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Scopolamine-induced alterations in predatory behaviour pattern in cats

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Abstract. Predatory behaviour in its full pattern (i.e., following the mouse, killing it and consuming the carcass) was tested in semi-natural conditions in cats. Consumption of minced horse meat was tested as well. Centrally acting scopolamine hydrobromide injected i. p. did not suppress predatory motivation, since following the prey and killing it were preserved. The executory phase of predation (i.e., the killing grip) was severely disturbed and the consumption of the mouse as well as meat was totally inhibited. Peripherally acting scopolamine methylnitrate administered into another group of cats under the same conditions generally did not affect predatory pattern, though meat and mouse consumption was disturbed to some extent. It is concluded that central muscarinic involvement in predatory behaviour in cats is limited to sensorimotor control of jaw movements.

Key words: predatory motivation, mouse-killing, eating, sensorimotor disturbances, scopolamine, cat

INTRODUCTION

Considerable evidence has been presented that cholinergic mechanisms are involved in the mediation of predatory behaviour. Cholinomimetics and cholinolytics were found to produce opposing effects on mouse-killing in rats. Either systemic or central administration of a variety of cholinergic agonists (arecoline, pilocarpine, oxotremorine) can facilitate or initiate the killing response, whereas cholinergic blockers (scopolamine, atropine) inhibit spontaneous muricide (Bandler 1970, Bell et al. 1985, Mc Carthy 1966, Smith et al. 1970, Vogel and Leaf 1972, Yoburn et al. 1981). Cholinergic involvement was also reported in feline predatory behaviour. In cats which do not kill spontaneously, i. p. injections of cholinomimetics elicit a biting attack, while pretreatment with muscarinic antagonists prevents this attack (Bernston and Leibowitz 1973). Scopolamine was found to raise significantly the threshold current eliciting predatory attack from the hypothalamus (Katz 1981).

The classical tricyclic antidepressants exhibiting varying degrees of anticholinergic side effects specifically inhibit muricide (Goldberg and Horowitz 1978, Horowitz et al. 1966), therefore the muricidal rat is commonly used to screen drugs for antidepressant action (Da Vanzo 1970, Strickland and Da Vanzo 1986). In cats the inhibitory action on predatory attack elicited by hypothalamic stimulation has been reported by Dubinsky and Goldberg (1971).

The consensus of these results supports the notion of a cholinergic link in the brain system that controls predatory behaviour.

There are, however, some contradictory findings, which make the picture less clear. No positive correlation was found between brain acetylcholine level and predatory aggression (Consolo and Valzelli 1970). On the other hand, Mandel et al. (1979) demonstrated higher choline acetyltransferase activity in the brain of spontaneous killers than non-killers, but no difference in acetylcholinesterase activity.

With regard to antimuricidal action of cholinergic antagonists, Horowitz et al. (1965) reported that atropine is not effective in rats, except at doses that produce motor debilitation or even general suppression of behaviour (Albert 1980). Various cholinergic agents have been reported to be inactive in the facilitation or inhibition of predatory behaviour in cats and ferrets (Leaf and Wnek 1978, Leaf et al. 1978, Meierl and Smith 1982). Also the antidepressant imipramine, in spite of its anticholinergic action, does not inhibit spontaneous predatory behaviour in cats. On the contrary, the chronically administered drug facilitates predatory attack in non-killers (Zagrodzka et al. 1987), as well as predatory dominance in cats previously submissive in predatory competition (Zagrodzka et al. 1985). Moreover, newer antidepressants without anticholinergic activity, such as mianserin, trazodone and bupropion, suppress mouse-killing in rats. Inhibition of killing attack by many commonly used antidepressants is thus not necessarily related to their anticholinergic activity (Strickland and Da Vanzo 1986).

The purpose of the present study was to examine the effect of centrally acting muscarinic antagonist scopolamine hydrobromide, at a dose found to be efficient in blocking hypothalamically elicited biting attack (Katz 1981) on predatory behaviour of cats that spontaneously kill mice.

Unlike other investigators, we attempted to study the full pattern of the spontaneous feline predatory act (i.e., approach, killing and eating the prey) in order to find possible disturbances in the motivational and/or executory level. Semi-natural conditions (to the extent that is available in the laboratory) were provided to allow the animal to display its complete repertoire of behaviours during interaction with a prey object.

