

was 15 min at 31°C. A small sample of tissue was rapidly snipped from the right lobe of the liver for glycogen estimation. This was determined by the method of Good, Kramer, and Somogyi (8), employing the Nelson's adaptation of the Somogyi method for glucose (9).

Table 1 shows that while the rats lose about 12 percent of body weight during the 48-hr fast and liver glycogen decreases to 5 percent of the original value, the glucose-6-phosphatase activity is increased by 60 percent.

These experimental data have been repeatedly confirmed in this laboratory in the course of other experiments involving various periods of fasting, and it was found that the difference between the glucose-6-phosphatase activity of normal and 48-hr fasted animals is significant also when expressed on nitrogen or liver weight-to-body weight ratio basis (10).

The afore-cited data demonstrate that the glucose-6-phosphatase activity increases in the liver of fasting mice and rats. A survey of the literature on the effect of fasting on liver enzymes shows that most

enzymes decrease under the described conditions here. The increase of glucose-6-phosphatase activity during fasting can, therefore, be considered as a physiological adaptive change to the stress of fasting during which the glycogen stores of the liver are depleted.

References and Notes

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1. P. Fantl, M. N. Rome, and J. F. Nelson, *Australian J. Exptl. Biol. Med. Sci.* **20**, 121 (1942); P. Fantl and M. N. Rome, *ibid.* **23**, 21 (1945).
2. M. J. Swanson, *J. Biol. Chem.* **184**, 647 (1950).
3. C. De Duve *et al.*, *Bull. soc. chim. biol.* **31**, 1242 (1949); H. G. Hers and C. De Duve, *ibid.* **32**, 20 (1950); H. G. Hers *et al.*, *ibid.* **33**, 21 (1951).
4. A. D. Chiquoine, *J. Histochemistry and Cytochemistry* **1**, 429 (1953).
5. G. T. and C. F. Cori, *J. Biol. Chem.* **199**, 661 (1952).
6. G. G. Duncan, *Diseases of Metabolism*, (Saunders, Philadelphia, ed 3, 1952), p. 53.
7. This project has been supported by grants from the Cancer Research Society. The valuable assistance of Vilma Jansons is gratefully acknowledged.
8. C. A. Good, H. Kramer, and M. Somogyi, *J. Biol. Chem.* **100**, 485 (1953).
9. N. Nelson, *ibid.* **153**, 375 (1944).
10. Unpublished.

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Communications

Inherited Jaundice in *Peromyscus*

This is to report (1) a mutation in *Peromyscus maniculatus* (2) that is typically associated with neonatal jaundice, chronic splenomegaly, anisocytosis, polychromatophilia, and reticulocytosis. Affected mice usually become jaundiced during their first postnatal 24 hr. The duration of the yellow color varies but it is not, as a rule, perceptible after the second postnatal day. The intensity of the color varies from deep to pale yellow, and in some individuals it may not be sufficiently marked to make identification possible upon first inspection.

The yellow color is succeeded by pallor, which may be very pronounced and may last until the fifth postnatal day when, in any event, it would be masked by the outgrowing pelage. All of more than 50 jaundiced young that were examined on successive days became pallid if they survived, and this symptom, because of its duration, is more useful than the neonatal jaundice in the identification of affected individuals.

All of 30 newborn mice recorded as jaundiced and/or pale had splenomegaly when, to test the point, they were posted at 3 mo of age or later. A much larger number of mice examined at an age when affected individuals would be jaundiced or pale, and recorded as not affected, had spleens of normal color and proportions. The normal spleen in the deer mouse is a slender leaflike organ that is pale red or dark red in color. Jaundiced mice develop a spleen that is very dark red or black in color and about double the size of the normal spleen in each dimension. There is variation in the size and color of both normal and hyper-

trophied spleens, but intergradation in size or color has not yet been observed.

The erythrocytes of adult splenomegalic mice vary in size and shape and this erythrocytic variability is an immediately obvious feature upon microscopic examination of either blood smears or the vascular areas of tissue sections. Blood smears of affected mice, stained by the Osgood-Wilhelm technique (3), have, in the 22 cases examined, exhibited a significantly higher proportion of reticulocytes than was found in the blood of normal individuals, of similar age, prepared at the same time. The reticulocyte counts (4) are shown in Table 1.

Table 1. Percentages of reticulocytes in smears made from jaundiced mice between 3 and 6 mo of age and of normal mice 3 mo old.

