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Pilocarpine, Food Deprivation, and Induction of Mouse Killing by Cats[1]

RUSSELL C. LEAF AND D. J. WNEK

Rutgers University, New Brunswick, NJ 08903

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LEAF, R. C. AND D. J. WNEK. *Pilocarpine, food deprivation, and induction of mouse killing by cats.* PHARMAC. BIOCHEM. BEHAV. 9(4) 439–444, 1978.—Effects of three treatments that induce mouse killing by rats were examined with cats. Food deprivation induced about 50% killing after 24 hr and almost 100% killing by 72 hr. Pilocarpine (at doses of 1.0 mg/kg that produced marked side-effects, and whether or not methyl atropine pretreatment blocked those side-effects) and chlordiazepoxide (at doses of 1.0–2.0 mg/kg) did not induce any killing. Pilocarpine produced a dose-related inhibition of spontaneous mouse killing (as it does in rats), but this was antagonised by methyl atropine. The side-effects of pilocarpine and chlordiazepoxide did not seem to account for their failure to induce killing. In contrast with food deprivation, the mechanisms by which pilocarpine and chlordiazepoxide induce killing in rats may not have homologs in cats.

Food deprivation Satiation Pilocarpine Chlordiazepoxide Mouse killing Aggression Predation

THE KILLING of animals of one species by those of another is common. Particular species often serve as prey for a number of predators. Mice, for example, are killed by other rodents, including rats, by various carnivores, including cats, and by animals of other orders and species, as well. Not all individuals of a given predatory species kill those of a particular prey spontaneously, however, even when the manner and pattern of killing by those who do is highly stereotyped. Despite the fact that some individuals may not kill a particular prey, most killing behaviors are usually presumed to be innately determined and species-specific [8,21]. Species-specific patterns may be present but not spontaneously evident in all individuals. If many, but not all, animals of one species typically kill those of another the nonkilling individuals may have a natural tendency to kill that is not expressed. In support of such a notion, nonkilling individuals can be induced to kill by experimental treatments that produce unusual conditions that are rare or absent in nature. The particular techniques that induce killing (e.g., extreme food deprivation, drug administration, brain lesions or stimulation) may reflect the degree to which certain neural and behavioral processes (e.g., those that control feeding) naturally control tendencies to kill.

Further, the similarities between or differences among treatments that induce killing in animals of different species may reflect the degree to which particular neural and behavioral processes are homologous. Relatively little experimental effort has been devoted to comparative studies of induction of killing. Comparative studies are desirable, however, in order to evaluate the hypothesis that homologous mechanisms that control patterns of killing are common in nature [14, 20, 21].

Deliberate induction of killing by nonkilling individuals has been studied most extensively with respect to the killing of mice by rats (see [2, 3, 16, 25, 27]) for reviews of various techniques and their results [1]. Three techniques for inducing killing are particularly important for interpreting the experiments described below. Rats that do not spontaneously kill mice can be induced to do so if they are food deprived [4,30] or given drug injections of pilocarpine [39,42] or chlordiazepoxide [19]. The killing induced by these treatments is somewhat similar, at least in the patterns and locations of fatal bites, to that which occurs spontaneously. Further, after rats are induced to kill, they sometimes do so spontaneously without further food deprivation or drug treatment. These observations suggest that at least some deprivation and drug treatments may stimulate natural mechanisms that control spontaneous killing [2,27].

Many cats spontaneously kill mice in a manner which is similar to that typical of rats [4, 5, 33, 44]. Both species (and other mouse predators, as well) kill mice by forcefully biting, in the upper back and neck region, so that they break the cervical spinal cords of their victims. The similarities in the killing patterns of diverse predators may have arisen either through different, parallel, evolutionary or learning processes or they may have been caused by common, homologous, genetic mechanisms. The common features of the killing behaviors of rats, cats, and other predators tend to favor the common heredity hypothesis. Killing by diverse, often otherwise highly dissimilar, species may, therefore, be largely controlled by homologous neural systems [7,21]. This hypothesis about killing is similar to currently accepted conceptions about the mechanisms that control other motivated behavior patterns, such as those of feeding and drinking [6].

