ship was traveling at between 19.6 and 21 knots. Many observations of short duration were made on 1 to 30 dolphins at a time as they were swimming with the ship, but after about 2 minutes at these speeds they dropped behind; this suggested that to sustain such swimming speeds was beyond their capability. Four large groups of dolphins were observed in calm seas, swimming at 14 to 18 knots for periods of 8 to 25 minutes. Details of these observations are given in Table 1. In at least two of these sightings the dolphins never came close to the ship or seemed to be deflected from their course by the presence of the ship. These data support the conclusion that dolphins 6 to 8 ft long can swim at a sustained speed of about 18 knots.

William Von Winkle, of the U.S. Navy Underwater Sound Laboratory, reported (*3*) that a school of blackfish (probably *Globicephala melana*) had been observed circling a Navy vessel, which was cruising at 22 knots, for several days at a time. They would pass the ship, go way out in front, and go back in the wake to look for food. They were 12 to 15 ft long. On the basis of model similiarity, a dolphin of this size, twice the length of the smaller animals, should have a muscle weight (and presumed power output), per square foot of surface area, twice as great. If the coefficient of surface drag were constant, this would lead to a speed $2^{1/3}$ ($= 1.26$) times as great, since the propulsive power increases as the cube of the speed; 1.26×18 knots (the sustained speed of the smaller, common dolphin) equals 22.7 knots, which comes very close to the speeds maintained by the blackfish.

The one killer whale *(Orcinus orca)*—actually a dolphin—that was observed from the *Monterey* traveled at speeds of 20.6 to 30 knots. If we assume that it was of average length, it should have been about 3 times the length of the 5- to 8-ft dolphin. On the basis of model similarities, this killer whale should be able to swim $3^{1/3}$ times faster than the 18 knots, or 1.44×18, or 26 knots. This value is about the speed at which the whale swam around the ship but less than the 30 knots at which it approached the ship. After playing around the ship for 20 minutes at a sustained speed greater than the 20.6 knots at which the ship was sailing, the killer whale continued on its original course at about the speed of the ship.

We suggest that under the assumption of a constant ratio of muscle power to muscle weight, the sustained speeds that have been observed imply that the coefficient of surface friction remains approximately constant for dolphins from 6 to 22 ft long. It seems probable that the capacity of these mammals for sustained work can be estimated more closely than their capacity for short-term high rates of work. Such estimates, together with the speed data of Table 1, should provide an improved basis for calculating the value of the surface drag coefficient.

Table 1. Observations made from the *Monterey* on sustained swimming ability of dolphins.

Approx. No. of dolphins	Date (1958)	Speed of dolphins (kn)	Length of time at observed speed (min)	Position of ship	Speed of ship (kn)	Comments
50	2 Nov.	18	8	32°S, 176°W	19.6	Some dolphins rode the bow wave. Large and small dolphins in group. Smaller ones came closest to ship.*
500	25 Oct.	14–16	20	34°S, 158°E	19.5	Group was traveling in same direction as ship when it was overtaken and passed. Group was about 0.25 mile away at closest approach.*
200	31 Oct.	18	10	34°10′S, 167°05′E	21	Some of the dolphins approached side of vessel but stayed 200 yd off.*
200–300	31 Oct.	17–18	25	34°15′S, 167°45′E	21	Dolphins were sighted several miles ahead of ship in calm sea. They remained about 0.5 mile away from vessel but swam on same course as vessel.*
1	12 Oct.	20.6–30	20	03°30′S, 141°45′W	20.6	Killer whale (about 20 to 25 ft long) approached ship at about 30 kn, swam back and forth around bow, veered away at about speed of ship.

* Dolphins were white-sided and about 6 to 8 ft long.

