(24°C) and again in a cooler solution. By means of a suction electrode[6] injury was produced, and the monophasic curves were recorded when the galvanometer was connected to this electrode and to another electrode several centimeters distant in the solution. Table II shows that when the cycles were long the monophasic curve of the cool muscle, measured from the top of its ascending limb to its end, had nearly double the duration of the curve derived from the uncooled ventricle. When the cycles were short, however, the monophasic curves were practically equal in duration at both temperatures. This experiment was typical of four. It explains the effect of rate increase in the uninjured heart, namely, that the decrease in the net QRS-T area is due to a change in the relative times required for repolarization, in the sense that the time becomes more nearly the same in all parts of the muscle.

This evidence indicates that in the human heart, beating slowly, the large gradient which is usually present (and which accounts for the normal T-wave directions) is due to the slower repolarization of some parts of the muscle than of other parts. When the rate is very rapid, the time course of repolarization is everywhere nearly the same, and the gradient is, therefore, reduced in magnitude.

6 Wiggers, H. C., *Am. J. Physiol.*, 1938, **123**, 215.

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Form Variation in Group C Salmonella Strains.*†

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The occurrence of strains of the Newport group (VI, VIII) which lacked antigen VI, the O component possessed in common with the Choleræ-suis (VI, VII) and Carrau-Onderstepoort (VI, XIV . . .) groups, occasioned a closer study of the O antigens of group C Salmonella cultures. Kauffmann[1] demonstrated that antigen VI is divisible into at least 2 fractions which he designated VI_1 and VI_2. The majority of cultures of *S. choleræ-suis* contained only VI_1 antigen, although a few contained only VI_2. The remaining strains of the VI, VII group which were studied contained both VI_1 and VI_2. On the contrary, cultures of the Newport group (VI, VIII) contained only VI_1. Hormaeche, Peluffo, and Ricaud de Pereyra[2] found that antigen VI was more complex than had been generally realized and that it was composed of at least 3 principal fractions and additional minor antigens. Further, they remarked that they had found its distribution rather irregular. Also, in routine typing it has been observed that cross agglutination between the VI, VII and VI, VIII groups was quite variable. The lack of antigen VI in certain cultures and the irregularities noted in its behavior suggested that the antigen, or a portion thereof, was subject to the form variation or O variation which Kauffmann[3,4] found in antigen I and in a fraction of antigen XII. Accordingly, cultures of *S. newport* and *S.*

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† The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the Kentucky Agricultural Experiment Station.

1 Kauffmann, F., *Z. f. Hyg.*, 1937, **120**, 177.

2 Hormaeche, E., Peluffo, C. A., and Ricaud de Pereyra, V., *J. Bact.*, 1944, **47**, 323.

3 Kauffmann, F., *Acta path. et microbiol. Scand.*, 1940, **17**, 135.

4 Kauffmann, F., *J. Bact.*, 1940, **41**, 127.

TABLE I.
Form Variation in VI, VIII Types.

Antigens	Serums VI_1, VI_2, VIII					Serums VI_1, VIII						Serums I, VI, XIV, XXV					
	20	40	100	200	400	100	200	400	1000	2000		20	40	100	200	400
Newport 563, colony 14 (±)	±	±	±	±	±	+	+	+	+	±		±	±	±	+	+
Newport 563, colony 21 (++)	+	+	+	+	+	+	+	+	+			+	+	+	+	+
Oregon 1511, colony 2 (±)	±	±	±	±	±	+	+	+	+	±		±	±	±	+	+
Oregon 1511, colony 9 (++)	+	+	+	+	+	+	+	+	+			+	+	+	+	+

