elimination is the following. The evacuation of the rectum of its contents is a reflex action which is partly a function of the sacral division of the parasympathetic system. In addition to reflex response produced by local distention of the rectum, involuntary defecation may result from emotional discharges over the sacral nerves (Lindsley, 1951). The role of the sympathetic system, as in many physiological functions other than defecation, is usually the reverse of the parasympathetic. Thus the sympathetic tends to cause retention of contents in the rectum, while the parasympathetic tends to promote evacuation of contents by causing relaxation of the sphincters and contraction and propulsive motility of the rectal walls. The foregoing account suggests that the greater emotional elimination of the MR strain in the open field is associated with a functionally active part of the parasympathetic system, i.e., the sacral division.

Furthermore, the relationship between the autonomic nervous system, in particular the parasympathetic division, and ulceration has long been established (Cushing, 1932). Recently, Francois & Sines (1961), using an antiserum to a nerve growth protein which selectively reduces the number of cells in the sympathetic chain ganglia of newborn rats, found that the severity of restraint ulceration in rats so treated was greater than in control groups. They interpreted this result as "further support for the notion that parasympathetic dominance is involved in the susceptibility of the rat to immobilization induced stomach lesions." It is known that sympathetic influences are inhibitory to gastric function (Cannon, 1915). Hence, if the sympathetic activity were predominant in the MR rats, ulcer severity would have been lower in this strain. This was not born out by the result of the present study. From what has been cited it may be concluded that (1) the greater emotional elimination of the MR strain appears to reflect greater parasympathetic activity of the sacral division under certain stressful conditions, and that (2) the greater parasympathetic discharge may partly account for the predisposition of the MR strain to ulcer formation.

Follow-up studies wherein the MR and MNR strains were tested for stress-induced ulceration confirmed the findings of the present report. It is worth noting that a comparison of data obtained recently from about 150 Sprague-Dawley and Wistar rats with those of the Maudsley strains indicated clear higher ulcer susceptibility in the MR rats noticeable in both the incidence and character of lesions. Of particular interest in this regard was the location of ulcers. While the ulceration of the Maudsley strains developed mainly in the antrum, no lesions ever appeared in this part of the stomach of Sprague-Dawley and Wistar strains.

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NOTES

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Blood pH as a possible hunger cue: Negative evidence¹

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An investigation was made into the role of blood pH in the regulation of food intake. Rats were each subjected to five treatments: acid injection, base injection, two saline injections, and no-injection control. Eating behavior was monitored directly after these treatments. The negative results indicated that blood pH probably does not serve as a hunger cue.

The inability of present theories of hunger regulation to account for all of the available data suggests that perhaps some chemical cue plays a yet undiscovered role in

hunger regulation (Smith, 1966). Hydrogen ion concentration of the blood (usually talked of in terms of its pH or negative logarithmic function) is here investigated as that possible chemical cue.

Upon ingestion of foodstuffs, the parietal cells of the stomach's lining produce a concentrated HCl secretion which enters the stomach and, at the same time, bicarbonate ions which enter the blood. It has been shown that blood leaving the stomach during gastric secretion is significantly more alkaline than the entering blood, thus accounting for the post-meal "alkaline tide" of blood pH found in humans (Hubbard, 1927) and in dogs (Gyorgy & Kleinschmidt, 1927).

Thus, while blood pH and eating do

Table 1

Amounts of HCl and NaHCO₃ necessary to change blood pH 0.2 acidic and basic respectively with respect to body weight. Calculated from Ruch & Patton (1965).

Body Weight	HCl Injection	NaHCO ₃ Injection
400 g	5.45 cc	9.20 cc
420 g	5.72 cc	9.66 cc
440 g	5.99 cc	10.12 cc
460 g	6.25 cc	10.57 cc
480 g	6.51 cc	11.02 cc
500 g	6.78 cc	11.48 cc

covary, apparently no attempt has been made to suggest it as a causal factor in hunger. There are several reasons why a blood pH mechanism may be considered tenable in food intake regulation. It is likely in the regulation of food intake that some blood-borne mechanism is involved and blood pH shifts are of such a direction, magnitude, and time course as to suggest themselves as a possible mechanism. Too, it has been noted that there are centers in the nervous system sensitive to pH variations. Also of relevance in this regard is the finding that cancer patients show normal appetites as long as they possess the ability to secrete HCl in their gastric juice, but lose their desire to eat as soon as this ability is lost (Gilmour, 1958). Finally, the finding that exercise leads to a marked increase in the acidity of the blood may be related to the increased ingestion of food that must follow if an energy balance is to be maintained.

