

References and Notes

1. The soil samples were supplied by R. L. Woodward of the Robert A. Taft Sanitary Engineering Center, U.S. Public Health Service, as part of U.S. Air Force Special Weapons Center, Kirtland Air Force Base (New Mexico), project No. 7801.
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4. We wish to acknowledge the invaluable assistance of S. A. Reynolds, Oak Ridge National Laboratory, Oak Ridge, Tenn., for the suggestion that the peaks might indicate the presence of Sc^{46} .
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Degree of Obesity and Serum Cholesterol Level

Abstract. No significant correlation was found between the serum cholesterol level and weight, weight corrected for frame size, or thickness of the fat shadow in medical students (mean age, 22 years).

Among the variables thought to affect serum cholesterol levels in normal subjects, the degree of obesity has been of particular interest because the amount of stored fat is accessible to dietary control. The literature, however, is divided on the relationship of body fat to cholesterol level. Moreover, reports have been variously based on anthroposcopic "endomorphism" ratings (1, 2), weight or relative weights (3), indices of body build (4), or infrascapular fat folds (1, 5) and not, to our knowledge, on direct radiographic measurements of the outer-fat shadow.

In the study reported here we compared the thickness of the fat-plus-skin shadow, measured at the level of the tenth rib on standard posteroanterior teleoroentgenograms (6), with serum cholesterol levels, determined, by a modification of the Bloor method (7), in blood samples obtained at the time

Table 1. Correlations between various measures of obesity and total serum cholesterol in healthy young men.

N	Mean body wt. (kg)	Serum cholesterol (mg/100 ml)*	Correlation (r)
<i>Body weight</i>			
159	74.8	225.2±36.3	0.033
<i>Body weight/chest breadth</i>			
134	73.4	226.0±37.8	0.126
<i>Lower thoracic fat</i>			
125	73.2	222.5±34.2	-0.030

* Mean ± standard deviation.

the roentgenograms were made. Less direct measures of relative obesity included weight, and weight expressed in relation to the bony-chest diameter, as measured on the films. Replicability was 0.95 for the fat measurements (8) and 0.92 for serum cholesterol (7).

Subjects included the 159 white male medical students in the classes of 1958, 1959, and 1960 of the Johns Hopkins School of Medicine, for whom body weight, cholesterol level, and posteroanterior chest plates were obtained at the time of admission. The mean height for the group was 178.9 cm, the mean weight was 74.8 kg, and the mean age was 22 years. The cholesterol range was 140 to 386 mg/100 ml with a mean of 225 mg/100 ml, and the lower-thoracic fat range for the subjects in whom the fat-plus-skin shadow could be accurately measured was 2 to 15 mm, with a mean of 7.4 mm.

As shown in Table 1, the correlation between serum cholesterol level and weight in the series of 159 men was not significantly different from zero at the 5-percent level of confidence ($r = 0.13$). In a restricted subsample, from which subjects whose radiographs were not suitable for the chest-breadth measurement had been excluded, the correlation between serum cholesterol and weight corrected for build was similarly low ($r = 0.13$). Finally, the correlation between serum cholesterol and lower-thoracic fat, measured on 125 radiographs technically suitable for the purpose, was -0.03 . It is noteworthy that the mean cholesterol levels and their standard deviations in the total sample and in the partial samples were very nearly the same. The ranges for the three groups were identical.

There are many factors in addition to nutritional status which have been shown to be related to the height of the serum-cholesterol level of healthy persons—in particular, age, sex, race, heredity, endocrine patterns, habits of smoking and exercise, and degree of emotional stress. We have reported positive correlations between several of these factors and high cholesterol levels among the Johns Hopkins medical students (9). The lack of relationship between the amount of body fat and the serum cholesterol level demonstrated in the present study supports the view that the nutritional status of healthy young men such as medical students is considerably less important in determining the cholesterol level than other biologic factors (10).

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10. This research was supported in part by grant H-1891 and in part by grant M-1260 from the National Institutes of Health. The assistance of Joan A. Haskell in making the fat measurements is gratefully acknowledged. The other data are part of a long-term study of the precursors of hypertension and coronary heart disease.

