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The Role of Colony Organization on Pathogen Transmission in Social Insects

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Social organisms are especially vulnerable to pathogens due to the homogeneity of the colony, and the close proximity and extensive interactions among its members. However, the social organization of these groups also offers the potential to provide an effective barrier against the transmission of pathogens within the colony. Social insects with their elaborate colony organizations provide an ideal model system to develop and test this hypothesis. While the different elements of colony organization are generally assumed to be products of ergonomic selection, in this paper we address how the same elements could influence the transmission of pathogens. By developing a simple model, we explore how three parameters of colony organization, division of labor, interaction network and colony demography could influence the transmission of pathogens. We find that heterogeneity among individuals in terms of division of labor alone has little effect on the spread of an infection in the colony and the scenario is indistinguishable from one in which all the individuals are homogeneous. However, division of labor, combined with heterogeneity in the interaction network and demographic schedules reduce the spread of an infection.

Introduction

Animal species that live socially are particularly vulnerable to pathogens (micro- and macroparasites) due to the close proximity and extensive social interactions among individuals. These social groups provide pathogens with a large homogeneous environment for sustenance and excellent opportunities for transmission. A large number of studies support this idea that there is a positive relationship between the size of a social group and parasitism (Brown & Brown, 1986; Moore *et al.*, 1988; Hieber & Uetz, 1990; Davies *et al.*, 1991). However, a number of studies also show that the relationship between group size and parasitism can in fact be negative (Duncan & Vigne, 1979; Helle & Aspi, 1983; Rutberg, 1987; Poulin & FitzGerald, 1989) as a result of encounter-dilution (Turner & Pitcher, 1986) or selfish herd (Hamilton, 1971) effects. Côté & Poulin (1995) showed that the relationship between group size and parasitism is positive both in terms of prevalence (number of infected individuals in a group) and intensity (number of pathogens per individual) for only those pathogens that are transmitted via direct contact between host individuals. In contrast, the intensity of infection decreases with group size for pathogens that are mobile and do not require direct contact between hosts for transmission.

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Social Insects

Social insects constitute the most superlative example of a social group in which individuals live in close cooperation in large, dense colonies resembling superorganisms. In contrast to other social groups, the homogeneity of most of these groups in terms of both physical and genetic environment, and the close contact among the individuals make them especially susceptible to infestation from pathogens. This idea is supported by the fact that honeybees, which form large colonies, are host to a number of diseases and entire colonies of bees are sometimes wiped out by epidemics. However, the highly elaborate social organization of insect colonies could also act as a barrier to the invasion and spread of a pathogen. Therefore, it can be expected that insect societies offer a rich arena for the coevolution of host–parasite strategies in social contexts. Thus far, this idea has received little attention except in the extensive work of Schmid-Hempel (1995, 1998).

While it is generally assumed that colony organization in social insects has been shaped by factors related to ergonomic optimization (Oster & Wilson, 1978), we address the issue of whether pathogens had any selective role in its evolution and how the observed colony organization could influence transmission dynamics.† The three key features of colony organization in social insects are (1) division of labor, the existence of different sets of individuals for the performance of different tasks, (2) interaction network, the pathways along which individuals exchange food and information, and (3) colony demography, the ratio of different types of individuals in the colony and their life history schedules.

Division of Labor

In social insects, division of labor results in different individuals doing different tasks based on their morphology (physical polyethism) or their age (temporal polyethism). Such caste-based task performance may make some individuals more vulnerable to infection than others and introduce heterogeneity in an otherwise homogeneous colony. This resulting heterogeneity could pose a barrier to the successful transmission of pathogens and reduce the risk of infection to a colony.

PHYSICAL POLYETHISM

In an insect colony with physical polyethism, individuals varying with respect to their morphology perform different tasks. Such polyethism may limit the exposure of the entire colony to pathogens by allocating a single, specific group of individuals to the performance of hazardous tasks and keeping them isolated from other individuals (Hart & Ratnieks, 2001). The size of an individual worker is also known to influence its susceptibility to pathogens, and smaller or larger individuals may be more susceptible to infection (Kermarrec *et al*., 1990; Müller *et al*., 1996). The preferential infestation of the larger (and therefore costlier) morphs has led to the suggestion that such parasite-induced costs may select against polymorphism (Feener, 1987, 1988). Keller (1995) has, however, proposed that polymorphism could be favored by selection to counter parasite pressure since it adds heterogeneity to the genetically homogeneous colony.

