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	Ob- served	Ex- pected	Chi- square	Р
Jaundiced and/or pale Not jaundiced	38 40	39 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.
- 61 Ofegon Graduate School.
 F. B. Sumner, Am. Naturalist 49, 688 (1915); L. R. Dice, Am. Naturalist 74, 289 (1940).
 E. E. Osgood and M. M. Wilhelm, J. Lab. Clin. Med. 19, 1120 (1900) 2.
- 3. 1129 (1933-34).
- The reticulocyte counts were made through the kindness 4. of Osgood and Koler at the University of Oregon Medical School.

25 October 1954.

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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10 October 1954.

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-

Table 1. Serologic results on tuberculous serums and their fractions. Fractions are labeled according to Nichols and Deutsch (4).

		Titer of		
Serum	Serum	Hemag-	Comple-	
No.	fraction	glutina-	ment-	
		tion	fixation	
		test	test	
4	Whole serum	Neg.	Neg.	
	Sup. A	Neg.	Neg.	
	Ppt. B	Neg.	Neg.	
	Ppt. C_2	Neg.	1:32	
5	Whole serum	Neg.	Neg.	
	Sup. A	Neg.	Neg.	
	Ppt. B	Neg.	Neg.	
	Ppt. C_2	Neg.	1:256	
6	Whole serum	Neg.	Neg.	
	Sup. A	Neg.	Neg.	
	Ppt. B	Neg.	1:2	
	Ppt. C ₂	Neg.	1:16	
7	Whole serum	Neg.	Neg.	
	Sup. A	Neg.	Neg.	
	Ppt. B	1:8	1:1	
	Ppt. C ₂	Neg.	1:16	
8	Whole serum	Neg.	Neg.	
	Sup. A	Neg.	Neg.	
	Ppt. B	Neg.	Anticompl.	
	Ppt. C_2	1:8	1:8	
9	Whole serum	Neg.	Neg.	
	Sup. A	1:64	Neg.	
	Ppt. B	Neg.	Anticompl.	
	Ppt. C ₂	Neg.	1:8	
10	Whole serum	1:20	Neg.	
	Sup. A	1:8	Neg.	
	Ppt. B	1:8	Anticompl.	
	Ppt. C ₂	1:16	1:8	
11	Whole serum	Neg.	1:16	
	Sup. A	1:4	Neg.	
	Ppt. B	1:16	1:2	
	$Ppt. C_2$	1:8	1:16	

tinating and complement-fixing antibodies, yielded fractions that gave a positive complement-fixation test. Fractions of three of these serums (No. 7-9) reacted also in the hemagglutination test. Further, fractionation of two tuberculous serums (No. 10 and 11, Table 1) giving a positive reaction in one or the other test furnished proteins manifesting antibody activity of the type seemingly absent in the whole serum.

As a control, five nontuberculous serums were subjected to the same procedures. None of their fractions exhibited antibody activity in the tests mentioned.

These results demonstrate that the absence of antibodies in the tuberculous serums investigated (6) was only an apparent one and that fractionation of the serum proteins unmasked antibody activity. The problem whether a substance suppressing antibody was present in these serums or whether fractionation accomplished dissociation of an antibody-antigen complex is under investigation.

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References and Notes

- 1. G. Middlebrook and R. J. Dubos, J. Exptl. Med. 88, 521
- (1948)W. Hentel and G. D. Guilbert, J. Lab. Clin. Med. 39, 426 2.
- (1952). W. H. Hall and R. E. Manion, J. Clin. Invest. 30, 1542 3. (1951)
- J. C. Nichol and H. F. Deutsch, J. Am. Chem. Soc. 70, 80 4. (1948)

5. S. Raffel, personal communication.

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Availability of Crystalline DL-a-Lipoic Acid

Studies conducted in this and other laboratories during the past decade have culminated recently in the isolation of an extremely active biocatalyst that has been designated α -lipoic acid (1) and Protogen-A (2). This substance is a growth factor for several microorganisms and has been shown to participate in the oxidative decarboxylation of pyruvic and α -ketoglutaric acids.

Although a-lipoic acid has been identified and obtained synthetically in racemic form (3-5), it is not generally available for biological research. In the belief that the nutritional and therapeutic value of this biocatalyst will be assessed only when it is made readily available to interested investigators, we have devoted our time recently to developing an improved synthesis of $DL-\alpha$ -lipoic acid. As a result we have on hand a significant quantity of the crystalline substance which we wish to make available to those interested in exploring its potentialities. Requests for samples should be sent to the address given below.

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References and Notes

- $\frac{1}{2}$
- L. J. Reed et al., J. Am. Chem. Soc. 75, 1267 (1953).
 E. L. Patterson et al., ibid. 76, 1823 (1954).
 C. S. Hornberger, Jr., et al., ibid. 75, 1273 (1953).
 M. W. Bullock et al., ibid. 76, 1828 (1954).
- 3.
- The racemic form is designated DL-a-lipoic acid (3) and 5. 6-thioctic acid (4).

13 October 1954.

