in further fear reduction, as opposed to the return of fear which is more typically found, suggests a potentially more important role for relaxation. It stresses the usefulness and importance of assisting clients in developing strategies that assist them in gaining a degree of personal control over their anxious arousal state. These strategies include training in relaxation, but also likely extend to other procedures, such as interpersonal problem solving training, that are useful in managing the sources of anxious arousal.

These results suggest that through a process of emotional spill-over (Rachman, 1990) fear levels may vacillate following treatment, given changes in the individual's state of anxious arousal. Arming individuals with this knowledge may prove to be a powerful strategy to assist in relapse prevention (Cameron, 1978; Marlatt & Gordon, 1980). The purposes of advising individuals of the relationship between anxious arousal and fear response are twofold. Firstly, they can respond to increases in anxious arousal by implementing procedures that effectively control anxious arousal. Secondly, if the individual is forewarned that increases in fear following increases in anxious arousal are to be expected and that the fear should decrease as the anxious arousal dissipates he or she will be less

likely to catastrophize the significance of the increased fear.

The results suggest that exposure is best conducted under calm conditions but that long term maintenance of fear is best accomplished when exposure is conducted in an anxiously aroused state and follow-up occurs in a calm state. These two findings, although seemingly incongruent, can likely be combined.

It is suggested that exposure should optimally occur under calm conditions so as to facilitate the habituation of fear. Once the fear has reduced significantly, it may be beneficial to expose the individual to the feared stimulus under conditions of increasing anxious arousal. This procedure will allow the individual, in a controlled manner, to confront the feared stimulus while in an anxiously aroused state, and facilitate the innoculation of the individual against increases in fear given the experience of anxious arousal in the future.

Although the results do not address the issue of whether self-efficacy is the primary mediator of behavior, they indicate that the relationship between anxious arousal and self-efficacy may not be robust. In any event, they do not detract from the fact that there is a relationship between self-efficacy and fear behavior. Clinically, self-efficacy ratings continue to be a useful source of information for the clinician regarding the client's perception of his behavioral capabilities.

CONCLUSIONS

The results of the current study will be summarized and several avenues of research that merit further exploration will be suggested.

The first set of conclusions concerns the effects of anxious arousal on fear, fear reduction, and the return of fear, and the ability of the two theories of habituation (dual process theory: e.g., Groves & Thompson, 1970; cortical theory: e.g., Whitlow & Wagner, 1984) to explain these results. Firstly, within-session changes in anxious arousal level did not influence either measure of fear (i.e., self-report or heart rate response). This inability of anxious arousal to affect fear levels is especially problematic for dual process theory as the role of 'sensitization' (i.e., anxious arousal) in determining response is a defining feature of the theory.

Experiencing anxious arousal during fear reduction results in less reduction of subjective fear (i.e., greater fear following fear reduction) than occurs given exposure under relatively calm conditions. Although this finding was predicted on the basis of both the revised version of dual process theory that was developed earlier and by cortical theory, the finding regarding heart rate response is problematic for the two theories. Specifically, heart rate response upon exposure to the feared stimulus was less, not greater as was predicted, given conditions of anxious arousal compared with relatively calm conditions. This

effect, and the discordance between the two measures of fear, is not explainable by either of these two theories.

Return of subjective fear was experienced by all of the subjects except for those who experienced fear reduction while in an anxious state and assessment in a calm state. These subjects experienced a substantial decrement in self-reported fear at follow-up. This pattern of results can be only partially explained on the basis of dual process theory, and seems inconsistent with predictions made on the basis of cortical theory.

The results were considered in reference to the emotional processing model of fear (e.g., Rachman, 1980). The current findings are congruent with reasoning derived from this model, give empirical support to several of the predictions derived from it, and suggest several refinements to the model. Specifically, the disruptive effect of anxious arousal during fear reduction is predicted by this model. Although the particular pattern of results regarding the return of fear were not predicted, they are easily explained based on this theory. The discordance between heart rate response and self-reported fear, which is problematic for the two habituation models, is congruent with the emotional processing model of fear.

Neither dual process theory nor cortical theory were able to account for these results to a satisfactory extent. Although the theories are not invalidated by the results of the current study, they have a limited ability to predict

and explain fear responses in human subjects. Given how far removed the stimuli and responses of the present study are from those on which the two theories of habituation were derived, it is not surprising that they had difficulty in accounting for these findings. As was suggested earlier, the complex nature of fear likely precludes a single explanatory mechanism of fear reduction. Although fear reduction may resemble habituation processes that have been observed in other contexts, the processes underlying the phenomena may differ. Consequently, the theoretical explanations will necessarily differ as well.

