COMMON SOURCE EPIDEMICS II:
TOXOPLASMOSIS IN ATLANTA

RONALD SHONKWILER
School of Mathematics,
Georgia Institute of Technology,
Atlanta, GA 30332, U.S.A.

MAYNARD THOMPSON*
Department of Mathematics,
Indiana University,
Bloomington, IN 47405, U.S.A.

A discrete time stochastic model formulated for the study of common source epidemics (Shonkwiler and Thompson, 1982) is implemented to study an outbreak of toxoplasmosis in Atlanta, Georgia, in 1977. A computer simulation program is described and conclusions are drawn on the basis of the simulations. Also, the detailed empirical data are organized to illuminate the roles of visitation and location patterns, and the variation in the empirical epidemic curve with different reporting schemes.

1. Introduction. In an earlier report (Shonkwiler and Thompson, 1982) a mathematical model for common source epidemics was formulated and an application was sketched. Here we describe that application in greater detail. The incident to be studied is an outbreak of toxoplasmosis, whose epidemiology has been reported (Teutsch et al., 1979) and which is reviewed briefly in Section 2.

In Section 3 we organize and analyze the data of the epidemic in ways which facilitate implementation of our model. Several parameters which are important in the subsequent simulations are identified and discussed.

Our common source epidemic model (hereafter referred to as the CSE model) is specialized to the toxoplasmosis setting in Section 4. We describe our methods for experimental parameter identification and selection, and the computer program for implementing the model.

The results of the simulations are discussed in Section 5. Values for system parameters are discussed and questions of sensitivity are considered. In Section 6 we summarize the conclusions and comment on unresolved questions.

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2. The Setting. Toxoplasmosis is a parasitic disease of vertebrates of worldwide distribution [Feldman (1974) and Remington (1974) are useful surveys]. In adult humans it may be asymptomatic or symptomatic. A common clinical manifestation is the lymphadenopathic form, but other forms, ranging to acute infections of the central nervous system, have been described (Slim, 1956). Recent infection can normally be detected serologically, but in immunologically deficient hosts it may be impossible to determine whether infection has been acquired recently or an infection acquired earlier (perhaps even congenitally) has been reactivated.

Although some aspects of the transmission of infection remain obscure, three specific methods have been proposed: (1) ingestion of cysts of the causative micro-organism, the protozoan parasite *Toxoplasma gondii*, in undercooked infected meat; (2) ingestion of oocysts of the organism directly from contaminated air or by transfer from hands to food; or (3) congenital transmission across the placenta.

The second method, which is the method of interest here, is described in a review article by Jacobs (1974). In this case an intestinal form of *T. gondii* develops in domestic and feral cats. Asexual development begins soon after ingestion of *T. gondii* cysts by the cat and shedding of oocysts begins 3–5 days after ingestion of the cysts. The shedding period is variable, but intervals of approximately 2 weeks (during primary infection, much shorter during reinfection) are commonly cited (Dubey, 1976; Frenkel, 1974). A single cat may shed several million oocysts, each of which sporulates in 1–5 days and then serves as a source of infection. The oocysts remain viable for periods of up to at least a year, but they are vulnerable to desiccation in low humidity environments (Dubey *et al.*, 1970).

In the autumn of 1977 an outbreak of toxoplasmosis occurred among those who visited a riding stable in Atlanta, Georgia. The facility consists of a large indoor arena surrounded with stalls for the permanent boarding of horses, offices, storage rooms, etc. (Figure 1). The floor of the arena consists of extremely dry silt which has been ground to a powder, partially by the mechanical action of the horses' hoofs. This same action generates a substantial amount of dust during periods of heavy use. Analysis of data collected by epidemiologists at the Centers for Disease Control indicates that the ingestion of infected meat could not have been the primary source of the disease (Teutsch *et al.*, 1979). A pet cat was known to defecate in the silt of the arena near the office in the west end. Visitors to the arena who spent most of their time near that area had the highest incidence of infection. The analysis of the data suggests that oocysts were the source of the infection. There are other instances in which infections have been circumstantially attributed to the ingestion of oocysts (for instance, Miller *et al.*, 1972).