CARL L. JOHANNESSEN
Department of Geography, University of Oregon, Eugene
JAMES A. HARDER
Department of Civil Engineering, University of California, Berkeley

References and Notes

1. J. Gray, *J. Exptl. Biol.* **13**, 192 (1936).
2. S. Brody, *Bioenergetics and Growth* (Reinhold, New York, 1945).
3. W. Von Winkle, personal communication to J. A. Harder, 9 Jan. 1959.

12 July 1960

Interpeduncular Nucleus and Avoidance Conditioning in the Rat

Abstract. Rats trained to make a jumping response to the onset of a visual stimulus lost the habit after damage to the interpeduncular nucleus of the midbrain. It was noted, however, that the majority of the operated animals showed perfect retention of the "fear" response to the conditioned stimulus.

Thompson and Massopust (*1*) have demonstrated that damage to the interpeduncular nucleus of the mesencephalon abolishes a previously established brightness discrimination in the rat. More recently it has been found that this nucleus also participates in mediating a kinesthetic discrimination as well as an auditory habit (*2*). Because of the paucity of anatomical, physiological, and psychological data related to the interpeduncular nucleus, its function in learning and retention is unknown. Some enlightenment was obtained in the study reported here, in which the effects of lesions in the interpeduncular nucleus on retention of an avoidance response were studied (*3*). Of particular interest was the finding that although damage to this nucleus profoundly disturbed the subsequent performance of the avoidance response, the fear-producing properties of the conditioned stimulus were spared.

Nineteen adolescent albino rats were trained to make a jumping response to the presentation of a visual stimulus. The apparatus, which consisted of an enclosed box made of Plexiglas, was located in a light-proof, sound-attenuated room. The box measured 6 inches on either side and was 10 inches high. The floor of the box was made into a grid composed of bronze rods. A space of ½ inch existed between the top of the walls and lid of the box.

This space permitted the rat to grip the edge of the walls with its forefeet and raise itself, thus escaping or avoiding the charged grid. The box was mounted between two 100-watt frosted light bulbs.

A trial consisted of a 5-second presentation of the light, followed by a combination of light and shock until the subject succeeded in making the appropriate response. If the subject made a conditioned response (gripping the edge of the walls during the initial 5-second presentation of the light), the light was immediately turned off and the shock postponed. Twenty to twenty-five trials were given daily, with an intertrial interval ranging from 30 to 150 seconds (mean, 90 seconds). Training was terminated when the subject reached the criterion of nine conditioned responses within a series of ten trials. Approximately 4 hours after learning, 12 animals were subjected to electrolytic lesions in the interpeduncular nucleus, while the remaining seven received only sham operations (controls). After a recovery period of 7 days, all subjects were required to relearn the avoidance response under the same conditions as those described in original learning. After the experiment, histological verification of the location of the lesions was accomplished (*4*). Details of the surgical and histological procedures may be found elsewhere (*1*).

Five of the 12 experimental animals had less than 20 percent damage to the interpeduncular nucleus. These animals achieved a mean trial savings score of 82 percent which is comparable to the retention performance of the controls (mean of 85 percent). That minimal damage to the interpeduncular nucleus has no effect on retention is in agreement with previous findings (*1*, *2*). The remaining seven experimental rats had lesions destroying from 35 to 85 percent of the nucleus; these rats earned an average savings score of −61 percent (Fig. 1 *A*). This retention score is significantly inferior to that of the controls beyond the .01 level. However, observations of these animals on the first day of the retention test indicated that some relevant memory of the conditioning situation was present. On the first trial of the retention test, five of the seven experimental animals made definitive "fear" responses to the presentation of the light prior to the onset of shock (see Table 1). These responses consisted mainly of squeaking, which was sometimes accompanied by abrupt changes in respiration and upward orientation of the head. In four rats, these fear responses elicited by the light occurred during every trial on the first day, but were never followed by a conditioned jumping response. These observations are not readily explicable on the basis of a motor disturbance. The unconditioned response (jumping in response to the onset of shock) exhibited by the experimental animals was executed as smoothly and with the same latency as that exhibited by the controls. Similarly, these data are not accountable in terms of a heightened sensitivity to the light (*5*). No disturbances in alertness or wakefulness were apparent in any of the experimental animals.

