

## Effects of incisor-cutting on muricidal behavior induced by olfactory bulbectomy in rats

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### Abstract

Muricidal behavior in rats is composed of two main components, attacking and killing performance. Since a large number of mice could be killed by rats during behavioral experiments, research has been limited in the past decade possibly because of ethical considerations. In preliminary studies, we found that the rat incisors play a key role in muricidal behavior in rats, so, in the present study, we cut off the incisors and assessed the following parameters of muricidal behavior: attack latency, first attack site, lethal attack site, attack frequency, total attack duration and mean attack duration. If after incisor-cutting (IC) rats still tried to demonstrate muricidal activity, but failed to kill the mouse, this would be an ideal model for studying the mechanisms of muricidal behavior. Since muricide can be induced in rats by olfactory bulbectomy (OBX), young adult male Wistar rats with OBX displaying muricidal behavior were tested for muricidal activity 4 h after IC, then every 24 h for 3 days. At 4 and 28 h after IC, only 9% and 36% of rats killed mice, but these values rose to 73% and 82% 52 and 76 h after IC, respectively. At 4, 28 and 52 h after IC, there was no significant difference in attack latency, first attack site, lethal attack site or mean attack duration between IC-treated rats (both killers and nonkillers) and sham-operated controls, while the attack frequency was obviously increased in IC nonkiller rats, and a significantly longer total attack duration was seen in both IC killer and nonkiller rats compared to controls. Since IC treatment increases attack frequency and prolongs the total attack duration without affecting other basic components of muricidal behavior in rats, these results suggest that the killer rats treated with IC may provide a suitable model for research on muricidal behavior. © 2002 Elsevier Science Inc. All rights reserved.

**Keywords:** Incisor-cutting; Muricidal behavior; Olfactory bulbectomy; Rats

### 1. Introduction

Muricidal behavior in rats is a very complicated aggressive behavior. Several neurotransmitter systems [8,14,15] and hormonal functions [7,11] are reported to be involved and muricidal behavior is affected by external factors, such as light conditions, feeding and social behavior [4,6].

The duration of muricidal behavior is very short. However, the performance of this behavior is quite stereotyped irrespective of whether it occurs spontaneously or is induced

by experimental manipulations [6]. When a mouse is introduced into a rat's home cage, the rat starts chasing, attacking and biting the mouse. During this period, the attacks toward mouse may be stopped by the mouse defending itself, but finally the rat bites the mouse and kills it. With an experienced killer rat, this whole process takes only a few seconds and it is therefore difficult to investigate neurotransmitter changes in the rat brain during muricidal behavior. Until now, behavioral data in most studies on muricidal behavior have been limited to determining changes in the percentage of killers after certain brain surgery procedures [12] or drug treatments [13]. Few studies have focused on behavioral components per se, such as changes in attack latency [2] or killing latency [3]. In addition to the extremely short duration of this behavior, ethical consider-

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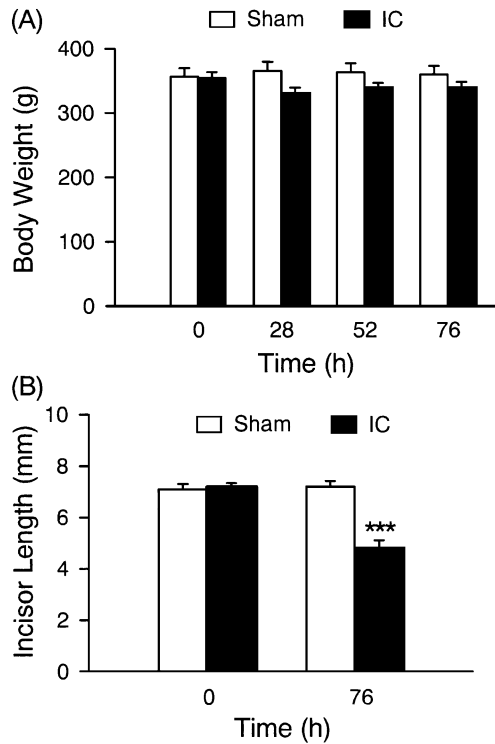


Fig. 1. Effects of IC on body weight (A) and incisor length (B) in rats. \*\*\* $P < .001$ , compared with the sham-operated control group. Time 0 h indicates IC surgery or sham operation.

ations about animal protection in recent years may have hindered research into muricidal behavior. Thus, the mechanisms of this behavior are still unclear.