Emotional processing models of fear seem very promising in their ability to predict and explain fear behavior. Although less parsimonious than single explanatory models of fear, this class of models are empirically based and theoretical predictions based on them can be empirically evaluated.

The predicted relationship between anxious arousal level and self-efficacy level (e.g., Bandura, 1986) was not found. Although these results do not preclude such a relationship, further theoretical refinement regarding the effect of anxious arousal on self-efficacy are necessary. The impact of anxious arousal on self-efficacy may not be a robust one.

This study is the first empirical documentation of the disruptive effects of anxious arousal on the reduction of subjective fear. This new finding has theoretical and

clinical implications. Further examination of this finding will likely prove fruitful. The finding that assessment in a calm state following fear reduction while in an anxiously aroused state blocks the return of fear is also novel. Return of fear is the typical finding in research of this type. Further research should attempt to identify other methods to prevent the return of fear.

In the current study, the amount of return of fear was assessed through an analysis of residual gain scores. This analysis allowed an evaluation of the magnitude of change across the follow-up interval while avoiding the difficulties inherent with the use of raw change scores. It is expected that analyses of this type will be used in subsequent evaluations of the return of fear. It is proposed that similar analyses be undertaken in evaluations of relapse in areas other than fear; typically, the assessment of clinical relapse is studied and reported in categorical terms. The extension of the residual gain analysis to the assessment of clinical relapse will introduce welcome precision.

There are several limitations to this study. The first limitation concerns the anxious arousal manipulation. Although the manipulation was shown to be effective in increasing subjective and physiological indices of anxious arousal, other forms of experimentally induced anxious arousal such as threat of negative evaluation or watching stressful movies may not have the same effects. Further

research is necessary in order to determine the generality of these findings. A related issue concerns the relationship between the anxious arousal produced in the current study and the experience of chronic levels of anxious arousal experienced by clinically anxious individuals. As noted by Barlow (1988), although the two share common features, experimentally produced forms of anxious arousal should not be confused with clinical presentations of anxious arousal. It is tempting to extrapolate these findings to the clinical setting, however, this must await further research that examines this question.

A second limitation concerns the feared stimulus that was used in the current study. Snake fear is an appropriate model in examining fear. However, it would be useful to replicate these results using other feared stimuli.

The study would have been improved by the addition of a second follow-up session in which all of the subjects were exposed to the feared stimulus while experiencing equivalent levels of non-anxious arousal. This would allow statements to be made regarding the durability of the effects that were noted during the first follow-up session.

Several directions for future research are indicated by the current study. Firstly, it is necessary to replicate these results using alternate forms of anxious arousal. It may be that a different pattern of results would emerge given other sources of anxious arousal such as threat of negative evaluation. Although more robust effects may be

found with higher levels of anxious arousal, researchers are limited by ethical constraints regarding the magnitude of anxious arousal that can be induced.

One very profitable avenue may be to compare the relative impact of the two major components of anxious arousal (worry vs. physiological arousal) on fear. Research in the area of the return of fear has focussed on the important role of autonomic arousal as indexed by heart rate. However, other theory and research (e.g., Barlow, 1988; Sarason, 1984, 1985) suggests that the worry component of anxious arousal may have a detrimental impact on fear. As well, the closely related finding that experiencing worry results in an inflation of subjective fear but reduced heart rate response, without causing a corresponding increase in baseline heart rate (Borkovec & Hu, 1990), also indicates that worry may play an important role in fear processes.

Research examining the effect of anxious arousal on predictions of fear would likely increase our understanding of fear. Predictions of fear and the relationship between prediction and the actual experience of fear have an impact on later predictions of fearful behavior (Rachman & Lopatka, 1986a, 1986b).

The current study has several important clinical implications that were discussed in the previous section. It would be very useful to examine the extent to which these findings generalize to clinical settings, populations, and problems. If these results do generalize, several important

implications regarding the management of anxiety-based problems follow.

A great deal of research has been generated based on self-efficacy theory, however, there has been a lack of attention to the construct of anxious arousal and its relationship to self-efficacy. The results of research that has been conducted are inconsistent. Several boundary conditions regarding anxious arousal were suggested. It would be useful to evaluate these hypotheses in subsequent research.

In summary, several important questions regarding the effects of anxious arousal on fear, fear reduction, the return of fear, and self-efficacy were addressed. Several additional questions were posed and issues were raised. Further examination of these questions and issues will likely prove fruitful in increasing knowledge of fear and fear related processes.

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APPENDICES

APPENDIX A

FEAR SURVEY SCHEDULE

FEAR SURVEY SCHEDULE

Please check (✓) the appropriate level of fear for each item.

