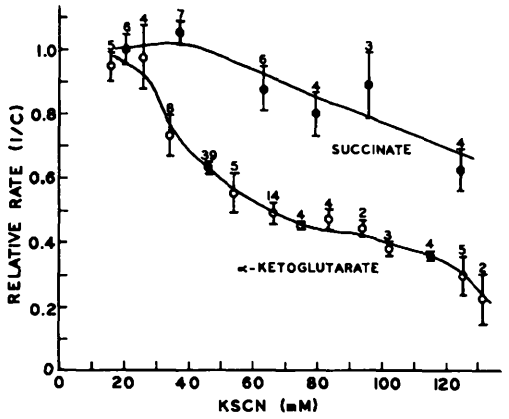

FIG. 4. Inhibition of oxygen consumption by high concentrations of SCN. Conditions are as given for Fig. 1. To enable averaging of data, the results for each 10 m*M* change in KSCN concentration have been grouped, and the average value of *I/C* plotted against the average KSCN concentration for that group. Bars represent ± one SE; the numbers are the number of determinations. Lines are drawn by eye.

If the inhibition of acid secretion in gastric mucosae is to be attributed to a direct effect of SCN^- on mitochondrial respiration, these experiments require either: (a) that the mitochondria in the gastric mucosa are more than 10 times as sensitive to SCN^- than are rat liver mitochondria, or (b) that a rapid and efficient mechanism exists for concentration SCN^- in the cytoplasm of the tubular cells. In addition, the effects of SCN^- are continued in the stomach, and show no recovery with time as is seen in isolated mitochondria. The concentration of SCN required to inhibit respiration of isolated mitochondria is more than 10-fold greater than that required to inhibit gastric acid secretion.

It therefore seems unlikely that the effects of SCN^- on the gastric acid secretion are explained by inhibition at the mitochondrial level as has been suggested. It appears that effects should be sought at the level of the acid secretory mechanism, as originally postulated.

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Effects of Inanition on a Nutritional Index and Cold Tolerance in Cats* (33415)

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Alterations in the ability to regulate body temperature have been shown to result from changes in the nutritional state (1–4). Thus, some measure of fatness or thinness is needed to insure that the changes found during cold exposure can be attributed solely to the environment. We have attempted to determine if the approach used by Von Pirquet (5) in the human or its adaptation to the dog by Cowgill and Drabkin (6) would be useful in the cat.

Methods. Changes in response to cold exposure were followed in six cats (two males

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and four nonpregnant females) as they were subjected to inanition over an extended period. The cold exposure tests consisted of placing the animals, restrained on their right side in a comfortable hammock, at an ambient temperature of 5 ± 0.5° for 3 hr. After the animals were placed in the hammocks for the control cold exposure tests, stem lengths (tip of nose to base of tail) were measured with an anthropometer and reported as the mean of three consecutive measurements. The animals were weighed before exposure, and rectal temperatures (T_r) were recorded at a room temperature of 24° and at the end of each hour in the cold. Body temperatures were measured with a calibrated thermistor probe inserted 10 cm into the rectum, and read directly from a portable bridge. Resting oxygen consumption rates, expressed as ml of O_2/kg/min, were taken at 24° and 5° on the control day. For these determinations each cat was confined in a sealed, small animal metabolic chamber and the O_2 consumption was measured by a closed circuit system. The most stable 6-min slope was selected from a 30-min test period to calculate the rate of O_2 consumption. Following the control measurements with the cats on regular laboratory cat food, the animals were restricted to a diet of 50 ml of evaporated milk diluted with 50 ml of water for a daily total of 69 kcal. Each cat was then tested at 5° each week following the above protocol. The terminal test for cold tolerance in any animal occurred when the T_r fell to 35° or lower. The day following the final test for cold tolerance, resting oxygen consumption rates were again measured at 24° and 5°. Statistical procedures were taken from Steel and Torrie (7).

