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## BRIEF REPORT

# Effect of Food Deprivation upon Electrically Elicited Predation in the Cat[1]

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The present experiment examined the effects of food deprivation upon predatory attack elicited by electrical stimulation of lateral hypothalamic sites in the cat. Latencies for attack initiation and for object contact were assessed under *ad libitum* feeding conditions and compared, in a within-subjects design, to attack latencies following 72 hr of food deprivation. Both initiation and object contact latencies were significantly reduced by food deprivation, suggesting a relationshhip between food deprivation and predation. Latencies recovered to initial baseline levels following the reinstitution of *ad libitum* feeding conditions.

Behavioral evidence from a variety of species and several methods of food restriction suggests the possible existence of an inverse relationship between feeding and prey killing in naïve animals. For example, food-deprived rats and hamsters both show facilitated prey killing upon initial exposure to prey in comparison to nondeprived subjects (Malick, 1975; O'Boyle, 1974;

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Paul, 1972; Polsky, 1974; Rossi, 1975; Whalen, 1964). It might tentatively be concluded from these studies that central feeding and predatory circuits share at least some common neural elements.

Despite the above evidence, however, most attempted physiological demonstrations of a relationship between feeding and predation have heretofore produced only inconclusive results (O'Boyle, 1974; Polsky, 1974; Rossi, 1975). For example, although considerable anatomical overlap between central feeding and predatory loci has been demonstrated (Hutchinson and Renfrew, 1966), these sites are nonetheless dissociable by differing stimulation requirements and by functionally specific areas failing to show behavioral overlap (Flynn *et al.*, 1970; Hutchinson and Renfrew, 1966; King and Hoebel, 1968). Likewise, although hypothalamic lesions tend to produce dramatic disruption of both feeding and predation, the rates of recovery of these functions differ considerably (Karli *et al.*, 1969). There exists, however, another possible means of physiologically demonstrating a relationship between feeding and predation. It might be shown, for example, that food restriction altered predatory behaviors elicited by electrical stimulation of hypothalamic sites; such a demonstration would offer direct evidence to this point. The only previous experiment employing this strategy, in fact, suggested the existence of a relationship between electrically stimulated ranacide and food deprivation in spontaneously killing rats (Desisto and Huston, 1971). This report, however, noted only marginally significant increases in approach likelihood under conditions that caused significant increases in killing behavior and employed nonorthogonal measures of both variables. The present report expands previous observations to two orthogonal measures of attack in cats which did not spontaneously kill.

The subject pool for the experiment consisted of six adult female cats, weighing 2.0 to 3.5 kg, obtained from a local supplier (Bio Medical Associates, Inc., Freidensburg, Pennsylvania). All subjects were maintained on *ad libitum* food and water during baseline testing. Following establishment of a stable attack baseline[2] all subjects were food-deprived for 72 hr. Subjects were then tested and subsequently returned to *ad libitum* feeding conditions. Normal day/night cycles of 14 hr daylight/10 hr darkness were maintained by natural and artifical lighting.

Subjects were anesthetized via intraperitoneal injection of 35 mg/kg of sodium pentobarbital (Nembutal). Each subject was stereotaxically implanted with 12 stainless steel electrodes 0.25 mm in diameter insulated to the tip. In addition, two indifferent electrodes were attached to stainless steel screws in

[2]In addition to attack latencies, all cats were initially examined for stimulus-bound feeding. Two subjects showed stimulus-bound eating at their respective attack thresholds. All other subjects could be induced to attack and bite food (as typically reported by Flynn *et al.*, 1970) with more intense stimulation.

the skull for purposes of monopolar stimulation. All electrodes were aimed at the lateral hypothalamic and the perifornical areas (Snider and Neimer, 1964). Surgery was performed under aseptic conditions. At the close of surgery, 150,000 units of Bicillin was administered intramuscularly.

During testing for predatory attack, subjects were maintained in a specially constructed apparatus that allowed for continual testing of the subject with a minimum of interference from the experimenter. The aggression apparatus consisted of a 50 X 32 X 111-cm wooden box with a central barrier 7.5 cm in height and a grid floor made of aluminum dowels. Apertures at either end of the apparatus allowed an attack object (a small stuffed toy 14 cm in length resembling a squirrel) to be maintained in constant irregular motion at the end of the box distal to the subject by means of one of two motor-driven rods. A 2HBA-1 Unimax microswitch with a pressure requirement of 28 g and a release requirement of 21 g was mounted at the end of each rod. An attack object was attached to each microswitch by nylon fishing line.

To assess changes in approach, two sets of photocells (Sigma Models 8-L-3 and 8-P-3) were mounted 14 cm above and 4 cm distal to the central barrier, along the width of the box. Doors of the apparatus consisted of one-way mirrors through which visual observations of attack could be made. A constant level of 20.0 db of white noise was maintained throughout the experiment via a Grason-Stadler white-noise generator.