In our preliminary study on muricidal behavior, we found rats mainly use their sharp incisors to bite mice to death and it would therefore be more difficult for them to kill the mouse if they lacked incisors. However, the effects of incisor-cutting (IC) on muricidal behavior in rats have never been investigated. Adler and Bermant [1] reported that the mounting frequency in rats is significantly increased when the penis of rat is anaesthetized and cannot undergo erection. Based on similar mechanisms in sexual behavior, killer rats without sharp incisors are unable to successfully kill mice, therefore, we would expect that, after IC, the attacking performance of the muricidal behavior might be preserved and even enhanced in rats. If this hypothesis is proved to be correct, the killer rat lacking incisors may be a useful animal model for investigating the mechanisms of muricidal behavior.

In the present study, we induced muricidal behavior in rats by olfactory bulbectomy (OBX). The OBX-treated rats that showed killer activity then underwent IC surgery and parameters of muricidal behavior were recorded and assessed at different time-points after IC. The parameters measured consisted of attack latency, first attack site, lethal attack site, attack frequency, total attack duration and mean attack duration.

## 2. Methods

Male Wistar rats weighing  $350 \pm 50$  g were used in this study. Bilateral OBX was performed as described in the previous study [5]. Briefly, rats were anaesthetized using sodium pentobarbital (10 mg/kg ip) plus ketamine (45 mg/kg ip) and placed in a stereotaxic instrument, and a skin incision was made to expose the skull overlying the bulbs. Two 2-mm diameter holes were drilled in the skull (5.0 mm rostral to the bregma and  $\pm 1.5$  mm lateral to the midline), and the olfactory bulbs were removed by suction. During recovery after OBX, the rats were kept individually in plastic cages. All rats were housed in an animal room with a 12-h light–dark cycle (lights on at 2200 h). Food and water were provided ad libitum. All experimental procedures involving laboratory rats in the present study conformed to the NIH Guide for the Care and Use of Laboratory Animals.

Five days after the OBX surgery, killer rats were selected by testing muricidal behavior every other day. We introduced a mouse treated with pentobarbital (3 mg/kg ip) into rats' home cages, and all tests were performed between 1400 and 1700 h. Rats displaying muricidal behavior within 5 min during at least three consecutive tests were used in the following experiments.

Basal levels of muricidal behavior in all killer rats were recorded, and then the rats were randomly divided into two groups. Both groups were then slightly anaesthetized by ether at the beginning of dark cycle and the rats in the experimental group ( $n = 11$ ) had all upper and lower incisors cut off at the gum line with a dental cutting tool, while the sham-operated control group ( $n = 11$ ) was left with intact incisors. The behavioral parameters of muricide were then recorded for 20 min at 4, 28, 52 and 76 h after IC.

To record the parameters of muricidal behavior (attack latency, first attack site, lethal attack site, attack frequency and total attack duration), a naive mouse was placed in the rat's home cage. An attack was defined as continuous body

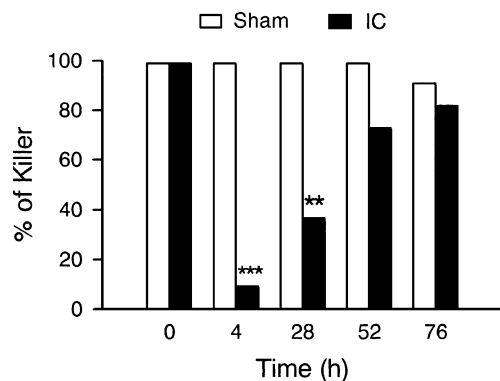


Fig. 2. Effects of IC on the percentage of rats that kill mice during a 20-min observation period of muricidal behavior. \*\* $P < .01$ , \*\*\* $P < .001$ , compared with the sham-operated control group at the given time-point. Time 0 h indicates IC surgery or sham operation.

