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THE EFFECT OF IMIPRAMINE ON PREDATORY BEHAVIOR AND LOCOMOTOR ACTIVITY IN CATS

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Abstract. The behavior toward mouse was studied under and after chronic imipramine treatment in two groups of cats — non-killers and killers. Imipramine facilitated predatory behavior in the non-killers but not in the killers, which is in contrast to results obtained on rats. Imipramine produced a marked decrease of locomotor activity of non-killers tested in open field. The inhibition of locomotion did not interfere with the occurrence of killing behavior. It was concluded that imipramine selectively facilitates the neurophysiological mechanism of predatory behavior, which in cats might be connected with the reward system.

INTRODUCTION

We have suggested previously that predatory behavior in cats is a complex mechanism possessing its own motivational basis (4). Predatory behavior is, however, related to both aggressive and alimentary behaviors and therefore considered by some authors as "food getting" (19), or more commonly as a class of aggression (12, 22, 25). It is known that mouse-killing may occur without eating and still be a part of the predatory sequence. Moreover, some clear evidence that there are separate neural mechanisms for food intake and predatory behavior was found (3, 27, 29). The relationship of mouse-killing to aggression seems to be more complicated — no one can deny that in order to obtain prey, the killing act is necessary. It was shown that the predatory act in cats is distinguishable from affective aggression (e.g. 3, 27) but there still re-

mains the question whether and/or to what extent neural mechanisms and neural substrates for predatory behavior are totally included within the aggressive system, partially overlapping or quite independent. An additional objection seems to derive from species differences. There are many facts indicating that muricide reaction in rats is not equal to feline predatory behavior and therefore they might differ in motivational, neurophysiological and biochemical mechanisms.

It was found by Horowitz et al. (9), and then confirmed in many other studies (see 6), that imipramine selectively blocks mouse-killing in rats. Muricide behavior is presently one of the most commonly used animal models to screen drugs for their antidepressant action. To the best of our knowledge the effect of chronic imipramine administration on predatory behavior in cats has not been studied before by other authors. The aim of the present experiment was to investigate the cat-mouse interaction in killers and non-killers under imipramine treatment.

In some animals locomotion in the open field was studied as well in order to find whether there is a correlation between the cat's general mobility and its ability to kill the prey. The dose of imipramine effective in the suppression of mouse-killing in rats was found to produce a decrease of motor activity in the open field (5). Therefore the suppression of muricide response in rats could be considered as a result of a general inhibition of motor behavior.

MATERIAL AND METHODS

Animals. The experiment was performed on adult male cats weighing 3.5-4.5 kg, housed individually and fed with standard food, i.e., meat soup with cereal and vegetables and milk. In the pretest period the cats were selected according to their predatory abilities. Originally we were interested only in non-killers. They formed a group of 10 animals. Additionally a group of 5 good killers was included in the experiment.

Predatory test. Each animal after 24 h of food deprivation was placed in an experimental compartment (180×180×180 cm). After 5 min a freely moving white mouse was thrown through the window placed 143 cm above the floor. The cat's behavior toward the mouse was observed during 20 min. The latency of killing and consuming the mouse, as well as playing with it or lack of interest were noted. The predatory test was performed for each cat separately 9 times before the imipramine treatment, 9 times during and 9 times after the treatment.

Open field. In seven non-killer cats the spontaneous motor activity in the open field was measured for 20 min. A squareshaped compartment was divided into 9 equal parts (30 cm×30 cm). The experimenter

observed the locomotor activity of the cat on a TV screen, counting the number of crossing through each of the nine parts of the field.

The open field test was performed on every other day 9 times before the treatment, 9 times during and 9 times after the treatment.

Imipramine treatment. Imipramine (Imipraminum Polfa) was administered to both killers and non-killers intramuscularly in chronic conditions during 3 weeks, in the following doses: 12.5 mg first week, 25 mg second week, 12.5 mg — third week. The injection was performed at least 4 h before the testing by a person who did not deal with the animal during the proper experiment.

