Pharmacology Biochemistry & Behavior, Vol. 21, pp. 5–7, 1984. © Ankho International Inc. Printed in the U.S.A. 0091-3057/84 $3.00 + .00

Dissociation of Prey Killing and Prey Eating by Naloxone in the Rat[1]

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Received 3 October 1983

WALSH, M. L., R. WHITE AND D. J. ALBERT. *Dissociation of prey killing and prey eating by naloxone in the rat.* PHARMACOL BIOCHEM BEHAV **21**(1) 5–7, 1984.—Mouse killing rats matched for killing latency and prey eating were injected (IP) with 0.5, 2.0, or 5.0 mg/kg naloxone or 0.9% saline. Naloxone did not significantly inhibit prey killing or alter prey killing latency at any dose but did reduce prey eating by 50% at the two higher doses. The dissociation of prey killing and prey eating by naloxone is consistent with other evidence that these two behaviors are separate components of predation in rats.

Mouse killing Naloxone Predation

NALOXONE appears to alter agonistic behavior in some circumstances but not others. It suppresses aggression towards an intruder by isolated mice [12,18]. However, in rats there is neither a suppression of aggression toward an intruder [15,20] nor a decrease in aggressiveness toward rats introduced as intruders into the home cage [19]. Naloxone increases defensive behavior of both rats [7] and mice [17] tested in a shock-induced fighting situation, but does not alter the frequency of mouse killing by rats [10]. This variety of results obtained with different forms of agonistic behavior is not necessarily contradictory since there is evidence that defensiveness, social aggression, and predatory killing are mediated by separate neural systems [1].

A surprising aspect of the effects of naloxone on agonistic behavior is its failure to suppress mouse killing [10]. This is unexpected since naloxone has been consistently found to inhibit eating and drinking in laboratory rats [6, 8, 9, 10, 11, 13, 21]. One of the reasons that mouse killing is considered predatory is that eating quickly follows the kill [1, 3, 16]. The effectiveness of naloxone in suppressing feeding but not killing suggests that this drug may dissociate the killing and eating components of predatory behavior.

The purpose of the present experiment was to further investigate naloxone's effect on mouse killing by using a range of dosages and by recording latency to kill as well as whether or not a kill occurred. In addition we recorded the amount of the prey consumed during a 2 hr period after the kill to determine whether eating of the prey is also attenuated.

METHOD

The subjects were 44 male hooded rats from Charles River, Canada, weighing 350 to 450 g. They were all animals that had been found to be mouse killing rats in the course of other experiments. Most of these rats had killed only one mouse during a mouse killing screening test. During the course of this experiment, they were housed individually and had free access to food and water. The colony area was on a 12/12 hr light/dark cycle and testing was done during the light period.

Behavioral Testing

On Day 1, all animals had a weighed adult albino mouse (about 30 g) dropped into their living cage. The latency to kill by each animal was recorded. The dead mouse was left in the cage for 2 hr following the kill. At the end of this time, the remains were weighed and the amount eaten calculated.

Those animals that killed within 2 min were retained in the experiment. This selection procedure was used in order to maintain a high level of homogeneity in the subject population. The animals were then divided into four groups which were approximately balanced for latency to kill and amount of the prey eaten.

On Day 5, the animals in the four groups were each given one of the following treatments 20 min prior to a second mouse killing test. One group was injected with 5.0 mg/kg naloxone hydrochloride (Endo Pharmaceuticals). A second group was injected with 2.0 mg/kg of naloxone, while a third group received 0.5 mg/kg of naloxone. The fourth group was injected with sterile, 0.9% saline, 1 ml/kg. All injections were given intraperitoneally. The concentration of the solutions was adjusted so that the animals in each group were injected with approximately the same volume of solution. The mouse killing and prey eating tests carried out on Day 5 were identical to those done on Day 1, with one exception. The maximum interval allowed for killing was 10 min instead of 2

[1]Supported by a grant from the National Science and Engineering Research Council of Canada. Naloxone was generously supplied by Endo Pharmaceuticals.

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TABLE 1

THE EFFECT OF VARIOUS DOSES OF NALOXONE ON MEAN LATENCY TO KILL (SEC)

Naloxone mg/kg	N	Pretest Day 1	Drug Day 5	Posttest Day 9
0.5	7	64 (29)	23 (11)	10 (2)
2.0	8	51 (16)	25 (14)	98 (69)
5.0	13	33 (12)	14 (5)	56 (33)
Saline	12	44 (26)	8 (3)	19 (14)

SEM in parentheses.

min in order to allow for a drug effect on latency to kill.

On Day 9, a final mouse killing and prey eating test was carried out. The testing procedure was identical to that on Day 5.

