

Fear and Phobia: A Critical Review and the Rational-Choice Theory of Neurosis

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Abstract

This article reviews the empirical status of theories of fear and phobia. Psychoanalysis received little support, as findings tend to refute its basic assumption that phobia results from repressed material. Although conditioning has its weaknesses, it appears to be the strongest explanation of simple phobia. Findings raise question as to whether interoceptive conditioning can account for the development of panic disorder and agoraphobia, as these disorders develop in the absence of environmental conditioning events. A significant body of research supports Clark's claim that catastrophic misinterpretation of bodily sensations are involved in both the development and treatment of panic disorder and agoraphobia. However, the causal relationship between the two remains unclear. Likewise, while biological factors certainly increase the vulnerability to developing fear and phobia, findings have not yet confirmed that these behaviors are controlled by biological mechanisms. A new theory, the Rational-Choice Theory of Neurosis (Rofé, 2010; Y. Rofé & Rofé, 2013), which preserves the psychoanalytic claim that bizarre phobias need to be explained within a theory that accounts for neuroses by one set of theoretical concepts, was used to resolve the theoretical confusion in this field.

Keywords: agoraphobia, biological models, cognitive models, conditioning, panic disorder, psychoanalysis, Rational-Choice Theory of Neurosis, simple phobia

1. Introduction

Two major theories that had a significant impact on the scientific investigation of fear and phobia, psychoanalysis and behaviorism, were initiated by Freud (1909) and Watson, and Rayner (1920). The case studies illustrating each of these rival theoretical approaches, Little Hans and Little Albert, respectively, have become symbols of each theory in the field of fear and phobia. Although both theories continue to dominate the field, a major theoretical change was made in the Pavlovian concept of conditioning to address various fundamental difficulties encountered by behaviorism. The revised form of the theory includes Seligman's (1971) preparedness theory, Jacobs and Nadel's (1985) stress induced recovery of fear and phobia, and neo-conditioning theories (Barlow, 2004; Bouton, Mineka, & Barlow, 2001; Forsyth & Eifert, 1996; Wolpe & Rowan, 1988). Meanwhile, cognitive models of conditioning (Bandura, 1971; Brewer, 1974; Davey, 1989a; Mackintosh, 1983), Clark's (1986) misinterpretation theory, as well as biological approaches such as the nonassociative account (Menziez & Clarke, 1995a; Poulton & Menziez, 2002a), and Klein's (1993) suffocation alarm theory, were proposed as alternative theoretical explanations for fear and phobia.

Thus, while in the beginning of the last century the dispute regarding the cause of fear and phobia was limited to psychoanalysis and behaviorism, today this field of research is characterized by considerable theoretical confusion. In fact, investigators are still puzzled over the exact processes by which these behaviors develop. This article presents a comprehensive review of the main existing theories of fear and phobia, and examines the extent to which they advance our understanding of the underlying causes of these debilitating behaviors. This review will explore whether these theories are capable of integrating the pertinent, yet seemingly incompatible, findings from the rival schools of thought into their own theoretical framework. In the discussion section, it will be indicated that despite serious weaknesses, conditioning theory appears to be the strongest explanation of simple phobia. The Rational-Choice Theory of Neurosis (Rofé, 2010; Y. Rofé & Rofé, 2013), was suggested as the best explanation of bizarre phobia (e.g., chocolate phobia or panic disorder).

1.1 Psychoanalysis

In psychoanalysis, phobias are conceptualized either as a defense against a repressed, anxiety-provoking impulse, as in the case of Little Hans whose horse phobia was attributed to the Oedipal conflict (Freud, 1909), or as the expression of a repressed, anxiety-provoking event, such as childhood trauma with water (Bagby, 1922) or trains (Leonard, 1927). Essentially, phobias do not differ from other types of symptom formations, as “the only thing all phobias have in common is the defensive use of avoidance. They share nothing else, either dynamically or genetically, which distinguishes them from any other class of symptoms” (Compton, 1992, p. 425). For example, both agoraphobia and panic disorder are attributed to early-repressed anxieties relating to separation experiences (De Poderoso, Julian, & Linetzky, 2005; DelMonte, 1996; Frances & Dunn, 1975; Gassner, 2004; Rhead, 1969; Vandereycken, 1983). Even specific phobias, which usually pose merely a minimal disruption to the individual’s daily functioning, such as spider, snake, and dog phobias are accounted for in psychoanalytic terms (Abraham, 1927; Compton, 1992; Moss, 1960; Newman & Stoller, 1969; Tyson, 1978). For example, Spurling (1971) noted, “most investigators seem to agree that the spider is a representative of the dangerous (orally devouring and anally castrating) mother and that the main problem of these patients seems to center around their sexual identification and bisexuality” (p. 493). Similarly, Lewis (1981) indicated with regard to the relatively high prevalence of phobias of snakes, spiders and heights that these phobias might be metaphors of “forbidden emotional states” (p. 84).

However, there is little evidence to validate the psychoanalytic theory of phobias. First, psychoanalysis relies on case studies of poor scientific quality to support its theoretical position. For example, in criticizing Freud’s (1909) case of Little Hans, which is still used today to illustrate the psychoanalytic theories of phobias (Blum, 2007; Wakefield, 2007), Wolpe and Rachman (1960) demonstrated that it does not meet even minimal scientific requirements. They noted that Freud saw Little Hans only once, and that Little Hans’s father, who was Freud’s student, and as such was strongly biased, supplied the material upon which Freud built his theory. Similarly, Bagby (1922) reported two case studies of phobias, but it is unclear whether he actually saw the patients (Rofé, 1989, pp. 140-141). Case studies remain the major tool of investigation when attempting to demonstrate the repressed etiology of phobia (Gassner, 2004; Wolitzky & Eagle, 1999). However, this method is not sufficient to substantiate psychoanalytic hypotheses, because case studies are given to subjective interpretation (McNally, 2003; McNally, Clancy, & Barrett, 2004; Piper, 1999). Furthermore, Grünbaum (1986) showed, in his thorough critique of Freud’s work, that “clinical data tend in any case to be artifacts of the analyst’s self-fulfilling expectations, thus losing much of their evidential value” (p. 217).

Second, when more controlled research efforts were employed to examine whether phobia emerges from childhood trauma, as assumed by psychoanalysis, findings were inconsistent. For example, while some investigators found that separation anxiety is significantly related to the development of panic disorder and agoraphobia (Bandelow et al., 2002; Laraia, Stuart, Frye, Lydiard, & Ballenger, 1994; Silove et al., 1995), others could not find confirmation for this hypothesis (Aschenbrand et al., 2003; Doerfler, Toscano, & Connor, 2008; Peter, Brückner, Hand, & Rufer, 2005; Thyer, Himle, & Fischer, 1988; Thyer, Nesse, Curtis, & Cameron, 1986). Moreover, the memory of separation anxiety in these studies was examined by self-report scales, which indicate that subjects were, in fact, aware of these events (Bandelow et al., 2001; Zitrin & Ross, 1988). However, according to psychoanalytic theory, it is the subject’s unawareness of the specific childhood trauma, which is said to cause the development of fear and phobia (De Poderoso et al., 2005). Thus, even if separation anxiety was truly linked to agoraphobia and panic disorder, the psychoanalytic theory would not be confirmed.

Third, there has been heated controversy in recent years regarding the very existence of repression, the “cornerstone on which the whole structure of psychoanalysis rests” (Freud, 1914, p. 16). While some investigators reported that people forget their traumatic experiences, as claimed by the psychoanalytical theory (Anderson & Green, 2001; Brown, Schefflin, & Whitfield, 1999; Cheit, 1998; Chu, Frey, Ganzel, & Matthews, 1999; Erdelyi, 2006), most studies obtained contradictory evidence, i.e. trauma enhances memory (e.g., see review by McNally, 2003; McNally et al., 2004; Piper, Lillevik, & Kritzer, 2008; Piper, Pope, & Borowiecki, 2000). They also demonstrated that the studies cited in support of psychoanalytic theory suffer from serious methodological flaws. In an attempt to resolve this controversy, Rofé (2008) claimed that psychoanalysis portrays repression as a multidimensional concept, which, aside from claiming that people forget traumas, assumes (1) the existence of the unconscious, which stores the repressed memories and controls their manifestation in the form of neurotic symptoms; (2) that repression has a pathogenic impact on daily activities; (3) that neurotic symptoms are the direct result of the repressed memories; and (4) that recovery is obtained through the lifting of repression. In reviewing relevant literature, none of these components received empirical

validation, which led Rof  to conclude that there is little scientific justification for maintaining the psychoanalytic concept of repression.

These findings question not only the psychoanalytic hypothesis linking fear and phobia to repressed material, but also the validity of the entire psychoanalytic theory of psychopathology. However, the abandonment of Freud's theory may leave a theoretical vacuum, as some unusual phobias may become incomprehensible in its absence. For example, it is difficult to see how rival theories can account for the spontaneous onset of unusual phobias of chocolate and vegetables (Rachman & Seligman, 1976), AIDS (Glass, 1993), rat germ contamination (Brandt & Mackenzie, 1987), insect phobia (Jacobs & Nadel, 1985) and hydrophobia (Bagby, 1922). In all these cases, patients are unaware of the underlying causes of their radical behavioral change, and there is no observable variable that could account for this behavioral shift. This claim can also be applied to frightening, obsessive thoughts, such as when someone develops extremely hostile thoughts towards loved ones with no ability to account for the underlying causes (Fenichel, 1946; see also case studies by Horowitz, 2004, pp. 169-186; McAndrew, 1989). Thus, we need either to revise the psychoanalytic theory so that it may account for these behaviors, modify other theories so that they may address this issue, or consider an alternative approach that would be able to integrate these exceptional cases.

In conclusion, research which directly examined the psychoanalytic hypothesis that fear and phobia are the consequence of repressed childhood trauma has provided little support for this claim. Moreover, the increasing number of studies questioning the very existence of repression, which constitutes the foundation of the psychoanalytic theory of pathological behavior, including fear and phobia, further challenges this theory. Nevertheless, the abandonment of Freud's theory leaves a theoretical vacuum, which is seemingly accountable by psychoanalysis alone. Therefore, as long as an alternative explanation is not given for those cases of unusual phobia, the understanding of fear and phobia will remain incomplete.

1.2 Behaviorism

1.2.1 Pavlovian Paradigm

According to the Pavlovian conditioning theory, any conditioned stimulus (CS) should lead to the development of fear when it is accompanied by an unconditioned stimulus (UCS) that naturally provokes anxiety. Watson and Rayner (1920) maintained that the conditioning model accounts for the development of fears and phobias, and later behaviorists generalized this concept to explain the development of neuroses (Eysenck & Rachman, 1965; Wolpe, 1952, 1958; Wolpe & Plaud, 1997).