Several lines of evidence from neuroanatomical studies support the notion that homologous biological mechanisms

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control the killing patterns of diverse species [26]. With rats and cats, in particular, physiological manipulations of both amygdaloid and hypothalamic brain structures have had similar effects on killing. In both species, amygdaloid lesions have eliminated mouse killing [13, 15, 35, 43] and electrical stimulation of the hypothalamus has evoked it [9, 17, 22, 40]. These treatments seem to be effective by changing thresholds for, or by evoking, normal motivational processes [32]. Thus, killing in these two species could be controlled by homologous mechanisms because comparable biological manipulations have had the same effects on killing by both species.

The studies reported here represent another attempt to predict about mouse killing from rat to cat. Surprisingly, methods for inducing killing by rats did not, in all cases, prove effective with cats. Food deprivation was effective, as described below, but injections of pilocarpine and chlordiazepoxide were not. These results suggest that there may be previously unsuspected biological dissimilarities between killing by rats and cats. We argue, therefore, that it would be prudent to question the extent to which the biological mechanisms that control mouse killing by rats and cats are, in fact, homologous.

EXPERIMENT 1: EFFECTS OF FOOD DEPRIVATION ON CATS THAT DID NOT SPONTANEOUSLY KILL MICE WHEN FOOD SATIATED

Rats that do not spontaneously kill mice can be induced to kill by food deprivation [30,41]. Readiness to eat is not the only factor in induction of killing, however. When rats are exposed to mice and do not kill them, habituation blocks the kill-inducing effects of later deprivation [16,28]. Similarly, deprivation-induced killing experience can induce killing in a variety of circumstances that did not previously evoke it [29,31]. Thus, it seems that food deprivation is a necessary, but not sufficient, condition for inducing killing in rats that do not spontaneously kill.

Experiment 1 was designed to examine whether mouse killing could be induced in domestic, laboratory-bred, cats by food deprivation procedures like those that are effective with rats. A number of cats that did not kill mice while they were satiated were divided into two groups. One group was then deprived of food. Both this deprived and the other, still satiated, group were tested repeatedly for mouse killing. Deprivation and satiation conditions were then reversed for the two groups, and further testing was carried out after the reversal.

METHOD

Animals

Three adult male and 12 adult female domestic laboratory-bred cats that did not spontaneously kill a mouse during a pretest, described below, were selected for this experiment.

Apparatus and Procedure

Prior to experimentation, the cats used here were separated by sex and housed with others (not all of which were subjects in this study) in groups of 4–8 in a number of chain-link pens. Each pen had a 243.8 by 121.9 cm floor area. The pens contained tiered shelves, open stainless steel cages and resting mats. Lights in the cat colony were on from 8:00 a.m. to 11:00 p.m. daily. All cats were food satiated for at least one week prior to the first killing test. The satiation schedule provided ad lib access to tap water and Purina Cat Chow plus daily dietary supplements of varied canned commercial fish and meat cat foods.

Each killing test was carried out by placing the cat with a single adult male Swiss-Webster albino mouse in a stainless steel cage similar to the ones in the home pen, but located outside that pen. Each time a cat killed a mouse during these tests the mouse was removed immediately and replaced by an additional mouse. The number of mice killed during each test was recorded.

The cats were separately housed in their stainless steel testing cages throughout the experiment. Each of 33 cats was given seven successive, daily, 0.5 hr mouse killing tests. Ad lib water and Purina Cat Chow were available on the first day. The first killing test served as a selection pretest. None of the 15 cats used for this experiment killed a mouse during that test.

After the selection pretest the cats were divided into two groups. One group was first deprived and later satiated (Group DS), while the other was first satiated and then later deprived (Group SD). Group DS consisted of 8 cats. DS cats were deprived of all food for three days, during which they continued to have ad lib access to tap water; they were then satiated for the final three days. Group SD consisted of 7 cats. SD cats continued on the satiation regimen, including Purina Cat Chow and supplementary canned food, for the first 3 days; they were then deprived for the final 3 days. In order to make test conditions identical for the two groups, no food or water was present during the 6 final 0.5 hr daily test periods whether or not cats were otherwise deprived or satiated.

