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Acidification of Bacterial Suspensions Containing Formaldehyde.*

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It is well known that the rate of bacterial autolysis as measured either by morphological disintegration or by chemical determination of the accumulating split-products, varies widely among the pathogens. No mention could be found, however, of this process being accelerated by an aldehyde.

Certain strains of the gram-negative diplococci (thrice washed in saline) have been found to become acid in suspensions containing appropriate concentrations of formaldehyde. While most of these observations were made on bacterial suspensions equivalent to 0.25% dry weight of organisms, the reaction was found to be roughly proportional to the concentration of bacterial material. The acidification was measured by the frequency with which small additions of N/10 NaOH were required to bring the reaction of the suspensions back to pH 7.2. It was most rapid in concentrations of 0.7-2% formaldehyde, was much slower (about one-eighth the maximum) in 4% and failed to occur in 8% formaldehyde. It was more rapid at 37°C. than at room temperature.

No acidification took place when, before adding formaldehyde, the suspension was boiled for 1 minute, heated to 75°C. for 5 minutes, or 60°C. for 20 minutes. A temperature of 50°C. for several days did not inhibit the action noticeably. The presence of 10% alcohol reduced the action and 20% alcohol prevented it altogether. Heavy metal showed an inhibitory property, *e. g.*, 0.1 mg. of silver nitrate per 10 cc. inactivated the mixture. 0.001 mg. potassium cyanide per cc. of suspension prevented the reaction.

Suspensions which had been rendered alkaline to phenolphthalein for a few hours failed to become acid. A suspension started at pH 7.2, to which no alkali was added during 7 days at 37°C., developed an acidity of pH 3.2.

Accompanying the acidification of the formolized suspensions is an increase in the non-protein nitrogen as compared with the control suspensions. This was determined on the sodium tungstate-tungstic acid filtrates.

These observations suggest that the phenomenon is an autolytic

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digestion of cellular proteins accelerated by combination of formaldehyde with the protein-split products. It occurred in the case of 5 meningococcus strains of 7 investigated, one gonococcus of 5 examined, and a catarrhalis strain. It was not observed in the case of a single strain each of Pneumococcus (rough), Type III Pneumococcus (smooth), Staphylococcus, nor *Streptococcus hemolyticus.*

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Meteorological Influences on Leukocyte Curve.

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When leukocyte counts are made at short time intervals (5 or 10 minutes) in human subjects or in experimental animals the fluctuations afford an excellent index of the autonomic balance of the splanchnoperipheral system.[1] In addition to this tide, the general leukocytic level will reflect the functional status of the bone marrow and the lymphatic system.

In the course of observations on various constitutional types and their reactions to the environment, we have made daily leukocyte counts under morning basal conditions. These revealed several interesting facts. First, that the leukocytic level is rather characteristic of the constitutional type, the pyknic in general having the most deficient bone marrow and the higher leukocytic levels.

Second, that the fluctuations in the level occur simultaneously in all individuals. While the pyknic may react more vigorously, the leptosome and the asthenic show a synchronous increase or decrease in the level, although the fluctuations may be of minor degree in such persons as might be anticipated from their general somatic reactivity.

Third, that these fluctuations are meteorologically conditioned. Inasmuch as the meteorological environment in its many components offers many individual factors that influence the organism (temperature, humidity, pressure, ionization, etc.) an arbitrary identifica-

[1] Petersen, Wm. F., Müller, E. F., and Boikan, Wm., *J. Infect. Dis.*, 1927, **41**, 405; Petersen, Wm. F., and Müller, E. F., *Arch. Int. Med.*, 1927, **40**, 575; Arquin, S., PROC. SOC. EXP. BIOL. AND MED., 1927, **25**, 97; Müller, E. F., Petersen, W. F., and Hölscher, R., PROC. SOC. EXP. BIOL. AND MED., 1930, **27**, 544.