Table 1. Percent damage to the interpeduncular nucleus and performance scores for seven rats.

Rat No.	Damage (%)	Trials		Trial savings (%)
		Learning	Relearning	
3	85	20	60	−200*
10	35	53	95	−79*
15	45	74	61	18*
17	35	46	17	63
33	45	53	60	−13*
42	45	21	48	−129
79	55	43	81	−88*

* The animal exhibited fear responses on the first trial of the retention test.

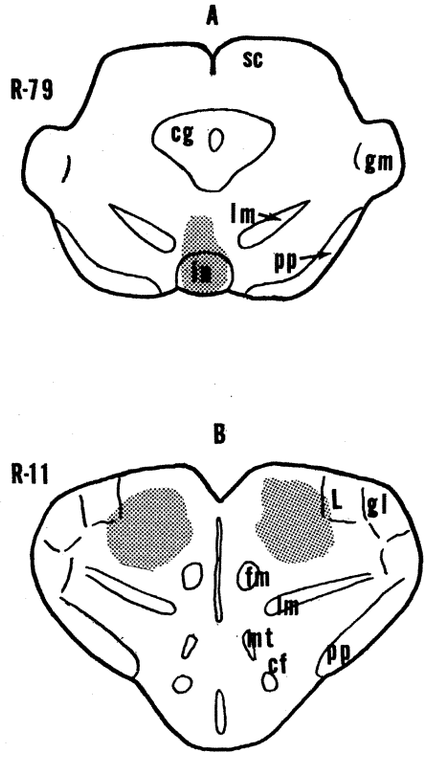

Fig. 1. Camera drawings of sections showing lesions (stippled area) in the interpeduncular nucleus (*A*) and the posterior thalamic region (*B*). Symbols: *cg*, central gray; *cf*, column of the fornix; *fm*, fasciculus retroflexus; *gl*, lateral geniculate nucleus; *gm*, medial geniculate nucleus; *in*, interpeduncular nucleus; *L*, lateral nucleus; *lm*, medial lemniscus; *mt*, mammillo-thalamic tract; *pp*, basal peduncle; *sc*, superior colliculus.

These observations are intelligible in terms of the two types of learning that take place in avoidance conditioning (*6*). The first type, frequently referred to as "visceral" conditioning, occurs early in training and is manifested by such responses as vocalization, urination, defecation, sudden motor jerks, and crouching. These responses evoked by the conditioned stimulus clearly reflect the animal's anticipation of pain. In the present experiment these anticipatory reactions were quite noticeable, in most cases, by the tenth learning trial. The second type of learning ("skeletal" conditioning) constitutes the adaptive response which effectually allows the animal to avoid the pain. This response, in the present experimental situation, became stabilized after about 50 trials. Presumably, lesions in the interpeduncular nucleus disrupt the neural pathways involved in this second type of learning, while sparing those pathways involved in the first type.

I subsequently endeavored to determine whether damage to other brain areas critical for visual performance would result in the same differential effect. The visual cortex was first investigated. Of the six rats with virtually total bilateral ablation of the visual cortical areas, three showed anticipatory fear responses to the onset of light on trial one. (One additional rat began exhibiting fear responses on trial two). However, these results are not altogether comparable to those obtained with lesions in the interpeduncular nucleus, since extirpation of the visual areas only partially impaired the conditioned jumping response. The animals with cortical damage earned an average-savings score of 49 percent. This score is significantly inferior to that of the controls ($p = .05$), but is significantly superior to the performance of the seven animals with lesions destroying at least 35 percent of the interpeduncular nucleus ($p = .05$).