Table 1  
Effects of IC on the first attack site

	Sham control				IC killer				IC nonkiller			
	Neck	Thorax	Head	Other	Neck	Thorax	Head	Other	Neck	Thorax	Head	Other
0 h	6/11 <sup>a</sup>	2/11	3/11	0/11	6/11	2/11	2/11	1/11				
4 h	3/11	6/11	2/11	0/11	1/1	0/1	0/1	0/1	6/10	3/10	1/10	0/10
28 h	5/11	6/11	0/11	0/11	4/4	0/4	0/4	0/4	5/7	2/7	0/7	0/7
52 h	6/11	4/11	1/11	0/11	5/9	2/9	2/9	0/9	1/2	0/2	0/2	1/2
76 h	8/11	1/11	2/11	0/11	6/9	2/9	1/9	0/9	2/2	0/2	0/2	0/2

<sup>a</sup> The numerator denotes the number of rats choosing the neck, thorax or head as the first attack site, and the denominator is the total number of rats tested.

contact between the rat's forepaws or snout and any part of the mouse's body, and the end of an attack was denoted by the appearance of nonaggressive behavior, such as grooming, feeding or exploration. The attack latency was calculated as the time between the introduction of the mouse into the rat's home cage and the rat's first attack response. The attack frequency was recorded as the number of attacks in the 20-min observation period. The total attack duration represented the total time spent in attack during the behavioral observation period. These parameters were manually recorded with a timer. The mean attack duration, the mean time spent in each attack, was calculated from the total attack duration and attack frequency. The area of the mouse's body first attacked by the rat was designated as first attack site and the critical areas bitten by the rat, subsequently leading to the death of the mouse, were denoted as lethal attack site.

When a mouse intruder encounters a rat in the rat's home cage, the mouse usually immediately rears up on its hind legs and uses its forepaws to defend itself by boxing the rat. According to Miczek [9], aggressive behavior in rats includes biting, boxing and conflict postures, all of which were counted as attack actions in the present study. However, when a rat encounters a mouse in the rat's home cage, it usually displays similar behavior induced by nonaggressive body contact, and this would also be counted as attack behavior in our study.

Changes in body weight and incisor length were evaluated by two-way repeated measures ANOVA, followed by Scheffé's test. The percentages of rats killing mice during a 20-min observation period were compared by Fisher's exact

probability test. Since some IC-treated rats were still able to kill mice during the behavioral observation period while others could not, it was not appropriate to pool the behavioral data from these two groups; so IC-treated rats were further classified as IC killers and IC nonkillers. Differences in attack latency, attack frequency, total attack duration and mean attack duration between these subgroups of IC-treated rats were analyzed using Mann–Whitney *U* test. Since the number of rats in each IC subgroup fluctuated daily, these four behavioral parameters were compared between the sham-operated control group and the IC subgroups using either the Mann–Whitney *U* test or Student's *t* test. A *P* value of <.05 was taken as statistically significant. Data were expressed as the mean ± S.E.M.

### 3. Results

#### 3.1. Changes in body weight and incisor length

The body weight of the IC rats slightly decreased by about 7% at 28 h after IC, then gradually recovered (Fig. 1A). The basal incisor length of rats was  $7.2 \pm 0.1$  mm and the cut incisors grew back to  $61 \pm 3\%$  of this basal length by 76 h after IC (Fig. 1B).

#### 3.2. Recovery of killing ability

The ability of rats to kill mice was significantly suppressed by IC. As shown in Fig. 2, at 4 and 28 h after IC, only 9% (1/11,  $P < .001$ ) and 36% (4/11,  $P < .01$ ), respectively, of rats killed mice during the 20-min observation period, while 73% (8/11) and 82% (9/11) killed mice at 52 and 76 h after IC, respectively.