Food intake was tested as well, since predatory behaviour in cats possesses a strong alimentary component.

In another group of cats, scopolamine methylnitrate, a quaternary muscarinic antagonist that does not readily cross the blood-brain barrier, was used according to the same procedure in order to find out whether and to what extent the peripheral action of the drug might affect the predatory behaviour.

MATERIAL AND METHODS

Animals

The experiment was performed on 16 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. Only cats that spontaneously killed the mouse were included in the experiment.

Predatory test

Each animal, after 24 h of food deprivation, was placed in an experimental compartment (180 x 180 x 180 cm). The construction of the compartment (i.e., wooden bars under the roof) enabled animals to escape safely, the size of the compartment allowed the cat to display its full pattern of predatory behaviour as following the prey, running for it, jumping etc. After 3–5 min a freely moving white mouse was thrown through the window placed 140 cm above the floor. The cat's behaviour toward the mouse was videotaped for 20 min and quantified according to the ethogram chart. The following behavioural parameters were noted: (1) the latency of cat's approach to the mouse, (2) the latency of killing, (3) the latency of consuming the prey, (4) the duration of consumption, (5) the manner of consumption (starting with the head or other parts of the mouse body, intervals in the consumption process), (6) playing with a live or dead mouse. Lack of interest or passive observation of the mouse were noted as well. The predatory test was performed for each cat 10 times before the scopolamine treatment on every other day. On the day of injection, the predation was tested 25 min (1 trial), 35 min (2 trial), 45 min (3 trial), 2 h (4 trial) and 24 h after the treatment.

Meat consumption

At the end of each pre-injection predatory test each cat was presented with a small (150 g) ball of raw minced horse meat. On the day of treatment, the meat ball was offered to the cat 20 min, 30 min, 40 min and 115 min after the injection. On the next day, a meat ball was offered at the end of the predatory test.

Scopolamine treatment

Scopolamine hydrobromide (SHBr), Herbapol, Poland was administered intraperitoneally at a dose of 1.0 mg/kg to 10 cats.

Scopolamine methylnitrate (SMetN), Sigma, USA was administered at the same dose and under the same conditions to another group of 6 cats. Both drugs were dissolved in 0.9% NaCl and applied in 1 ml volumes.

No saline-treated group was included in this experiment, because saline injections were found ineffective on predation in our earlier study (Zagrodzka and Jurkowski 1988).

For the statistical analysis one-way analysis of variance (ANOVA) and Duncan's multiple range test were used.

RESULTS

Control predatory behaviour and meat consumption

During ten pre-injection sessions, all cats approached the mouse immediately within 1 s (Table I). Nine of them in successive trials waited for the mouse under the window, five of them climbed the wall impatiently. The mean latency of killing for all cats was 14.3 $\pm$ 2.63 (Mean $\pm$ SEM), which means that within a few seconds the cat caught the mouse with his paws by the head and in a characteristic killing grip broke its neck.

Playing with the mouse either alive or dead was never observed. The animals always first consumed the head and thereafter the rest of the carcass. No intervals in the process of eating were observed.

The latency of consuming the prey was 1 to 5 s. The time of consumption varied between 35 s and 2.5 min. All cats used in this experiment were able to eat up to six mice during 3 h. All cats ate the meat-ball within a second, immediately after it had been presented.

Effect of SHBr treatment on predatory behaviour

Within the first hour (respectively 25 min, 35 min, 45 min after the injection of SHBr) three mice were offered to the cat, the fourth one – 2 h after the injection. In all cats, except two, the latency of approach to the successive mice remained unchanged i.e., it was immediate (Table I). When the mouse was held by the experimenter, the cat climbed to it very efficiently, stood on his hindlimbs trying to get the mouse with the forepaws. Cat SHBr1 exhibited longer latencies upon all 4 trials and cat SHBr5 only once, in the third trial.

