## **Classical Conditioning of Pain-Elicited Aggression**

Abstract. Unconditioned aggression between paired animals in response to electric shock has been previously demonstrated. In this study, with the use of classical Pavlovian conditioning procedures, aggression was produced between paired rats as a response to a tone stimulus.

Paired animals fight when subjected to painful stimulation (1, 2). This behavior occurs within many species and between members of different species (3). Despite only minimum success in earlier attempts to develop conditioned fighting by pairing painful stimuli, such as electric shocks, with neutral stimuli (4), we have successfully conditioned the pain-aggression response in rats.

Eighteen young, adult, male Long-Evans hooded rats were subjects. A plastic experimental chamber clear measuring  $38 \times 39 \times 36$  cm had, as its floor, 23 rods, 3.18 mm in diameter and spaced 11.11 mm apart. The chamber was housed in a larger sound-attenuated chest and had a clear observation window. Electric shocks and electrically generated tones of 60 db at 1320 cy/ sec were programmed by apparatus in a separate room. Initially it was established that the sound stimulus would not elicit fighting through 1000 repetitions, nor would a pseudoconditioned response occur to the sound stimulus when it was first presented immediately after 1000 electric shocks given at 10second intervals. We paired the neutral auditory stimulus, hereafter referred to as the conditioned stimulus (CS), and the painful electrical unconditioned

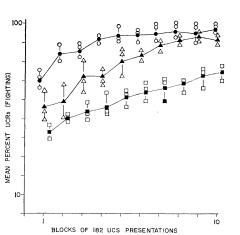


Fig. 1. Individual (open symbols) and mean (solid symbols) percentages of UCR's (fighting) are shown for each block of 182 presentations of UCS at each of three different shock intensities. UCS, unconditioned stimulus; UCR, unconditioned response. Circles, 2.0 ma; triangles, 2.5 ma; and squares, 3.0 ma. stimulus (UCS). The nine pairs of animals were given 2000 pairings of the tone with the shock, one of three shock intensities being delivered to each of three different groups of three pairs of animals. Duration of the CS was 1.0 second. One-half second after onset of the CS this stimulus was joined by the UCS, both terminating simultaneously after 0.5 second. The onset-to-onset interval between trials with the CS was 10 seconds. Each 11th presentation was the CS alone, this being a test for the development of the conditioned aggressive response. Fighting responses were recorded by an observer who scored any striking or biting movement made by either animal toward the other. Usually such responses were made from a stereotyped fighting posture on the hind legs, which the animals typically maintained through most of each session.

Rates of pain-elicited fighting and the shock intensities bore an inverse relation to one another (Fig. 1). A nonparametric analysis of variance (Kruskal-Wallis; H = 7.19, P < .005) indicated highly significant differences be-

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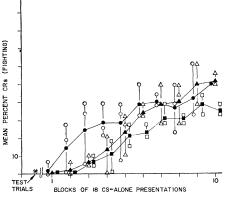


Fig. 2. After initial test trials demonstrated that no fighting was developed by the tone alone and that no pseudoconditioning developed (see arrow), tone (CS) plus shock (UCS) trials were instigated. Individual (open symbols) and mean (solid symbols) percentages of conditioned fighting are shown for each block of 18 presentations of CS alone for each of the three shock intensities used. CS, conditioned stimulus; CR, conditioned response. Circles, 2.0 ma; triangles, 2.5 ma; and squares, 3.0 ma.

tween total fight responses to the different shock intensities.

The complex nature of pain-elicited fighting is reflected in the change, over 2000 trials, from an initially low response rate of 53 percent to an average of 88 percent. This change appears to be largely the result of the extinction of competing responses. As trials progressed, the escape behavior, having no effect upon stimulus events, gradually became less frequent. Although fighting also had no effect upon stimulus events, it increased substantially as trials continued. The fact that unconditioned fighting to 2-ma shock was consistently higher is commensurate with earlier findings which show that 2 ma is the optimum shock intensity for producing pain-aggression in rats (2).

Figure 2 shows that the same relation exists between intensity of shock and rate of conditioned fighting as between intensity of shock and rate of painelicited fighting. This is consistent with the common observation (5) that optimum intensities of the UCS for eliciting unconditioned responses are also optimum for the development of conditioned responses. Comparison of Figs. 1 and 2 shows that less overlap occurred at each intensity of the UCS among the unconditioned responses; still, nonparametric analysis of variance (Kruskal-Wallis; H = 5.67, P < .05) of shock intensity and group differences in total conditioned fight responses indicated differences beyond chance expectation.

In addition, animals were observed to fight at a higher rate when they were close together and facing one another at the time of the onset of shock, in accord with earlier observations (2). It was further observed that, at the onset of shock, if one animal was in a fighting posture, there was considerably greater likelihood that his partner would attack him than if he had all four feet on the grid floor.