oregon, the O antigens of which are delineated as VI_1, VIII, were plated and individual colonies examined by slide agglutination with VI_1,VI_2,VII and VI_1,VIII serums. The latter serum was used only to check the agglutinability of the individual colonies and all were agglutinated equally well by it. The behavior of the colonies in VI_1,VI_2,VII serum divided them into 2 groups. Some colonies were agglutinated quickly and completely (++) while in others there was only a trace of agglutination (±) after several minutes. In the particular strains examined the numbers of VI± and VI++ colonies were nearly equal. The remains of the colonies were picked to agar slants and alcohol-treated antigens prepared by the method of White.[5] Tube tests yielded the results given in Table I. It is evident that in *S. newport* and *S. oregon* the content of antigen VI_1 is quite variable. In order to ascertain the stability of the two forms, plates were streaked from the agar slant cultures which had been used to prepare the alcohol-treated antigens. Two cultures of each form of *S. newport* and *S. oregon* were plated. Twenty colonies on each plate were tested and it was found that 5 to 20% of the colonies had reverted to the opposite form. Thus it may be seen that *S. newport* and *S. oregon* contain VI± and VI++ forms and that each form reverts naturally to the other. The same type of variation was found in a non-motile VI,VIII culture received from Major K. S. Wilcox.

After the above results were obtained, *S. hartford, S. mikawashima, S. oranienburg,* and *S. montevideo* of the VI_1,VI_2,VII group were examined in a similar manner. Colonies were tested with VI_1,VIII and VI_1,VI_2,VII serums. Again it was found that VI± and VI++ forms were recognizable. The results obtained with representative single colony cultures are given in Table II. These forms also were found to revert to the opposite form under ordinary conditions of culture. Absorptions were done with a number of VI± forms derived both from VI, VII, and VI,VIII cultures. In general it may be said that absorption of a VI,VII serum with a VI±

[5] White, P. B., Med. Res. Counc. Gt. Brit., Spec. Rpt. Ser. No. 103, 1936.

TABLE II.
Form Variation in VI, VII Types.

Antigens	VI_1, VIII					Serums VI_1, VI_2, VII						I, VI, XIV, XXV					
	20	40	100	200	400	100	200	400	1000	2000		20	40	100	200	400	
Hartford 3015,	+					+	+	+	+			+	+	+	+	+	+
colony 9 (±)	+		±			+	+	+	+			+	+	+	+	±	
Hartford 3015,		+	+	+	±	+	+	+	±			+	+	+	+	+	±
colony 16 (++)	+	+	+			+	+	+	+			+	+	+	+	+	+
Montevideo 623,	+		+			+	+	+	+			+	+	+	+	+	+
colony 1 (±)		±				+	+	+	+			+	+	+	+	+	+
Montevideo 623,	+	+	+			+	+	+	+			+	+	+	+	+	+
colony 9 (++)						+	+	+	+			+	+	+	+	+	+
Mikawashima 45,	+	+	+			+	+	+	+			+	+	+	+	+	+
colony 4 (±)						+	+	+	+			+	+	+	+	+	+
Mikawashima 45,	+	±	+	±		+	+	+	+			+	+	+	+	+	+
colony 7 (++)						+	+	+	+			+	+	+	+	+	+
Oranienburg 9945,	+	+	+			+	+	+	+			+	+	+	+	+	+
colony 1 (±)						+	+	+	+			+	+	+	+	+	+
Oranienburg 9945,	+	±	±	±		+	+	+	+			+	+	+	+	+	+
colony 7 (++)	+	+	+	+		+	+	+	+			+	+	+	+	+	+

form derived from a VI,VIII culture removed all agglutinins for itself and other similar forms but left traces of agglutinins for all VI++ forms derived from VI,VIII cultures. Considering the amount of reversion that occurs in the VI± forms, one would not expect a large residue of VI_1 agglutinins to be left in the serum. Similar absorption tests with VI± forms derived from VI,VII cultures in which VI,VIII serum was used gave comparable results.