None of the animals failed to kill the prey. Nevertheless, one-way ANOVA showed significant effect of SHBr injection on latency of killing ($F(3.27) + 5.56$, $p<0.01$). Duncan test revealed the increase of killing latency during three trials in the first post-drug hour ($p<0.01$) as well as in the trial performed in 2 h after the injection ($p<0.05$). There were no significant differences between pre-drug trials and the trial 24 h after the injection (Table I, Fig. 1). The killing grip, in normal cats precisely directed to the nape of the prey and strong enough to kill at once, was no longer performed in the usual stereotyped manner. All mice were killed with drops of blood visible, which is never observed in normal cats. Some of them were crushed to death, more

TABLE I

Scopolamine Hydrobromide effect on latency of approach and killing (in s) for individual cat

Cat	Before injection (mean latency of 10 trials)		1 h after injection (mean latency of 3 trials)		2 h after injection (1 trial)		24 h after injection (1 trial)	
	Approach	Killing	Approach	Killing	Approach	Killing	Approach	Killing
$SHBr_1$	1	15	21	130	1	5	1	12
$SHBr_2$	1	20	1	130	1	150	1	18
$SHBr_3$	1	4	1	27	1	10	1	2
$SHBr_4$	1	34	1	52	1	30	1	9
$SHBr_5$	1	13	216	277	15	120	1	14
$SHBr_6$	1	10	1	612	1	7	1	20
$SHBr_7$	1	6	1	122	1	50	1	1
$SHBr_8$	1	12	1	22	1	20	1	1
$SHBr_9$	1	15	1	75	1	30	1	20
$SHBr_{10}$	1	14	1	110	1	28	1	13
Mean ± SEM		14.3 ± 2.63		155.7 ± 55.69		45 ± 16.01		11 ± 2.38

were bitten. The killing act was effective and relatively quick, although slower than in non–treated cats, awkward and not typical for usual feline predatory behaviour.

After SHBr injection, the cats used to carry the prey for a long time or even slept with it in the mouth. They never allowed the experimenter to take the mouse away, defending it with their forepaws.

One cat (SHBr1) in one trial only consumed the mouse (Fig. 2). Attempts to eat, however, were present. Cats used to sniff and lick the dead mouse, few of them sucked the head of the prey. They sometimes tried to start eating with the mouse's limb, usually when it was pointed slightly upward, making the access easier. This was never successful.

Cat SHBr3 started to eat immediately after killing the first mouse offered, but he stopped 5 min later after consuming the head of the prey and in next trials he no longer tried to eat, in spite of a strong interest in the mouse body (sniffing and licking).