Results. The results of the cold tolerance tests are shown graphically in Fig. 1. Rectal temperatures at 24° are shown as closed circles, those after three hr at 5° are open circles and the nutritional index as crosses. Animals in the cold maintained core temperature at a net mean cooling rate of 0.2° (—0.2 to 0.8)/hr until the final week when the mean rate rose to 2.9° (0.9 to 5.8)/hr and the T_r fell to 35° or below. In several of these tests it will be seen that on occasion the final T_r was higher than it had been in the previous week. Since the smooth progression of weight losses was interrupted at these points, it was suspected that the animals at times received additional food.

Mean rates of O_2 consumption are shown by the bar graph of Figure 2. In the initial tests, the O_2 consumption at 5° increased an average of 39.2% over the value of 24°. In the final test, taken when the cats could no longer maintain their body temperature at 5° the O_2 consumption at 5° was increased 56% over the O_2 consumption at 24°. The mean increase in O_2 consumption in the final test was not significantly different (5% level) from that in the initial tests. Mean rates for the two males on the reduced diet were 59.3% and 49.1% higher than the mean of the females at room and cold temperatures, respectively, and although the sample size was inadequate for statistical analysis, there appeared to be a trend towards a sex difference in O_2 consumption in these animals.

Table I is a summary of some of the physical characteristics of the six cats before, and at the time of impaired temperature regulation. Total body surface area, calculated from body weight using the equation of Vaughan and Adams (8), showed a mean decrease of 677 cm^2. The nutritional index, listed in the last two columns, was developed by Von Pirquet (5) as the ratio of weight$^{1}/^{3}$(g) to stem length (cm). The largest index (Table I) at which any of the cats' T_r dropped to 35° or lower in the test at 5° was 0.26.

Discussion. Since, in the final test in the cold, oxygen consumption was at least equal to that found earlier, the marked loss in body temperature cannot be ascribed to a failure in heat production. Likewise, it is apparent that the exposure to cold for 3 hr once a week was inadequate to develop acclimatization to cold in these animals. Lowered insulation values due to loss of subcutaneous fat, was undoubtedly a factor in the marked loss in T_r in the final test, but this would not explain the dramatic differences between the final tests and the previous ones. A failure in peripheral vasoconstriction could explain the results but the mechanism would be hard to postulate.