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Neural and Hypophyseal Colloid Deposition in the Collared Lemming

Abstract. Feral and captive lemmings from Churchill, Manitoba, are subject to a unique pathological process in which a colloidal material is deposited in blood-vessel walls at scattered points through the central nervous system. Destruction of nervous tissue at these foci is progressive, and colloidal masses in the vascular lumina of the hypothalamus appear to become fixed in the capillaries of the hypophyseal anterior lobe. Inflammatory reactions are never associated with the lesions, and the latter are larger and more numerous in older animals in warmer environments.

A current study of the microanatomical and physiological characteristics of the collared lemming (*Dicrostonyx groenlandicus* Traill) has revealed a unique and previously unknown pathology which is probably a significant factor in the behavior and population fluctuations of these ecologically significant arctic rodents.

Lemmings used in this study consist both of animals fixed in 10-percent neutral buffered Formalin immediately after capture at Churchill, August 1953 and July 1954, and captives and their progeny perfused and fixed in Bouin's fluid. Observations on the behavior, reproduction, growth, diseases, and reproductive and endocrine organs of these animals have been presented (1). Captive lemmings were raised and maintained usually in an artificially and constantly lighted room at 24°C (21° to 27°). Groups of animals were periodically transferred to darkness and a temperature of 23°C for 4 months, or to a thermostatically controlled deep-freeze cabinet with a transparent lid and forced circulation of air. After 10

to 14 days of decreasing temperature the lemmings were kept at -9° to -6° C, either in light for several days or in darkness for up to 4¼ months in apparent good health. Three animals were subjected to temperatures of 32° to 33° C for 1 to 2 days. At autopsy, weight, blood counts, and smears were made.

Serial paraffin sections of the brains of 16 of the best-preserved captives, which appeared to be healthy externally, were stained by a variety of methods (2) and demonstrate a pathological process without any signs of disease organisms or inflammatory responses (3). The characteristic and progressive lesions may be somewhat arbitrarily divided into five types: (i)

segments of capillaries and venules with thickened endothelia containing granules staining with azure, periodic-acid Schiff, and Casella techniques; (ii) segments of capillaries and venules with similarly staining fibrous or granular deposits radiating into the surrounding neuropil as well as forming aldehyde fuchsin- and periodic-acid Schiff-positive spheroidal and amorphous intravascular colloid masses (Fig. 1,A); (iii) small (5 to 20μ) colloid bodies staining with aldehyde fuchsin and Bauer techniques and scattered in the neuropil, especially in the white matter of cerebellum and tegmentum, and probably resulting from lesions of types i and ii; (iv) spheroidal and amorphous colloid bodies detached in the ventricles

of the brain and in veins, and originating at least in part from type ii lesions (Fig. 1,B); and (v) focal lesions of the type ii category but expanded to 120 to 160μ in diameter and containing centrally a metachromatic (Giemsa stain), aldehyde fuchsin-positive, insoluble lipid-containing material. Lesions of types i to iv are most numerous in lemmings in higher environmental temperatures, with the exception of certain of the oldest of the cold-treated females, which have many of these lesions also. Type v lesions are usually found only in animals over 6 months old. Brain lesions similar to some of these but generally less severe have been observed in lemmings fixed at capture.

The anterior lobes of the hypophyses of all of the captives contain intravascular colloid bodies staining with aldehyde fuchsin and resembling those of the type iv brain lesions. When these are counted along with the parenchymal cells of the pars distalis in sections 7μ in thickness, they are found to form 0.7 to 4.5 percent of the elements at environmental temperatures of -9° to 6° C, 2.0 to 6.0 percent at 21° to 27° C, and 3.7 to 7.8 percent at 32° to 33° C. All but three of the 25 animals fixed at capture at Churchill show similar hypophyseal colloid bodies (Fig. 1,C), but usually less abundantly than the captive series at 21° to 27° C. It appears likely that the hypophyseal colloidal emboli descend via the hypothalamo-hypophyseal portal system, since they are not found in the hypophyseal posterior lobe, and formative stages are not seen in hypophyseal endothelia.