Table 2  
Effects of IC on the lethal attack site

	Sham control			IC killer		
	Neck	Thorax	Head	Neck	Thorax	Head
0 h	8/11 <sup>a</sup>	0/11	3/11	5/11	3/11	3/11
4 h	5/11	2/11	4/11	1/1	0/1	0/1
28 h	5/11	1/11	5/11	4/4	0/4	0/4
52 h	7/11	0/11	4/11	4/8	0/8	4/8
76 h	6/11	1/11	3/11	7/9	0/9	2/9

<sup>a</sup> The numerator denotes the number of rats choosing the neck, thorax or head as the lethal attack site, and the denominator is the total number of rats tested.

Table 3  
Proportion of rats attacking the nonlethal site

	Sham control	IC killer	IC nonkiller
0 h	0/11 <sup>a</sup>	1/11	
4 h	0/11	1/1	9/10
28 h	0/11	0/4	6/7
52 h	0/11	4/8	2/3
76 h	1/11	4/9	2/2

<sup>a</sup> The numerator denotes the number of rats attacking nonlethal sites, and the denominator is the total number of rats tested.

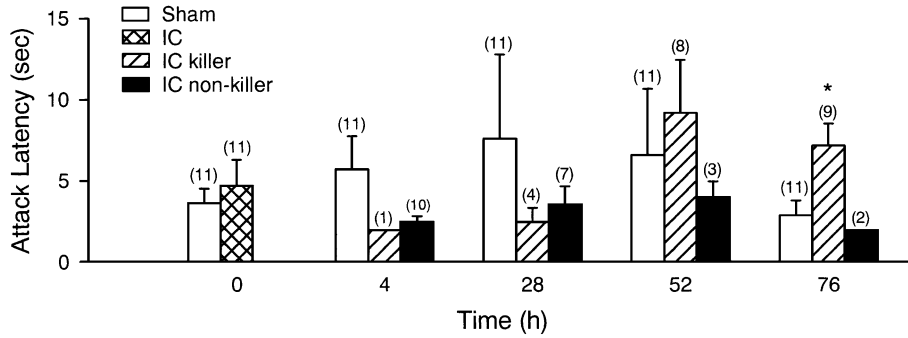


Fig. 3. Effects of IC on the attack latency of rats. \*  $P < .05$ , compared with the sham-operated control group at the given time-point. The number of rats tested at the different observation points is indicated in parentheses. Time 0 h indicates IC surgery or sham operation.

3.3. Attack site

The head, neck and the dorsal region of the thorax were the main body areas first attacked by sham-operated rats, with more than 70% choosing the neck and dorsal part of the thorax as their first attack site. There was no obvious difference in first attack site between IC-treated rats and sham-operated controls (Table 1). Table 2 shows that the lethal attack site for both sham-operated and IC killer rats was the same as their first attack site. As shown in Table 3, only 1 of 11 rats before IC treatment (IC killers at 0 h) showed to attack other body parts than neck, thorax and head of the mouse (nonlethal sites). However, more than 50% of rats after IC attacked the nonlethal sites. One of the eleven rats in the sham-operated control group that failed to kill the mouse at 76 h after sham operation also showed to attack the nonlethal sites during the observation period (Table 3).

3.4. Attack latency

As shown in Fig. 3, the basal attack latency (at 0 h) was  $4.2 \pm 0.9$  s. The statistically significant difference in attack latency between the sham-operated control group and IC

killer rats was only at 76 h, at which time-point the attack latency of IC killer rats was significantly longer than that of control animals ( $t_{19} = 2.61, P < .05$ ). No obvious differences in attack latency were seen either between the sham-operated controls and IC nonkiller rats or between IC killer and nonkiller rats during the observation period.