RESULTS AND DISCUSSION

Naloxone injections did not suppress mouse killing (Chi Square Contingency Test, $\chi^2=6.4$, $p>0.05$). Of all the animals tested, 3/11 injected with 2.0 mg/kg and 1/14 injected with 5.0 mg/kg of naloxone did not kill within the 10 min time interval allowed on the injection day. All 7 animals injected with 0.5 mg/kg of naloxone and all 12 animals injected with saline did kill within the allotted 10 min. Since the four animals that did not kill also failed to kill within the allotted 10 min interval on the postinjection test four days later (Day 9), we inferred that their killing behavior was simply eratic and excluded their behavior from further data analysis.

Naloxone did not alter latency to kill (Table 1). A two-way analysis of variance indicated that there was no significant tendency for the saline or the various doses of naloxone to effect latency to kill differentially, $F(6,72)=1.52$, $p>0.05$. The very high latencies to kill obtained for two groups on the third test day are due to large increases in the kill latency for one animal of each group. In order to evaluate the possibility that variability in one or more groups was masking a significant drug effect, t-tests for correlated samples were done comparing each groups kill latencies for Days 1 and 5. None of the differences was significant. The analysis of variance also indicated no significant differences between groups, $F(3,36)=2.21$, $p>0.05$, or test days, $F(2,72)=2.1$, $p>0.05$. The fact that the group effect approached significance appears due to slight differences between groups which persisted over the test days; a similar tendency in the days effect appears due to the tendency of the kill latencies for all groups to decline over days.

In contrast to the absence of an effect of naloxone on mouse killing latency, its effect on prey eating was robust. There was a significant suppression of prey eating by naloxone (Fig. 1; $F(2,72)=35.2$, $p<0.01$) and a significant group × test interaction, $F(6,72)=6.7$, $p<0.01$. Individual comparisons within groups between the first and second

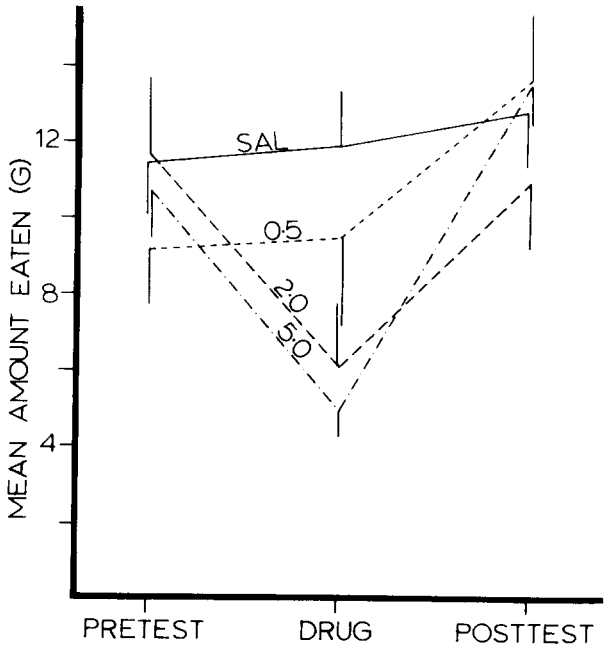

FIG. 1. Mean amount of prey eaten following each mouse kill by animals injected with either 0.9% saline or naloxone. SEM is indicated at each point.

mouse killed indicated that the suppression of eating was significant with the 2.0 and 5.0 mg/kg dose ($p<0.005$) but not the 0.5 mg/kg dose of naloxone (t-test for correlated samples).

In agreement with previous evidence [10], naloxone did not decrease the mouse killing frequency at a dose of 2.0 mg/kg. The present results demonstrate that a substantially higher dose (5.0 mg/kg) also does not suppress the killing behavior nor does it increase the latency to kill. On the other hand, in agreement with other findings, both the 2.0 and 5.0 mg/kg dose of naloxone suppressed eating [6,9]. This suppression of eating occurs in spite of the fact that prey eating is a highly motivated consummatory behavior. A rat that is sated with lab chow will still eat part or all of a prey it has just killed (Albert and Walsh, unpublished experiments). This was indicated in the present experiment by the prey eating data of the control animals (Fig. 1).

Prey killing and prey eating are known to be separable components of predatory behavior. In the rat, the most striking illustration of this separability is the observation that rats will kill available prey far in excess of what they can eat [2, 4, 5]. Also, prey killing is reinforcing as is indicated by the observation that the killing continues to occur even in the absence of an opportunity to eat the prey [1, 14, 22]. The present evidence with naloxone suggests that the neural substrates mediating prey killing and prey eating are pharmacologically distinctive.

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