TEMPORAL POLYETHISM

Temporal polyethism is almost universally characterized by the performance of on-nest tasks by young workers and off-nest tasks by the older ones (Wilson, 1971). Using an ergonomic approach, it has been argued that such a pattern would be selected for because a worker can increase its availability to the colony by deferring the performance of the riskier off-nest tasks to the older, less valuable ages (Wilson, 1968; Jeanne, 1986). However, Schmid-Hempel (1995) has suggested that this pattern of age polyethism could also result from older individuals posing a greater risk inside the colony, since the probability that a worker is infected by a disease could increase with age if acquiring pathogens were a positive function of time.

In species with temporal polyethism, the caste structure could be categorized as either discrete

† We would like the readers to note that our treatment of colony organization excludes reproductive division of labor or eusociality. Although a number of authors have considered the role of pathogens on eusociality, the issue is not within the scope of this paper.

or continuous (Wilson, 1976). In species with discrete castes, individuals perform sets of similar tasks while in species with continuous castes, individuals take on mainly one task at a time. It has been argued that a discrete caste structure increases ergonomic efficiency since similar tasks are spatially clustered. However, the broader task repertoires of individuals in such a discrete caste structure could also increase the probability of pathogen transmission in the colony. For example, if a cleaner bee also tends brood, it has a high probability of transferring a pathogen from infected cells to healthy brood. Given this scenario, it is interesting to note that the primarily discrete caste structure of honeybees has exceptions in that individuals performing cell cleaning (Seeley, 1982) or hygienic tasks such as removing infected brood from the colony (Arathi *et al*., 2000), are extremely specialized and do not perform any other tasks.

In species showing age-based division of labor, the speed at which individuals progress through the age polyethism schedule is also likely to have a strong effect on pathogen transmission. Once a worker grows old and switches to tasks outside the nest, a resident pathogen that infects only the brood or younger workers will have fewer opportunities to find other susceptible hosts and its transmission within the colony will be slower. This is why the precocious foraging observed in honeybees infected by the protozoan, *Nosema apis* (Hassanein, 1953; L'Arrivee, 1963; Wang & Moeller, 1970) or the sacbrood virus (Bailey & Fernando, 1972) could be a host adaptation to reduce the rate of disease transmission within the colony. Such a role for the speed of age polyethism schedule in pathogen transmission has also been proposed by Schmid-Hempel & Schmid-Hempel (1993) to explain their demonstration that rapidly multiplying variants of the trypanosome *Crithidia bombi* show a higher within-colony transmission than slower ones in the bumblebee, *Bombus terrestris*.

Interaction Network

A key parameter of all epidemiological models, but one that is very difficult to measure, is the rate of transmission (Anderson, 1982; Anderson & May, 1979, 1981; May & Anderson, 1979). In a social group, the rate of transmission is primarily determined by the frequency of interactions among individuals. The presence of any mechanism that limits such interactions can reduce the spread of a pathogen, and thus be favored by natural selection. However, interactions are also crucial to the integrity of social groups and cannot be completely selected against.

Within a group, interactions are rarely random or homogeneous and varying degrees of heterogeneity arise due to differences in spatial distribution, occupational role or social status of various individuals. Several authors have noted that such within-group heterogeneity can increase the thresholds of occurrence and persistence for an epidemic (Mollison & Levin, 1995). This probably results from a partitioning of the group into several "islands" among which the frequency of interactions is lower than that within them. Research in graph theory has shown that such small differences in a few connections in a network can result in large changes in flow patterns of the entire network (Watts & Strogatz, 1998). Such heterogeneity in interactions could also give rise to a hierarchical structure within a social group in which case a few individuals at the top of the hierarchy may have a key influence on the spread of an epidemic. Knowledge of such critical control points of an epidemic process may provide important means for designing disease management programs for social species.