The major significance of the study is knowledge of the conditioning phenomenon itself. The finding that organisms respond aggressively to what outwardly may appear to be neutral stimuli, in accordance with their past experiences of pain, suggests a possible explanation for some cases of "apparently unprovoked" aggression.

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## **Inhibitory Centers in Sexual** Behavior in the Male Rat

Abstract. Small lesions placed near the diencephalic, mesencephalic junction, in either the lateral or medial mammillary region, resulted in an increase of copulatory behavior. This increase was expressed both by increased numbers of copulation plugs formed per 14-day interval and by increased percentage of days on which copulation occurred. Inhibitory structures thus form an essential part of the circuitry involved in mediation of sex behavior in the male.

A number of reports indicate that deficiencies in mating behavior occur following destruction of localized regions of neurons within the diencephalon. There is general agreement that lesions in the preoptic or anterior hypothalamic area result in a decrease or abolition of overt sexual response in the male (1, 2). More posterior lesions involving the posterior hypothalamus and mammillary bodies have produced findings of loss of sex behavior (2) and findings of no change in sexual performance (3).

Reexamination of the problem of specific areas involved in overt sexual behavior was essential for two reasons. (i) The reported observations are in disagreement. (ii) Various studies in related "control systems" operating in the hypothalamus indicate that the results obtained could be a function of the metal material which was used in the electrode employed in making the lesion (4).

We observed the following effects presumably due to the destruction of discrete areas within the nervous system. Lesions in the mammillary region placed medially or laterally resulted in an increase in production of copulation plugs and in days on which copulation occurred (nine animals). Slightly smaller lesions in the same area (nine animals) resulted in an unchanged level of copulation following lesion. After lesions in the middle hypothalamus dorsal to the arcuate nucleus a gradual decline in copulatory level occurred. This decline in copulatory level paralleled a decline found in the control, unlesioned animals.

Rats are normally reared, from the time of weaning at 21 days of age, in groups segregated by sex. When a large mature male was caged with a single stimulus female for a period of 8 weeks, the peak of sexual activity usually occurred during the first 14-day interval. This was true when measured both as number of copulations per 14day interval and the percentage of days on which copulation occurred. During the following 6-week period a gradual decline in total sexual activity was observed. This finding suggests that an adaptation occurs to the situation-that is, the constant presence of a stimulus female is no longer novel, so sex activity declines. Presumably if an experiment were extended over a sufficiently long time interval a steady state would eventually be established, which would represent the basic sex drive for a rat of a particular strain and age when caged with the same partner, who was in a constantly receptive state. Therefore it is important to realize that the control condition is not a steady state (Table 1A). A parallel decline in copulatory activity occurred following lesions in the dorsal area of the middle hypothalamus (Table 1B). The placement of lesions dorsal to the mammillary complex which extended into the habenula and overlying cortex also resulted in a similar decline in copulation (Table 1C). These observations are all in marked contrast to the mammillary lesions which resulted in a steady state or increasing levels of copulatory output (Table 1D). This strongly suggests that the dorsal medial hypothalamus is not important for the mediation or maintenance of copulatory behavior. Similarly, those structures dorsal to the mammillary area, including structures in and around the habenula, appear to be unessential for sexual behavior in the male; in contrast, the diencephalic mesencephalic juncture at the level of the mammillary bodies does contain structures which appear to exert an inhibitory control on the level of copulatory behavior. The overt sexual behavior expressed must therefore depend on the balance of activity between an integrative and activational (facilitory) system likely localized in the preoptic and anterior hypothalamic regions and a regulatory (inhibitory) system located in the region of the mammillary bodies.

Large males of the CFE strain (5) with a body weight of 370 to 410 g at the beginning of the experiment were used. The actual subjects were chosen by the criterion of having shown a minimum of 4 nights of mating during the original 14-day observation period. All animals were kept one pair per cage; thus the male had free access to a receptive female at all times. Receptivity of the female was assured by subcutaneous placement of two blobs

Table 1. Copulatory records for males, showing successive 14-day totals for copulation plugs and, in parentheses, percentages of nights on which copulation ocurred.

