IDENTIFICATION OF SILENT INFECTIONS
IN SIR EPIDEMICS

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A deterministic model for an SIR epidemic with silent infections is investigated. It is shown for the model studied that the extent to which silent infections are present may be determined from data concerning only those individuals with symptomatic infection.

1. Introduction. A silent infection occurs when an individual is infected without noticeable symptoms by an agent of disease. This phenomenon was investigated by Kemper (1978) for epidemics of the SIS type, in which recovery from infection provides no immunity to future disease attacks. Cooke (1979) extended those results to more general SIS models. Silent infections have also been documented for a number of acute infectious diseases which do confer immunity. Examples of poliomyelitis and the various strains of influenza come first to mind. Considering the ongoing interest in such SIR diseases, the establishment of a basis for the analysis of the role of silent infection in SIR epidemics is desirable.

The aim of this report is the investigation of a deterministic model for an SIR epidemic in which silent infections contribute to the spread of disease. In analogy to the conclusions of Kemper (1978), these results suggest the possibility of identifying the existence and extent of silent infections through analysis of infections with attending disease, data for which is more likely to be obtainable.

2. An SIR Epidemic Model with Silent Infections. Several SIR models, including a very basic one, are discussed by Hethcote (1976). The model on

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which attention is focused in this study is given by the following system of ordinary differential equations:

\[
\begin{align*}
S' &= -(r_1I_1 + r_2I_2)S \\
I_1' &= \beta(r_1I_1 + r_2I_2)S - \gamma I_1 \\
I_2' &= (1 - \beta)(r_1I_1 + r_2I_2)S - \gamma I_2 \\
R &= 1 - S - I_1 - I_2
\end{align*}
\]

(1)

with initial conditions \( S(0) = S_0 \in (0, 1), I_1(0) \geq 0, I_2(0) \geq 0 \) and \( S_0 + I_1(0) + I_2(0) = 1 \), where \( r_1, r_2, \gamma, \) and \( \beta \) are positive constants with \( \beta < 1 \).

Except for the fact that infectives recover with immunity, the epidemiological assumptions reflected in equations (1) are identical to those described in Kemper (1978) and the reader interested in the detail of the assumptions is referred there. A discussion of the validity and importance of a number of these assumptions can be found in Kemper (1980). Briefly, \( S, I_1, I_2 \) and \( R \) represent fractions of the population which are, respectively, susceptible, infected with symptoms, infected without symptoms, and recovered with immunity (or otherwise removed from the disease transmission process). The independent variable is time \( t \), which has been suppressed in equations (1).

The model above has appeared in Kemper (1980) as a model for the study of the “superspreader effect” in infectious disease transmission. The following result is essentially identical to Theorem 2 of that reference.

**Lemma 1.** For the model given by equations (1) with \( \lambda = \beta r_1 + (1 - \beta)r_2 \), the infectious contact number (as usual) is \( \sigma = \lambda/\gamma \) while the infective potential is given by

\[ J(t) = r_1I_1(t) + r_2I_2(t). \]

If \( \sigma S_0 \leq 1 \), then \( J(t) \) decreases to zero. 
If \( \sigma S_0 > 1 \), then \( J(t) \) increases to a maximum value equal to \( J(0) + \lambda S_0 - \gamma(1 + \log \sigma S_0) \) and then decreases to zero.

In either case the susceptible fraction \( S(t) \) decreases with limiting value \( S_\infty \) equal to the unique root in \( (0, 1/\sigma) \) of the equation \( S_\infty = (1/\sigma) \log (S_\infty/S_0) + S_0 + (1/\lambda)J(0) \).

The practical application of the model is dependent on the degree to which observable data can be used to identify one or more of the model’s parameters. Since data is normally available only for actual cases of illness