enced after about the sixth day of starvation, but this was regarded as involving a depression of appetite rather than hunger. Cannon did not appear to distinguish between hunger and appetite in connection with prolonged starvation.

In further studies of the effect of fasting 10–41 days by human subjects (*8–11*), it was found that hunger persisted throughout the fasts, whether it was considered as the pangs produced by the periodic gastric contractions or as a centrally produced impulse to eat (*12*). The kymographic record of a period of gastric hunger contractions obtained on the fortieth day of fasting was published in 1927 (*9*), and data concerning the periodic gastric motor and secretory activity obtained without the use of an inflated balloon in the stomach, as well as data regarding sensations experienced during fasts of 33 and 41 days, were published in 1944 (*11*). In dogs and rabbits, the periodic gastric hunger contractions were likewise found to persist during prolonged starvation (*5, 6*). Some rats, after fasts of 15–25 days, were so voracious that they died, apparently from overeating, within a few hours after being supplied with food (*13*). Their stomachs were greatly distended with food, but evidently little was digested and absorbed. Hibernating animals resume eating after prolonged abstinence from food, presumably because of hunger.

In man, hunger sensations nevertheless usually appear to become modified or less acute after the first few days of fasting. The complete disappearance of hunger in some hunger-strikers and sick individuals who resort to the fasting cure cannot be regarded as normal. Observations made by the junior author (*14*) indicated that the acute hunger sensations experienced during the first few days of fasting after living on a mixed or high carbohydrate diet are chiefly due to carbohydrate starvation or a lack of adaptation to living on a purely carnivorous diet, such as one actually lives on while fasting. Evidence that hunger is influenced considerably by carbohydrate starvation or the blood sugar level was previously obtained by Bulatao and Carlson (*15*) in a study on dogs, and more recently by Mayer and Bates (*16*) in a study on rats.

Hoelzel (*11*) also found that the contractions of the fasting stomach were felt only when the contents of the stomach were less than about 5 cc, and that the emptying of the fasting stomach became complicated by increasing reverse peristalsis or regurgitation of the duodenal contents after the first few days of fasting. Under such circumstances, gastric pangs of hunger were not felt, but the desire to eat was experienced, with increased restlessness, general weakness, and, eventually, some degree of nausea. Janowitz and Grossman (*17*) apparently did not regard such "hunger sensations" as related to the periodic fasting gastrointestinal activity. Hunger, or the desire, impulse, or drive to resume eating, normally increases again after the initial acute sensations decrease. Advocates of the fasting cure regard the return of hunger (normal, natural, or instinctive hunger) after the early disappearance of "hunger" ("habit-hunger" or "false appetite") as evidence that health has been restored by fasting and that eating should be resumed. The normally increasing desire to resume eating or the return of hunger with the prolongation of fasting appears to be mainly due to protein starvation. Protein starvation alone was found to give rise to the most acute type of epigastric hunger sensation (*11*). In rats and mice protein starvation produced peptic ulcers (*13*). In short, hunger in some form normally seems to persist about as long as life is maintained.

A. J. CARLSON
F. HOELZEL

Department of Physiology
University of Chicago

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Escherichia coli in the Intestine of a Wild Sea Lion[1]

LITTLE literature is available concerning the presence of *Escherichia coli* in the intestinal tract of marine mammals. Coliforms have been found in polar bears (*1*) but were not confirmed to be *E. coli*. ZoBell (*2*) states that *E. coli* does not appear to be a normal inhabitant of the intestines of seals in captivity. Recent work by the authors, however, suggests that *E. coli* is commonly found in the intestines of captive seals maintained in either fresh or sea water. This organism is a very common contaminant of stored marine fish (*2*) and may be introduced into the seal's alimentary system by the feeding of such material. Therefore, when a wild sea lion (*Zalophus californianus*) was captured near the Scripps Institution of Oceanography, an attempt was made to determine whether *E. coli* might be present under natural conditions.