RESULTS

Predatory behavior before imipramine treatment

Group I — non-killers. The non-killers selected to this group did not kill the mouse in any of the experimental sessions (Table I and Fig. 1). A few of them — cats NK8 and NK2 — displayed some interest in the mouse, i.e., they observed it, sometimes followed it, but never caught and killed the prey. Cat NK7 used to play with the mouse very intensively, chasing it and striking with the paw, but effective killing was never observed. The other cats ignored the mouse completely — at the first encounter in every session they usually sniffed the mouse and

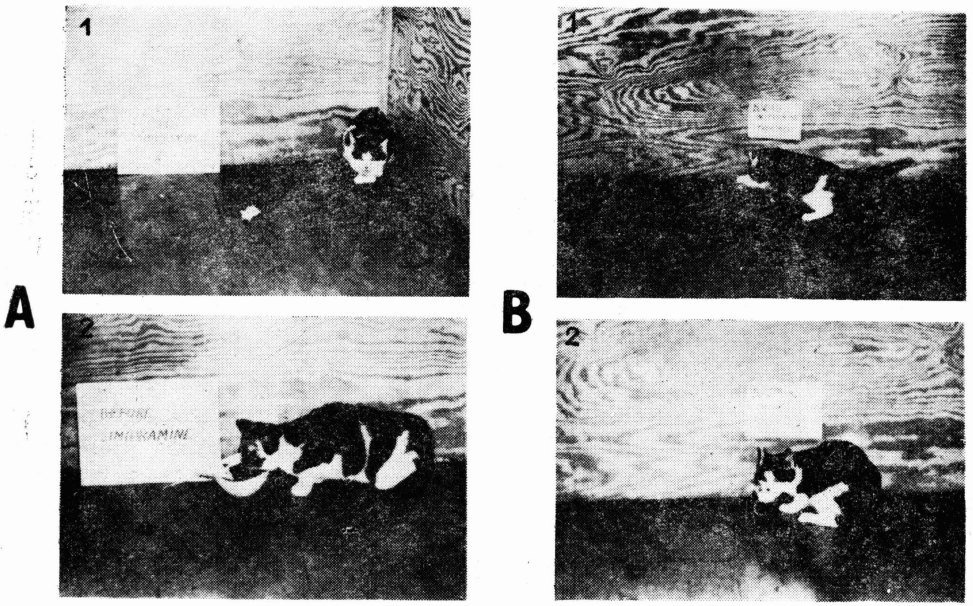

Fig. 1. Cat NK3 in a predatory situation. *A*, before imipramine treatment. *B*, under imipramine treatment.

TABLE I

The effect of imipramine treatment on non-killer cats in a predatory situation. −, lack of killing attack; +, delayed killing attack (within 20 min); $\overset{+}{+}$, fast killing attack (within 1 min); $\overset{+}{\overset{+}{+}}$, immediate killing attack (within 1 s)

Cat	Before treatment	During treatment			After treatment		
		Week 1	Week 2	Week 3	Week 1	Week 2	Week 3
			+++	+++	++	+++	+++
NK 1	−	−−+	+++	+++	−++	++	+++
			+	+++	+	+	++
			+	+++	+++	+++	+++
NK 2	−	−−−	−++	+++	+++	+++	+++
					++	+++	+++
			+++	+++	+++	+++	+++
NK 3	−	−−−	+++	+++	+++	+++	+++
			+++	+++	+++	+++	+++
			+++	+++	+++		
NK 4	−	−−+	++	+++	+++	+++	+++
				+			
				+++	+++		+
NK 5	−	−−−	−++	+++	++	−−+	+++
NK 6	−	−−−	−−−	−−−	−−−	−−−	−−−
NK 7	−	−−−	−−−	−−−	−−−	−−−	−−−
NK 8	−	−−−	−−−	−−−	−−−	−−−	−−−
NK 9	−	−−−	−−−	−−−	−−−	−−−	−−−
			++	+++	+++	+++	++
NK 10	−	−−−	− +	+++	+++	+++	++
				+++	+	++	++

then walked away. A 20 min session was mostly spent on drowsing or sitting motionless in a sphinx position. No signs of affective arousal were observed.

Group II — killers. In four cats the effective attack occurred immediately. That is, within 1 s of mouse presentation the cat caught it with his paws by the head and in a characteristic killing grip broke its neck, then began to eat it. In one cat, K5, the mean latency of attack was significantly longer, although he never failed to kill the prey during the experimental session (Table II). This cat used to play with the mouse, chase it etc., however there were sessions in which he killed the mouse immediately without playing.

Open field before imipramine treatment

Seven cats from NK group were submitted to the open field test. The individual differences in locomotor activity were observed. Three cats were rather inactive in the open field (NK6, NK9, NK10). They

TABLE II

The effect of imipramine treatment on predatory behavior in killer cats. +, delayed killing attack (within 20 min); ++, fast killing attack (within 1 min); $\overset{+}{+}$, immediate killing attack (within 1 s)

Cat	Before treatment (mean latency)	During treatment Week 1	Week 2	Week 3	After treatment Week 1	Week 2	Week 3
	+	+++	+++	+++	+++	+++	+++
K 1	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
K 2	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
K 3	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
K 4	+	+++	+++	+++	+++	+++	+++
	+	+++	+++	+++	+++	+++	+++
			+	+	+		+
K 5	+	+++	+++	+++	+++	+++	+++
				+			+

mostly sat in the middle of the experimental compartment or on the bar supporting the wall grill. The mean number of crossings for these cats was less then ten. Vocalization was very often observed in cats NK4 and NK9 occasionally also in other animals. Some of the cats used to jump on the wall grill a few times during the experimental session — especially active in this respect were cats NK5 and NK4.