RESULTS

Food deprivation clearly induced killing. The percentage of cats that killed mice and the mean number of mice that each cat killed increased with successive days of deprivation, as shown in Fig. 1. By the end of the fourth test, 7 of the 8 food deprived DS cats had killed at least 1 mouse, but only 2 of the satiated SD cats had done so ($\chi^2 = 5.40$, $df = 1$, $p < 0.05$). The fact that two SD cats did kill suggests that the single pretest used here did not provide a very strict test for tendencies to kill spontaneously. When the DS cats were subsequently food deprived, during the final 3 test days, all 7 killed mice. Further, the mean numbers of mice killed (even when means were calculated only for those cats that killed at least once on a given test day) were higher for deprived than satiated killers, and mean kills per cat increased monotonically with increasing days of food deprivation. In sum, food deprivation consistently led to initiation of killing by cats that had not previously killed, increased rates of killing by those cats that were killers, and the percentage of cats that killed increased monotonically as deprivation levels increased. In the cat, as in the rat [30] food deprivation consistently induced mouse killing.

One difference between the cats in this experiment and rats is evident. Cats induced to kill by deprivation did not consistently continue to kill when they were subsequently satiated. As Fig. 1 indicates, most of the group DS cats that had killed when deprived did not consistently do so when satiated. Further, every cat in group DS killed less mice during the tests when they were satiated than they had during the tests when they were deprived ($p < 0.01$, sign test). In

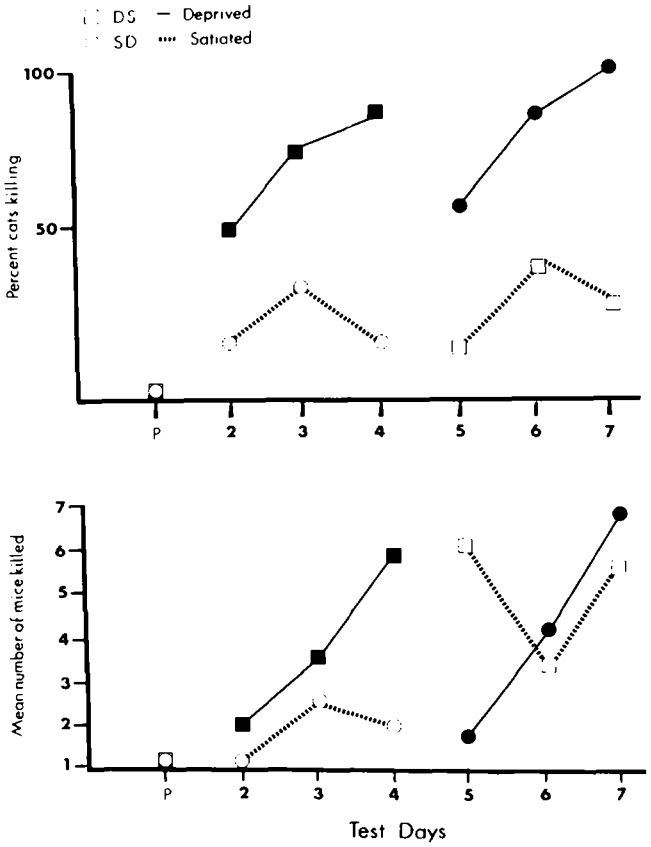

FIG. 1. Killing by cats during Experiment 1. The upper graph shows results from all the cats, including actual values during the selection pretest. The lower graph shows results only for those cats that killed at least once on a given day. The pretest values shown in the lower graph represent theoretical minimum values for these selected animals, none of which actually killed during pretests. The number of cats represented at each point on the lower graph can be calculated from the corresponding point immediately above it on the upper graph.

contrast, rats usually continue to kill when satiated after initial killing is induced by deprivation [30].

Food deprivation, thus, had approximately the same effect on cats as had been found with rats. Satiation, in contrast, seemed to strongly inhibit killing only in cats. Both results suggest that, in cats as in rats, readiness to kill is strongly influenced by readiness to eat.

EXPERIMENT 2: EFFECTS OF A SERIES OF PILOCARPINE AND CHLORDIAZEPOXIDE INJECTIONS ON FOOD-SATIATED NONKILLING CATS

Even when rats are not food deprived, they can readily be induced to kill mice if they are repeatedly injected with pilocarpine [11,39]. Readiness to eat may, nevertheless, influence killing induced by pilocarpine. Amygdaloid brain lesions that produce aphagia delay the onset of killing. In addition, killing is facilitated if side effects of pilocarpine that inhibit readiness to eat, such as emesis and anorexia, are blocked with methyl atropine or habituated [42]. The relationship between killing induced by pilocarpine and that induced by food deprivation is, thus, not entirely clear. There are both similarities between and differences among the effects of these two methods [2].