One week after surgery, animals were tested for response to stimulation. Only sites that consistently yielded directed attack culminating in biting directed at the neck, and from which affective display was absent, were selected for additional testing. During initial testing and throughout the course of the experiments, stimulation consisted of monophasic 1.0-msec duration pulses, delivered unilaterally through a 2-$\mu$F capacitor in series with the animal to provide for reversal of current. Pulses were delivered 150 pulses per second in a 10-sec train, with 120- to 150-sec intervals between stimulations. All stimulation was delivered by a Grass SD-9 stimulator and monitored across a 100-ohm resistor in series with the subject, on a 122-A Hewlett-Packard oscilloscope. Current levels were determined immediately prior to each stimulation by the presentation of a single 1.0-msec pulse.

Based upon initial protocols, subjects were tested for attack behaviors in the aggression apparatus. A single threshold level of current was employed for *ad libitum*, deprivation, and recovery testing sessions for a given subject. Threshold current was defined as the lowest level of stimulation for the elicitation of at least one attack episode from two consecutive stimulations (attack was defined as approach culminating in terminal object contact) and was determined during the initial protocol by an ascending series of stimulations beginning at 0.05 mA and increasing in 0.05-mA steps. Two stimulations were administered at each level.

Two measures of approach were obtained for each stimulation. The first measure was the latency from stimulation onset to initial movement as measured by barrier crossing (initiation). The second measure was the latency from initial movement to object contact (locomotion). All latencies were timed electronically using Lehigh Valley 412-01 timers operated by the photcells and microswitches of the apparatus. In the event that a subject failed to cross the central barrier, initiation and locomotion scores were recorded as 10 sec each. In the event of barrier crossing without object contact, the locomotion score was recorded as 10 sec.

All subjects were initially tested under *ad libitum* feeding conditions. Following initial testing, all subjects were food-deprived for 72 hr and retested. All cats were subsequently allowed up to 7 days of *ad libitum* feeding and were then retested.

At the close of the experiment all subjects were sacrificed via an overdose of Nembutal. Subjects were perfused first with physiological saline and subsequently with 10% formaldehyde in saline. Histological examination of individual brains using the photographic technique of Skinner (1971) revealed that all placements were located in the lateral hypothalamic area.

All data were analyzed as within-subjects difference scores, using the Student's $t$ test (Dixon and Massey, 1969).

An index of the success of the food manipulation may be found in the significant decline of the mean weight of the subjects. Under *ad libitum* feeding, mean weight was 3.3 ± 0.1 kg; after 72 hr of food deprivation, mean weight was 2.6 ± 0.1 kg [$t(5) = 4.23$, $P < 0.02$]. Restriction of food also had significant effects upon approach latencies, as may be seen in Fig. 1. All

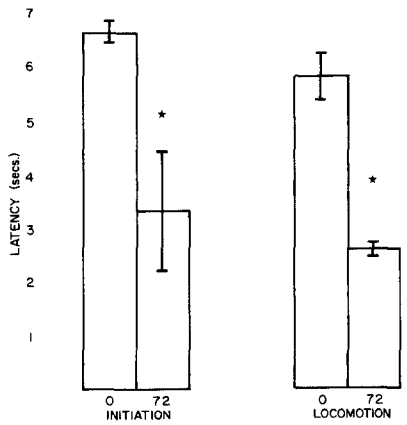


Fig. 1. Effects of food deprivation upon approach behaviors. 0 = *ad libitum* feeding; 72 = 72 hr of food deprivation; ★ = $P < 0.05$, within-subjects $t$ test, two-tailed criterion.

results are presented as means and standard errors of the mean. It may be seen that 72 hr of food deprivation caused reductions in both initiation and locomotion latencies at threshold current. Both differences are significant [$t(5) = 2.58$ and 3.31 for initiation and locomotion, respectively; $P < 0.05$].[3]

To simplify the presentation of data, recovery data are not presented graphically. For no subject, however, did predeprivation-postdeprivation latency scores differ significantly. Postdeprivation initiation and locomotion scores were 6.25 ± 3.83 and 5.34 ± 3.31, respectively. ($t$ scores for difference scores were −1.40 and −1.68, respectively. At the time these measurements were taken mean body weight had increased to 3.1 ± 0.9 kg.)

The present results may be taken as an extension of previous findings with regard to feeding effects upon predation. In addition. they suggest that food deprivation affects the approach behaviors in predation as well as in killing itself. These results are consistent with a variety of behavioral manipulations (Malick, 1975; Paul, 1972; Polsky, 1974) pointing to an induction of prey-killing in naïve subjects. The present results suggest that such increases are not unique to between-subjects designs and may be obtained for within-subjects designs, through the use of brain stimulation.

Finally, it might also be noted that previous reports (Flynn *et al.*, 1970; Karli *et al.*, 1969) have suggested a physiological dissociation of feeding and predatory mechanisms. Although feeding and predation are in part dissociable by stimulation requirements and anatomical fine structure, such partial dissociations do not preclude some degree of relationship, as previous results as well as the present findings demonstrate.

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[3]It might be objected that the present results are explicable in terms of an increase in general activity rather than a specific increase in attack behavior. It should be noted in this regard that visual observations of all subjects under food deprivation indicated no spontaneous barrier crossing. Rather, the cats remained calm and immobile until stimulated, at which point they attacked.

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