3.5. Attack frequency

Under basal conditions, all rats killed mice during the first attack; so only one attack was observed (Fig. 4). In the sham-operated control group, the attack frequencies at all four observation points were not significantly different from the basal level. No significant difference in attack frequency was seen between IC killer rats and sham-operated controls, whereas therefore also shown in this figure the attack frequency of IC nonkiller rats was significantly higher than that of sham-operated control rats at all four observation points ( $t_{20} = 10.89, P < .001$  at 4 h;  $t_{17} = 8.88, P < .001$  at 28 h;  $U = 2.57, N_1 = 11, N_2 = 3, P < .05$  at 52 h;  $U = 2.17, N_1 = 11, N_2 = 2, P < .05$  at 76 h after IC) and significantly higher than that of IC killer rats at 28 h ( $U = 2.65, N_1 = 4, N_2 = 7, P < .01$ ), 52 h ( $U = 2.35, N_1 = 8, N_2 = 3, P < .05$ ) and 76 h ( $U = 2.12, N_1 = 9, N_2 = 2, P < .05$ ) (Fig. 4).

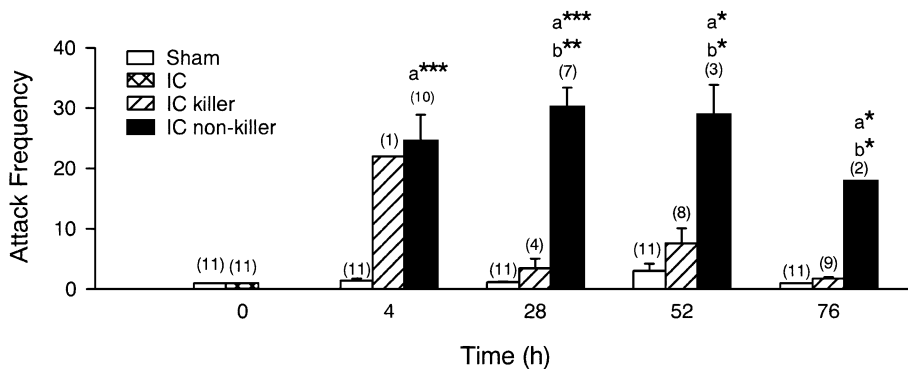


Fig. 4. Effects of IC on the attack frequency of rats during the 20-min observation period. \*  $P < .05$ , \*\*  $P < .01$ , \*\*\*  $P < .001$ ; (a) compared with the control group; (b) compared with the IC killer group at the given time-point. The number of rats tested at the different observation points is indicated in parentheses. Time 0 h indicates IC surgery or sham operation.

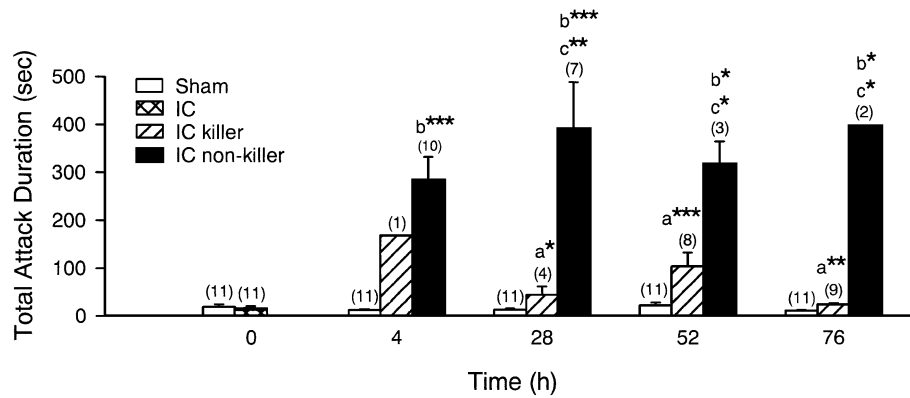


Fig. 5. Effects of IC on total attack duration of rats during the 20-min observational period. \* $P < .05$ , \*\* $P < .01$ , \*\*\* $P < .001$ ; (a) and (b) compared with the sham-operated control group; (c) compared with the IC killer group at the given time-point. The number of rats tested at the different observation points is indicated in parentheses. Time 0 h indicates IC surgery or sham operation.

