This explanation seems plausible enough when we compare a densely populated area like Auckland, where free circulation of infection is probable at all times, with country districts, where there must normally be little interchange of infecting agents. But what of the towns lying between city and country—Otahuhu, Papatoetoe, Papakura—where germ exchange must be considerable, and yet the toll of the epidemic has been highest of all?

The answer will be found, I think, in the history of these places. Between 1936 and 1945 their aggregate populations increased by 39 per cent. and they are still growing rapidly. It seems probable that the greater part of this growth is due to influx not from other urban districts, but of people from country areas.

If these people with the low immunity of rural dwellers were subjected to the rapid circulation of a virus at epidemic virulence before they had had time to acclimatize themselves to town life, one would expect the incidence of positive cases amongst them to be high.

The suggestion, then, is that what happens to any population during a poliomyelitis epidemic depends largely on the degree of immunity acquired during the preceding "silent" interval. This, in turn, I have assumed to be dependent on population density. When the circulation of a virus is a matter of only a small proportion of individuals being affected at a time, even minor differences of population density might influence its range. During an epidemic, however, the proportion of the population responsible for spreading the virus must increase enormously; this is what we should expect, and a similar process has actually been demonstrated during epidemics of cerebro-spinal meningitis. In a prolonged epidemic like the present, therefore, it is hardly likely that any ordinary degree of density variation could influence the total number of persons ultimately affected by the virus; but the number reacting unfavourably (positive cases) should be smaller in dense areas than in more sparsely populated districts, at least up to a point. If, however, dispersal is carried to extremes, sooner or later a state must be reached where mere distance between families prevents effective circulation of the virus, even under epidemic conditions.

Let us put the theory to the test, beginning with the broad divisions of the district mentioned above:—

	Persons Per Acre.	Cases Per 10,000 (to 30th April, 1949).
(*a*) Auckland urban area	6·0	8·0
(*b*) Otahuhu	5·6	13·2
Papatoetoe	3·2	17·5
Papakura	1·2	58·3
(*c*) Remainder (semi-rural)	0·05	15·4

It will be seen that as the density falls, the incidence rapidly increases, until we come to the very sparsely populated rural districts, when the incidence drops again. The result is so neat that the reader may wonder why I have bothered to drag in the hypothesis that an influx of country dwellers has boosted the incidence in group (*b*). The reason is that it would clearly be absurd to pretend that density reductions of the order shown could alone account for such enormous differences in incidence. Other factors must obviously be at work.