was also obtained. Finally the retina was fixed, stained and mounted, and a second map of the blood vessels made. By comparing the two maps the electrode sites could be related to the retinal structure.

According to the map, one electrode site was at the circle marked in Fig. 3. The optic nerve fibres are seen dragged aside as the tip moved over the artery. Apart from the two cells shown, there was none within a radius of 500 μ from the circle except for one at the zero of the scale. The large and smaller cells of Fig. 3 appear to be correlated with the large and smaller spikes of Fig. 2, which were recorded from this locality.

Conclusions. The retinal spikes arise from large ganglion cells rather sparsely scattered in the ganglion layer of the retina (Fig. 3). The cell has a wide-thrown ramification of branching filaments extending some 0·5 mm. in all directions. These cells appear to collect impulses from a wide region of the retina, and to be ill-adapted to discriminate form and colour.

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Role of Co-factors in the Inhibition of Bacterial Viruses by the Somatic Antigens of *Shigella sonnei*

During the course of an investigation[1] on the susceptibility of *Sh. sonnei* Phase I and II [2] and their phage-resistant variants to the *T* series of bacterial viruses, it was found that the Phase I micro-organism was lysed only by T_2 and T_6, whereas the Phase II bacillus was susceptible to T_3, T_4 and T_7 as well as T_2 and T_6. Solutions of the chemically purified and immunologically specific somatic antigens derived from Phase I and II bacilli[3] were incubated at 37° C. with approximately 2,000 particles per ml. of the appropriate viruses in an ammonium lactate medium (*F*)[4]. The bacteriophages employed were kindly supplied by Dr. Mark Adams, of New York University. Fresh stocks of the T_3 and T_7 viruses were prepared in nutrient broth using *E. coli* B as the host cell. T_2 and T_6 were prepared in *F* medium, and the T_4 virus in *F* medium containing 5 micrograms of l-tryptophane per ml. When 0·1 ml. of the mixture was assayed on nutrient agar by the Hershey poured-plate technique[5], there was no evidence that the lipocarbohydrate–protein antigens, even in concentrations as high as 0·1 mgm. per ml., would inhibit the lytic action of the viruses. This indicated that the purified antigens were incapable of preventing adsorption of the virus by the bacterial cell. These observations appear to be contrary to those of other investigators[6], who found that extracts of a variety of micro-organisms, which presumably contained immunologically active carbohydrates, have the power of preventing the adsorption of bacterial viruses to the host cell.

Further investigation has now revealed that the somatic antigen of Phase II *Sh. sonnei* in the presence of broth does inhibit the lytic action of T_3, T_4 and T_7 on the homologous micro-organism. On the other hand, the same antigen fails to inhibit the action of T_2 and T_6. It appeared that some constituent of the broth plays an essential part in the selective inhibition of T_3, T_4 and T_7 by the Phase II antigen. Anderson[7] has shown that l-tryptophane serves as a co-factor in promoting the absorption of T_4 and T_6 on *E. coli*. It seemed possible that this amino-acid might likewise be involved in the combination of T_3, T_4 and T_7 with the cell-free somatic antigen derived from *Sh. sonnei* Phase II.

Inhibition of bacterial viruses T_3, T_4 and T_7 by the somatic antigen of Phase II *Sh. sonnei*

Bacterial virus tested	Medium used	Final concentration of Phase II antigen in virus–antigen mixture*		
		0·1 mgm.	0·01 mgm.	0·001 mgm.
T_3	*F*	0	0	0
	Broth	99	65	12
	F plus 0·2 mgm. l-tryptophane per ml.	0	0	0
T_4	*F*	0	0	0
	Broth	99	96	93
	F plus 0·2 mgm. l-tryptophane per ml.	98	85	63
T_7	*F*	0	0	0
	Broth	98	93	81
	F plus 0·2 mgm. l-tryptophane per ml.	0	0	**0**

* The figures represent the percentage of virus inactivated. *F* = ammonium lactate medium.

When 0·2 mgm. of l-tryptophane per ml. was added to *F*, as little as 1 microgram per ml. of the Phase II antigen inhibited T_4, but had no effect upon T_3 and T_7, as can be seen from the accompanying table. Since the latter viruses are inhibited by comparable quantities of the Phase II antigen in the presence of broth, it would appear that in these instances still other co-factors are involved.

The antigen of Phase I *Sh. sonnei* caused no inhibition of T_2 and T_6 when tested with Phase I micro-organisms, or of T_3, T_4 and T_7 when tested with Phase II bacilli, even in the presence of broth. It is evident, therefore, that the inhibitory action of the Phase II antigen is highly specific, and that a co-factor is required in order for it to block combination of T_3, T_4 and T_7 with the host cell.

These experiments leave certain facts still unexplained. It is not yet known whether the virus combines with the protein, the lipid or the carbohydrate constituent of the somatic antigen; but work now in progress should establish this point. The nature of the co-factors present in broth which permits T_3 and T_7 to be inhibited by the antigen is not yet known. It would appear, however, that in the case of Phase II *Sh. sonnei* the substance which combines with T_3, T_4 or T_7 is a constituent of the lipocarbohydrate–protein complex distributed at the bacterial surface, and that this combination occurs only when an essential co-factor is present.

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