Chlordiazepoxide seems to induce killing in a manner that differs somewhat from that of both pilocarpine and food deprivation. It is most effective when it is first administered, and repeated injections tend to be less effective. Further, only a small proportion of all nonkilling rats can be induced to kill with chlordiazepoxide, and dose levels are quite critical [19]. Extrapolation of comparable methods from rat to cat is therefore more difficult with chlordiazepoxide than with pilocarpine or food deprivation.

Experiment 2 was designed primarily to examine the possibility that pilocarpine would induce mouse killing by cats, and to provide a secondary test for chlordiazepoxide induction. A single group of nonkilling cats was given pilocarpine injections before each of a series of killing tests. Methyl atropine pretreatments were also used at moderate and low dosages in order to facilitate induction of killing. The injection series was continued until an adequate number of injections of habituation, based on rat data and on observations of side effects, was given. A rest period, free of drug injections, followed the pilocarpine series. Finally, tests with chlordiazepoxide concluded the induction attempts.

METHOD

Animals

Three adult male and three adult female laboratory-bred cats that did not spontaneously kill a mouse during a pretest, described below, were selected for this experiment.

Experimental Design, Apparatus, and Procedure

The cats were separated by sex and housed with others in groups of 4–8 in separate rooms. Each room was 243.84×304.80 cm in floor area and 243.84 cm in height. Each room contained tiered shelves, open stainless steel cages, and resting mats. The same schedules of lighting and food satiation that had been used for Experiment 1 were used throughout Experiment 2, for all cats.

The procedure for killing tests was also the same as that used for Experiment 1, except that each test during Experiment 2 lasted for 2 hr and tests were given only every third day (to permit recovery from the acute effects of the drug injections). The first test served as a selection pretest. None of the cats used for Experiment 2 killed a mouse during that test.

After the pretest, cats were always tested under various drug conditions. Drug injections were always administered intraperitoneally (IP) in 0.1 cc/kg solutions of 0.9% NaCl. An injection of 1.0 mg/kg pilocarpine HCl (pilocarpine) was given to each cat for each of 13 tests, and a pretreatment injection of atropine methyl nitrate (methyl atropine) was also administered before the fourth through the twelfth of these tests. The methyl atropine, which served to at least partially antagonize peripheral side effects of pilocarpine, was given at 0.25 mg/kg before the fourth test, increased in dose to 0.5 mg/kg before the fifth test, and then successively halved in dose on each test from the sixth through the twelfth tests (0.25, 0.125, 0.063, 0.031, 0.016, 0.008, and 0.004 mg/kg, respectively). The successive reductions in methyl atropine dose paralleled increasing tolerance to the peripheral side effects of pilocarpine.

A ten day interval, free of drug administration and killing

tests, followed the last test with pilocarpine. After this interval two tests with chlordiazepoxide were given, with a three day interval between them. Doses of 1.0 and 2.0 mg/kg Chlordiazepoxide HCl (chlordiazepoxide) were administered before the first and second such test, respectively.

RESULTS

The results of Experiment 2 were simple and clear. No cat ever attacked or killed a mouse.

The failure to kill was apparently not due to gross perceptual or cognitive deficits. The cats all continued to orient appropriately to objects in their environment, including mice, and they seemed to behave normally most of the time. They were actuely ill during the first hour of the initial sessions with pilocarpine, but methyl atropine seemed to block the most evident side-effects of the drug, such as emesis and lethargy. The cats all habituated to pilocarpine's side-effects, as well. During the last tests with pilocarpine, when little or no protective methyl atropine was given, no overt emesis or lethargy was apparent. The only observable side-effects of chlordiazepoxide was some intermittent, brief, unsteadiness of gait. In sum, both pilocarpine and chlordiazepoxide were biologically active but neither drug seemed to have sustained toxic effects.

Pilot observations with a higher dose of pilocarpine, 4 mg/kg, showed severe toxic (convulsions, coma, death) and debilitating (failure to eat, drink, walk) effects throughout the whole test period. It seems likely, therefore, that a highly robust kill inducing effect, like that found with rats, was not missed with cats because the dose levels of Experiment 2 were inadequate.