	I am not at all fearful of:	I am slightly fearful of:	I am moderately fearful of:	I am extremely fearful of:	I am terrified of:
Snakes					
Cats					
Birds					
Spiders					
Worms					
Dogs					
Insects					
Horses					
Heights					
Enclosed Spaces					

Name:

Phone Number and Best Time to Call:

If you are interested in participating in a Fear Study supervised by Dr. S. Rachman please fill in your name and phone number. You will receive course credit for participating (instructor permitting).

APPENDIX B

MOOD SCALE

Mood Scale

Instructions: Please place a slash (/) anywhere along the continuum you feel is appropriate. Please indicate how you are feeling right now.

How anxious do you feel?

0 ——————————————————— 100

I am not at all anxious — I am extremely anxious

How sad do you feel?

0 ——————————————————— 100

I am not at all sad — I am extremely sad

How agitated do you feel?

0 ——————————————————— 100

I am not at all agitated — I am extremely agitated

How happy do you feel?

0 ——————————————————— 100

I am not at all happy — I am extremely happy

How relaxed do you feel?

0 ——————————————————— 100

I am not at all relaxed — I am extremely relaxed

How apprehensive do you feel?

0 ——————————————————— 100

I am not at all apprehensive — I am extremely apprehensive

APPENDIX C

SELF EFFICACY SCALE

S-EA Questionnaire

Instructions: Please place a slash (/) anywhere along the continuum you feel is appropriate. Please indicate how you are feeling right now.

How confident are you that you will be able to _____ _____?

0 100

Not at all confident — Totally confident

APPENDIX D

CONSENT FORM

Consent Form
Fear Study

We are conducting a research project on factors affecting fear levels and would welcome your participation. The experiment will consist of two sessions, four weeks apart. The first session will be about one hour in length and the second will be about 20 minutes in length.

In the first session you will be asked to spend some time focusing on bodily sensations of muscle tension or relaxation. In addition, some people who participate in the experiment may experience a series of completely harmless, but painful, electric shocks. Shocks will be given at current intensities that cannot be detected and will increase each time in small increments. At any time during the sequence of shocks, simply saying "Stop" will end the series of shocks and no further increases will be delivered. Following the series of increasing shocks, a further series of shocks may be received. These will be within the tolerable range and will be delivered in a random order of intensities and at random intervals. All subjects will also be asked to slowly approach a live, harmless garter snake. You will be asked at various points throughout each session to report your level of fear, mood, and confidence. Throughout all of this, your heart rate will be recorded using a small monitor that clips onto your ear. The second session is similar in structure to the first session, however, it is only about 20 minutes in length.

If for any reason you wish to withdraw from the experiment you are free to do so at any time without jeopardizing your class standing. However, we hope that you will be willing to participate. After completion of the study all participants will be given the opportunity to learn the outcome of the research as well as receive course credit for participation.

In addition, please note that all information collected during the course of this study is kept strictly confidential and access to it will be restricted to Dr. S. J. Rachman or Gene Flessati, M. A. For further information, you may contact Gene Flessati, Department of Psychology, UBC (telephone:).

If you have any questions about the procedures outlined above, please feel free to ask.

I have read the attached information, consent to participate in this research, and have received a copy of this consent form.

Signature: _____

Name: _____

Student #: _____

Date: _____

APPENDIX E

RESIDUAL GAIN SCORE ANALYSIS

A residual gain score analysis was conducted to assess the amount of change in self-reported fear between time 4 and time 6. Analysis of raw change scores formed by subtracting the SUDS score at time 4 from the SUDS score at time 6 is inappropriate as these scores are related to any random measurement error. Unlike raw change scores, residualized gain scores have no correlation between pre-test (i.e., SUDS score at time 4) and post-test (i.e., SUDS score at time 6) scores. Residualizing the change score removes the portion of the change across the follow-up interval that could have been predicted based on linear regression of the SUDS scores at time 4. Refer to Cronbach and Furby (1970) for a discussion of the use of change scores. In this analysis, SUDS score at time 6 was regressed on SUDS score at time 4. The score that would be predicted at time 6 (from the SUDS score at time 4) was then computed. The residual SUDS score at time 6 was computed as the difference between the SUDS score at time 6 and the predicted SUDS score at time 6. Thus, a positive residual score indicates that subjects reported greater fear at time 6 than would be predicted from the subjects' reported fear at time 4. Conversely, a negative residual score indicates that less fear was reported at time 6 than would be predicted from the subjects' reported fear at time 4. This analysis is equivalent to analysis of covariance using the

SUDS scores at time 6 with SUDS score at time 4 as the covariate. The mean residual gain score for each group is equivalent to the adjusted mean obtained from the ANCOVA for that group minus the mean SUDS score at time 6 for the entire sample. It was decided to present the data in this fashion because residual gain scores are more easily interpreted than mean adjusted heart rate response scores.