The natural occurrence of this pathology in lemmings at Churchill, one of the most southern points in the geographic range of this species, and the augmentation of the lesions in moderately warm environments is interesting in terms of evidence for a metabolic limitation in these animals which may restrict their dispersal and influence their vigor and reproductive potential. It appears that adaptations for life in an arctic climate in these animals are associated with a peculiar vulnerability in warm environments (4).

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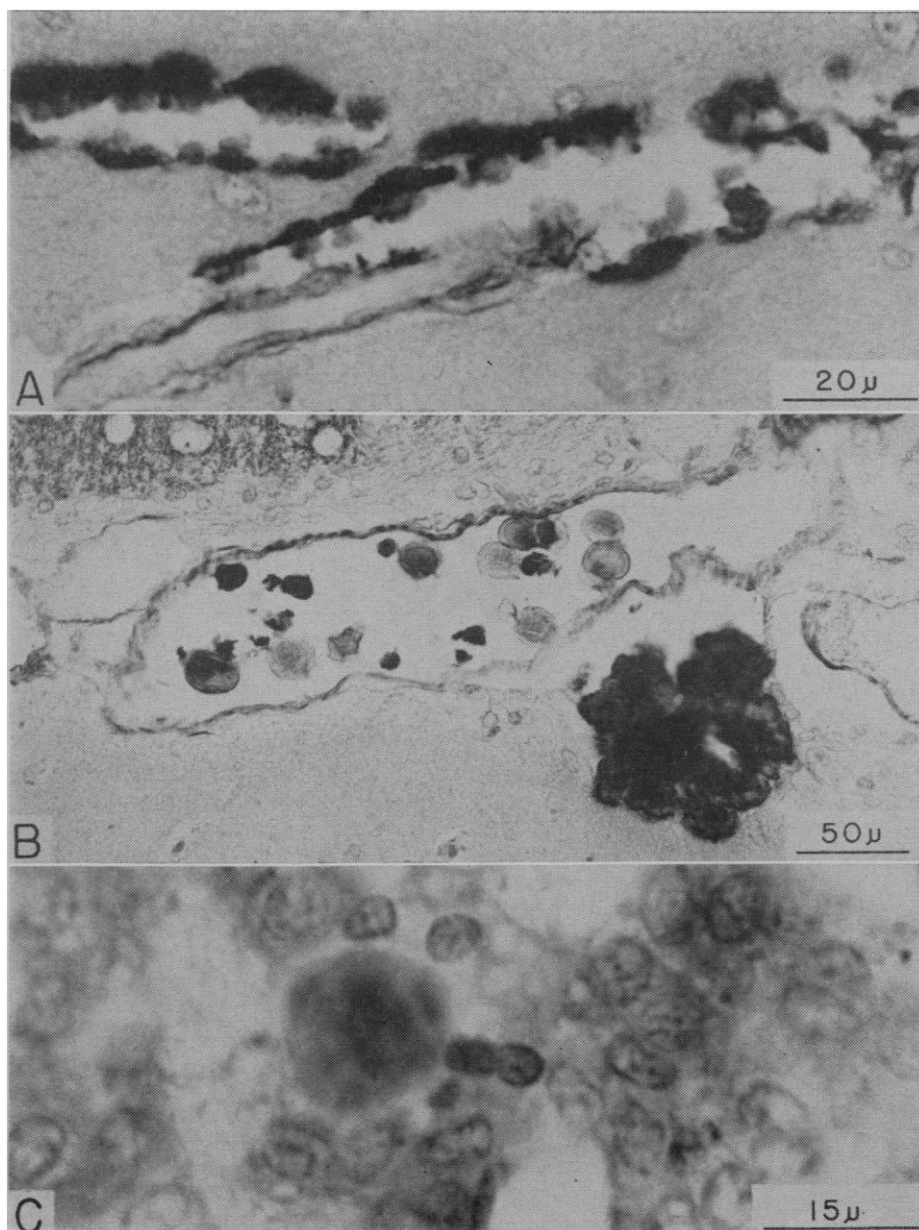


Fig. 1. (A) Type ii lesion spreading from the endothelia of a cerebral vessel (Casella technique and hematoxylin stain). (B) Detached and invasive colloid bodies in meningeal vessels (Oil red O and hematoxylin stain). (C) Hypophyseal colloid body in a feral lemming (periodic-acid Schiff and hematoxylin stain).