### 3.6. Attack duration

As shown in Fig. 5, the total attack duration in IC killer rats was significantly longer than that in sham-operated controls at 28 h ( $U = 2.48$ ,  $N_1 = 11$ ,  $N_2 = 4$ ,  $P < .05$ ), 52 h ( $t_{18} = 3.11$ ,  $P < .001$ ) and 76 h ( $t_{19} = 3.91$ ,  $P < .01$ ). A statistically significant difference in total attack duration was also seen between IC nonkiller rats and the sham-operated controls at all observation points ( $t_{20} = 5.76$ ,  $P < .001$  at 4 h;  $U = 4.61$ ,  $N_1 = 11$ ,  $N_2 = 7$ ,  $P < .001$  at 28 h;  $U = 2.57$ ,  $N_1 = 11$ ,  $N_2 = 3$ ,  $P < .05$  at 52 h;  $U = 2.17$ ,  $N_1 = 11$ ,  $N_2 = 2$ ,  $P < .05$  at 76 h). Furthermore, the total attack duration in IC nonkiller rats was also longer than that in IC killer rats at 28 h ( $U = 2.65$ ,  $N_1 = 4$ ,  $N_2 = 7$ ,  $P < .01$ ), 52 h ( $U = 2.45$ ,  $N_1 = 8$ ,  $N_2 = 3$ ,  $P < .05$ ) and 76 h ( $U = 2.12$ ,  $N_1 = 9$ ,  $N_2 = 2$ ,  $P < .05$ ). In contrast, there were no differences in mean attack duration between these groups at all observation points except at 76 h after IC, at which time-point mean attack duration in IC nonkiller rats was significantly longer than in sham-operated controls ( $U = 2.17$ ,  $N_1 = 11$ ,  $N_2 = 2$ ,  $P < .05$ ) and IC killer rats ( $U = 2.12$ ,  $N_1 = 9$ ,  $N_2 = 2$ ,  $P < .05$ ) (Fig. 6).

## 4. Discussion

In the present study, we demonstrated that the IC treatment causes not only an increase in attack frequency and prolongation of the total attack duration without affecting other basic components of muricidal behavior in rats, but also a decrease in the number of mouse killed by the killer rats during the behavioral experiment. These results suggest that the IC-treated killer rats may provide a suitable model for research on muricidal behavior.

In a study of intraspecies aggression in rats, Miczek et al. [10] found that *d*-amphetamine altered the temporal pattern of the aggressive behavior, while ethanol changed its sequential pattern. However, when the percentage of rats attacking intruders was considered, there was no significant difference in the effects of these two drugs on aggressive behavior. Thus, we should use as many different parameters as possible for behavioral analysis. As mentioned previously, it is difficult to study muricidal behavior, because few parameters can be successfully recorded, due to its short duration. Therefore, the effects of treatments on muricidal

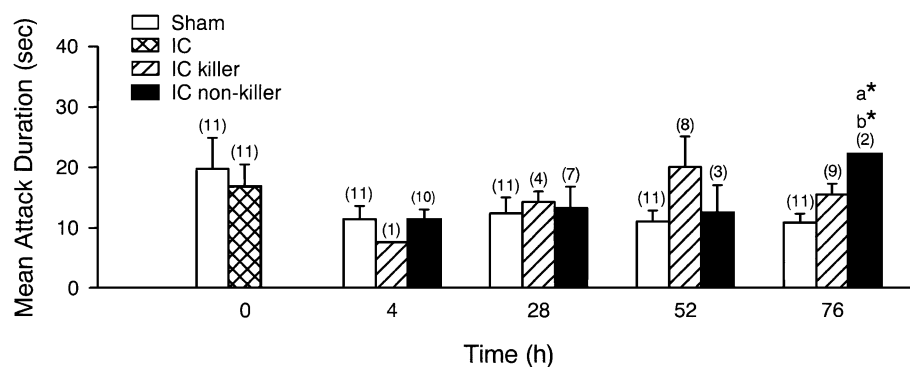


Fig. 6. Effects of IC on mean attack duration in rats during the 20-min observational period. \* $P < .05$ , (a) compared with the sham-operated control group; (b) compared with the IC killer group at the given time-point. The number of rats tested at the different observation points is indicated in parentheses. Time 0 h indicates IC surgery or sham operation.

behavior are usually limited to comparing changes in the percentage of killers [12,13].