The specimen, an adult female, was sacrificed, and samples were removed aseptically from the stomach and from the initial, central, and terminal sections of

[1] Contribution from the Scripps Institution of Oceanography, New Series No. 559.

the intestines. Difco Levine EMB agar, prepared both with fresh and with sea water, was used as the isolating medium. The medium was poured into plates and streaked with the samples from the sea lion.

After 24 hr incubation, at both 27° and 37° C, the EMB plates were examined, and the presence of coliform organisms was established in both the lower end of the small intestine and in the entire large intestine. The stomach appeared to be sterile. Typical coliform organisms appeared on the sea-water medium, but the colonies on fresh-water medium lacked their differentiating metallic sheen. All other features, including routine differential media, indicated that the organisms were *E. coli*. The isolated *E. coli* cultures grew as well, at either 27° or 37° C, on sea-water as they did on fresh-water medium; hence these organisms may have been indigenous to the sea or were more resistant forms from terrestrial contamination. *E. coli* will normally tolerate limited exposure to sea water, but the conventionally known strains are reportedly either killed quantitatively or diluted to virtual extinction by sea water in full strength (*2*).

CARL H. OPPENHEIMER
ARTHUR L. KELLY

Scripps Institution of Oceanography
University of California, La Jolla

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Autohemagglutinins in the Serum of Patients with Acute Lupus Erythematosus

A RECENT note by Schleicher (*1*) described a test for acute lupus erythematosus, in which Group O Rh-positive erythrocytes were agglutinated by the patient's serum in a saline solution of egg albumin. The agglutination disappeared at room temperature and at 37° C but reappeared at refrigerator temperatures.

Fjelde (*2*), using a modification of Schleicher's procedure, has reported detecting the erythrocyte aggregation factor in the serum of 17 out of 17 patients with the acute form of lupus erythematosus. The factor was not identified.

The similarity of the tests used by Schleicher and Fjelde to those usually employed to detect autohemagglutinins in the serum of patients presenting primary atypical pneumonia syndromes, hemolytic syndromes, and occasional other disorders (*3*) prompted us to investigate the matter further. In addition, we had previously noted that autohemagglutinins active at refrigerator temperatures were frequently present in the serum of patients suffering from acute lupus erythematosus.

Samples of blood were obtained from 7 patients with acute disseminated lupus erythematosus, 2 with noncongenital hemolytic syndromes, 3 with miscellaneous disorders—including 1 patient with pneumonitis presumed to be of viral origin—and 20 persons whose serum did not contain autohemagglutinins.

The method used in our laboratory seldom gives positive results with serum from normal persons; it consists of serial dilutions of serum in 0.9% sodium chloride solution, ranging from 1:4 through 1:2048, to which is added 0.1 ml 2% suspension of washed O Rh-positive erythrocytes. The tubes are refrigerated overnight and examined for the presence of agglutination. The foregoing test was performed on samples of serum from all the cases. Venous blood was allowed to clot at room temperature, and the serum was removed and used at once, or was frozen for use in the near future.

The method described by Schleicher was also used on duplicate samples and at the same time. To 1 ml refrigerated 10% egg albumin in 0.9% sodium chloride solution, 0.5 ml serum was added, and the fluids were mixed. One drop (0.05 ml) of a 10% suspen-

TABLE 1
RESULTS OF TESTS

Case	Clinical diagnosis	Cold agglutinin titer	Schleicher test	Schleicher test after absorption with O Rh + erythrocytes in the cold
1	Acute lupus erythematosus with hemolytic anemia	1:8	+	
2	" " " " " "	1:32	+	Negative
3	Subacute lupus erythematosus	Negative	Negative	
4	" " "	"	"	
5	Acute lupus erythematosus	1:8	+	"
6	" " "	1:16	+	"
7	" " "	1:16	+	"
8	Noncongenital hemolytic syndrome	1:8	+	
9	" " "	1:32	+	"
10	Hyperglobulinemia with multiple serologic abnormalities	1:8	+	"
11	Acute leukemia with indeterminate lesion in lung	1:16	+	
12	Pneumonitis	1:32	+	
13–32	Normal controls	Negative	Negative	