a pleomorphic, Gram negative bacillus, motile and spore-bearing. A detailed description of the special technique employed in its isolation and cultivation, together with a more complete account of its biological characters, will be given in a subsequent publication.

From cultures of this bacillus it has been possible to extract a soluble principle which, in the absence of the living cell, decomposes this specific carbohydrate. The decomposition of the specific polysaccharide is indicated by the appearance of reducing sugars in the hydrolyzed mixtures and by the simultaneous disappearance of serological specificity. The rate of reaction and the total amount of specific substrate decomposed appear to bear a quantitative relationship to the concentration of the active principle.

The active substance present in the sterile bacterial extracts is heat labile, being destroyed by exposure to a temperature of 60° to 65° C. It is extraordinarily specific in its action against the polysaccharide of pneumococci of Type III, since the capsular carbohydrates of the specific types of Friedländer's bacillus, and even those of pneumococci of Types I and II, are unaffected. The fact that the reacting substance is a product of living cells, that it is specific and heat labile and that its action seems to conform to the laws of enzymatic reactions strongly supports the view that the active principle is of the nature of a specific enzyme.

The addition of an active extract to media does not inhibit growth or cause lysis of pneumococci; however, organisms of Type III, when grown under these conditions, are not specifically agglutinable in immune serum of the homologous type. That the *function* of elaborating the type-specific substance is not destroyed, however, is shown by the fact that pneumococci so treated continue to produce the capsular polysaccharide when transferred to a medium devoid of the active hydrolyzing agent. These two facts, namely, the decomposition of the specific carbohydrate removed from the pneumococcus cells and the hydrolysis of the specific capsular substance as rapidly as it is formed in growing cultures are evidence that the active principle is directed against this single, specific component rather than against the cell as a whole.

Previous studies on infection with pneumococci have led to the view that the invasiveness of these organisms is conditioned, in part at least, by the presence of the cell capsule. Since, early in the present work, the experimental evidence pointed to the fact that only the capsular material of the cell is vulnerable to the attack of this enzyme, it was tempting to determine whether the course of pneumococcus infection in a susceptible animal might not be favorably influenced by the injection into the animal of this specific enzyme. This possibility seemed more likely, since it was found that the activity of the enzyme *in vitro* is not inhibited or retarded by the presence of fresh animal serum. Repeated experiments with various preparations of sterile extracts containing the specific enzyme have demonstrated that the active principle has a distinct and specific protective action in mice experimentally infected with pneumococci of Type III. The protection afforded is type-specific, being effective only against pneumococci of this particular type. The protective value of the enzyme is destroyed by heating the bacterial extracts at 70° C. for 10 minutes. The capacity of any given preparation to protect animals against infection bears a definite relationship to its power to decompose the specific polysaccharide *in vitro*.

In addition to its protective action, the active principle has been found to exert a specific prophylactic and curative effect on experimental Type III pneumococcus infection in mice.

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