The Pavlovian paradigm of fear was largely based on animal studies, which support this theory consistently (Hulse, Egeth, & Deese, 1980; Rachman, 1977, 1990b). This theory also received significant support among humans, as fear often develops in accordance with conditioning principles (Di Nardo et al., 1988; Field, 2006; Kuch, Swinson, & Kirby, 1985; Lutch, 1971; Yule, Udwin, & Murdoch, 1990). However, this theory has encountered several fundamental difficulties. One problem is that people often fail to develop conditioned fear when exposed to anxiety-provoking situations. For example, aversive experiences with dogs (Di Nardo, Guzy, & Bak, 1988) or intense combat trauma (Rachman, 1990a; Saigh, 1984a, 1984b, 1985) do not usually cause conditioned fear. Similarly, in a laboratory analogue of electrical aversion therapy, Hallam and Rachman (1976) found that no conditioned fear developed in response to the aversive event. Likewise, Marks and Gelder (1967) reported that most of their patients were indifferent to conditioned stimuli employed in electrical aversion therapy (for similar results, see also Bancroft, 1969; Hallam, Rachman, & Falkowski, 1972).

Another fundamental difficulty concerns cases in which phobias develop in the absence of observable learning experiences. For example, very young children display spontaneous fear and phobia, such as snake, spider, and height phobias, upon first contact with these stimuli (see review by Poulton & Menzies, 2002a). Similarly, agoraphobia and panic disorder (Jacobs & Nadel, 1985; Mathews, Gelder, & Johnston, 1981), and other types of severely disruptive phobias (e.g., of trains, insects and chocolate; Jacobs & Nadel, 1985; Leonard, 1927; Rachman & Seligman, 1976) usually develop in the *absence* of observed environmentally noxious events which could justify these abrupt behavioral changes. Another issue lies in the difficulties of the conditioning model to account for the unequal distribution of different types of fear and phobia (Rachman, 1977, 1990a, 1990b). For example, snake phobia is far more prevalent than dental or injection phobia, despite the higher frequency of negative experiences with the latter (Agras, Sylvester, & Oliveau, 1969; Rachman, 1990a, 1990b).

It is also unclear why children display greater specific phobias and fear (e.g., fear of animals, darkness, and water; see Flatt & King, 2008; King, Eleonora, & Ollendick, 1998; Marks, 1987a; McNally & Steketee, 1985), but show the opposite pattern with regard to agoraphobia and panic disorder (e.g., see DSM-IV-TR, American Psychiatric Association, 2000; Goodwin et al., 2005; Rosenbaum, Pollock, Otto, & Pollack, 1995; Wittchen &

Essau, 1993). Given the fact that previous non-aversive experiences inhibit conditioned fear (Davey, 1989a, 1989b; Field, 2006; Mineka & Cook, 1986; Mineka & Zinbarg, 2006; Rachman, 1990a, pp. 181-182), one would expect, from the perspective of conditioning theory, that children, who have had less extensive fearless experiences with harmless stimuli than adults (e.g., public places), would be more vulnerable to develop clinical phobias. Another puzzling issue concerns the high prevalence of fear and phobia among mentally retarded children (Gullone, 1996; Gullone, Cummins, & King, 1996; Muris, Merckelbach, De Jongh, & Ollendick, 2002; Ramirez & Kratochwill, 1997) and less educated people (Arnarson, Gudmundsdottir, & Boyle, 1998).

It may also be important to note that some investigators found that the majority of phobic subjects attributed their fears to conditioning events when using self-report questionnaires (Merckelbach, Arntz, & De Jongh, 1991; Merckelbach, De Ruiter, Van den Hout, & Hoekstra, 1989; Öst & Hugdahl, 1981, 1983, 1985). However, when a different type of self-report scale was employed, this effect was obtained only for a minority of subjects (see review by Poulton & Menzies, 2002a). Moreover, test-retest after a period of one year showed the retrospective report of fear onset to be quite unstable, and hence, unreliable (Taylor, Deane, & Podd, 1999).

In summation, studies consistently demonstrate that fear among animals develops in accordance with conditioning theory, and some studies show that some of human fears and phobias develop in accordance with this theory. However, this theory suffers from fundamental problems, which indicate that, in its present form, it is unable to account for the human development of fear and phobia. Thus, while the conditioning paradigm is obviously important, this theory must be substantially revised in order to address these issues.

1.2.2 Preparedness Theory

Seligman (1971) proposed a revised form of the classical conditioning theory, termed preparedness theory, which suggests that species are biologically programmed to be more easily conditioned to stimuli that endangered their existence throughout evolution, such as spiders and snakes. Consistent with preparedness theory, superior conditioning and resistance to extinction were demonstrated for fear-relevant stimuli (e.g., pictures of snakes or spiders), as compared to fear-irrelevant stimuli (e.g., pictures of flowers or geometric shapes; see reviews by McNally, 1987; Mineka & Öhman, 2002a; Öhman & Mineka, 2001). While some studies have failed to replicate these findings, this might have been the result of inappropriate conditioning methodology (see review by Öhman & Mineka, 2001).

Some studies also examined differences in arousal levels elicited by phylogenetic fear-relevant stimuli (e.g., slides of snakes and spiders) and ontogenetic fear-relevant stimuli (e.g., slides of handguns or electric outlets). Here too, there is some inconsistency among investigators. While some studies found no differences between the two types of stimuli (Davey, 1995; Davey & Craigie, 1997; Davey & Dixon, 1996; Honeybourne, Matchett, & Davey, 1993; Lovibond, Siddle, & Bond, 1993), others reported that both arousal and conditioning are more intense to phylogenetic fear-relevant stimuli than to ontogenetic fear-relevant stimuli (Mühlberger, Wiedemann, Herrmann, & Pauli, 2006; Öhman & Mineka, 2001). Findings also show that fear-relevant stimuli capture subjects' attention and are perceived more rapidly than fear-irrelevant stimuli (Öhman, Flykt, & Esteves, 2001; Waters & Lipp, 2008). Additionally, subjects were more distracted by fear-relevant animals (e.g., spider) than non-fear-relevant animals (e.g., horse; Waters & Lipp, 2008).

Preparedness theory has certainly received significant empirical support, and made an important contribution in addressing the shortcomings of the classical conditioning theory to account for the unequal distribution of fear and phobia. However, it is doubtful whether findings support Seligman's evolutionary concept of fear and phobia. From an evolutionary perspective, some stimuli have endangered our species' existence (e.g., various predatory animals and poisonous plants) at least as much as snakes and spiders, but have not resulted in phobias (Davey, 2002; Delprato, 1980). An alternative explanation would suggest that snake and spiders stimulate arousal due to their specific characteristics and features, in terms of shape and movement, rather than by their dangerous evolutionary characteristics. This is consistent with findings indicating, "Ugly, slimy, speedy or sudden-moving animals are experienced as less approachable and more fear-provoking than animals without these qualities" (Bennet-Levy & Marteau, 1984, p. 40). Evidence also suggests that the disgusting nature of some animals may be an important causal factor in fear acquisition, independent of their potential danger (Davey, 1992a, 1993, 1994; Davey, Forster, & Mayhew, 1993; Jain & Davey, 1992; Matchett & Davey, 1991; Ware, Jain, Burgess, & Davey, 1994). For example, spiders can be classified not only as "fear-evoking animals" (e.g., rats and snakes), but also as "disgust-evoking animals", such as caterpillars, maggots, and slugs (Davey, 1992a). A cross-cultural study of fear of animals also showed that the emotion of disgust becomes attached to different animals in different cultures, suggesting that disgust of certain animals could be, at least in part, the consequence of socialization (Davey et al., 1998).

Regardless of the theoretical explanation of preparedness, it seems that this concept can only promote our understanding of simple, but not clinical, phobia. Although some investigators reported that specific fears (e.g., fear of loud noises, fear of bees and wasps) made up the majority of fears among clinical subjects (De Silva, 1988; De Silva, Rachman, & Seligman, 1977; Zafiropoulon & McPherson, 1986), others could not replicate these findings (Merckelbach, Van den Hout, Hoekstra, & Van Oppen, 1988). Further, it is difficult to accept Öhman, Dimberg and Öst's (1985) proposal that agoraphobia can be seen as a prepared phobia of open spaces, which increase the vulnerability to predators. Such a suggestion does not explain why both panic disorder and agoraphobia are more prevalent among adults, whereas they are very rare among children (DSM-IV-TR, American Psychiatric Association, 2000; Goodwin et al., 2005; Rosenbaum et al., 1995; Wittchen & Essau, 1993). In this regard, McNally (1987) noted, "the task confronting fear theorists is not to explain the rapid acquisition of phobias. Rather, the task is to explain the wide variability in the rate of fear acquisition and to account for age-related vulnerability to acquiring certain fears" (p. 296).

In conclusion, studies support the idea that certain phobias particularly snake and spider phobias, are more easily conditioned than others are, which accounts for the unequal distribution of fears and phobias. However, the interpretation of these findings is controversial, since apart from possible evolutionary significance, snakes and spiders differ from irrelevant stimuli both in terms of their perceptual characteristics and in terms of their cultural acceptability. Either one of these components could be responsible for the more rapid conditioning of these stimuli. Moreover, Seligman's theoretical approach has little clinical utility, as it cannot account for the development of clinical fears and phobias, such as agoraphobia and panic disorders.

1.2.3 Interoceptive Conditioning

As stated, panic disorder and agoraphobia usually develop in the absence of an observable noxious event (UCS; Jacobs & Nadel, 1985). In an attempt to address this issue, neo-conditioning investigators claim that the intense bodily sensations (e.g., hyperventilation) experienced during the first panic attack are the UCS of interoceptive conditioning, and thus accounts for the development of these disorders (Barlow, 2004; Bouton et al., 2001; Carter & Barlow, 1995; Forsyth & Eifert, 1996, 1998; Mineka & Oehlberg, 2008; Mineka & Zinbarg, 2006; Öst & Hugdahl, 1981, 1983, 1985; Wolpe & Rowan, 1988). As noted by Wolpe and Rowan (1988), "just as electrically elicited anxiety becomes conditioned to contiguous stimuli to produce experimental neurosis...so may panic anxiety become conditioned to contiguous stimuli to produce panic disorder" (p. 446). The initial attack is attributed to a false alarm resulting from stress in interaction with biological/genetic, personality/temperamental or experiential factors (Barlow, 2004; Bouton et al., 2001; Mineka & Oehlberg, 2008).