Predatory behavior during and after imipramine treatment

Group I — non-killers. Six cats (NK1, NK2, NK3, NK4, NK10) started to kill the mouse during imipramine administration. Two cats started to attack in the first week of the treatment, four others — in the second week (Table I and Fig. 1). The onset of predatory behavior was usually sudden (except cats which used to show some interest in the mouse in the pretreatment period) with the effective killing grip precisely directed to the nape of the prey. The latency of the first attack i.e., the time between mouse presentation and killing was longest as compared to next sessions. Only one cat (NK3) killed the mouse immediately within 1 s, beginning with the first session. In the remaining animals the latency of attack decreased with consecutive sessions. All cats consumed the prey. Sometimes they did it immediately after the killing, sometimes they played with the dead mouse for a while. The time of consumption of the prey was different in different cats.

Four cats did not change their behavior toward the mouse during imipramine treatment (Table I). Cat NK7 remained very much interested in the mouse. Two times during the treatment an awkward attempt of grasping the prey was observed, but effective killing never occurred. Cat NK8 observed the mouse passively, usually sniffing it 2-3 times during the session. Cat NK6 and NK9 did not display an interest in the mouse. They usually drowsed.

After the imipramine treatment the predatory behavior persisted in each of the cats, which became killers. In three cases (NK1, NK4, NK5) the latency of attack increased in the second and third week after the treatment. These cats played with the mouse chasing it, striking etc. before killing. The interest in the prey was very strong from the moment of mouse presentation. In two cases (NK1, NK5) the lack of predatory attack, but not lack of interest in the mouse during 20 min observation occurred in some sessions. In two cats (NK2, NK3) the predatory attack occurred immediately in every session after the treatment, in one (NK10) the latency of attack was slightly longer — up to 5 s (Table I).

All cats were tested occasionally in a predatory situation for 3-4 months after the experiment — they always killed the prey within 20 min. Cats on which imipramine had no influence with respect to their killing behavior were tested systematically twice a week for 4 months and a predatory attack was never observed.

Group II — killers. Imipramine treatment has no significant effect on cats selected before as good killers. The latency of attack of cat K5, which previously often played with the mouse decreased slightly.

Cat K3, which in the pre-treatment period killed the mouse immediately, twice during the imipramine administration played with the mouse for a few seconds before killing it (Table II). In some animals the latency of consuming the prey (i.e., time between killing and eating) was prolonged, because cats played with the dead mouse. No changes in killing behavior were noted during the 3 weeks' period of post-treatment observation.

Open field during and after imipramine treatment

Imipramine decreased significantly ($P < 0.05$, Student t-test) the locomotor activity in the open field (Fig. 2). This effect was independent from the releasing or not releasing of the predatory attack (Fig. 3). In three cats (NK4, NK5, NK10) which became killers the motor activity in the open field was inhibited as strongly as in cats which remained indifferent towards the mouse. Vocalization in the open field was pronounced generally in all cats. These cats which used to jump on the

bar, did it at the beginning of the session and stayed there. On the first day after the drug administration had been discontinued, the locomotor activity increased in all cats. In some of them it even overpassed the pre-treatment level. Mean number of crossings measured during 3 weeks after the last imipramine injection was the same or higher then the baseline in all cats except one.

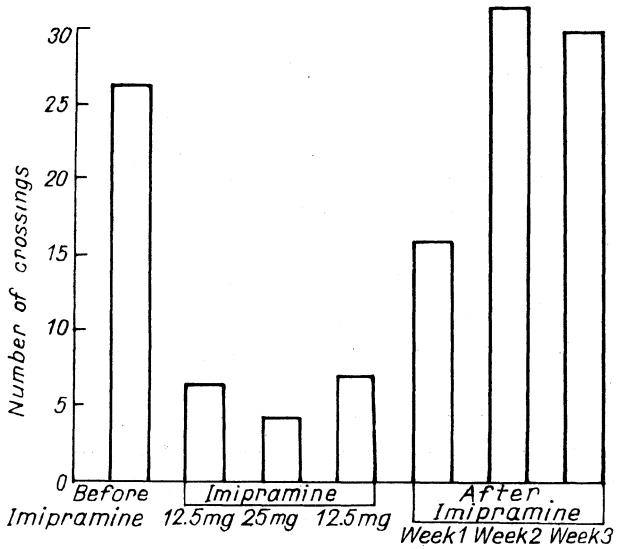

Fig. 2. The effect of imipramine on locomotor activity. Bars indicate the mean number of crossings of seven cats tested in the open field during the pre-treatment, treatment and post-treatment periods.