In social insects, the elaborate colony organization is maintained by a vast network of interaction pathways and it has been proposed that the design of this network aids in the optimization of colony ergonomy by facilitating parallel operations of the workers (Oster & Wilson, 1978; Jeanne, 1986). Moreover, the lack of any strict hierarchy in the network facilitates exchange of information since any individual could potentially interact with any other (Wilson & Hölldobler, 1988). However, such a diffuse interaction network also has a high potential for transferring pathogens across colony members and therefore pathogens could have played a role in selecting the exact design of the network. Schmid-Hempel (1998) has shown that the same features of work organization which increase the

ergonomic efficiency and reliability of the colony also increase the rate of pathogen transmission within the colony and therefore opposite selection pressures could have been at work in the evolution of the network design.

Trophallaxis, the exchange of liquid food in a social insect colony (Wilson, 1971), is known to be a process in which any one individual distributes food to a large number of other individuals (Nixon & Ribbands, 1952; Free, 1957; Wilson & Eisner, 1957; Wallis, 1961; Markin, 1970; Korst & Velthuis, 1982; Moritz & Hallmen, 1986; Crailsheim, 1998). However, trophallaxis is not entirely uniform across all colony members and differences do exist depending on the age, task role, dominance status and hunger level of a worker (Wallis, 1962; Brian & Abbott, 1977; Howard & Tschinkel, 1980; Sorensen *et al*., 1981, 1985). At a colony level, the collection of food and its distribution in the colony is influenced by colony size, food type, amount of stored food and weather conditions (Fewell & Winston, 1996; Riessberger & Crailsheim, 1997). The factors that influence the distribution of food are also known to have synergistic interactions among themselves (Howard & Tschinkel, 1981). The enormity of the trophallactic network and the multifactorial influences on it suggest that the dynamics of social exchange could be complex in a social insect colony and produce similarly complex patterns of pathogen transmission.

There has been several demonstrations that trophallaxis is an effective pathway of pathogen transmission in colonies of social insects. Bailey & Gibbs (1964) describe that chronic bee paralysis virus is spread in honeybee colonies via trophallaxis. Jouvenaz (1986) assigns an important role to trophallaxis for the transmission of pathogens in colonies of fire ants. Transmission via trophallaxis is also important for all brood diseases since the brood in social insect colonies is completely dependent on the workers for food and care. The brood disease American Foulbrood (*Paenibacillus larvae*) in honeybees is known to be transmitted within the colony through spores passed among the workers during trophallaxis.

Allogrooming, the removal of foreign particles from the body surface of a nestmate, is another mechanism that plays a similar role in pathogen transmission as trophallaxis in social insect colonies. Although it seems that allogrooming can function to reduce the pathogen load of the infected individual that is groomed, the activity could also result in the transfer of the pathogen to the one that performs the grooming. This possibility may be well exploited by a pathogen to transmit itself among colony members. For example, in a honeybee colony infected with the chronic bee paralysis virus, the nestmates lick and chew the body of the infected workers. Whether the allogrooming is elicited by an infected worker to reduce its pathogen load or evoked by the virus to spread itself in the colony remains to be known.

Colony Demography

The size and density of the host population are two critical epidemiological parameters that determine the persistence and transmission of a pathogen in a population since these parameters determine the frequency of interactions among individuals (Anderson & May, 1986). In social insects, colony size is a highly dynamic variable and the proportion of different castes, frequency of different behaviors and many other traits vary with colony size (Wolf & Schmid-Hempel, 1990; Schmid-Hempel *et al*., 1993; Pacala *et al*., 1996). Although a larger colony probably has a higher ergonomic efficiency, it also has a higher probability to contact and transmit a pathogen among its members. Larger colonies with bigger workforces are likely to have higher contact rate with pathogens outside the colony. In addition, the pathogen would spread faster within such larger (and therefore more dense) colonies due to a higher contact rate between infected and non-infected individuals. Such opposing selection pressures resulting from ergonomic and pathogenic considerations could lead to the evolution of intermediate colony sizes. The observation that *Nosema* has a only a moderate effect in honeybee colonies of intermediate size while both large and small colonies are more severely affected (Jeffree & Allen, 1956) is a pointer toward such a possibility.