In accordance with this new conception, studies have successfully produced conditioning effects in which the UCS was prolonged exposure to CO₂-enriched air, while the CS was either a fear-relevant stimulus (e.g., fear-relevant images; Forsyth, Eifert, & Thompson, 1996; Forsyth & Eifert, 1998; Stegen et al., 1999) or a brief exposure to CO₂-enriched air (Acheson, Forsyth, Prenoveau, & Bouton, 2007). In accordance with the claim that panic disorder patients are more vulnerable to conditioning due to genetic, temperamental, or experiential vulnerability (Bouton et al., 2001), experimental evidence shows that panic patients demonstrate greater resistance to extinction in comparison with a non-clinical sample (Michael et al., 2007).

However, there is an enormous gap between panic disorder in everyday life and panic-like effects in the laboratory. Unlike laboratory studies, where the conditioned response is easily extinguished and has little psychological effect (Harrington, Schmidt, & Telch, 1996; Prenoveau, Forsyth, Kelly, & Barrios, 2006; Perna, Cocchi, Politi, & Bellodi, 1997), panic disorder severely disrupts the individual's daily functioning and is highly resistant to extinction (Eifert, 1992; Jacobs & Nadel, 1985; Seligman, 1988). While one may argue that panic disorder is not easily eliminated because the UCS (prolonged, intense bodily sensations) reinforces the CS (brief bodily sensations) of each new attack, neo-conditioning investigators have yet to prove such a claim.

The interoceptive conditioning theory attributes the first panic attack to stressful events (Barlow, 2004; Bouton et al., 2001). However, since the first panic attack does not eliminate the stressor, it is unclear why stress by itself is not sufficient to account for the subsequent panic attacks. Why is the conditioning concept necessary?

The neo-conditioning theorists also attribute all forms of phobia to conditioning, and view agoraphobia as the by-product of panic disorder (Barlow, 2004; Bouton et al., 2001; Mineka & Zinbarg, 2006). However, Wittchen et al. (2008) found that in a substantial number of cases, agoraphobia is "a clinically significant disorder that exists independently of panic attacks and panic disorders" (p. 153; see also Fava, Rafanelli, Tossani, & Grandi, 2008; Goodwin et al., 2005; Hayward & Wilson, 2007). In fact, agoraphobia may serve as a predictor of the

development panic disorder (Bienvenu et al., 2006). It is difficult to see how these findings can be integrated within the interoceptive conditioning theory.

An additional problem is that, inconsistent with conditioning theory, “hyperventilation does not invariably lead to a panic attack in PD [panic disorder] patients (as a UCS should)...” (Sanderson & Beck, 1989, p. 581). For example, only 23% of panic patients responded with panic attacks following laboratory induction of hyperventilation (Gorman et al., 1988a). Similarly, in examining hyperventilation among panic disorder patients in a natural setting, Hibbert and Pilsbury (1989) noted that their findings do not support the hypothesis that hyperventilation causes panic attacks or contributes to their severity. Hyperventilation may be better understood as a consequence of panic (p. 805). Other researchers reported additional inconsistent findings (Gorman et al., 1994; Nardi et al., 2004; De Ruiter, Garssen, Rijken, & Kraaimat, 1992).

Additionally, assuming that panic disorder is the consequence of conditioning, as claimed by interoceptive theory, it is difficult to see how such a theory can integrate the large body of findings indicating that misinterpretation of bodily sensations constitute a critical factor in the development of panic disorder (Austin & Richards, 2001; Clark, 1986, 1988), especially given the fact that changing patients’ beliefs regarding these sensations through cognitive therapy yields positive results even without exposure (Arntz, 2002; Clark, 1999; Salkovskis, Clark, & Hackmann, 1991).

In conclusion, neo-conditioning theorists significantly revised the Pavlovian paradigm in their attempt to resolve the difficulties they encountered in accounting for the development of panic disorder and agoraphobia. The most significant change is the suggestion that bodily sensations can serve as a UCS. However, while the interoceptive conditioning account resolves the problem of the lack of environmental observable trauma in the etiology of these disorders, it has also encountered fundamental problems, which challenge its validity.

1.2.4 Stress-Induced Recovery of Fear and Phobia

Jacobs and Nadel (1985) suggested a theory, which integrates findings from conditioning, psychoanalytic and biological studies to account for the onset of fear and phobia in adulthood without any discernable conditioning event. Attention is drawn to the facts that these fears develop in the absence of UCS, that subjects are unaware of the conditioning event and that stress precedes the development of these deviant behaviors. Jacobs and Nadel (1985) claim that conditioning must have occurred during infancy, and that unawareness is due to infantile amnesia resulting from the immaturity of the hippocampus. Based on animal research, they also suggest that stress produces hormonal changes, which cause the reinstatement of the conditioned fear established at the infancy stage. Once developed, “conditioned fear might never be eliminated using traditional extinction or counter-conditioning procedures” (p. 525).

Jacobs and Nadel employed this theoretical approach to account for the development of panic disorder and agoraphobia. While their attempt to integrate different models may be the right method to gain a genuine understanding of fear and phobia, it is doubtful whether their specific integrative theory is an appropriate answer for this issue. First, the existence of repression has been seriously challenged, if not altogether refuted (see reviews by Piper et al., 2008; Rofé, 2008). Moreover as stated above, no significant link has been found between childhood trauma and the development of panic disorder or agoraphobia. Hence, Jacobs and Nadel’s claim regarding “the tyranny of childhood” (p. 513) in the etiology of fear and phobia has received no empirical support. Second, the authors present neither research nor clinical evidence from human subjects demonstrating that the critical conditioning event had ever taken place during infancy. Finally, the contention that stress precipitates the “recovery” of this event later in life has never been supported.

2. Conclusion

The classical theory of conditioning has significantly changed since the original Pavlovian conception. Changes suggested by preparedness and interoceptive conditioning, which take into consideration variables such as genetic predisposition, personality factors, and learning history, minimize the difficulties this theory has encountered throughout the years. However, while advocates believe that this theory can account for the etiology of all types of fear and phobia, it seems that at this stage, its explanatory power is proven only with regard to specific phobias. The applicability of this theory to panic disorder and agoraphobia is still doubtful in light of fundamental difficulties, which question its validity.

2.1 Cognitive Theories

2.1.1 Conditioning Model

Although the cognitive account of conditioning agrees with behaviorism that fear and phobia develop out of learning experiences, advocates of this approach claim that conditioning results in the association between

stimulus-stimulus (S-S) rather than stimulus-response (S-R) (Davey, 1989a; Mackintosh, 1983). This hypothesis was supported by experimental studies showing that conditioned fears fail to develop when subjects are unaware that the CS signifies an aversive event, and that extinction is readily obtained when they are assured that the UCS will no longer follow the CS. (Bandura, 1971, 1977; Brewer, 1974; Davey, 1989b; Dawson, Schell, & Banis, 1986).

These findings led Bandura (1977) to propose the self-arousal theory, which states that conditioned fear is aroused through fear-provoking thoughts. He states, "For individuals who are aware that certain events forebode distress, such events activate fear-arousal of thoughts, which in turn produce emotional responses" (p. 69). Thus, conditioning is a matter of belief that past contingencies remain in effect and the more severe the effects are expected to be, the stronger the emotional arousal will be. A similar theoretical position was expressed by Beck (Beck, 1976; Beck, Emery, & Greenberg, 1985) in his perceived danger theory of phobia.

Davey (1989a, 1992b) suggested the concept of "latent inhibition", which refers to previous non-aversive exposures to CS, to account for cases of conditioning failure despite exposure to traumatic incidents. As proof, Davey (1989b) presented evidence that prior painless dental treatment inhibited the acquisition of dental phobia. With regard to the emergence of phobias in the absence of trauma, Davey (1989a) claimed that the CS-UCS association, a process termed "sensory preconditioning", might have been established at an earlier stage, when the UCS was only minimally aversive. At a later stage, an event inflates the negative effect of the UCS, in absence of the CS, resulting in an intensified fear towards the CS. For example, a person may witness a death caused by a heart attack (UCS) while riding a bus or train (CS) without developing observable fear. However, later exposure to the death of a relative brought about by heart attack may inflate the negative impact of the UCS and give rise to an acute anxiety toward public transportation (for additional examples see Davey, De Jong, & Tallis, 1993). In examining this hypothesis (White & Davey, 1989), a visual stimulus (CS) was associated with an innocuous 65dB tone (UCS). Then the level of aversion to the "UCS" was inflated, in the absence of the CS, by increasing the intensity of the tone to 115dB. Results showed that the CS aroused a significant conditioned response (skin conductance response) only after the inflation procedure, but not at the initial stage.

It is difficult, however, to see how Davey's concept can account for the development of agoraphobia and panic disorder. Considering the concept of latent inhibition, whereby a few painless treatments prevented the development of dentist phobia, one would expect that numerous fearless experiences with supermarkets and other public places would have immunized adults from developing agoraphobia and panic disorder, regardless of any preconditioning experience. It is also doubtful whether Davey's concept of sensory preconditioning is applicable to non-situational panic disorder, in the absence of agoraphobia, whereby the manifestation of panic is independent of any specific environmental stimuli (Marks, 1987b; Jacobs & Nadel, 1985).

Moreover, from a clinical standpoint, Davey had to demonstrate that agoraphobia and panic disorders are consequences of an inflation of earlier traumatic experiences. This idea is reminiscent of the Freudian concept of repression, particularly in its learning version, as formulated by Dollard and Miller (1950), which states that a previous fearful event, of which the subject is not aware, increases the individual's vulnerability to develop pathological behavior at a later stage. However, as previously stated, findings tend to refute the existence of this Freudian concept.

Furthermore, although some investigators succeeded in replicating White and Davey's (1989) aforementioned experimental findings regarding Davey's inflation theory (Hosoba, Iwanaga, & Seiwa, 2001), others obtained negative results (De Jongh, Muris, & Merckelbach, 1996; De Jongh, Merckelbach, Koetshuis, & Muris, 1994). Additionally, even if the inflation hypothesis had been consistently supported, this would not have necessarily been applicable to agoraphobia and panic disorder, as fundamental differences exist between laboratory-conditioned fears, which are easily extinguished when subjects are notified that the CS will no longer be followed by the UCS, and clinical phobias, which are highly resistant to extinction (Jacobs & Nadel, 1985; Seligman, 1988).

Additional problems concern the cognitive assertion that awareness is a necessary condition for the development of conditioned fear. Although much evidence is consistent with this theoretical position, some studies suggest that conditioning without awareness may occur (see review by Lovibond & Shanks, 2002; see also Clark, Manns, & Squire, 2002; Weike, Schupp, & Hamm, 2007). A more fundamental problem concerns the cognitive position that fear should, in fact, be extinguished once subjects become aware that the UCS no longer accompanies the CS (Davey, 1989a). Although this effect is typically observed in laboratory studies (Brewer, 1974; Davey, 1989b; Dawson et al., 1986), this is not the case in natural settings. As noted by Seligman (1971) "telling a phobic, however persuasively, that cats (CS) won't do him any harm, or showing him that the UCS doesn't occur when

cats are around is rarely effective” (p. 311). Similarly, as stated by Rachman (1990a) fears in everyday life “can arise and persist in the acknowledged absence of any threat or danger” (p. 74). Resistance to extinction is particularly puzzling with regard to agoraphobia and panic disorder, where patients resist abandoning their fears even after experiencing numerous non-reinforced exposures to the feared stimulus (Jacobs & Nadel, 1985; Seligman, 1988).