It should be emphasized that the results outlined above apply only to that portion of antigen VI which has been designated as VI_1. The VI,VIII cultures contained only VI_1 and the VI,VIII serums used to test the VI,VII strains contained only agglutinins for VI_1. While it is not intended to enter into an extended analysis of antigen VI, it is desirable to know whether the variation described affects only VI_1 or the whole VI complex. This was easily determined by testing ± and ++ forms of VI,VII and VI,VIII cultures with serum derived from *S. onderstepoort* (I, VI, XIV, XXV) which contains agglutinins both for fractions VI_1 and VI_2. These tests are included in Tables I and II. It is evident that the ± and ++ components of the VI,VIII cultures, which contain only VI_1, react in *S. onderstepoort* serum as they do in VI,VII serum. On the contrary both the VI± and VI++ forms of the VI,VII types, which contain both VI_1 and VI_2, react strongly in *S. onderstepoort* serum. It may be said, therefore, that a colony which contains VI_1 and VI_2 antigens may possess only a ± complement of VI_1 but still have a full complement of VI_2. Further, 100 colonies of a culture of *S. choleræ-suis* var. *kunzendorf*, the O antigens of which were VI_2, VII, were tested with *S. onderstepoort* serum. All the colonies reacted equally well and there was no indication that any O variation occurred. While the results with one strain are not conclusive, they strongly suggest that VI_2 is not variable. Certainly variation in VI_1 and VI_2 does not go hand in hand.

Certain cultures of group C seem to be totally and permanently lacking in antigen VI. Among such cultures are *S. amherstiana* (VIII:1,v-1,6 . . .) of Edwards and Bruner[6]

and the recently described *S. virginia* (VIII:d) of Saphra and Seligmann.[7] In addition a culture received from the Virginia State Department of Health had the formula VIII:d-1,2,3 . . . Also two cultures of *S. newport* isolated from a snake by Dr. W. R. Hinshaw had only antigen VIII. Non-motile variants isolated from the same animal had identical O antigens. It is well known that antigens that are subject to form variation may be totally lacking in certain cultures. For instance, Kauffmann demonstrated variation involving antigen I in a number of types of groups A and D. Cultures of these types are found which contain no I antigen. Likewise Kauffmann found that a fraction of antigen XII (XII_2) was subject to variation and that it was totally lacking in a strain of *S. typhi*, a species which ordinarily contains the antigen. Therefore, it is to be expected that VI_1 would be lacking in certain VI_1,VIII strains. These probably represent loss variants of cultures with typical O antigens.

Summary. It was found that antigen VI_1 was subject to a reversible form variation similar to that reported by Kauffmann for antigens I and XII_2. This variation occurred both in the *S. choleræ-suis* and *S. newport* groups. Colonies which were deficient in VI_1 had a normal content of VI_2. So far as is known the latter antigen is not subject to form variation.

[6] Edwards, P. R., and Bruner, D. W., *J. Immunol.*, 1942, **44**, 319.

[7] Saphra, I., and Seligmann, E., PROC. SOC. EXP. BIOL. AND MED., 1945, **58**, 50.

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A new Salmonella type: *Salmonella pensacola.**†

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Salmonella pensacola, the type to be described, was isolated from a severe case of acute gastro-enteritis in a laboratory worker and was forwarded to the writers by Mrs. Mildred Galton. The source of infection was not known. On preliminary examination, the culture proved to be a motile rod which possessed the usual cultural and biochemical characteristics of the Salmonella group. Hydrogen sulfide was produced and Jordan's tartrate was fermented but indol was not formed nor was gelatin liquefied. Acid and gas were formed in 24 hours from glucose, rhamnose, xylose, arabinose, maltose, trehalose. dulcitol, mannitol, sorbitol, and inositol. Cellobiose was fermented after 6 days incubation. Lactose, sucrose, raffinose, and salicin were not attacked. Dextro-tartrate, levo-tartrate, mucate, and citrate were utilized but meso-tartrate was not attacked.

On serological examination the organism was found to be a member of group D of the Kauffmann-White classification and in absorption tests it completely removed agglutinins from *S. gallinarum* serum. The O antigens of *S. pensacola* are IX, XII. When the H antigens were examined it was found that *S. pensacola* was flocculated in low dilution by the majority of serums derived from forms related to *S. enteritidis, i.e.*, the monophasic forms containing antigen g. However it was agglutinated in high dilution only by serums derived from *S. california* (IV,XII:g,m,t . . .) and *S. oranienburg* (VI,VII:m,t . . .) In these two serums it was agglutinated to titer. When

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