DETERMINISTIC AND STOCHASTIC MODELS FOR RECURRENT EPIDEMICS

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1. Introductory remarks

In spite of the brilliant pioneering work of Farr, Hamer and Ross, and of important later studies by Soper, Greenwood, McKendrick, E. B. Wilson and others (see references and also the historical survey by Serfling [31]), a quantitative theory of epidemics in any complete sense is still a very long way off. The well-known complexity of most epidemiological phenomena is hardly surprising, for not only does it depend on the interactions between "hosts" and infecting organisms, each individual interaction itself usually a complicated and fluctuating biological process, but it is also, and this is a further point to be stressed, a struggle between opposing populations, the size of which may play a vital role. This last aspect is essentially one that can only be discussed in terms of statistical concepts. Greenwood (see p. 15, [16]) has remarked that "the epidemiologist's unit is not a single human being, but an aggregate of human beings"; however, even this remark omits to stress the second population of infecting virus or other parasitic invaders, and a much more comprehensive statement by Greenwood and his co-authors will be found in *Experimental Epidemiology* (see pp. 7–11, [17]). From the time of Ross at least, the importance of studying the nature, density and mode of transmission of the infecting agent has been recognized, although reliable information of this kind is often comparatively meagre. It should also be realized that the virus or bacterial populations may be in a continuous genetic or other biological state of flux. One need merely recall, for example, the existence of different strains of influenza virus, or the evidence for strains of different virulence in experimental epidemiological studies (see section 6, [17]). Considerable care is of course necessary not to confuse such variation in the virus with variation in resistance of the susceptible population, or with variation in the facility of transmission, especially when one remembers the severity of, say, a first epidemic of measles introduced into an isolated community, or asks what unambiguous evidence there is for intrinsic rise or

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