In an attempt to resolve this problem, Bandura (1971, 1977) claimed that fear-arousing beliefs become so powerful that they are not easily subject to voluntary control. In support of his claim, Bandura noted that acrophobics display fear when found in tall buildings, because they are unable to quell horrendous thoughts despite the safety of their situation. However, findings inconsistent with this claim indicate that perception of danger is, in fact, a weak predictor of acrophobic behavior (Menzies & Clarke, 1995b; Williams, Turner, & Peer, 1985; Williams & Watson, 1985) and that agoraphobics are rarely preoccupied with thoughts of danger when confronted with their feared situation (Williams, Kinney, Harap, & Liebmann, 1997). Hence, as noted by Williams et al. (1997), “rarity of danger thoughts poses an explanatory challenge for all cognitive theories of phobia and especially for the perceived danger theory” (p. 511).

Cognitive theory would expect negative cognitions to precede the experience of fear. However, although negative thoughts preoccupy the individual’s attention when exposed to the stimuli that they fear (De Jongh, Muris, Schoenmakers, & Ter Horst, 1995; De Jongh, Muris, Ter Horst, & Duyx, 1995; De Jongh & Ter Horst, 1993; Kent & Gibbons, 1987; Thorpe & Salkovskis, 1995), some studies indicate that negative cognitions follow, rather than precede, the experience of fear (Wolpe & Rowan, 1988). Moreover, negative thoughts may be a direct consequence of the fear (“If I feel anxious, there must be danger”; Arntz, Rauner, & Van den Hout, 1995), and fear may also be experienced in the absence of negative cognitions (Barlow, Brown, & Craske, 1994; Fleet et al., 2000; Kushner & Beitman, 1990; Williams et al., 1997).

In conclusion, it does not seem that the suggestions of the cognitive theory of conditioning, such as that awareness of CS-UCS is a necessary condition for the acquisition of phobia, latent inhibition and sensory preconditioning, can promote our understanding of agoraphobia and panic disorder. Adding these difficulties to those of the Pavlovian model raises serious doubts as to whether the conditioning model is an appropriate approach for understanding these disorders. Nevertheless, cognitive factors were incorporated within the contemporary theory of conditioning of human fear (Bouton et al., 2001; Mineka & Zinbarg, 2006), and there seems to be little doubt that these factors affect the acquisition of simple fear and phobia.

2.1.2 Misinterpretation Theory

Clark’s (1986, 1988) cognitive theory suggests that panic disorder results from catastrophic misinterpretation of benign bodily sensations of unclear or ambiguous origin, and that educating patients to interpret their bodily sensations realistically is an effective therapeutic intervention. This theoretical approach received significant empirical support for both the development (Austin & Richards, 2001; Cox, Endler, & Swinson, 1995; Goldberg, 2001; Margraf & Ehlers, 1991; Richards, Edgar, & Gibbon, 1996; Schneider & Schulte, 2007; Westling & Öst, 1995) and treatment (Beamish et al., 1996; Beck, 1988; Clark, 1988, 1999; Gelder, Clark, & Salkovskis, 1993; Hecker et al., 1998; Ollendick, 1995; Westling & Öst, 1995) of panic disorder.

These studies, however, do not necessarily confirm a causal relationship between cognitions and panic attacks. As noted by Rachman (1990a), catastrophic misinterpretation of thoughts may be “mere accompaniments of a fundamentally biological disorder or accompaniments of conditioned panic reactions” (pp. 129-130). Findings suggest that panic attacks may occur in the absence of catastrophic thoughts as well; for example, a number of investigators found that panic attacks occur during the non-dreaming stages of sleep, where catastrophic thoughts are absent (Craske & Barlow, 1989; Craske & Rowe, 1997; Hauri, Friedman, & Ravaris, 1989; Lesser, Poland, Holcant, & Rose, 1985; Ley, 1988; Mellman & Uhde, 1989). Although Schredl, Kronenberg, Nonnell and Heuser (2001) reported that nocturnal panic attacks are closely related to dreams and nightmares, they too reported that a subgroup of patients in their study experienced panic attacks in the absence of dreams. Furthermore, subjects may display diurnal panic attacks which are not preceded by catastrophic cognitions (Kenardy, Fried, Kraemer, & Taylor, 1992; Kenardy & Taylor, 1999; Rachman, Lopatka, & Levitt, 1988; Zucker et al., 1989).

Additionally, the theory does not address cases of “non-fearful panic disorder” or “non-cognitive panic”, which occur in the absence of fear or catastrophic cognitions (Barlow et al., 1994; Beitman et al., 1987; Beitman, Mukerji, Russell, & Grafing, 1993; Bringager et al., 2008; Fleet et al., 2000; Kushner & Beitman, 1990; Rachman, Levitt, & Lopatka, 1987; Rachman, Lopatka, & Levitt, 1988). A more fundamental problem concerns the development of agoraphobia. Considering cognitivists’ position that agoraphobia is a by-product of panic

disorder (Clark, 1986; Gelder, 1989), it is difficult to see how this theory can account for a substantial number of cases in which agoraphobia develops independently of panic attacks or panic disorder (Fava et al., 2008; Wittchen et al., 2008) and that agoraphobia without panic attack can in many cases predict the onset of panic disorder (Bienvenu et al., 2006).

A possibility was raised that fearful thoughts are sometimes “missing” because the catastrophic misinterpretation of bodily sensations takes place so quickly that panic patients may not be aware of their existence (Clark, 1988; Rachman et al., 1987). McNally (1990) noted, however, that patients would remember thoughts of having heart attacks, even if they were momentary. Furthermore, this claim renders Clark’s theory untestable. As noted by McNally (1990), if patients could become unaware of such self-producing catastrophic thoughts, “then what would constitute evidence against the hypothesis that catastrophic misinterpretation necessarily precedes panic attacks?” (p. 407; see also McNally, 1999, p. 8).

Cognitive theory also provides no explanation as to the mechanism, which makes patients, think irrationally or non-adaptively. While psychoanalysis attributes this effect to the unconscious (De Poderoso et al., 2005; Shilkret, 2002), conditioning theories to learning experiences (Bouton et al., 2001; Davey, 1989a), and biological models to adverse neurochemical factors (Barlow, 2004), cognitivists provide no explanation for why a minority of people suddenly begin to think irrationally.

It is also difficult to see how cognitive theory may account for the therapeutic efficacy of both antidepressants (Den Boer, 1998; Westenberg, 1996) and interoceptive exposure (Arntz, 2002; Beck & Shipherd, 1997; Beck, Shipherd, & Zebb, 1997; Hecker et al., 1998), despite the absence of corrective information that supposedly educates patients to interpret their bodily sensations rationally (Beck, 1988; Clark, 1988). It is additionally unclear why cognitive therapy (Van den Hout, Arntz, & Hoekstra, 1994; Williams & Falbo, 1996) is ineffective for treating agoraphobia, which cognitive theory believes to have the same etiology as panic disorder (Barlow, 2004; Gelder, 1989).

In addressing the efficacy of drugs, Taylor (2000) argued that when patients are informed of potential side effects, they reinterpret their bodily sensations as a sign that the medication is exerting its influence, in place of the previous perception that the sensations herald impending disaster. However, if this explanation were true, then any substance, which is perceived as a drug, should have a similar effect when patients are made aware of expected side effects. However, so far there is no evidence in support of this claim. As an alternative explanation, Taylor suggested that drugs increase patients’ psychological confidence in their own abilities to cope with the arousal-related sensations. Here too, however, a placebo should have a similar effect to the actual drug, since the crucial therapeutic factor is patients’ expectations and beliefs. Regarding the efficacy of interoceptive exposure, Arntz (2002) noted that “the hypothesis of Clark and his coworkers... that irrespective of the type of treatment, reduction of panicogenic beliefs is necessary for (stable) symptom reduction to take place, is not true” (p. 339).

In conclusion, findings consistently indicate that catastrophic cognitions accompany the experience of most panic attacks, and that changing these cognitions plays an important role in the treatment of panic disorder. However, the causal relationship between these cognitions and panic disorder remains unclear. Although in many cases catastrophic cognitions precede the experience of panic attacks, other evidence indicates that panic attacks may occur in the absence of negative cognitions. It is also doubtful whether this theory can account for the development of agoraphobia in the absence of panic disorder, and whether it can integrate the therapeutic effects of both interoceptive exposure and drug treatments into its theoretical framework.

2.1.3 Anxiety Sensitivity Theory

Anxiety sensitivity (AS) theory assumes the existence of a stable predisposition towards anxiety-related bodily sensations and/or emotions, which stem from the belief that these experiences have harmful physical, psychological or social consequences (Reiss, 1991; Reiss & McNally, 1985; Reiss, Peterson, Gursky, & McNally, 1986). Findings show that high AS individuals tend to implicitly associate anxiety-related symptoms with negative consequences (Lefavre, Watt, Stewart, & Wright, 2006), and that this tendency increases the risk of developing panic disorder (Grant, Beck, & Davila, 2007; Li & Zinbarg, 2007; Schmidt & Lerew, 2002; Schmidt, Lerew, & Jackson, 1999). Similar results were found in a longitudinal study of adolescents (Hayward, Killen, Kraemer, & Taylor, 2000; Hayward, Killen, & Taylor, 2003; Hayward & Wilson, 2007; Schmidt, Zvolensky, & Maner, 2006). Studies also report a significant relationship between AS and other anxiety disorders, such as OCD and social phobia (Anderson & Hope, 2009; Calamari et al., 2008; Rector, Szacun-Shmizu, & Leybman, 2007). Further studies demonstrate that apart from various anxiety disorders, AS facilitates the development of other anxiety-related behaviors. For example, AS has been found to be significantly related to smoking and substance abuse, as these behaviors enhance the individual’s ability to cope

with the unpleasant sensations resulting from stress (Battista et al., 2008; Bonn-Miller, Zvolensky, & Bernstein, 2007; Comeau, Stewart, & Loba, 2001; Feldner et al., 2008; Zvolensky et al., 2009).