The combined implications are that changes in the hydrogen ion concentration of the blood seem at least tenable as a possible satiation and/or hunger cue. The "alkaline tide" after eating would imply a possible satiating effect of lower H⁺ concentrations while the exercise data would indicate a hunger effect associated with higher H⁺ concentrations. The experimental test of this hypothesis was to independently manipulate blood pH and note concomitant eating behaviors in rats.

SUBJECTS

Sixteen large (400-500 g) male Wistar rats, 150-300 days old, served as Ss. Of these, 10 were used in the data analysis. The reasons for discarding the six unused animals were as follows: two did not eat after the operation, one because of equipment malfunction, and three had their venous catheters come loose.

PROCEDURE

Subjects were individually housed in Skinner boxes and trained to bar press for 45-mg food pellets on a FR 1:1 schedule. The Ss were then placed on an automated feeding schedule in which the white noise signaled 50-min eating periods twice daily, spaced 12 h apart. Water was made available at all times. After the response rate was stabilized (about 8 days), they were

operated on and a two-way Silastic venous catheter was inserted into the external jugular vein and pushed 1 or 2 mm into the right atrium of the heart. The other end of the catheter (of PE-50 tubing) was brought around under the skin and exteriorized at the back of the neck so that it protruded about 3 cm. A PVP-heparin mixture was used to fill the catheter when it was not in use to prevent blood from clotting in it.²

Each S was given a total of five treatments, on five consecutive days. Two of these conditions were acid and base injections. The amounts of osmolar (.167 M) acid (HCl) and osmolar (.167 M) base (NaHCO₃) that were necessary to change the pH downward 0.2³ for the acid injection and upward 0.2 for the base injection according to body weight were calculated (Ruch & Patton, 1965). (See Table 1.) Since the amounts of base were greater than the amounts of acid required for equivalent but opposite pH shifts, two isotonic saline control conditions were instituted. One saline injection control matched the amount of HCl injected and the other matched the amount of NaHCO₃ injected. All injection solutions were at body temperature. The fifth and final treatment was a no-injection control condition.

Four days after the operation, the experimental sequence began. Just prior to the normal evening eating period, a 25-cm extension of PE-50 tubing was connected to the catheters. Then a .25-cc sample of mixed venous blood was withdrawn into a plastic syringe coated with heparin. In an injection condition the animal received an injection of HCl, NaHCO₃ or saline via a Lambda pump at .6 ml/min. Thus the injection periods lasted from 9-18 min. After the injection was completed, or after a delay of 5 min in the no-injection condition, another blood sample was collected as before. At meal time the white noise signal was activated and Ss' evening meal began. The number of bar presses during the next 50 min was recorded. After this meal another blood sample was collected. The blood samples were stored and cooled and within 1 h were analyzed for pH on an Instrumentations Laboratory pH meter.⁴

RESULTS AND DISCUSSION

The averaged blood pH curves revealed that the shifts due to the acid and base injections were real and close to predicted values: The observed average pH shifts were 0.25 for acid and 0.23 for base. Variability was slight. In fact, immediately after injection, the pHs of the animals in the acid and base conditions were entirely out of the range of the control animals' pHs. After 50 min the pH shift was still very evident in the base condition but the fact that blood sampling apparently lowers pH slightly

Table 2

The average number of 45-mg food pellets consumed in relation to experimental condition.

Condition	Average Number of Pellets Ingested
Acid Injection	104.1
Base Injection	93.8
No Injection	109.2
Saline Control (Acid)	81.0
Saline Control (Base)	82.5

obscures the difference in the acid condition. This is probably not crucial since nearly all of the Ss completed their eating within the first 20 min of the 50-min eating periods.

The analysis of the eating data was via a 5 by 5 Latin square design independently replicated twice (Edwards, 1960). The analysis revealed no significant differences between any of the treatments ($F = 1.252$, $p > .05$, $df = 4/32$), thus supplying evidence against any pH chemical cue in hunger regulation. It is possible to argue that the acid and base injections made the animals sick and unable to eat normally, but the eating data revealed that animals in the no-injection and saline conditions did not eat significantly more food (see Table 2). It thus seems that rats are able to regulate their food intake in spite of slight variations in blood pH rather than because of them.

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NOTES

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- Technique developed by J. D. Davis.
- This pH shift is somewhat greater than a pH shift due to eating but of a magnitude comparable to that developed in strenuous exercise. A pH shift of this magnitude was selected to keep the shifts equal. While the shifts are sizable they are merely at the extremes of the normal range and do not cause a noticeable sickness. In addition, pilot work revealed that pH shifts of this size could be maintained for 1 h.
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