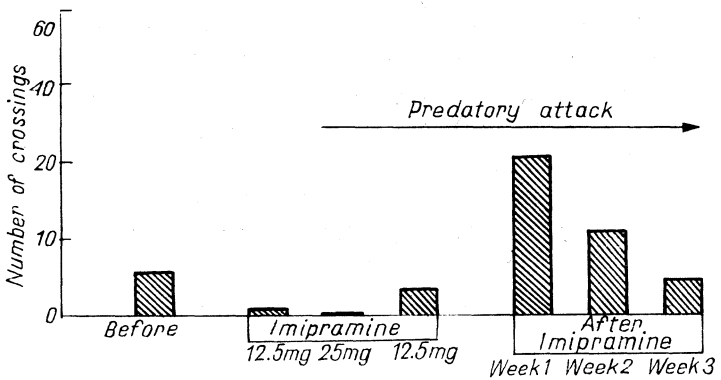

Fig. 3. The effect of imipramine on locomotion and killing behavior in cat NK10. Note the onset of the effective predatory attack in the period of zero activity in the open field.

General behavior. An increase of vocalization, pupil dilatation and hypersensivity to noise were observed in all cats. Vomiting under imipramine treatment occurred only once in a homecage.

During the injection the cats struggled, tried to escape miaowed and hissed, but generally handling in the experimental situation did not become harder.

DISCUSSION

Our results indicate that there are significant species differences concerning the imipramine effect on mouse-killing behavior. We found that imipramine not only does not suppress mouse-killing in killers, but it may even produce or activate mouse-killing in non-killers, and this effect was maintained for an extended period of time after treatment discontinuation.

In none of the five cats selected before as good killers has imipramine inhibited killing, which is in agreement with our previous studies on killers participating in a predatory competition under imipramine treatment (30).

The predatory test used for non-killers showed that six out of ten cats started to catch and kill the mouse in the course of imipramine treatment. Moreover, the predatory attack, once initiated, continued to appear in a predatory situation, even when checked 3-4 months after the treatment. The onset of the first predatory attack during imipramine treatment was usually sudden, only in few cases preceded by some interest in the mouse. The killing grip was in every case perfectly performed, i.e. directed to the nape of the prey and strong enough to kill at once. It is known from the Flynn experiments (3, 27) that the fixed pattern of prey killing is present in the central nervous system, even when the animals do not usually use it. But as Leyhausen (18) pointed out, electrostimulation activates only the motor aspect of the instinct movement, not its propensity component. Our results might indicate that imipramine prods an atrophic propensity mechanisms into continuous automatic function, since the predatory behavior once initiated, is preserved. The predatory attack occurred in two cases in the first week of the imipramine treatment, in four others, in the second week. The latency of attack decreased during successive experimental sessions, which might be connected with the cumulating effect of imipramine.

Four cats out of ten did not kill the mouse neither under nor after the imipramine treatment. No-killing of a genetic origin cannot be excluded in these cases, however, it seems more likely that the individual

differences in susceptibility to the drug or the degree of atrophy of the predatory propensity produced that effect. These cats were tested regularly with mice for 4 months after the imipramine treatment and they never killed. This indicates that the constant training in adult cats fails to evoke predatory behavior, which is in agreement with Leyhausen's observations (18). Therefore the occurrence of mouse-killing in six cats during imipramine treatment seems to be an effect of the drug, not the training. As mentioned in the introduction, it has been proved by many authors that imipramine inhibits the muricide response of rats. Our finding on cats is therefore in obvious contrast to results obtained on rats. The contradiction might be due to several reasons, such as various manners of application (acute versus chronic), different doses of the drug, species differences and probably others.

It has been found that both acutely and chronically administered antidepressant drugs decrease rat muricide behavior (26). As far as doses are concerned, it is known from Horowitz's experiments (9) that imipramine, in contrast to tranquilizers, blocked-mouse-killing in rats at dose levels that does not debilitate. The median effective doses of imipramine producing the inhibition of mouse-killing were several times lower that doses affecting rotarod performance.

The dose of imipramine administered to cats in our experiment was slightly lower than Horowitz and other authors used to suppress muricide reaction in rats.