Initial index values ranged from 0.32 to

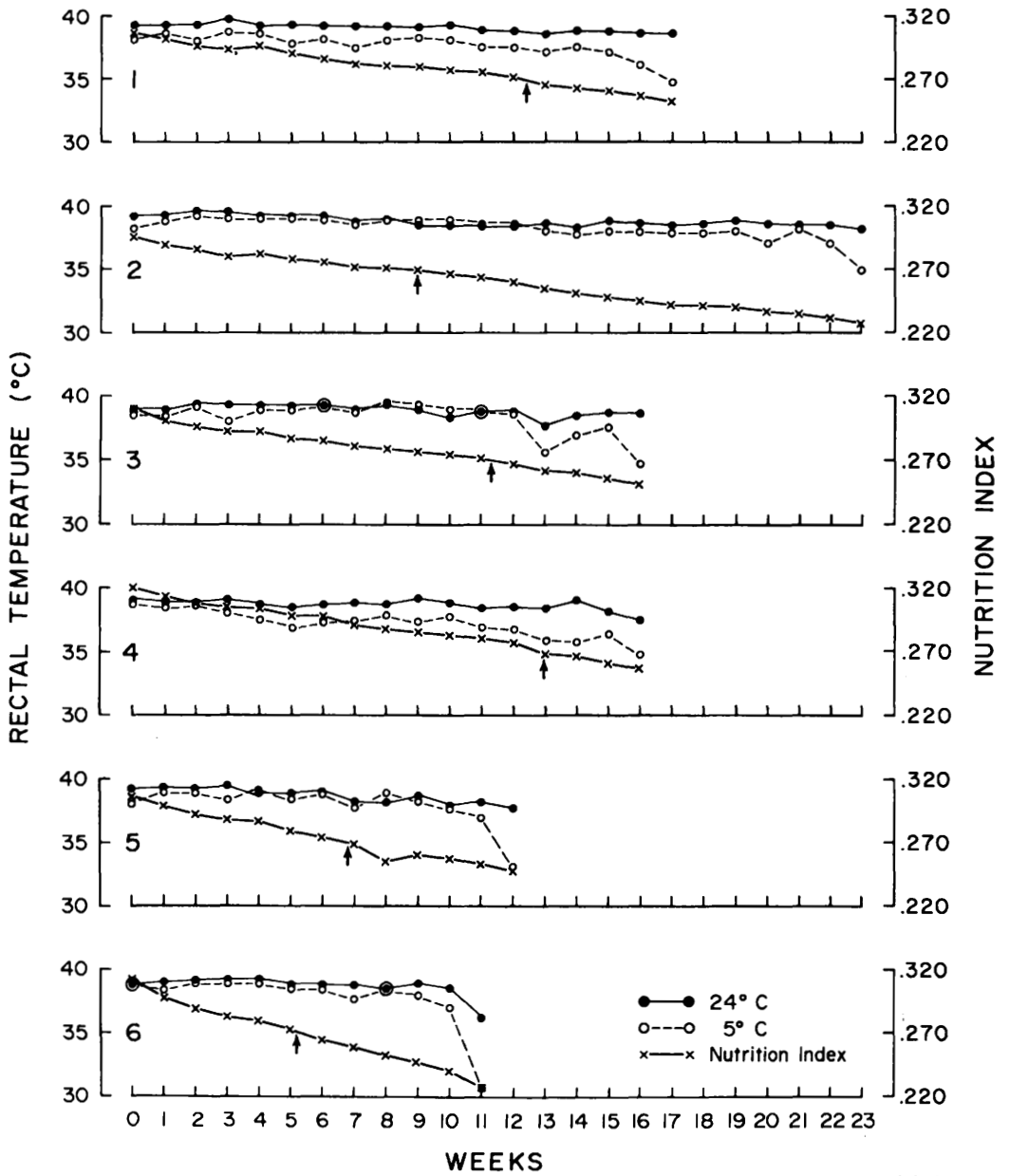

FIG. 1. Initial and final temperature in tests for cold tolerance and changes in nutritional index as inanition progressed. Index levels of 0.27 are marked by arrows.

0.30 (Table I), which is slightly larger than the values of 0.31–0.29 proposed by Cowgill and Drabkin (6) for dogs in a good average state of nutrition. In their nomogram, index readings below 0.29 indicated thinness, whereas those greater than 0.31 denoted some degree of obesity. One cat was available which had been prepared for another experiment and which appeared quite obese. It had a weight of 3600 g and a stem length of 46.8 cm and thus an index of 0.33. This is very comparable to the value of 0.34 found in obese dogs (9). Similarly, the critical index values for our emaciated cats (0.27) agree with the 0.26 found for emaciated dogs (9). It is proposed that in the cat values of 0.33

TABLE I. Initial and Final Physical Characteristics of Subjects.

Cat no. and sex	Time (weeks)	Body wt.		$\frac{W_I - W_F}{W_I} \times 100$ (%)	Stem length (cm)	Surface area		Observed nutritional index $\frac{W^{1/3}}{L}$	
		Initial (g)	Final (g)			Initial (cm^2)	Final (cm^2)	Initial	Final
1 ♀	17	2850	1260	55.8	46.0	2003	1514	0.31	0.25
2 ♀	23	3230	1730	46.4	49.9	2151	1477	0.30	0.23
3 ♀	16	2990	1410	52.8	46.2	2058	1501	0.31	0.25
4 ♀	16	3540	1710	51.7	47.6	2271	1607	0.32	0.26
5 ♂	12	3890	1800	53.7	51.6	2407	1708	0.30	0.25
6 ♂	11	4120	2530	38.6	51.7	2497	1514	0.31	0.23
Mean ± SE	15.8 ± 1.7	3440 ± 210	1740 ± 180	49.8 ± 2.6	48.8 ± 1.1	2231 ± 80	1554 ± 36	0.31 ± 0.003	0.24 ± 0.005