Twenty four hours after SHBr injection, the predato-

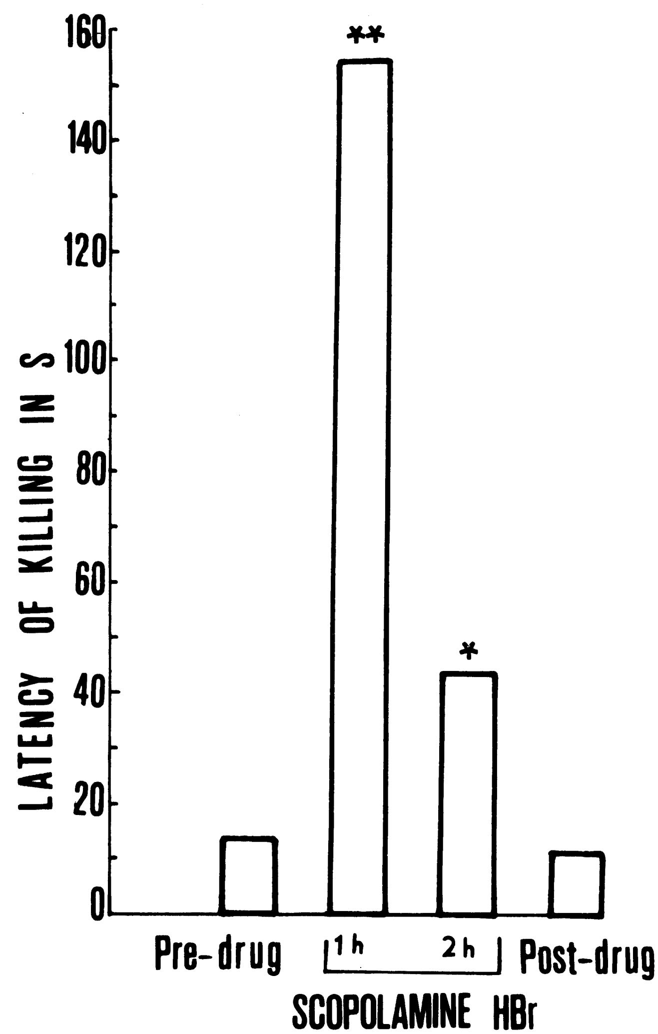

Fig. 1. The effect of scopolamine HBr on mouse-killing. The bars indicate latency of killing (in s) for the whole group in the pre- and post- injection periods and 24 h after scopolamine treatment. Note that scopolamine induced significant (** for $p<0.01$) increase in latency of killing during three trials in the first post-injection hour (25 min, 35 min, 45 min,) as well as in the trial performed 2 h after the injection (* for $p<0.05$).

ry behaviour returned back to normal (Fig. 2). Cats approached the mouse within the first second, killed the prey with a short latency and consumed the carcass immediately afterwards in less than 100 s.

Effect of SHBr treatment on meat consumption

Cats approached the meat–ball immediately, sniffed and licked it, finally tried to take a piece into the mouth. Usually the meat dropped back on the floor. They repeated the attempts for a few minutes, sometimes with a break, then walked away. In four cases, when the meat had been consumed, the act of eating was far from normal, very long (3–4 min for one 150 g ball of soft minced meat), and clumsy. On the next day all cats consumed the meat ball immediately, as they had done before the injection (Fig. 2).

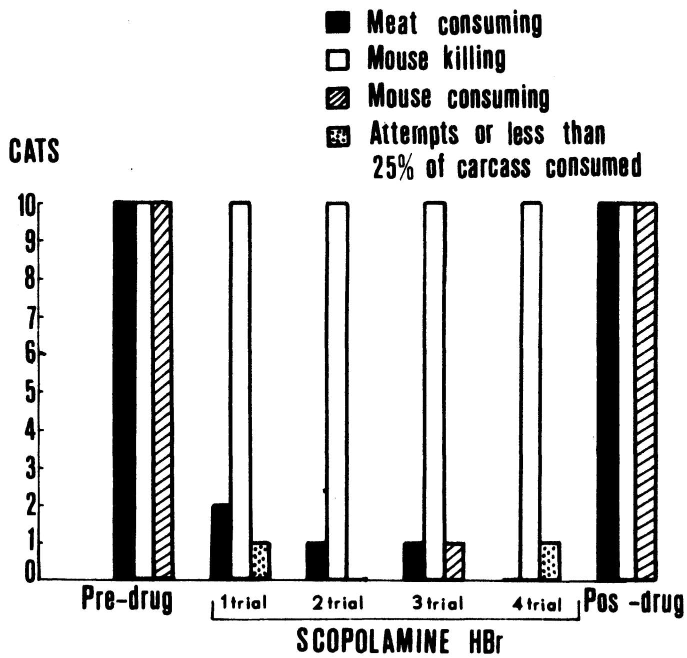

Fig. 2. The effect of scopolamine HBr on killing, eating the mouse and consuming the meat. The bars indicate the number of cats performing these activities in the pre- and post- injection periods. Predatory tests were performed 25 min after the injection (1 trial), 35 min (2 trial), 45 in (3 trial), 2 h after the injection (4 trial).

Effect of SMetN treatment on predatory behaviour

Within the first hour after the drug administration, all animals approached each successive mice immediately. All 24 mice offered to SMetN treated cats were killed effectively in the stereotyped manner with the strong, precisely directed killing grip. The killing latency was significantly longer only in cat SMetN4 2 h after the injection. The cat played with the mouse before killing it.

Consumption began no later then before treatment, but in 7 cases out of 24, mice were eaten only partially (Fig. 3). The duration of consumption was significantly longer (from 3 min 30 s to 8 min). All cats displayed some difficulties in mastication and swallowing during the whole test.

After the twenty four hours all observed parameters of predatory behaviour returned to predrug level. Difficulties in eating the prey disappeared.