Although AS is an important factor in predicting panic disorder, it does not provide a comprehensive account of this disorder: as noted by Bouton et al. (2001), only a small percentage of the variance (2%-16%) is accounted for in the disorder's development (Li & Zinbarg, 2007; Schmidt et al., 1999). Additionally, the mechanism by which AS exerts its influence is not yet clear, and various investigators account for its effect on the development of panic disorders with either the cognitive theory of panic disorder or interoceptive conditioning (McNally, 1999; Taylor, 2000; Zvolensky et al., 2006). Thus, while AS enhances our understanding of some central factors, which directly affect the development of panic disorder, it lends little information to help resolve the dispute between cognitivists and behaviorists regarding the mechanism by which panic disorder develops.

2.2 *Biological Models*

Advancements made in biological research increased the conviction that the biological approach may provide a better means for the understanding of deviant behavior. As stated by Reich (1982), "American psychiatry has in fact undergone a significant shift from an emphasis that was primarily psychological to one that is more clearly biological ... in every sphere of psychiatric enterprise" (p. 189). Likewise, Telch (1988) noted that "biological theories of panic and agoraphobia have shown a major increase in popularity" (p. 507). The biological perspective becomes particularly important in light of the growing dissatisfaction with the existing psychological theories of fear and phobia. This theoretical approach consists mainly of the non-associative account, which addresses the development of specific fears and phobias, and other models, which address the causes of panic disorder and agoraphobia.

2.2.1 The Non-Associative Account

The non-associative account observed that certain specific phobias (e.g., spider phobia and acrophobia) develop upon initial contact with the feared stimuli, in the absence of observable learning experiences (see review by Menzies & Clarke, 1995a; Menzies & Harris, 2001; Poulton & Menzies, 2002a). These findings were interpreted as evidence that both humans and animals have been biologically programmed through evolution to fear stimuli that might endanger their existence.

This theoretical approach was subject to a number of criticisms (Davey, 2002; Kleinknecht, 2002; Marks, 2002; Mineka & Öhman, 2002b; Muris et al., 2002b). One critique concerns the authenticity of retrospective reports (Kleinknecht, 2002; Mineka & Öhman, 2002b). The retrospective reports, however, were supplemented with prospective longitudinal studies (Poulton & Menzies, 2002a), as well as children's reports that were confirmed by their parents (Craske & Waters, 2005; Graham & Gaffan, 1997; Merckelbach, Muris, & Schouten, 1996; Merckelbach & Muris, 1997; Poulton & Menzies, 2002a). A recent study also found data that 7- to 18-month-old children display fear when observing films of moving snakes (DeLoache & LoBue, 2009). It would seem that the non-associative account's basic premise, which fear develops in the absence of observed learning experiences, is indisputable.

Nevertheless, the non-associative account is problematic for other reasons. For example, the lack of recollection of learning events is also observed with phobias that are certainly not evolutionarily significant, such as fear of injection (Kleinknecht, 1994), public speaking (Hofmann, Ehlers, & Roth, 1995) and driving (Ehlers, Hofmann, Herda, & Roth 1994; Taylor et al., 1999). Moreover, evolutionarily, there are some stimuli which have endangered the species' existence (e.g., various predatory animals and poisonous plants), yet have not resulted in common phobias (Davey, 2002; Delprato, 1980). By contrast, spiders are viewed as an evolutionary biological phobia by the non-associative account, despite the fact that "only 0.1% of the 35,000 varieties are dangerous to human beings" (McNally, 2002, p. 170). Furthermore, heritability of evolutionarily relevant fears tends to be low (Kendler et al., 1992) or at best moderate (Kendler, Karkowski, & Prescott, 1999; Kendler, Myers, Prescott, & Neale, 2001). At any rate, as noted by Kendler et al. (1999), environmental experiences still "play an important role in the development of phobias" (p. 539). Hence, these findings do not necessarily confirm the evolutionary basis of these fears and phobias.

Some investigators also claim that "evolutionary-relevant phobia" may be the consequence of interoceptive conditioning, caused by sudden terror or disgust sensitivity (Davey, 2002; Mineka & Öhman, 2002b) which is viewed as a substitute for the UCS. However, this claim is just as speculative as the non-associative account, as it has no supporting empirical evidence. Moreover, conditioning theorists have not yet accounted for the origin of the subjects' intense arousal. As noted by McNally (2002), "advocates consider sudden terror in the presence of the to-be-feared stimuli as conditioning events that explain the subsequent phobia ... This formulation, of course, begs the question of why the person experienced terror in the first place" (p. 170; see also Poulton &

Menzies, 2002b, pp. 198-199). An additional problem is that “a single stimulus can hardly serve as both the CS (i.e., neutral stimulus) and the UCS (i.e., fear-provoking stimulus) in the one event” (Poulton & Menzies, 2002b, p. 199).

In conclusion, there seem to be sufficient findings to support the basic claim of the non-associative account that certain specific fears and phobias emerge spontaneously upon first contact with the feared stimuli. However, the interpretation of these findings is controversial. While the non-associative account attributes the spontaneous fear to evolutionary/biological causes, learning theorists claim that this effect may be the result of conditioning. All the same, even if the non-associative account is valid, its theoretical proposal is applied to a very narrow range of fear and phobia. As acknowledged by Poulton and Menzies (2002b) themselves, “the non-associative model of fear acquisition postulates the existence of a limited number of innate evolutionary-relevant fears, while emphasizing conditioning modes of onset for evolutionary-neutral fears” (p. 197).

2.2.2 Biological Approach to Panic Disorder and Agoraphobia

The biological approach to panic disorder and agoraphobia bases its theoretical claims on four major groups of empirical findings: (1) Studies indicating that patients with panic disorder and agoraphobia suffer from structural brain abnormalities, such as in the temporal lobe, the basal ganglia, the cingulate, and the brainstem (Asami et al., 2008; Barlow, 2004; Ham et al., 2007; Han et al., 2008; Lee et al., 2006; Stein, 2008); (2) Studies showing that genetics play a significant role in the etiology of panic disorder and agoraphobia (Chantarujikapong et al., 2001; Hettema, Neale, & Kendler, 2001; Kendler et al., 1999; Kendler et al., 2001; Nocon et al., 2008; Stein, 2008); (3) Findings obtained using the paradigm of the biological challenge, whereby panic attacks can be experimentally induced in panic disorder patients using a variety of chemical substances (e.g., sodium lactate infusion and CO₂ inhalation; see Abrams, Schruers, Cosci, & Sawtell, 2008; Barlow, 2004); (4) Research demonstrating that drug therapy is an effective intervention in the treatment of these disorders (Furukawa, Watanabe, & Churchill, 2006; Mavissakalian & Ryan, 1998; Westenberg, 1996).

While these findings are impressive, and need to be taken into consideration for understanding panic disorder and agoraphobia, the extent to which biological factors are involved in the development of these disorders is unclear. Advocates have yet to prove that these disorders are uniquely associated with certain brain abnormalities, or that the existence of specific brain damage is a necessary condition for their development. As noted by Barlow (2004), “At the present time, there is no evidence for any specific biological marker; nor, for that matter, is there evidence for any important neurobiological differences between patients with panic disorder and individuals without panic” (p. 221). Similarly, genetic studies indicate that the concordance rates among monozygotic twins vs. dizygotic twins are 31%-42% vs. 0%-17% (Barlow, 2004), which point to the limitations of genetic influence and leaves much room for environmental/psychological factors. At any rate, it does not seem that biological factors on their own can account for the development of these disorders.

As for the biological challenge, biologists have not yet succeeded in identifying biological mechanisms that could account for the psychological impact of chemical substances (see review by Barlow, 2004). Barlow (2004) correctly pointed out that “It... seems safe to say at this time that there is no single underlying biological mechanism of action that can account for these diverse provocation procedures” (p. 178). Panic attacks can be provoked by such a wide variety of chemical substances (e.g., adrenaline, epinephrine, isoproterenol, yohimbine and caffeine; Barlow, 1988, pp. 112-121; Nutt & Lawson, 1992; Rapee, 1995) that Gorman (1987) wondered if there is “any active agent that does not cause a panic” (p. 6). Some studies also suggest that the effect of biological challenges can be accounted for in cognitive terms, whereby patients catastrophically misinterpret physiological sensations induced by these chemical substances (Rapee, 1995).

Additionally, the therapeutic efficacy of drugs does not necessarily validate the biological etiology of panic disorder and agoraphobia. In considering this issue, Acierno, Hersen and van Hasselt (1993) stated that “it is an error in logic to assume that because drugs alleviate symptoms, their absence (or the absence of their metabolites, etc.) is the cause of those symptoms” (p. 563). Similarly, Margraf, Ehler, and Roth (1986) noted that “the efficacy of different methods for treating panic attacks must be determined independently of any inferred etiology of those attacks” (p. 563).

In conclusion, none of the aforementioned findings of the biological approach is sufficiently convincing to necessitate the biological account of panic disorder and agoraphobia. However, these findings must be taken into consideration in any further attempt to understand the etiology of these disorders.

2.2.3 Suffocation False Alarm Theory

A more systematic attempt to understand the development of panic disorder in biological terms was made in Klein's (1993) suffocation false alarm theory, which suggests that panic attacks among panic disorder patients are the consequence of a malfunctioning brain suffocation monitor that erroneously signals a lack of useful air remaining in the body. In support of his theory, Klein (1993) reviewed a variety of data to draw a causal link between respiratory difficulties and panic disorder (Preter & Klein, 2007).

Klein (1993) noted that CO₂ has a central importance in stimulating panic attacks among panic patients, because the elevation of its level in the arteries (PCO₂) erroneously activates the suffocation alarm system. A number of experimental studies are consistent with this hypothesis (Asmundson & Stein, 1994; Gorman et al., 1994; Maddock, 2001; Pine et al., 1994). For example, in Asmundson and Stein's study (1994), panic patients displayed significantly shorter duration in a breath-holding task than did control subjects. These results were interpreted as evidence that subjects attempt to avoid the activation of their hypersensitive suffocation alarms. Based on cognitive theory, however, Schmidt, Telch, and Jaimez (1996) claimed that these findings could equally be accounted for by cognitive concepts, i.e., panic patients discontinue breath holding due to a catastrophic misinterpretation of dyspnea sensations. The authors reached the same conclusion with regard to the findings by Gorman et al. (1994) and by Pine et al. (1994). This claim could also be generalized to Biber and Alkin's (1999) study that examined the sensitivity of panic patients to CO₂.

Moreover, Schmidt et al. (1996) reported that contrary to the suffocation false alarm theory, no significant difference in dyspnea was found between a biological-challenge procedure which reduced the risk of activation of the suffocation alarm system (hyperventilation) and a method which increased this risk (inhalation of 35% CO₂). This theory was further challenged by findings demonstrating that cognitive-behavioral therapy effectively eliminated CO₂-induced panic attacks (Schmidt, Trakowski, & Staab, 1997). While at the pre-treatment stage a significant majority of patients experienced panic during the inhalation of 35% CO₂, only a minority of subjects did so at the post-treatment assessment. Studies also refute Klein's distinction between panic patients with intense dyspnea, which he attributed to dysfunction of the suffocation alarm system, and patients with fear-like panic experiences with little or no dyspnea, which he claimed to be a result of maladaptive learning (McNally, Hornig, & Donnel, 1995; Taylor et al., 1996; Vickers & McNally, 2005). These and other studies indicate that cognitive variables are more appropriate for explaining relevant findings and serve as better predictors of panic disorder than pulmonary measures (Carr, Lehrer, & Hochron, 1995; Moore & Zebb, 1999; Rose, 1998).

