1. Introduction

In order to better understand complex natural phenomena, it is useful to recreate the phenomenon in question under controlled laboratory conditions. Although laboratory models may never be the real thing, they can provide scientists with new ideas and stimulate new research.

For this reason, several efforts were made to reproduce acute panic attacks. Typically, groups of panic patients were compared to controls with respect to their reactions to various, supposedly anxiogenic, pharmacological interventions. While biologically-oriented scientists focused their attention on non-situational panic, laboratory models of phobic anxiety were also elaborated upon. The latter, however, was generally carried out by stimulus-response psychologists and their models of phobia did not result from pharmacological interventions but rather from manipulating stimulus contingencies.

Although this chapter is devoted to panic, we will first briefly discuss models of phobias. This is not only to demonstrate, in a general sense, the power of experimental psychopathology, but also to show that experimental analysis of phobias may be directly relevant to understanding natural or artificial panic.

In discussing models of phobias and panic, empirical data will be presented that underscores the earlier statement that models are not substitutes for natural phenomena. Although changing some stimulus conditions may produce phobic behavior, it would be wrong to assume that biological factors are of little or no importance. Similarly, it will be argued that, while panic symptoms can be induced by certain pharmacological interventions, it would be wrong to conclude that panic disorder “is” a biological disorder to which behavioral data are of limited or no significance.

This paper touches upon such classical dichotomies as biological vs. psychological and endogenous vs. exogenous. Rather than rephrasing theoretical arguments about the unfruitfulness of such dichotomies, it will be argued that unequivocal data from the very field of experimental psychopathology of anxiety empirically shows that simple “endogenous vs. exogenous” etc. splits should be abandoned when it comes to explaining the complexities of human psychopathology.
2. Phobias

2.1. Some Behavioral Models

For the present purposes, a behavioral model of phobia is defined as any set of manipulations of stimulus contingencies that induce, maintain, and/or reverse phobic behavior.

From the voluminous work carried out in this area (see, for example, Abramson and Seligman 1977), a suitable example was selected, namely, Baum’s experiments on rats (Baum 1970, 1972a–b, 1973, 1976). Cages were constructed that contained a grid floor that could be used to deliver shocks to the rats and a safety ledge to which the animals could jump in order to avoid contact with the conditioned stimulus (CS) (the grid floor) and the unconditioned stimulus (the electrical shock). Delivering shocks a few seconds after the rats were placed on the grid floor quickly produced avoidance behavior, i.e. jumping to the ledge. Reminiscent of the irrationality of phobic fears was the persistence of avoidance behavior even when shocks were no longer given. This would seem to suggest that, in both men and rats, avoidance of an anxiety-provoking situation reduces anxiety and, moreover, that anxiety reduction reinforces avoidance behavior, even in situations where it is no longer even necessary. The resemblance to human phobias becomes even more apparent where treatment is concerned. Removal of the safety ledge prevented any effective conditioned stimulus avoidance and exposed the rats to the “fearful” stimuli. Such exposure in vivo resulted primarily in restlessness and in futile efforts to jump to the removed ledge but avoidance efforts extinguish. Strong shocks took approximately 30 minutes to extinguish avoidance; mild shocks, only 3 to 5 minutes. Extinction was facilitated (Baum 1973) by shortening the time interval between CS exposures (cf. massed practice), by having rats observing non-fearing fellows or rats undergoing the same procedure (cf. modelling procedures) and by forcing the animals to explore the grid floor cage (cf. concentrating on the objective environment as a coping skill to handle agoraphobic panic).

Baum’s work thus illustrates how changing environmental cues may produce, maintain and/or reverse “phobic” avoidance in rats. The last two points are particularly interesting, as there is such an obvious resemblance to human phobias; indeed it is likely that anxiety-reduction resulting from avoidance is a powerful maintaining factor, both in experimental phobias of rats and in clinical phobias and compulsions (Rachman and Hedgson 1980). As for reversing phobic avoidance, it has been demonstrated time and again that prolonged exposure in vivo is an effective treatment approach to situational anxiety (Emmelkamp 1982).

Does this prove that biological determinants play no role in experimental or clinical phobias?

2.2. Biological Aspects of Behavioral Models of Phobias

Rhesus monkeys reared in the laboratory do not appear to get upset when confronted with real or toy snakes; those reared in the wild, however, do. They show clear signs of emotional disturbance and display persistent avoidance behavior (Mineka et al. 1980). Previously non-snake-fearing, laboratory-raised monkeys were made to observe the reaction of their parents, who had been raised in the wild, to simultaneously presented neutral and snake stimuli. One observation of the snake-fearing parental model was all that was needed to induce “fear” and avoidance in the laboratory-raised offspring (Mineka et al. 1983).

One could conclude then that snake phobias in rhesus monkeys are sometimes acquired vicariously; they may also be difficult to extinguish. An attempt was made at reducing avoidance behavior by enabling hungry monkeys to take food only from a box containing a toy snake. “Anxious” behavior, however, persisted across the seven flooding sessions and, at follow-up, 6 months after this exposure therapy, monkeys had relapsed (Mineka and Keir 1983).