ASSOCIATION OF MENINGOCOCCAL INFECTION WITH OTHER DISEASES.

Influenza.—In the Public Health Report it was stated that up to September the prevalence of influenza did not have a very marked effect on the incidence of meningitis in either camp.

The attached table shows the monthly incidence of meningococcal infection, and the number of influenza and measles cases reported:—

Month.	Trentham.			Featherston.		
	Pneumonic and Meningococcal Cases.	Influenza.	Measles.	Pneumonic and Meningococcal Cases.	Influenza.	Measles.
January	1	207	58	..	56	31
February ..	..	218	70	..	91	113
March	..	71	163	4	68	71
April	..	94	21	..	88	40
May	2 (?)	164	38	4	108	57
June	5 (?)	368	54	3	247	32
July	13	618	95	2	422	77
August	20	291	47	4	416	355
September ..	9	236	141	12	224	283
October	8	348	71	4	139	238
November ..	4	395	54	6	236	148
December ..	4	59	24	4	141	76

At Trentham influenza became very prevalent in June, when 5 cases of pneumonia following measles occurred—none, however, definitely meningococcal in origin—and no cases of meningitis. In July influenza rose to its height, and 5 cases of meningitis occurred. But influenza fell to one-half in August, and now 14 cases of meningitis arose. There was another rise in influenza in November, but only 2 cases of meningitis resulted. Similarly at Featherston, when influenza was at its height in July and August only 1 case of meningitis occurred in each month. There was not then any close connection between the prevalence of influenza and the spread of meningococcus.

Measles.—With measles there is much closer union, since we must accept the pneumonic complication as being in the majority of cases a meningococcal infection. Even so there is lack of co-ordination between the prevalence of the two diseases. Thus at Trentham measles was most prevalent in March, when there were no cases of meningococcal infection; but when that infection was at its height in August measles had dropped to a total of 47 for the month—a comparatively low figure. At Featherston there was a closer connection, measles being most prevalent from August to October, when there was also most meningococcal trouble, yet when the former dropped in November the pneumonic infection increased.

Taking the histories of 46 cases of meningitis in both camps we find that in 27 of these cerebro-spinal meningitis appeared without preliminary infection by influenza or measles; in 10 it was preceded by measles; in 9 it was preceded by an influenzal attack. Of the pneumonia cases some 8 of the earlier ones at Trentham are dubiously of meningococcal origin, but we can regard 22 cases at Trentham and 19 at Featherston as meningococcal infection following measles. In addition, some 4 cases of pneumonia following influenza were probably meningococcal, so that in all 45 cases of pneumonic infection occurred as a sequel to these two diseases. It must be admitted, then, that measles markedly, and influenza to a lesser extent, were strong factors in the spread of meningococcal infections. As we have seen, however, that the prevalence of such infections did not vary according to the prevalence of measles or influenza, it is apparent that meningococcal complications were a result of chance secondary infections rather than a direct outcome of these two diseases. This affords evidence adverse to the theory lately propounded that the meingococcus may be the cause of certain influenza, like epidemics, in the course of which it becomes worked up in virulence till it can produce infections of the cerebro-spinal type. If this were so we should expect meningitis and pneumonia to have varied directly with the intensity of the influenzal outbreaks. It seems more probable that measles and influenza act by making the individual more susceptible to the attack of the meningococcus, which may thereby be given the opportunity to change from a saprophytic existence in the naso-pharynx to a virulent infective agent. Doubtless, too, its virulence would be increased by passing through a series of individuals thus made receptive by the preliminary catarrhal diseases. The role played by the carrier would be explained by this theory. We know that carriers exist fairly commonly without producing any manifestations of meningococcal activity. But if such a carrier were to come in close contact with a group of persons made susceptible by catarrh-producing diseases the chances are that some of these persons would afford a suitable nidus for the meningococcus to develop its pathogenic properties. Three factors are required for an outbreak of meningococcal infection—a carrier, a susceptible person, and a close contact between the two. An epidemic of measles or influenza means an increased number of susceptible persons. The extent to which the existence of this prepared soil will lead to encouragement of the meningococcus depends on the chance of the presence of a carrier, and on what opportunity is permitted for contact close enough to enable the organism to be transferred from the throat of the carrier to those of the receptive individuals. Probably also opportunity