Further evidence that Klein (1993) considered to be support for his suffocation false alarm theory was the high frequency of panic disorder among patients with pulmonary diseases (Moore & Zebb, 1999; Nascimento et al., 2002; Roy-Byrne, Craske, & Stein, 2006; Shavitt, Gentil, & Mandetta, 1992; Valença et al., 2006). However, inconsistent with this theory, findings show that the high prevalence of panic disorder in asthmatic patients (9%), although higher than that of the general population, was very similar to the rate observed in non-asthmatic patients referred for a histamine provocation test (8.9%) (Van Peski-Oosterbaan et al. 1996). These authors also reported no difference in pulmonary function between asthmatics with and without panic disorder. The main difference between the two groups is a higher level of anxiety and depression among those with panic disorder (Dorhofer & Sigmon, 2002). Other investigators (Massana et al., 2001; Moore & Zebb, 1998, 1999) found similar results.

One last issue for consideration is Klein's (1993) claim that agoraphobia stems from the same psychological process as panic disorder, with agoraphobics experiencing more frequent and severe panic attacks, earlier onset and longer duration of illness. Aside from the aforementioned difficulties, the suffocation false alarm theory has an additional problem in accounting for a substantial number of cases of agoraphobia without panic (Fava et al., 2008; Goodwin et al., 2005; Hayward & Wilson, 2007; Wittchen et al., 2008; Wolitzky & Eagle, 1999).

In conclusion, there seems to be enough data indicating that people with pulmonary diseases are more likely to develop panic disorder and agoraphobia. However, findings cast serious doubts regarding the validity of Klein's suffocation alarm theory.

2.2.4 Mitral Valve Prolapse

Another attempt to account for the development of panic disorder and agoraphobia in biological terms was made by studies investigating the relationship between Mitral Valve Prolapse (MVP) and panic and agoraphobia disorder. MVP is a congenital cardiac abnormality whose symptoms include non-anginal chest pain, palpitations, dyspnea, light-headedness, and anxiety (Bowen, D'Arcy, & Orchard, 1991; Marks, 1987a). Several researchers found a relatively high prevalence of MVP among panic and agoraphobic patients, as compared to the general population (Bowen et al., 1991; Hamada, 1998; Katerndahl, 1993; Singh, 1996). This correlative data and the

general similarity of the symptoms led to the idea that MVP could be an important etiological cause of panic disorder and agoraphobia (Margraf, Ehlers, & Roth's review, 1988). However, as Margraf et al. (1988) showed, elevated prevalence of MVP was also reported for eating disorders (Kaplan et al., 1991), generalized anxiety disorder, and bipolar affective disorder.

Nevertheless, based on a meta-analysis of 21 studies, Katerndahl (1993) concluded that there appeared to be a significant relationship between MVP and panic disorder. Likewise, Gorman, Goetz, Fyer, King et al. (1988b), in re-examining this issue, reported that MVP is more common in those who suffer from panic and agoraphobia disorders. However, MVP in these patients was mild, was associated with neither thickened mitral leaflets nor small left ventricular size, and was not commonly conjunctive with moderate or severe prolepses. The authors concluded that although MVP is more common among these patients than in the general population, "there continues to be no evidence that MVP has any clinical relevance in the actual management of patients with PD/AgP [panic disorder or agoraphobia with panic attacks]" (p. 120). Additional indications call into question the causal relationship between MVP and panic disorder. Coplan, Papp, King, and Gorman (1992) claimed that MVP is the consequence rather than the cause of panic disorder. Accordingly, they found that panic disorder patients with MVP showed amelioration of prolapse on repeated echocardiograms after treatment for panic disorder. Indeed, a recent review article strengthened previous reports that the existing data are insufficient for determining a clear relationship between MVP and panic disorders, and that "if any relationship does actually exist, it could be said to be infrequent and mainly occur in subjects with minor variants of MVP" (Filho et al., 2008, p. 38). Further research may be needed to come to a firm conclusion regarding the relationship between MVP and panic and agoraphobia disorders, but its role in the development of these disorders seems at present to be negligible.

3. Discussion

3.1 Strengths and Weaknesses of Traditional Theories

Theories of fear and phobia have encountered fundamental difficulties in accounting for these behaviors. Although none of the existing theories provides a comprehensive account of the topic, each does provide its own unique contributions to the understanding of different types of fear and phobia. Upon completing the examination of the empirical status of the various theories, it is important to review the relative contributions of each theory, to specify the difficulties that have yet to be resolved, and to raise possible suggestions that may promote our knowledge of the factors involved in the development of fear and phobia.

When one examines the various types of fear and phobia, the reviewed literature indicates that specific phobia has received by far the most comprehensive explanation; behaviorism has successfully accounted for this phenomenon, outdistancing any other theory. Though this theory in its original form encountered some difficulties, advocates of this approach have made significant changes that adequately resolve these issues. One problem concerned the fact that people sometimes fail to develop conditioned fear despite exposure to an anxiety-provoking event (e.g. aversive experiences with dogs, painful dental experiences). In addressing this matter, Davey (1989a, 1992b) suggested the concept of latent inhibition, which refers to previous non-aversive exposures to the CS in order to account for cases of conditioning failure despite exposure to trauma (Mineka & Zinbarg, 2006).

Another challenge to conditioning theory concerned the claim of the non-associative account that childhood phobias often develop in absence of any observable conditioning event (Poulton & Menzies, 2002a). Conditioning investigators attributed these seemingly biological phobias to interoceptive conditioning caused by sudden terror or disgust (Davey, 2002; Mineka & Öhman, 2002b). McNally (2002) voiced reservations regarding this suggestion, asserting that interoceptive conditioning provided no explanation as to how spontaneous arousal occurs in response to such stimuli. Similarly, Poulton and Menzies (2002b) noted, "a single stimulus can hardly serve as both the CS (i.e., neutral stimulus) and the US (i.e., fear-provoking stimulus) in the one event" (p. 199). However, this argument does not necessarily pose a difficulty to conditioning theory. Studies indicate that both animals and humans are more likely to experience fear if the stimuli are novel or strange (Collard, 1967; Harlow & Zimmermann, 1959; Hebb, 1946; Marks, 1987a; Mussen, Conger, Kagan & Houston, 1984; Warr, 1990). While low or moderate levels of discrepancy between the subject's prior experiences (schema) and present stimuli may elicit attention and even excitement, a high discrepancy would be likely to produce fear (Mussen et al., 1984). Thus, when a young child first encounters stimuli such as spiders and snakes, the perception of this stimulus serves both as a CS and as a UCS because the unique shape, movement, and other perceptual characteristics of the stimulus are radically different from the individual's perceptual schema. This can cause a

spontaneous arousal as severe as an electric shock can (see case study by Merckelbach, Muris, & Schouten, 1996), thereby producing a conditioning effect.

The third problem concerned the distribution of fear and phobia and its inconsistencies with classic conditioning theory, such as the fact that snake phobia is far more prevalent than injection phobia despite the higher frequency of negative experiences with the latter (Rachman, 1977, 1990a). In an attempt to resolve this problem, Seligman (1971) suggested that organisms have been evolutionarily prepared to acquire certain fears more easily than others do. In accordance with this prediction, fear-relevant stimuli (e.g. pictures of snakes and spiders) were more easily conditioned than fear-irrelevant stimuli (e.g. picture of flowers and geometric shapes). Although these findings do not necessarily support Seligman's evolutionary claim, as they may be due to differences in structure, movement, and the subject's cultural background, this does not change the fact that some fears can be more easily acquired than others can. Considering these modifications, the classical conditioning theory seems to provide the most complete explanation for the development of specific fear and phobia.

Undoubtedly, cognitive and biological/genetic variables play an important role in the development of specific phobia. However, it does not seem feasible to account for the mechanism of phobia solely by cognitive theory, because unlike laboratory fear, phobia in real-life settings resembles a reflex-like response, in which the knowledge that a UCS is no longer present does not extinguish an individual's phobia. Similarly, although biological/genetic factors may also facilitate or inhibit the acquisition of fear and phobia by affecting, for example, emotional arousal (Mineka & Oehlberg, 2008), there is no evidence linking the variability of these behaviors to specific biological factors.

Unlike specific fear and phobia, there are inherent difficulties in the theories of the development of panic disorder and agoraphobia that have yet to be resolved. For example, the interoceptive conditioning theory has successfully demonstrated that similar to the CS in the conditioning paradigm, bodily sensations often trigger panic attacks among panic disorder patients (Barlow, 2004; Bouton et al., 2001), and exposure to interoceptive stimuli in the absence of cognitive intervention can result in significant therapeutic change (Beck & Shipherd, 1997; Beck et al., 1997; Hecker et al., 1998). However, it is hard to accept that the initial panic attack, attributed to stressful life events, serves as a substitute for a conventional UCS. If this is so, why is stress alone insufficient in accounting for the subsequent panic attacks? It is also unclear why hyperventilation, which subjects the individual to intense bodily sensations (CS), does not invariably lead to panic attacks among those suffering from panic disorder (Gorman et al., 1988a; Hibbert & Pilsbury, 1989; Nardi et al., 2004; Sanderson & Beck, 1989). A further challenge concerns the efficacy of cognitive therapy without exposure, which seems to alter the patient's belief rather than the S-R connection (Arntz, 2002; Clark, 1999; Salkovskis et al., 1991).

Cognitive theory has convincingly demonstrated that catastrophic beliefs are a central characteristic of patients with panic disorder, and that changing these beliefs has significant therapeutic effects (Clark, 1986, 1989, 1999; Austin & Richards, 2001; Schneider & Schulte, 2007). However, as noted above, the existence of catastrophic beliefs does not necessarily prove a causal relationship, especially because panic attacks may occur in absence of catastrophic thought (Craske & Rowe, 1997; Hauri et al., 1989; Ley, 1988; Mellman & Uhde, 1989).

Although the biological approach has provided a significant amount of evidence demonstrating the role of genetic and biological factors in the etiology of fear and phobia (Barlow, 2004; Mineka & Oehlberg, 2008), investigators have yet to present the necessary data to convincingly demonstrate that the development of panic disorder and agoraphobia is biologically determined. Further, the validity of Klein's (1993) suffocation false alarm theory is questionable; in fact, findings tend to be inconsistent with this theoretical position. Similarly, it is doubtful whether MVP plays any etiological role in the development of panic disorder and agoraphobia.

