influence of ultra-violet light to give the corresponding hydroxy-acids:

$$R.CH(NH_2).COOH \rightarrow R.CH(OH).COOH.$$

Here again, a certain resemblance exists between enzymatic and photochemical processes.

This photolysis of amino-acids is a general reaction. It is the primary process in absence of oxygen and precedes, therefore, the well-known photo-oxidation of amino-acids[3] into the corresponding aldehydes:

$$R.CH(NH_2).COOH \rightarrow R.CHO.$$

Histidin, for example, is reported by Szendrö[4] to give imidazol-acetaldehyde, on irradiation. We have been able to show that here, too, ammonia and a hydroxy-acid are formed primarily; the presence of the latter one is easily demonstrated by the colour reaction with 2.7 dihydroxy-naphthalene[5]. The reaction velocity is high: 115 c.c. of a 1 per cent aqueous solution of (-)-histidin, irradiated for two hours in the apparatus, described previously, gave 18·4 c.c. of $N/10$ ammonia, corresponding to a 25 per cent decomposition of histidin in the above sense. In presence of oxygen only, the hydroxy-acid is degraded into imidazol-acetaldehyde.

The histamin-like biological effects of an irradiated histidin solution[6] are due to imidazol-acetaldehyde according to Szendrö[4], and there is no need to assume a photolytic degradation of histidin into histamin proper. As imidazol-acetaldehyde resembles histamin physiologically, the possibility has to be kept in mind that the "histamin-precursor" identified by Barsoum and Smirk[7] with histamin on the basis of their biological resemblance is also imidazol-acetaldehyde.

It would be worth while to test the biological effects of imidazol-hydroxy-propionic acid.

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Sulphonamide Drugs and Pneumococcus Capsular Polysaccharides

THE remarkable curative effects on certain types of bacterial infection of drugs of the sulphonamide group[1] immediately opens up a new field of research into the mode of action of these drugs. In a bacteriological examination of organisms isolated from the peritoneal cavity during a course of treatment of an experimental pneumococcal infection in mice by 'M and B 693' (2-[*p*-aminobenzenesulphonamido]-pyridine), Whitby[2] reported that the "capsules became swollen and crenated and eventually disappeared". Hence it might seem that the drug has a direct destructive action on the pneumococcus polysaccharide-capsular material. It is well known that the capsule is a defence mechanism of virulent bacteria and, for example, in pneumococcal infections, is probably the decisive factor in determining the fate of the bacteria in the host. Any destructive action on capsular material by sulphonamide drugs would be comparable to the effects of the polysaccharide-splitting enzymes studied by Avery, Dubos and others[3]. In this work it has been clearly demonstrated that denudation of the organism of its capsule leaves the cell exposed to the phagocytic action of the body and usually the attack of pneumonia is repelled.

We have thought it of interest to ascertain whether solutions of sulphonamide drugs have any destructive action *in vitro* on purified capsular polysaccharides from Type I and Type II pneumococcus. Accordingly dilute solutions of the polysaccharides were separately incubated in physiological saline during several days at body temperature, with saturated solutions of sulphonamide, sulphonilamide (supplied by the Glaxo Laboratories, Ltd.) and 'M and B 693' (supplied by Messrs. May and Baker, Ltd.). The solutions were systematically examined for changes in specific rotation, increase in reducing power, change in viscosity and for loss in precipitin reactivity with homologous immune antisera.

We could clearly demonstrate that under the above conditions the drugs have no chemical action on the specific polysaccharides. It is likely, therefore, that the action of the drugs *in vivo* is not one of capsule destruction but one of capsule inhibition. Whilst this work was in progress, a recent investigation by McIntosh and Whitby[4] has thrown considerable light on the problem. These authors favour the idea that the mode of action of the drugs is in some measure bound up with the inactivation of the bacterial enzymes responsible for the food requirements of the organisms. In support and extension of these findings it is of interest to recall the work carried out in this department and elsewhere[5] on polysaccharide synthesis by bacteria. In the cases studied here it has been found that polysaccharide production is extremely capricious, for not only must the organisms have their appropriate food supply but a highly specific 'accessory growth factor', albeit in minute quantity, must also be available. It is possible that *in vivo* sulphonamide drugs can render unavailable to the organism the necessary 'accessory growth factor' and thus inhibit capsule formation; the body would then be able to rid itself of the invading bacteria by phagocytic action.

This tentative explanation would account for differences observed in the mode of action of various drugs on different groups of organisms, since 'accessory growth factors' are sharply specific for each group. A search in peptone broth or in blood for such growth-substances responsible for capsule production in the pneumococcus group, for example, might well prove a profitable study.

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