Finally, attention was focused on the posterior thalamic area of the brain (see Fig. 1 *B*). This region of the thalamus medial to the lateral geniculate bodies has been found to be highly critical for visual discrimination performance (*1*). Ten rats with bilateral lesions in the posterior thalamus revealed a mean trial-savings score of −150 percent, a score which is very significantly inferior to that of the controls. Of these ten rats, only one showed anticipatory fear responses to the light during the early trials on the first day of the retention test. By the use of Fisher's exact probability test, the frequency of fear responses shown by these animals was found to be significantly less than that exhibited by the seven animals with moderate dam-

age to the interpeduncular nucleus ($p = .05$).

The foregoing results strongly support the notion that two different neurological systems function in the retention of avoidance conditioning. The neural system involved in visceral conditioning probably contains the posterior thalamic region as one component, but does not include the interpeduncular nucleus or the visual cortex. In contrast, all three anatomical structures studied in this report are implicated with the neural system involved in skeletal conditioning, particularly the interpeduncular nucleus and the posterior thalamic area.

ROBERT THOMPSON*
Department of Psychology, George Peabody College for Teachers, Nashville, Tennessee

References and Notes

1. R. Thompson and L. C. Massopust, *J. Comp. and Physiol. Psychol.*, in press.
2. R. Thompson, *et al.*, *ibid.*, in press.
3. This study was supported in part by research grant M-2529 awarded to the Department of Psychology, George Peabody College, from the National Institute of Mental Health of the National Institutes of Health, U.S. Public Health Service.
4. I acknowledge the assistance of Rita Thompson for preparation of the histological materials.
5. A control group of six untrained rats with moderate damage to the interpeduncular nucleus (mean damage of 48 percent) did not reveal any squeaking responses to the onset of light during ten presentations in the conditioning apparatus.
6. O. H. Mower, *Learning Theory and Personality Dynamics* (Ronald, New York, 1950).
* Present address: Neuropsychiatric Institute, University of California Medical Center, Los Angeles.

13 June 1960

Possible Explanation of Fluoride-Induced Respiration in Chlorella pyrenoidosa

Abstract. Low concentrations of sodium fluoride significantly increase oxygen consumption and total phosphorylated nucleotides in respiring *Chlorella pyrenoidosa*. Measurements of gas exchange at several *p*H values indicate that the stimulation is probably related to the undissociated hydrogen fluoride concentration in the suspending media.

Although fluoride has long been regarded as an inhibitor of respiration, recent investigations have demonstrated that it will also stimulate respiration. The stimulatory effect is usually produced at low concentrations. This phenomenon has been reported for algae (*1*), yeast (*2*), seedlings (*3*), and leaf tissue (*4*), but little interpretation has been offered as to the processes involved. The investigation reported here was an attempt to relate the fluoride-induced increase in oxygen uptake with some phase of the metabolism of *Chlorella*.

Chlorella pyrenoidosa Chick, Emerson strain type D, was grown at 24 to 26°C in a modified Knop's solution with added micronutrients. Iron was supplied as the salt of ethylenediaminetetraacetic acid. Air enriched to 3 percent CO_2 was continually supplied to the cultures. Unilateral illumination was provided by daylight-type fluorescent tubes, which supplied 600 ft-ca (6480 lux). After 72 hours' growth in constant light the cultures were subjected to 12-hour cycles of light and darkness for 48 hours and were harvested 6 hours after the last dark period.

The cells were harvested by centrifugation at approximately 500*g* for 15 minutes. The packed cells were resuspended in a volume of distilled, sterile water equal to the volume of packed cells. Cell counts were made on the resuspended algae, and all measurements were referred to these cell counts. Manometric measurements of the gas exchange in respiring *Chlorella* suspensions were made in 15-ml Warburg vessels in total darkness at 25°C. After a 110-minute treatment with NaF in 0.022*M* phosphate buffer at *p*H 4.0 it was apparent that $1.05 \times 10^{-4}M$ NaF produced an 8-percent increase in oxygen consumption and $1.05 \times 10^{-3}M$ NaF produced a 60-percent increase, while at $1.05 \times 10^{-2}M$ the rate was reduced to less than 70 percent of that of the nontreated system.