In addition to the aforementioned specific difficulties challenging behavioral, cognitive and biological theories, these theories share several fundamental difficulties. One issue common to these theories is that panic disorder and agoraphobia are rarely found in children under the age of ten (DSM-IV-TR, 2000; Goodwin et al., 2005), as opposed to specific phobias (Flatt & King, 2008; King et al., 1998; Marks, 1987a; Stinson et al., 2007). Another concern is that while existing theories view agoraphobia as a by-product of panic disorder (Bouton et al., 2001; Mineka & Zinbarg, 2006; Klein, 1993), agoraphobia develops in the absence of panic attacks in a significant number of cases, and at times may even predict the development of panic disorder (Bienvenu et al., 2006; Wittchen et al., 2008).

Although the abandonment of Freud's theory seems inevitable, it is premature to disregard Freud's clinical intuition that in order to understand the etiology of fear and phobia, one must comprehend how unawareness occurs. This is especially true with panic disorder and agoraphobia, which as demonstrated above develop in the absence of conventional UCS. There would be a scientific justification to dismiss the psychoanalytic approach if

rival theories could integrate relevant findings into one cohesive theoretical framework. Moreover, it is difficult to see how we could understand the development of some bizarre phobias, such as phobia of chocolate and vegetables (Rachman & Seligman, 1976), train phobia (Leonard, 1927), rat germ phobia (Brandt & Mackenzie, 1987) and phobia of AIDS (Glass, 1993), in the absence of an alternative theory to psychoanalysis. In fact, the inability of patients to account for the underlying causes for their dramatic behavioral change motivated Freud to suggest his psychoanalytic theory. As noted by Shevrin and Dickman (1980), “the clinical phenomena that led to the assumption of unconscious processes often takes the form of a patient describing a bothersome condition that the patient can neither account for nor control” (p. 422). Similarly Woody (2003) noted “the unconscious is invoked to explain behavior that is remarkable or portentous and inscrutable: actions and thoughts that seem otherwise inexplicable – bizarre behavior...” (p. 190; see also Erdelyi, 1985). Even Rachman (1990a), a leading orthodox behaviorist, acknowledged that although psychoanalysis cannot serve as a comprehensive account of all human fears, “it may help us to understand some of the more unusual fears” (p. 204).

There are three striking differences between simple and bizarre phobia, which strengthen the possibility that these are two distinct psychological phenomena that require different theoretical explanations. First, panic disorder and agoraphobia (Michelson et al., 1998; Milrod, Leon, & Shear, 2004; Scocco, Barbieri, & Frank, 2007; Venturello, Barzega, Maina, & Bogetto, 2002; Wardle et al., 1997) and unusual specific phobia (Brandt & Mackenzie, 1987; Glass, 1993; Rachman & Seligman, 1976), are generally preceded by stressful-life events not uniquely associated with the deviant behavior (Jacobs & Nadel, 1985). In contrast, common specific phobias tend to develop either spontaneously at an early age, in the absence of a learning experience (Poulton & Menzies, 2002a), or after an anxiety-provoking event directly linked to the phobia (e.g., a dog bite or painful dental treatment may cause dog phobia or dentist phobia, respectively, but a dog bite is unlikely to cause dentist phobia). Evidence confirming a direct link between type of trauma and type of phobia was observed regarding various types of specific phobia, including dental phobia (Davey, 1989b; De Jongh, Fransen, Oosterink-Wubbe, & Aartman, 2006) dog phobia (Di Nardo, Guzy, & Bak, 1988; Hoffman & Human, 2003), and spider phobia (Mercklebach & Muris, 1997). Second, bizarre behaviors, such as panic disorder and agoraphobia, are extremely rare; most studies found rates between 1%-2% (American Psychiatric Association, 2000). In contrast, common specific phobias of all types have a relatively high prevalence of 7.2%-12.5% (American Psychiatric Association, 2000; see also Kessler et al., 2005; Stinson et al., 2007). Third, bizarre disorders generally develop in later years. As stated in the DSM-IV (American Psychiatric Association, 2000), panic disorder and agoraphobia tend to develop between late adolescence and mid-30s. In contrast, common specific phobia typically develops during childhood onset peaks at age five with a secondary peak at age 10, mean age of onset 9.7 (Stinson et al., 2007).

In conclusion, it is necessary to distinguish between simple and bizarre phobia in order to gain true insight in the mechanisms by which fear and phobia develop. It is also necessary to explain why patients with bizarre phobia are unaware of the underlying causes of the dramatic change in their behavior. While the behaviorist's conditioning paradigm appears to be the best explanation for simple phobia, a revised version of psychoanalytic theory, seems most suitable for the explanation of bizarre phobia.

3.2 *The Rational-Choice Theory of Neurosis*

The Rational-Choice Theory of Neurosis (RCTN) (Rofé, 2010; Rofé, & Rofé, 2013), is a revised version of Psycho-bizarreness Theory, presented in a book entitled *The Rationality of Psychological Disorders* (Rofé, 2000; Lester, 2002, Sarma, & Garfield, 2001). The basic assumption of this theory is that individuals are likely to adopt neurotic disorders, such as panic disorder, agoraphobia, OCD and conversion disorder, when confronted with an intolerable level of stress and other options, such as suicide, drug abuse, and antisocial behaviors, are unavailable or too costly. The basic ideas of this theory are summarized below.

3.3 *The Need to Reinstate Neurosis*

RCTN agrees with psychoanalysis that the concept of neurosis is essential for understanding the development and treatment of these disorders. Accordingly, it rejects the DSM-III's decision to remove neurosis (American Psychiatric Association, 1980) by claiming, “There is no group of conditions which together comprise the ‘neurosis’” (American Psychiatric Association, 1976, p. 11). First, this decision might have been affected by personal interest as the many task force members had one or more financial ties to pharmaceutical companies (Pilecki, Clegg, & McKay, 2011). As noted by Pilecki et al. since the psychoanalytic therapy refers to underlying conflicts which necessitate relatively long intervention, “There was a desire to remove neurosis from diagnostic terminology and focus instead on descriptions of severe pathology that were more rare and justifiable in terms of reimbursement” (p. 196). Second, although the DSM claimed to be neutral, in reality, medical models implicitly determined the diagnostic categories (Burstow, 2005; Follette & Houts, 1996). As claimed by Pilecki et al. (2011)

the DSM “has not provided a neutral collection of observation-based syndromes and it seems naïve to have ever expected such an outcome” (p. 199). Third, although the DSM’s statement that psychoanalysis did not provide operational criteria is true, there was not scientific justification to remove this concept. The DSM’s task force arrived to the conclusion that neurosis has no common characteristic because they searched for only one common diagnostic criterion. However, much research and clinical data that are reviewed elsewhere (Rofé, 2000; Rofé, 2015), indicate that neurosis is a multi-dimensional concept, which shares five major diagnostic criteria. All the five characteristics must be present in order to classify a certain behavior as neurosis. Some of this evidence is reviewed below.

(1) Impact on Attention and Daily Activities: Research shows that the neurotic symptom has a powerful distractive value, as it becomes the focus of the individual’s attention due to its significant time-consuming and deleterious effects on daily activities and quality of life. For example, 49% of eating disorder patients spend more than three hours each day on their eating disorder rituals, and 16% spend more than 8 hours (Sunday, Halmi, & Einhorn, 1995). Similarly, panic disorder also interferes with daily life and activities and results in a significant reduction in quality of life (Carpiniello et al., 2002; Hoffman & Mattis, 2000; Perugi et al., 1998; Welkowitz et al., 2004). Similar effects were observed with bizarre phobias, such as train phobia (Leonard, 1927), chocolate phobia (Rachman & Seligman, 1976), rat germ phobia (Brandt & Mackenzie, 1987) and insect phobia (Jacobs & Nadel, 1985). In contrast, deviant behaviors other than neurosis, such as specific/simple phobia, do not necessarily preoccupy attention and disrupt one’s daily activities. As noted by Stanley and Beidel (1993), individuals with simple phobias can arrange their lives so that their symptoms do not interfere with their daily functioning.

(2) Spontaneous Mode of Onset: The onset of neurotic disorders is in the absence of a contingent event that exclusively associated with the deviant behavior or can account for its development. Bizarre phobias, such as train phobia (Leonard, 1927), chocolate phobia (Rachman & Seligman, 1976), and insect phobia (Jacobs & Nadel, 1985) fulfill this criterion, as their onset occurs in the absence of a preceding frightening event (e.g., traumatic experiences with trains) that could account for the dramatic behavioral change. Similarly, conversion disorder (Blanchard & Hersen, 1976; Jones, 1980) and a variety of OCD bizarre ritual behaviors (Fenichel, 1946, p. 271; Neale, Oltmanns, & Davison, 1982; Rachman & Hodgson, 1980, p. 65; Samuels et al., 2002) occur in the absence of a contingent event that is uniquely associated with these behavioral changes. The absence of an exclusive preceding event that can account for the development of neurosis is also illustrated in Rachman’s (1978) puzzling question regarding the onset of agoraphobia: “One needs to know... why do they acquire [the fear] when on hundreds or thousands of previous exposures to the same set of stimuli, they remained unaffected?” (p. 196).

Although stress must precede the development of neurotic disorders and seems to be a necessary condition for this behavioral change, this factor alone cannot compel the development of a specific neurotic symptom. First, stress is an insufficient factor, since the same unbearable stress may lead to a variety of deviant behaviors other than neuroses, such as depression or suicide. Second, stress is not exclusively associated with a specific neurosis, as it may cause a variety of neurotic disorders. For example, intolerable levels of stress resulting from marital problems or other family conflicts may precede the development of obsessive ruminations (McAndrew, 1989), dissociative fugue (Masserman, 1946), or various types of conversion disorder (Brady & Lind, 1961; Blanchard & Hersen, 1976).

Rival theoretical camps often report findings that seemingly indicate that a certain event is associated with and constitute the cause of a given behavioral disorder. Such evidence, however, violates the criterion of spontaneous onset only if the specific event has been unequivocally proven a sufficient condition for the development of a specific disorder or exclusively associated with this disorder. For example, the etiology of panic disorder and agoraphobia is still disputable among traditional theories of psychopathology, such as cognitive (Clark, 1986, 1988), medical (Uhlenhuth, Leon, & Matuzas, 2006), and psychodynamic (De Poderoso, Julian, & Linetzky, 2005) theories.

