THE EFFECTS OF ASYMPTOMATIC ATTACKS ON THE SPREAD OF INFECTION DISEASE: A DETERMINISTIC MODEL†

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A deterministic model for a multi-agent disease epidemic with asymptomatic attacks is proposed and investigated. The limitations inherent in the assumptions of the model are discussed in connection with specific agents of disease. The mathematical treatment of the model is separated into analyses of the equilibrium situation and the transient behavior of the disease outbreak. Explicit formulas are derived for the number of susceptibles in the population as well as for the numbers of each type of infective—those with and without symptoms. These theoretical results are followed by a discussion of the practical considerations which must be taken into account to obtain useful information from the model.

1. Introduction. An asymptomatic attack by an agent of infectious disease occurs when that agent establishes itself in a host individual without producing any of the observable signs or symptoms which are commonly associated with a "case" of illness. For such an individual, neither the physical discomfort which is part of an active case of disease nor the social responsibility or moral sanction imposed by knowledge of being a potential spreader of the disease is available to restrict normal activities. Depending on the specific infectious agent involved, the individual may (often, unknowingly) effect the spread of infection to others.

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Many of these secondary infections may include full-blown cases of illness. In some disease outbreaks an understanding of this spread of infection by asymptomatically attacked individuals, also known as carriers, may be quite significant in determining the overall pattern of the outbreak.

The role of carriers in the spread of disease has been generally understood for many years—(see Ledingham and Arkwright, 1912). More recently, the laboratory identification of asymptomatically infected individuals has become possible for many disease agents (see, for example, the results reported in Finklestein and Gomez (1963) and Hardy and Watt (1945) on cholera and acute diarrheal disease, respectively). Moreover, the importance of such subclinical infection in the spread of disease has been demonstrated for a number of specific agents. The results on shigella reported in Nelson et al. (1968) are typical. A discussion of the general principles underlying the spread of disease, along with a glossary of epidemiological terms, may be found in Fox et al. (1972).

The laboratory identification of asymptomatically attacked individuals has obvious value in establishing disease control methods, but is often prohibited on a large scale by the considerable expense involved in mass testing to isolate (or otherwise identify) the agent in individuals who appear to be normal. Additionally, since the asymptomatic infections are often short-lived, such a mass testing program might be able to identify only those presently infected (assuming that the infection leaves no easily identifiable antibody titre). Moreover, the administrative difficulties to be overcome in order to carry out such a testing program on a large scale are likely to force rather long delays between the decision to carry out the tests and the tests themselves. Since outbreaks of infection above endemic levels can often be measured in weeks or a few months, such delays may preclude the use of testing in an outbreak situation. When the results of such mass testing are available, a large part of their value is derived from the resulting ability to generate estimates, in the various population groups, of the asymptomatic attack rates.

One of the aims of the mathematical model studied in Section 3 is a determination of the level of asymptomatic attacks from the gross behavior of a disease epidemic. That is, from the number of cases of actual illness, measured over time, we will be able to estimate the number and pattern of appearance of asymptomatic infections. If the estimation procedure can be validated by comparison with data gathered in actual outbreaks, these results might be helpful in reducing the expense of mass testing. Moreover, the results may be able to provide dependable estimates of asymptomatic attack rates where they may not have been available by any means previously.

The model presented in Section 3, while discussed in a general context,