Experiments at other *p*H values, from 4.0 to 7.0, did not yield comparable results when the rate of oxygen consumption was compared to the total fluoride concentration of the solution. However, if the oxygen consumption was plotted relative to the calculated concentration of the undissociated HF, the results at different *p*H values were comparable (Fig. 1). The respiratory quotient did not vary greatly in the treated system as compared to the control.

To further define the mechanisms of fluoride-induced stimulation of oxygen uptake, the phosphate metabolism-respiration relationship was investigated. Respiratory-rate measurements were made on algae suspended in a nonbuffered water system in which the *p*H was initially adjusted to 4.0 with HCl or KOH. Similar suspensions of algae were placed in 40-ml test tubes that were attached to the manometer supports, and from these, aliquots were removed for phosphate determinations. Fluoride was added as sodium fluoride at zero time.

Measurements of phosphorylated nucleotides were made with a modification of the Norit A method (*5*). This procedure excludes sugar phosphates and other phosphorylated metabolic intermediates. The determinations of inorganic phosphate, after hydrolysis, were made according to the Fiske-Subbarow method (*6*).

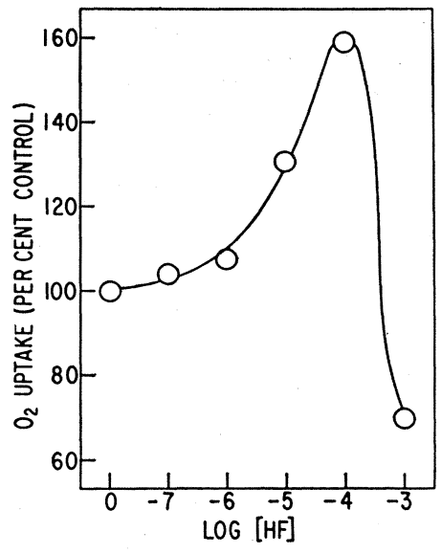

Fig. 1. Oxygen uptake in relation to the calculated HF concentration. The total fluoride concentration varied from 1.05×10^{-3} to $1.05 \times 10^{-1}M$ at *p*H 7.0, and from 1.05×10^{-4} to $1.05 \times 10^{-2}M$ at *p*H 4.0 in 0.022*M* phosphate buffer.

Concentrations of fluoride of 1.05×10^{-4}, 1.05×10^{-3}, and $1.05 \times 10^{-2}M$ increased oxygen consumption and at the same time increased the total phosphorylated nucleotides (Fig. 2). At $1.05 \times 10^{-4}M$ the oxygen consumption increased to 107 percent of that of the

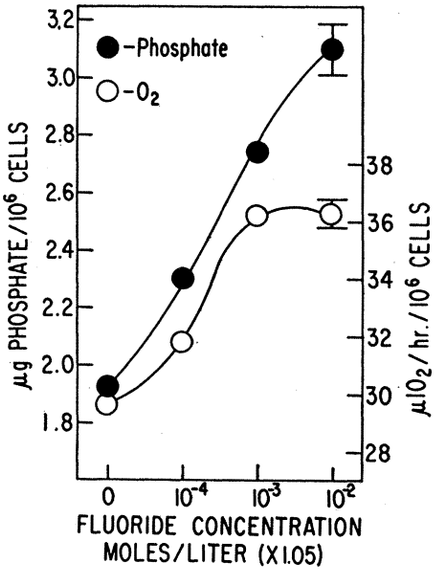

Fig. 2. Total nucleotide phosphate and rate of oxygen uptake in different concentrations of fluoride in the suspending solution. Undissociated HF concentrations were not calculated since this was a nonbuffered system. Bars at terminal points indicate 95-percent confidence limits.