(3) Unawareness: Patients displaying neurotic behaviors are oblivious to the underlying causes of their dramatic behavioral changes. Although patients may provide rational explanations for their symptoms, such as anorexics attributing their behavior to being overweight (Bruch, 1973) and compulsive cleaners to biological vulnerability to infection (Rachman & Hodgson, 1980), these do not reflect genuine awareness, as they are incompatible with reality. This criterion is undoubtedly one of the fundamental features of all neurotic disorders, and the one that motivated psychoanalysts to assume the existence of the unconscious. Examples of lack of awareness can be seen in virtually all cases of neurotic disorders, such as hysterical blindness (Brady & Lind, 1961), OCD (Horowitz, 2004, pp. 169-186; McAndrew, 1989; Neale et al., 1982) and anorexia nervosa (Bruch, 1978) and

panic disorder (Jacobs & Nadel, 1985; Leonard, 1927). Unawareness resulting from normal forgetting processes would not fulfill this criterion, such as when adults are questioned about their specific childhood phobias (McNally & Steketee, 1985).

(4) Rarity: All forms of neurotic disorders have low prevalence, ranging between 1%-3.5% (American Psychiatric Association, 2000).

(5) Social Judgment: Since neurotic behaviors appear irrational, these behaviors were stigmatized. Although this criterion has received little empirical attention, a number of investigators acknowledge the importance of this factor in the diagnosis of abnormality. As noted by Carson, Butcher, and Coleman (1988), “Almost by definition, however, abnormal behavior is behavior that is *unintelligible* to the vast majority of persons observing it” (p. 17). Similarly, Bandura (1969) stated:

The designation of behavior as pathological thus involves social judgments that are influenced by, among other factors, the normative standards of persons making the judgments... Psychopathology is characteristically inferred from the degree of deviance from the social norms that define how persons are expected to behave at different times and places. Consequently, the *appropriateness* of symbolic, affective, or social responses to given situations constitutes one major criterion in labeling “symptomatic” behavior (p. 3).

Thus, given APA’s statement that “classification should be based on shared phenomenological characteristics” (American Psychiatric Association, 1976, p. 11) and evidence indicating that neurosis share five common characteristics, neurosis need to be reinstate as a diagnostic category.

3.4 New Concept of Repression

RCTM agrees with Freud (1914) that repression is the key for understanding neurosis. However, RCTM preserves only the essence of this concept, defined by Freud (1915) as “Turning something away and keeping it at a distance, from the conscious” (p. 147). Moreover, Erdelyi and Goldberg’s (1979) claim that the unconscious was not a critical theme in Freud’s original conception of this notion is consistent with RCTM’s theoretical position. “In his very earliest writings (Freud, 1894/1962) repression was treated as a potentially conscious mechanism... at least at times, repression is a conscious, deliberate act” (p. 365; Erdelyi, 2006). Accordingly, RCTM defines repression as a *conscious coping mechanism that deliberately eliminates threatening stimuli from attention through the employment of distractive maneuvers*. This new conceptualization is consistent with the bulk of experimental studies that found that repression is nothing more than conscious distraction (see review by Holmes, 1974, 1990).

When people encounter stress exceeding their normal coping resources, some people intuitively feel that a certain neurotic behaviors are the best way and the least costly response to cope with their stressors. The major psychological benefit of these behaviors is repression: Stress-related thoughts become inaccessible because one’s attentions is heavily preoccupied. Thus, contrary to psychoanalysis, madness arises in response to current stress rather than historical trauma. Most importantly, *repression is the consequence, and not the cause, of neurosis*.

3.5 Choice of Symptoms

The choice of a specific symptom is determined by the individual’s **need to exercise control** over the stressor (e.g., soldiers during WWII developed conversion symptoms since they increased their ability to escape from combat stress, see Ironside & Batchelor, 1945; Mucha & Reinhardt, 1970), **the availability** of this behavior through various channels of information (e.g., media, peer group, and family; see Spanos, 1996; Spanos, Weekes, & Bertrand, 1985), and **cost-benefit analysis** (e.g., men are less likely to choose neurotic symptoms due to damage to their work abilities and social embarrassment).

3.6 A Clinical Example

One example demonstrating RCTN’s theoretical position is the autobiographical account of Leonard (1927), a poet, writer, and a professor at the University of Wisconsin. At the age of 36, he experienced a sudden panic attack when he saw a train while standing alone on a cliff overlooking a quiet lake. As described below, the onset of his symptom was irrational and ridiculous even to him:

Then on the tracks from behind ... comes a freight-train, blowing its whistle. Instantaneously diffused premonitions become acute panic. The cabin of that locomotive feels right over my head, as if about to engulf me ... The train feels as if it were about to rush over me... I race back and forth on the embankment. I say to myself (and aloud): “it is half a mile across the lake—it can’t touch you, it can’t; it can’t run you down... I rush back and forth on the bluffs: My God, won’t that train go, my God, won’t that train go away!” I smash a wooden box to pieces, board by board, against my knee to occupy myself against panic... (pp. 304-307)

The panic had such a dramatic impact on Leonard that when he was taken to his parents' home he declared, "Father and mother, this looks like the end. I guess I am dying" (p. 308). At this time, Leonard could not go a hundred feet beyond his parents' home. Later, when the university authorities threatened to dismiss him due to his long absence, Leonard moved with his parents to an apartment across the street from the university (pp. 328-329). In his self-hypnosis, conducted more than ten years after the onset of his symptoms, Leonard recalled a childhood trauma at the age of two where he was almost run down by a thundering train. Both Leonard, who had extensive knowledge of psychoanalytic theory, and advocates of psychoanalysis (Allport, 1929; Culler, 1930; Taylor & Culler, 1929, 1931; White & Watt, 1981), attributed Leonard's symptoms to repression of the train trauma.

While it would be difficult to explain this case by conditioning or cognitive theories, it is also difficult to accept the psychoanalytic interpretation. First, a large amount of studies shows that people remember rather than forget their childhood and current trauma (Piper et al, 2008; Rofé, 2008). Second, the train episode was recorded in a diary Leonard's mother started keeping for him three days after the incident. She noted that Leonard was "Talking a great deal about it ever since" (p. 16), which means that he remembered the event, rather than repressed it. Third, Leonard continued to travel by trains afterward, so even if a fear originally developed, it would have been extinguished through repeated exposure to locomotives. Leonard himself testified that he "Developed no specialized fear of locomotive... on the contrary, trains became a childish passion" (p. 24). Fourth, even if Leonard was subconsciously affected by the train episode, it is not fully clear why he developed agoraphobia rather than train phobia.

RCTN claims that Leonard deliberately adopted his panic-agoraphobic symptoms as a coping mechanism in response to an intolerable level of stress. The panic attack occurred at the age of 36, soon after his wife, the daughter of a highly respected family, committed suicide. The community regarded Leonard as demanding and self-centered and almost unanimously blamed him for her death. Leonard contemplated a great deal about the death of his wife, and it seems that the major function of his neurotic disorder was repression. Instead of being preoccupied with stress-related thoughts, such as his self-criticism of his role in his wife's death, he chose to make himself the victim of a serious disease. The intensive preoccupation with his symptoms enabled him to block the accessibility of stress-related thoughts and relieve his intolerable level of depression. The symptom meets Leonard's controllability needs. It distanced him from the community who ostracized him, and prevented him from resuming his work at the university, a preventative measure to avoid social rejection also by his colleagues. Later, when the university authorities threatened to dismiss him due to his long absence--when the controllability demands changed--he moved closer to the university to resume his teaching obligations. Leonard also adapted his agoraphobic symptoms in accordance to his unique personal needs; he was able to ride his bicycle and purchase merchandise downtown (pp. 343, 346) and even travel twice to Chicago and once to New York to meet his fiancé. He rationalized this violation of his "illness" by saying he could control his phobia because "the speed of the bicycle magnified my beat..." (p. 343).

Leonard's symptoms also fulfil the RCTN's diagnostic criteria. His agoraphobic symptoms severely disrupted his daily activities (1); the onset was spontaneous, in the absence of any event that could unquestionably account for the radical change in his behavior (2); he was unaware of the underlying cause for his behavioral change (3); his symptoms were extremely rare (4); Leonard diagnosed himself, immediately after the onset of his symptom, as suffering from illness. "I know I am in a critical condition" (p. 308) (5).

3.7 Unawareness

Although the understanding of patients' unawareness of the underlying causes for a dramatic change in their behavior is a prerequisite for the understanding neurosis, it is difficult to accept Freud's resolution for this matter for reasons specified previously. Instead, RCTN suggests that neurotic symptoms are conscious behaviors and that patients become unaware of their Knowledge of Self-Involvement (KSI) through a number of cognitive processes. Before specifying these processes, it is important to indicate that decisions made analytically, where the individual examines various options before reaching decision, and intuitively, made with little or no conscious awareness. Intuitive decision is "often experienced in the form of feelings (not words)..." (Hogarth, 2001, p. 9). "A defining property of intuitive thoughts is that they come to mind spontaneously, like percepts" (Kahneman, 2003, p. 699). Intuitive decisions are often made during stressful situations "when an in-depth analysis is not possible and the decision-maker must move quickly to a plausible solution" (Sayegh, Anthony, & Perrewe, 2004, p. 183), and they are based on the individual's experiential resources stored in the long-term memory and usually are no less rational than conscious deliberate decisions (Glöckner & Witteman, 2010; Sayegh et al., 2004). This process seems to be the way by which neurotic symptoms are chosen. For example, it seems that Leonard intuitively felt that panic and agoraphobic symptoms would be the best possible coping

strategy, based on his childhood traumatic experience, wide knowledge in the psychoanalytic theory and his unique controllability demands. However, the intuitive processes lack executive abilities and it is the conscious that decided to implement the intuitive suggestion after rapid cost-benefit analysis.

The unawareness of KSI is obtained through a number of factors that disrupt the encoding of this information and memory-inhibiting mechanisms that cause the forgetting of this information immediately after the adoption of the neurotic symptoms. Soon afterwards, patients develop self-deceptive belief that rationalize the redisplay of the symptom and prevent the retrieval of the KSI. Space limitations do not allow specifying these psychological processes, which are discussed in details elsewhere, also in reference to Leonard's case (Rofé, 2000, 2010, 2015; Rofé & Rofé, 2013). Although RCTN's unawareness model lacks supportive evidence of its own, it has clear advantage over Freud's unconscious. It integrate a large amount of research and clinical data, and most importantly this is the only theory that succeeded to integrate of all therapeutic methods pertaining to neurosis into one theoretical framework (Rofé, 2000, 2010; Rofé, 2015).

In conclusion, this article showed the traditional theories of fear and phobia cannot adequately explain these behaviors. A necessary condition for the understanding these behaviors is to make a sharp distinction between simple and bizarre phobia. While the Pavlovian paradigm seems to be the best explanation of simple phobia, the revised version of psychoanalytic theory, termed, the Rational-Choice Theory of Neurosis, appear to be the most suitable explanation of bizarre phobia.

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