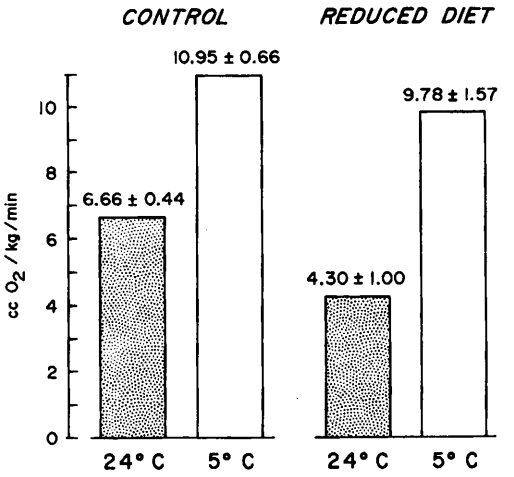

FIG. 2. Average oxygen consumptions at 24 and 5° initially and when the animals were no longer able to maintain normal body temperature when exposed to 5°.

or higher would indicate obesity, values of 0.32–0.30 would indicate normal, while values of 0.27 or lower would suggest considerable emaciation. In Fig. 1 arrows have been placed on each index curve where it fell to 0.27. In only one cat (no. 4) did the T_r drop below normal after 3-hr exposure to 5° as long as the index was 0.27 or higher. However, in all our animals marked disturbances in regulation against cold occurred when the index fell to 0.26 or lower.

These data indicate that the cats deprived of their normal caloric intake may lose from 38.6 to 55.8% of their body weight before their body temperature falls below normal levels in the cold. This result agrees with that of Clark (3) who reported that cats under similar conditions of temperature and diet lost from 42.5 to 52% of their body weight after 5.5–11 weeks. The weight loss he reported occurred more rapidly because his cats were fasted for a week to 10 days prior to restricting food.

The changes in ability to combat cold reported here are of at least the magnitude of those reported by Clark *et al.* (9) to result from damage to neural mechanisms. The position of such workers would be strengthened if they would also report nutritional index values, since damage to neural ther-

moregulatory mechanisms commonly also impair feeding mechanisms.

The following polynomial equation was derived in an attempt to predict the decrease in T_r for individual cats exposed to 5° from known values of the nutritional index: $\Delta T_r(°) = -50.77 + 343$ Index -585 Index^2. However, the confidence intervals showed the variability to be too great to provide anything but crude estimations of ΔT_r. These preliminary results merit further investigation of additional ambient temperature effects on a larger sample population.

Summary. A nutrition index ($W^{\frac{1}{3}}/L$) was applied to six healthy cats subjected to a restricted diet for a period of 11–23 weeks. During this period, they were exposed each week for 3 hr to an ambient temperature of 5° with T_r measured at the end of each hour. Thermoregulation was considered critically impaired when the T_r fell to 35° or below. In the final test, in each of the six cats, the T_r fell to 35° or below, and in each the nutritional index fell below 0.27 while the mean body weight loss of the six animals was 49.8%. It is suggested that in experiments in which integrity of the thermoregulatory mechanism is vital, use of the nutritional index would eliminate one variable. It is also suggested that the index provides an estimate for the degree of obesity.

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Glutathione Reduction: Studies Using Deoxyribonucleosides as Substrates* (33416)

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The coenzyme requirement of erythrocyte glutathione reduction remains unclear. In intact cells this reduction is linked primarily to reduced triphosphopyridine nucleotide (TPNH) (1). Disruption of cellular integrity links the reduction to reduced diphosphopyridine nucleotide (DPNH) (2). The purified enzyme catalyzing this reaction, glutathione reductase, can use both TPNH and DPNH as cofactors (3,4).

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Several investigators have attempted to solve this problem using lactate-generated DPNH and oxidized glutathione (GSSG) produced under a variety of conditions (2, 3). Some have suggested that the failure to obtain significant glutathione reduction was due to a failure to generate DPNH through the glyceraldehyde-3-phosphate dehydrogenase reaction (3).

The failure to observe significant glutathione reduction with glucose-6-phosphate dehydrogenase (Glc-6-*P*D) deficient erythrocytes has been used as evidence that glyceraldehyde-3-phosphate dehydrogenase-gener-