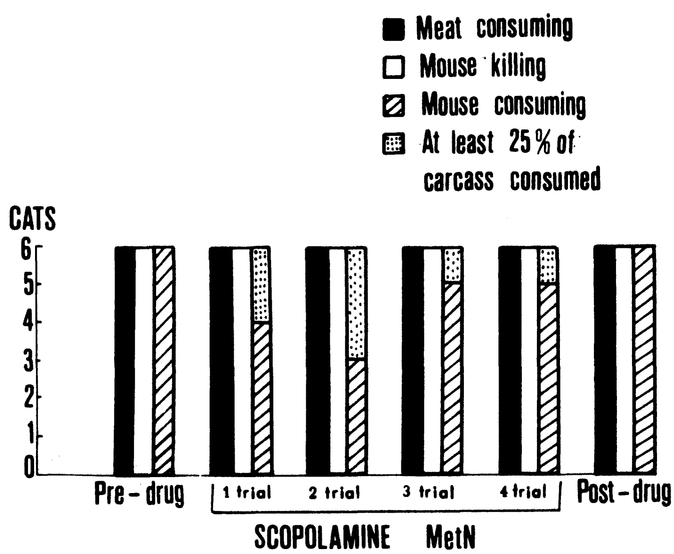

Fig. 3. The effect of scopolamine MetN on killing, eating the mouse and consuming the meat. The bars indicate the number of cats performing these activities in the pre- and post- injection periods and 24 h after SMetN treatment. Another denotations as in Fig. 2.

Effect of SMetN treatment on meat consumption

Cats approached the meat ball and started to eat it immediately, but with apparent troubles and finally consumed it very slowly (from 10 s to 2 min).

Twenty four hours later the consumption of meat ball lasted no longer than 1 s in all cats.

General behaviour

Pupil dilatation and dryness of the mouth were observed in all the cats. Some of them demonstrated tachypnea during the first hour after injection. Intensive vocalization started about 10 min after injection and disappeared and reappeared often during the observation period. In some cats intensive jaw movements, presumably resulting from dryness of the mouth, were observed in the first 15 min of the experiment. Generally, locomotion was not disturbed and cats were able to run, jump, to stay on their hindlimbs, to climb the wall. In two cats (SHBr1, SHBr5), however, it was noted that during locomotion the hindlimbs were spread apart in a wide stance.

Some cats drowsed a great deal during the experiment, some others walked around or sat in the sphinx position. Generally no significant change in mobility was noted.

Cats were reactive and attentive to visual and acoustic stimuli. They were easy to handle, never displayed aggression toward the experimenter.

DISCUSSION

Our results support the notion of a cholinergic link mediating predatory behaviour in cats. They show, however, that acetylcholine is not acting as a triggering factor, as suggested by some authors (Bell et al. 1985, Bertnson and Leibowitz 1973, Bertnson et al. 1976), but cholinergic involvement is limited exclusively to sensorimotor mechanisms connected with jaw movements that are necessary to accomplish the predation. Injection of the centrally acting muscarinic antagonist scopolamine hydrobromide, does not inhibit predatory behaviour either in terms of interest in the mouse or of killing it. It does, however, totally suppress the consuming of the prey as well as eating regular meat. Moreover, the killing act itself loses its stereotyped character.

Injection of the cholinolitic of predominantly peripheral action, scopolamine methylnitrate affects to some extent only the eating process.

A vast amount of research has been devoted to the study of the biochemical mechanisms of predatory aggression. Most of the data indicating cholinergic involvement in the control of muricide, concerns rats (Bandler 1970, Bell et al. 1985, Smith et al. 1970, Vogel and Leaf 1972). It should be pointed out here that, in spite of similarities in the killing pattern of diverse predators, there are many findings demonstrating that mouse–killing in rats and cats differs in many aspects, probably in motivational, neurophysiological and biochemical mechanisms. Leaf and Wnek (1978), on the base of their work, concluded that pharmacological mechanisms and processes controlling killing in rats do not have homologs in cats since the major phenomena that have been observed in rats are not evident in cats. Results of the present experiment and our previous studies are in agreement with this notion.