| trol,                                  | Days   | Days   | Days   | Days  |
|--|--|--|--|---|
| Days                                   | 1-14   | 15-28  | 29-42  | 43-57   |
|  |  | ······   |  |   |
|  | A. Contro  | ol males (n  | ıo lesion)   |   |
|  | 9(29)  | 5(29)  | 5(21)  | 7(21)   |
|  |  |  |  | 6(23)   |
|  |  |  |  | 10(38)  |
|  |  |  |  | 4(31)   |
|  |  |  |  | 12(54)  |
|  |  |  |  | 2(8)  |
|  |  |  |  | 2(8)  |
|  | 17(57)   | 5(21)  | 2(14)  | 9(36)   |
| . Mai                                  | les with le  | esions in m  | uiddle hvvo  | thalamus  |
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|  |  |  |  |   |
| 64)                                    | 21(93)   | 8(36)  | 6(36)  |   |
| С.                                     |  |  |  | and   |
|  | ov   | erlying cor  | tex  |   |
| 50)                                    | 8(43)  | 3(14)  | 2(14)  |   |
|  | 3(14)  | 7(36)  | 15(50)   |   |
| 50)                                    | 12(43)   | 16(43)   |  |   |
|  | 8(43)  | 9(36)  |  |   |
| 43)                                    | 7(36)  |  |  |   |
|  |  |  |  |   |
|  |  |  |  |   |
|  |  |  |  |   |
|  |  |  |  |   |
|  | 15(57)   | 13(50)   | 5(21)  |   |
| I                                      | D. Males v   | vith mamm  | illary lesio   | ns  |
|  |  |  |  |   |
| 29)                                    | 3(21)  | 11(64)   | 16(64)   |   |
|  | 12(50)   | 11(50)   | 26(86)   |   |
| 29)                                    |  |  |  |   |
| 29)<br>36)<br>36)<br>36)               | 12(50)   | 11(50)   | 26(86)   |   |
| 29)<br>36)<br>36)                      | 12(50)<br>19(79)   | 11(50)<br>25(86)   | 26(86)<br>25(93)   |   |
| 29)<br>36)<br>36)<br>36)<br>29)<br>29) | 12(50)<br>19(79)<br>18(43)   | 11(50)<br>25(86)<br>12(50)   | 26(86)<br>25(93)<br>10(43)   |   |
| 29)<br>36)<br>36)<br>36)<br>29)        | 12(50)<br>19(79)<br>18(43)<br>12(43)                               | 11(50)<br>25(86)<br>12(50)<br>9(43)  | 26(86)<br>25(93)<br>10(43)<br>12(36)   |   |
| 29)<br>36)<br>36)<br>36)<br>29)<br>29) | 12(50)<br>19(79)<br>18(43)<br>12(43)<br>9(50)                      | 11(50)<br>25(86)<br>12(50)<br>9(43)<br>10(43)  | 26(86)<br>25(93)<br>10(43)<br>12(36)<br>11(46)   |   |
|  | 36)<br>50)<br>71)<br>36)<br>29)<br>43)<br>57)<br>50)<br>29)<br>64) | 9(29)<br>11(36)<br>17(57)<br>7(36)<br>12(43)<br>13(64)<br>10(36)<br>17(57)<br><i>Males with le</i><br>36) 10(36)<br>50) 9(29)<br>7(1) 7(43)<br>36) 6(21)<br>29) 7(29)<br>43) 15(57)<br>57) 6(21)<br>50) 7(43)<br>36) 6(21)<br>29) 7(29)<br>43) 15(57)<br>57) 6(21)<br>50) 7(43)<br>36) 6(21)<br>29) 7(29)<br>43) 15(57)<br><i>C. Males with</i><br>ov<br>50) 8(43)<br>29) 3(14)<br>50) 12(43)<br>64) 8(43)<br>54) 7(29)<br>46) 4(14)<br>39) 13(36)<br>46) 15(57) | A. Control males (r        9(29)      5(29)        11(36)      8(29)        17(57)      17(50)        7(36)      8(43)        12(43)      13(43)        13(64)      7(43)        10(36)      7(21)        17(57)      5(21)        b. Males with lesions in m        36)      10(36)      5(21)        50)      9(29)      3(7)        71)      7(43)      8(29)        36)      6(21)      7(36)        29)      7(29)      4(21)        43)      15(57)      9(43)        57)      6(21)      7(36)        50)      7(43)      10(50)        29)      0(0)      3(7)        64)      21(93)      8(36)        C. Males with lesions in overlying cor      50)      8(43)        50)      12(43)      16(43)        64)      21(93)      8(36)        C. Males with lesions in overlying cor      50)      12(43)        50)      12(43)      16(43)        64) | A. Control males (no lesion)        9(29)      5(29)      5(21)        11(36)      8(29)      10(29)        17(57)      17(50)      14(36)        7(36)      8(43)      6(29)        13(64)      7(43)      5(14)        10(36)      7(21)      4(7)        17(57)      5(21)      2(14)        10(36)      7(21)      4(7)        17(57)      5(21)      2(14)        *      Males with lesions in middle hypo        36)      10(36)      5(21)      4(27)        50)      9(29)      3(7)      4(18)        71)      7(43)      8(29)      8(36)        36)      6(21)      7(36)      8(36)        29)      7(29)      4(21)      8(18)        57)      6(21)      5(21)      7(36)        50)      7(43)      10(50)      5(36)        29)      0(0)      3(7)      0(0)        64)      21(93)      8(36)      6(36)        C. Males with lesions in habenula overlying cortex      50) |