Species differences seem to deserve a special attention with respect to pharmacological manipulations. It has been shown by Hoffmeister and Wuttke (7) that aggressive behavior of the mouse and cat can be differentially influenced by psychopharmacological drugs. Since Karli's pioneer work (10) there has been a considerable debate as to whether mouse-killing in rats can be considered as predatory behavior, as it is in cats (e.g. 23, 25). Leyhausen (18) pointed out that the act of killing and other behavioral patterns involved in catching the prey are not homologous in cat and rat. Blanchard and Blanchard (1) in their review on rat's muricide response concluded that mouse-killing may represent not a predatory aggression, but something very similar to conspecific attack.

According to Kreiskott's definition (14), the killing behavior in rats is an instinct reaction of interspecific aggression toward smaller mammals. In Felidae the predatory behavior is at least partly dependent on alimentary mechanisms, since in nature it serves to supply food. The rat is a scavenger whose omnivorous nature is uncontested. The prey is neither a necessary nor a preffered source of food for the rat. Moreover, there is a very small percentage of genetic non-killers among cats (18),

whereas according to other studies (14) the percentage of spontaneously killing rats varies from 1% to 20% only (depending upon origin).

It seems reasonable to assume that mouse-killing in rats and predatory behavior in cats are two distinct reinforcing speciestypical behaviors, possessing their distinct motivational systems, which might involve similar elements, but are integrated in a different manner and linked with a different biological importance.

Although King and Hoebel's (12) conclusion that "rats kill the mice for the sake of the kill" might be too simplistic, the prevalence of aggressive component seems to be undoubted. In the cat's predatory behavior, the relation to hunger and the hedonistic value of getting food might play a crucial role.

The mechanism of imipramine action is not yet fully recognized (see 20). One of the commonly cited hypotheses is that the drug interacts with monoamine reuptake, causing the inhibition of noradrenaline and serotonine uptake (28). Imipramine enhancing central adrenergic functions acts probably on the positive reward system (2, 24). It seems likely that this mechanisms is involved in facilitating predatory behavior, which is hedonistic to a great extent. This is in agreement with our previous data on social dominance. It has been found that imipramine evokes a predatory competition and produces in the submissive cat a tendency to get the predatory dominance over the partner. We suspected then that imipramine might act on the positive reward system raising predatory motivation to the level sufficient for the competition with very good killers, which before were dominant (30).

The increase of intracranial self-stimulation after imipramine also supports this hypothesis. Predatory behavior in cats was elicited by electrostimulation from the lateral hypothalamus (3, 27). Horowitz et al. (8) found that imipramine increased rates of self-stimulation in cats with electrodes in LH. According to Lapin (17), the adrenergic mechanism is more important in cats and dogs on which peripheral adrenosensitization has been obtained, whereas the serotoninergic mechanism is more peculiar to rats.

Assuming that the aggressive component is the main dominating feature of the rat's muricide response, it might be supposed that the serotoninergic action of imipramine produce the inhibition of mouse-killing in rats. However, the relationship of various neurotransmitters to aggression is rather controversial, there is evidence that serotonine suppresses aggressive behavior (11, 13, 15, 16).

The interesting point of our study is that there is no relation between the imipramine — induced predatory behavior and the motor activity of previously non-killer cats. During imipramine treatment, whether

there was a facilitation of the predatory attack or not, a statistically significant dercease of locomotion in the open field was observed. It seems to indicate that imipramine acts specifically on predatory motivation, not through enhancing general activity or motor abilities.

Vogel et al. (26) suggest that the clue to the effects of antidepressants on motor activity might be found in a study that determined motor effects on the days of injection and on the next few days. With acute administration of tricyclic, imipramine decreased the spontaneous motor activity half an hour to 5 h after injection (21). With chronic administration, when motor activity was measured within a few hours after injection, again the activity was decreased by tricyclic antidepressants. On the other hand, the locomotor activity measured at least 12 h after the last injection was found to increase markedly. Vogel concluded that each single dose of tricyclic antidepressants (imipramine included) inhibited motor activity within a few hours of administration and that chronically administered antidepressants increased motor activity 12 to 24 h after each dose — presumably after the acute inhibitory effect wore off.

This suggestion is in agreement with our results, since the marked increase of motor activity was observed in our cats on the first day after ceasing the imipramine administration. In our cats however the increased activity lasted usually longer than 24 h.

Concluding, imipramine stimulates selectively the neurophysiological mechanisms subserving predatory motivation, possibly via the reward system, although biochemical processes involved in this phenomenon need further investigations.

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