The failure of pilocarpine to induce killing by cats is more surprising, given the rat data, than that with chlordiazepoxide. Chlordiazepoxide does not induce killing, at any dose level, in a very high percentage of rats [19]. Its ineffectiveness could be an artifact of the small sample size of Experiment 2. Pilocarpine, however, seems to be the most powerful inducer of killing by rats that is known. Repeated low doses not only induce killing by food-satiated, neurologically normal, nonkillers [11, 39, 42]; they even induce killing by rats that have amygdaloid brain lesions that block spontaneous killing [10]. Pilocarpine elicits killing more quickly and effectively than Δ^9-tetrahydrocannabinol [25]. In contrast to other kill eliciting drugs [1, 18, 24, 34, 38], it seems to be the only one that ever results in killing rates of 100% [39,42]

EXPERIMENT 3: EFFECTS OF PILOCARPINE AND METHYL ATROPINE ON CATS THAT SPONTANEOUSLY KILL MICE

Pilocarpine, in Experiment 2, did not induce any killing. Although unlikely, it is possible that its side-effects might have completely masked or blocked a tendency to induce killing. Inhibitory effects of the drug in rats do not block killing completely. Like the inhibitory effects of food satiation, however, they might be more important in cats than in rats. It is difficult to rule out this possibility and, as with any negative result, to conclude that pilocarpine lacks any kill inducing actions in cats.

An indirect test of the degree to which pilocarpine's side-effects might have completely blocked a kill inducing action is possible. In rats, both spontaneous and induced killing are similarly blocked by the peripheral side-effects of pilocarpine [12]. Both blocking actions can be antagonised by methyl atropine pretreatment [12,42]. It is possible to examine whether or not pilocarpine blocks spontaneous mouse killing by cats and, if so, whether the block can be antagonised by methyl atropine. Because the blocking action, in rats, seems independent of whether or not the killing is spontaneous or induced, its magnitude during tests of spontaneous killing, in cats, might indicate whether a kill inducing action of pilocarpine could have been concealed during Experiment 2.

Experiment 3 was designed to provide such a test. It examined the effects of pilocarpine, with or without methyl atropine pretreatment, on spontaneous mouse killing by cats. A group of cats that killed mice spontaneously was given injections before killing tests. A dose-effect function for the blocking effect was determined, and then the degree to which it could be antagonised by methyl atropine pretreatment was examined.

METHOD

Animals

Two adult male and three adult female laboratory-bred cats that spontaneously killed mice during every one of ten pretests, described below, were selected for this experiment.

Experimental Design, Apparatus, and Procedure

The cats were housed, fed ad lib, and tested every second day according to procedures identical to those used for Experiment 1. Each cat was pretested ten times for mouse killing. All cats selected for this experiment not only killed a mouse during every pretest but, by the end of the series of pretests, did so within 1 min after its presentation.

Drug Experiments 3A, 3B, 3C were conducted sequentially after the tenth pretest. Drug injections were administered IP in 0.2 cc/kg solutions of 0.9% NaCl, and the 0.5 hr killing test was given 20 min later. When two injections were given, the pretreatment injection was given 1 min before the second injection.

Experiment 3A examined the effects of 0.0, 0.25, 0.5, 1.0 and 2.0 mg/kg of pilocarpine. Doses were administered in a counterbalanced order taken from a 5×5 latin square.

Experiment 3B examined the effects of 2.0 mg/kg pilocarpine after pretreatment with 0.0, 0.125, 0.25, or 0.5 mg/kg methyl atropine. Different random orders of pretreatment doses were used for each cat.

Experiment 3C examined the effects of either 0.0 or 2.0 mg/kg pilocarpine after pretreatment with either 0.0 or 10.25 mg/kg methyl atropine. Different random orders of dose conditions were used for each cat.

RESULTS

Pilocarpine clearly blocked spontaneous killing. During Experiment 3A all five cats killed after the control saline injection. Only four killed after 0.25 mg/kg pilocarpine and the number of killing decreased 3, 2, and 0, respectively, at doses of 0.5, 1.0 and 2.0 mg/kg.

Methyl atropine partially antagonised the blocking effect of pilocarpine. During Experiment 3B, when all cats were given 2.0 mg/kg pilocarpine before every test, none killed after the control, saline, pretreatment. After pretreatment with 0.125 mg/kg methyl atropine one cat killed. The number killing increased to three at pretreatment doses of 0.25 and

0.5 mg/kg. Four of the five cats killed at least once after methyl atropine pretreatment, a significant number when compared to the effects of saline pretreatment ($\chi^2=6.67$, $df=1$, $p<0.01$). These findings are similar to those observed with rats [42].