Muricidal behavior in rats is composed of two main components, attacking and killing, and the incisors play a key role in the muricidal action. When they had no, or very short, incisors at 4 and 28 h after IC, 10/11 and 7/11 rats, respectively, failed to kill mice (Fig. 2). In contrast, 82% of rats killed at 76 h after IC (Fig. 2), at which time-point the incisors had recovered to approximately 60% of their original length (Fig. 1B). These results clearly indicate that killing ability in rats depends on the presence of incisors.

Since the percentage of killer rats decreased significantly at 4 and 28 h after IC, we will focus our discussion on the data collected at these two time-points in IC nonkiller rats. Although no remarkable changes in attack latency (Fig. 3), mean attack duration (Fig. 6), first attack site (Table 1) or lethal attack site (Table 2) were seen in these rats as compared to their sham-operated controls at these two observation points, the attack frequency (Fig. 4) and total attack duration (Fig. 5) increased significantly, as the IC nonkiller rats still attacked mice. Thus, IC suppressed the killing part of muricidal behavior, but preserved most of the basic attacking components of this behavior and, in fact, enhanced some of these. This model will therefore be helpful in analyzing muricidal behavior.

The IC-induced suppression of killing was reversible. Four hours after IC, only 9% (1/11) of rats killed mice during the observation period. However, 36% (4/11) of rats killed mice 28 h after IC, and these IC killer rats showed no differences in attack frequency and mean attack duration compared with control animals (Figs. 4 and 6), indicating total recovery of muricidal behavior. Even with cut incisors, four rats in this group were able to grip the cervical region of the mice and choke them to death. Seventy-six hours after IC, 82% (9/11) of rats recovered their killing ability (Fig. 2), and this may be related to the rapid recovery of their incisors, which grew back to about 60% of their original length within 76 h (Fig. 1B). In fact, killing ability was totally restored in all rats 100 h after IC (data not shown). However, the possibility that repeated testing might also participate in the recovery of killing ability after IC could not be excluded.

IC treatment did not affect the first attack site (Table 1). However, the proportion of rats attacking nonlethal sites was increased (Table 3), with more than 50% of IC-treated rats attacking nonlethal sites. Since the IC-treated rats could not kill rapidly, the mice had a greater opportunity to show defensive behavior, which included standing up and facing the rats, boxing with the forepaws or lying down with the neck and head on the floor with the tail pointing towards the rat (including kicking with the hind limbs). All these defensive postures seem to protect lethal attack regions of the body from the rat's mouth. When the rat could not attack these regions, it bit nonlethal sites to force the mouse to change its posture, then, if the mouse tried to run away and exposed the lethal sites, the rat took the opportunity to attack these sites.

In the present study, the food pellets were cut into small pieces for rat's easier chewing, and the body weight of rats after IC was not changed (Fig. 1A). Therefore, hunger, if any, could not be a major factor to affect the behavioral outcome. Since not all rats with OBX treatment display muricidal behavior and all killer rats used in this IC experiment were selected by a series of muricide behavioral tests after OBX, it implies that the bullectomy surgery on these killer rats in the current study should be adequate.

The advantage of using this IC-treated animal model to study muricidal behavior is the suppression of the mouse-killing components and the augmentation of the mouse-attacking components. Thus, this IC animal model provides the possibility of simultaneously investigating changes in neurotransmitters in the rat brain during muricidal behavior using microdialysis, a great improvement on the alternative of continuously introducing fresh mice so that the rat continuously expresses muricidal behavior during the observation period.

In conclusion, IC has little effect on the fundamental components of OBX-induced muricidal behavior, but can prevent the killing component and amplify the attacking component of this behavior. IC-treated killer rats may therefore be a suitable model for research on muricidal behavior.

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