Percentage	20	18	16	14	12	10	8	6	4	2	n
Normal mice										7	7
Jaundiced mice	2		1		6	3	2	1			15

A chi-square test of the difference in the number of mice in the two classes produced by the mating of normal hybrid mice with jaundiced mice is given in Table 2. The phenotypes of all parent mice and all of their offspring were identified by more than one person during the first postnatal day and, if necessary, on a succeeding day. Since 15 of the 19 broods contained young of both classes, comparison assisted separation into two classes. The data fit the assumption that the characteristics of the syndrome described here are brought about by a single recessive gene effect.

Table 2. Number of young in the two classes produced by mating jaundiced mice with nonaffected hybrids. Expected ratio 1:1. P based on one degree of freedom.

Class	Observed	Expected	Chi-square	P
Jaundiced and/or pale	38	39		
Not jaundiced	40	39	0.52	.95-.70

In addition to the data here tabulated we have a larger number of young produced by test crossing females, identified as probable hybrids by pedigree inspection and progeny test, with jaundiced males. These test crosses have produced 70 normal and 63 jaundiced or pale mice. Jaundiced mice, mated *inter se*, have produced 27 jaundiced or pale mice and two that were cachectic but without the typical jaundice or pallor.

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References and Notes

1. This research was assisted by a grant from the University of Oregon Graduate School.
2. F. B. Sumner, *Am. Naturalist* **49**, 688 (1915); L. R. Dice, *Am. Naturalist* **74**, 289 (1940).
3. E. E. Osgood and M. M. Wilhelm, *J. Lab. Clin. Med.* **19**, 1129 (1933-34).
4. The reticulocyte counts were made through the kindness of Osgood and Koler at the University of Oregon Medical School.

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New Etiologic Agent in Nonspecific Bacterial Vaginitis

This is a preliminary report of an investigation in progress dealing with the etiology and clinical manifestations of "nonspecific" bacterial vaginitis. The etiology of the condition has previously been ascribed to a large group of unrelated bacteria. An intensive search of the literature has not revealed evidence that a regularly appearing etiologic agent has been previously found to explain these infections. We have isolated a new bacterium that appears to be the causative agent in the vast majority of so-called "nonspecific" vaginitides.

The investigation includes a clinical and bacteriological study of 91 cases of bacterial vaginitis. A previously unidentified and unclassified organism belonging to the genus *Haemophilus* has been isolated in 81 of the 91 cases. Although the organism was predominant in each of the 81 cases from which it was isolated, it occurred in pure culture on one or more occasions in 62 of the 81 cases.

A compilation of the clinical signs and symptoms suggests that the infection resulting from this organism constitutes a specific disease entity. The discharge is usually gray in color, thin and homogeneous, odorous, and less acid than the secretions of a normal vagina. Itching and irritation, although occasionally

present, are not prominent symptoms. The infection has been established in normal (volunteer clinic) patients by direct inoculation of material from the vaginas of infected patients and by material from pure culture.

The organism has been found to be sensitive to the tetracycline group of antibiotics and to sulfonamides. These drugs have been administered orally and intravaginally with the infection being eradicated in the majority of cases.

Although it is felt that a diagnosis usually can be made by correlating clinical manifestations with microscopic findings, cultural methods are necessary for final proof of the infection. Stained smears of the discharge reveal tremendous numbers of small, pleomorphic, gram-negative bacilli. This new organism is extremely fastidious, and isolation has been achieved routinely only on proteose-peptone No. 3 agar containing 10 percent defibrinated sheep blood, incubated under increased carbon dioxide tension (candle jar). The cultural characteristics undoubtedly explain why the organism has escaped previous isolation and identification. A complete report [*Am. J. Obstet. Gynecol.*, in press] describes in detail the isolation and identification of the organism, the clinical manifestations of the entity, evidence of pathogenicity, and so forth. We feel that sufficient evidence is at hand to establish proof of a newly defined specific bacterial vaginitis, the etiology of which heretofore has not been recognized.

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Absence of Circulating Antibodies in Patients with Pulmonary Tuberculosis

Serodiagnostic tests of both the complement fixation and hemagglutination type have failed to detect circulating antibodies in a substantial number of patients with active tuberculosis. In recent studies evaluating the hemagglutination test (1) the number of negative reactors varied from 14 (2) to 44 percent (3). This is remarkable in view of the nearly 100-percent incidence of antibody formation in other granulomatous diseases, for instance, coccidioidomycosis or brucellosis.

Under the assumption that substances inhibiting antibody activity may be present, tuberculous serums were fractionated by the cold ethanol method (4), which does not accomplish complete separation of the various proteins. The fractions were then tested by both the hemagglutination and complement-fixation methods (5). The following results are available. The globulin fractions of three tuberculous serums (No. 1-3), like the whole serums from which they were derived, exhibited activity in both tests. Six serums (No. 4-9, Table 1), apparently devoid of hemagglu-