Methyl atropine alone did not block illing. During Experiment 3C all five cats killed after control saline injections whether they had received saline or methyl atropine pretreatments. None of the cats killed when pilocarpine followed saline pretreatment, replicating the finding of Experiment 3A. Two cats killed when pilocarpine followed methyl atropine pretreatment, replicating the partial antagonism observed during Experiment 3B. These findings, again, are similar to those observed with rats [13,42].

DISCUSSION

Food deprivation induced mouse killing by cats, as well as rats. In fact, changes in readiness to eat, which are probably the most important results of food deprivation, may influence readiness to kill more strongly in cats than in rats. Satiation after food deprivation blocked killing to a substantial extent in cats (Experiment 1), though it has relatively little effect on rats [30]. Both cats and rats eat most of the mice that they kill, if permitted to do so, and the adaptive value for both species of an effect of readiness to eat on readiness to kill is obvious.

The blocking effect of pilocarpine on spontaneous killing is predictable from, and supports, a conclusion that readiness to eat influences killing more in cats than in rats. Pilocarpine produces nausea and decreases readiness to eat even when emesis does not occur. When these effects were intense, at a dose of 2 mg/kg, spontaneous mouse killing by cats was completely blocked (Experiment 3). With rats, in contrast, high doses only partially block spontaneous killing [12]. Further, the partial antagonism of pilocarpine's side-effects produced by methyl atropine pretreatment was apparently as effective in cats as in rats (Experiment 3; [42]). Both rats and cats kill mice fairly frequently if pilocarpine's side-effects are partially antagonised by methyl atropine.

The failure of pilocarpine to induce *any* killing during Experiment 2 is, therefore, surprising. The doses of pilocarpine and methyl atropine used in that experiment should not, given the findings of Experiment 3, have completely blocked a kill inducing action. As noted earlier, in spite of the fact that its side-effects block killing, pilocarpine is the most potent inducer of killing by rats that has been discovered [10, 39, 42]. Pilocarpine's mechanism of action is poorly understood, but it clearly activates some central neural mechanism that controls mouse killing by rats. If pilocarpine does not activate a similar mechanism in cats it either has some different pharmacological action in cats than it has in rats or the mechanism is absent in cats. Pilocarpine is not known to act differently in cats than in rats, so the latter possibility must be taken seriously. The mechanism by which pilocarpine induces killing in rats may not have a homolog in cats.

The mechanism by which pilocarpine induces killing by rats may, in spite of its potency, be due to a physiological or pharmacological anomaly that is not generally present in other species. Early findings suggested that predatory mouse killing by rats might be induced because of cholinergic activation of amygdaloid brain mechanisms [18]. Some later findings are consistent with this interpretation [10,37], but there are several problems, as well. Arecoline and oxotremorine, which are usually more centrally active than pilocarpine as cholinomimetic agonists, do not induce mouse killing by rats [11,42]. Further, the kill inducing effects of intracerebral carbachol seem to be associated with, and possibly due to, very high levels of motor activity induced by these treatments [36]. Although the kill inducing actions of pilocarpine in rats can be partially blocked by scopolamine [42], the fact that arecoline and oxotremorine do not induce killing suggests that kill induction may be due to non-cholinomimetic, pharmacologically anomalous, actions. Further, it is now clear that pilocarpine can induce powerful behavioral effects (on drinking) in rats that are not antagonised by cholinergic antagonists [10]. These facts may, therefore, be a sign that unusual mechanisms or drug actions may influence killing by rats.

The failure of chlordiazepoxide to induce killing in cats tends to support, at least weakly, the notion that rats may be anomalous. The killing induced by chlordiazepoxide in rats, like that induced by pilocarpine, may not be due to stimulation of mechanisms that have a specific predatory function. It may, on the contrary, reflect changes in emotional or motivational processes of a general, nonspecific, type that only indirectly influence killing and also affect many other classes of behavior [18,19]. If so, studies of killing by rats may be relatively unsuitable for assessing general, typical, features of mechanisms that control natural killing. Cats, on the other hand, may provide a species with which anomalous, artifactual, results are less evident. Further research is obviously necessary to determine whether or not this suggestion is correct.

It seems likely, from the studies reported here, that there are important differences in the pharmacological mechanisms that control killing by rats and cats. It seems prudent, therefore, to be cautious in speculating about whether killing by rats and cats involves homologous mechanisms and processes. Further studies with cats, in particular, are necessary in order to determine the extent and significance of the differences between cats and rats.

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