Therefore, all the evidence pointing to a cholinergic link in the central control of predatory behaviour obtained in rats should be considered as valid only with respect to this species. There are pharmacological reports however, supporting the idea of a "cholinergic trigger" in predatory behaviour in cats (Bertnson et al. 1976, Katz and Thomas 1975, Katz 1981). On the other hand though, there is one study indicating that scopolamine does not suppress the killing act in cats (Leaf et al. 1978). By accurate, ethopharmacological observation we demonstrated, that SHBr injection, without inhibiting killing itself, alters the pattern of predatory behaviour in some of its components. The full predatory act in normal cats consists of preparatory (motivational) and consummatory (executory) phase. The latter one involves killing attack and consumption of the prey. These form a naturally integrated chain reaction. Before the killing grip is performed, predatory motivation is usually expressed in interest in the mouse, approaching it by means of crouching, running or jumping. The killing grip as well as the manner of consumption are stereotyped (Leyhaunsen 1979). It seems, that scopolamine injection did not affect the motivational phase, since all cats demonstrated strong interest in the mouse. Two cats (SHBr1, SHBr5) approached the prey at longer latencies, which might be attributed to motor disturbances or drowsiness.

Marked changes appeared in the executory phase. The killing grip was no longer bloodless. Some mice were crushed to death instead of to be bitten. The latency of killing was significantly prolonged. After the killing, cats were very much interested in consuming the prey, yet their attempts were ineffective, except in one case. The same was true of their meat intake. Only 3 successful attempts were noted after the scopolamine injection.

The question arises as to whether the disorders in killing, inhibition of consuming the prey, and consuming the meat were caused by the same mechanisms. It seems possible that sensorimotor disturbances connected with jaw movements are responsible for the alterations in the predatory pattern. The results of SMetN treatment indicate that central rather than peripheral action of scopolamine is responsible for inadequate killing grip and inhibition of consumption. It might be supposed that SHBr causes a blockade of muscarinic receptors in the brain areas that are involved in the control of orofacial movements, i.e., cortex, striatum, globus pallidus, substantia nigra, cranial nerve of motor nucleus (Lund and Enomoto 1988). These structures are known to possess a high concentration of muscarinic receptors (Rotter 1984).

The motivational component of predatory behaviour after the injection of scopolamine was not affected and therefore the approach and the interest in the mouse were not changed. The killing was performed effectively, however in highly disturbed manner. The consumption of the mouse and meat was almost totally inhibited. Some masticatory disturbances were observed also in scopolamine methylnitrate treated cats, which might be attributed to the fact, that SMetN to some extent penetrates to CNS. The possibility of overlapping peripheral and central action of both drugs cannot be excluded, however.

Our results are consisted with the research done by Berntson and Leibowitz (1973) and Zagrodzka et al. (1989) on the effects of muscarinic agonist arecoline on predation in cats. Stimulation of central muscarinic receptors evoked intensive biting, interpreted by Berntson (1973) as cholinergic facilitation of predatory behaviour. Ethopharmacological studies (Zagrodzka et al. 1989) revealed, however, that arecoline destroyed well established sequences of predatory behaviour in killers. The only component of the predatory pattern in killers, as well as in non–killers, observed after arecoline injection was vigorous biting directed toward the prey as well as toward a piece of styrofoam indicating again that cholinergic involvement in predatory behaviour is limited only to sensorimotor mechanisms connected with the orofacial area.

Our results may also contribute to the discussion on the alimentary mechanisms in predatory behaviour.

It is known (Leaf and Wnek 1978) that food–deprivation induces mouse–killing in cats, while satiation blocks it. Predatory behaviour sometimes is defined as food getting. On the other hand, there is evidence that alimentary and killing mechanisms are separate (Flynn et al. 1970, Fonberg and Serduchenko 1980). Fonberg and Zagrodzka (1982) on the basis of their experiments stated that predatory behaviour in cats possesses its own motivational system, closely connected to, but separate from feeding mechanisms. The present study confirms this point of view. In spite of a lack of an alimentary reward (mouse consumption) animals continued to kill the mice in successive trials. Disconnection of the killing – eating link did not suppress predatory motivation.

In conclusion, scopolamine alters the pattern of predatory behaviour with respect to the final, executory phase, but it does not suppress predatory motivation expressed by following, hunting and killing the prey. This effect is related to central not peripheral action of the drug. Changes in the executory phase might be due to the blockade of muscarinic receptors in the brain areas involved in the control of the jaw movements.

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