Although most studies regarding the relationship between colony size and parasitism in social

insects report a significant positive correlation (Strassmann, 1981; Wcislo, 1987; Matthews, 1991; Müller & Schmid-Hempel, 1993), reports of negative correlation (MacFarlane & Pengelly, 1974) or no correlation at all (Gamboa *et al.*, 1978) also exist. Such conflicting results stress the importance of considering the population dynamics within the colony in understanding the influence of colony size on parasitism. Since individuals of different castes may have different probabilities of contacting a pathogen, the ratio among these castes will be an important epidemiological parameter. The birth and death rates of various castes within the colony as well as the developmental rate at which individuals progress from one caste to another (in a system with temporal polyethism) will determine the number of susceptible and infected individuals in the colony and the dynamics of the infection. In honeybees, the incidence of chalkbrood disease has been shown to be influenced by both the size and the demography of the colony (Koenig *et al.*, 1987; Spivak, 1993).

In order to consider the dynamics of infection in a social insect colony with regard to some of the ideas discussed above, we have formulated a simple probabilistic cellular automata model. We believe that such spatially explicit models based on locally interacting individuals are particularly well suited to capture the scenario in a social insect colony. First, the model is developed for the more general scenario pertinent to a social group in which there is no heterogeneity and all the members of the group are alike. Next, heterogeneity is introduced into the group, especially with reference to a social insect colony, in order to model how differences among the members of a social group could modify the pattern in which a disease spreads in the colony.

A Cellular Automata SIR Model for the Spread of an Infection in a Social Group

The basic model is defined by a two-dimensional square lattice that is inhabited by the host population. Each individual of the host population is free to move within this lattice and interact with individuals at neighboring locations. Individuals can exist in either of the three epidemiological states: (a) susceptible (S) are those individuals which are free of the infection and can get infected; (b) infective (I) are those individuals which carry the infection and can transfer it to other individuals and; (c) recovered (R) are those individuals that have recuperated from the infection and cannot be infected. The infection is transmitted when there is an interaction between an I and an S during the free movement of individuals.

In our model, locations on the lattice are denoted by i, j and the state, C, of each non-empty location is defined by either an S, an I, or an R individual. In each successive time-step, individuals can move across m nearest-neighbor sites by random walking.

$$C_{i',j'}(t+1) = C_{i,j}(t), \quad i' = i \pm 0,\ m$$

$$\text{and } j' = j \pm 0,\ m. \tag{1}$$

If an S is on a nearest-neighbor site to an I, the S will become infected with a given probability p,

$$S_{i,j}(t) \rightarrow I_{i,j}(t+1)$$

$$\text{if } S_{i,j}(t) = I_{i\pm m, j\pm m}(t) \text{ and } p' \leqslant p, \tag{2}$$

where p' is a randomly drawn probability value.

For the sake of convenience, and to keep the host population size constant without explicitly invoking birth and death rates, the I becomes R after a certain number of time steps, t_{rec}, and the R are reinstated as S after a certain number of time steps, t_{reins}. The removal of I as R simulates the death of an infected individual and their reinstatement as S simulates the process of the birth of a newly susceptible individual

$$I_{i,j}(t) \rightarrow R_{i,j}(t+1) \quad \text{if } t \geqslant t_{rec}, \tag{3}$$

$$R_{i,j}(t) \rightarrow S_{i,j}(t+1) \quad \text{if } t \geqslant t_{reins}. \tag{4}$$

With these three classes of individuals, the population size, N, is given by

$$N = \sum_{i,j} S_{i,j}(t) + I_{i,j}(t) + R_{i,j}(t). \tag{5}$$

In order to simulate the model, the following parameter values are used as the default set and

using these values for all but the one under consideration, the effect of each of these parameters are evaluated one at a time by changing its value. Since the nature of these simulations is stochastic, each of the simulations is iterated 1000 times and the mean is depicted. The model is simulated on a 100×100 square lattice with the host population distributed randomly over it at the initial time step.

(i) The colony consists of 250 individuals or $N = 250$

(ii) The initial number of infectives, $I(0)$ is given by $0.01N$ or 1% of the colony is infected in the beginning.

(iii) Individuals can move one cell at each time step, i.e. $m = 1$.

(iv) If an infective (I) interacts with a susceptible (S), its probability p of transmitting the infection is 0.5.

(v) Infectives (I) remain in the infective class for ten time steps, i.e. $t_{rec} = 10$.

(vi) Recovereds (R) remain in the recovered class for ten time steps, i.e. $t_{reins} = 10$.

COLONY SIZE

In epidemiological literature, the concept of a critical population size is well established (Anderson & May, 1981; Cliff & Haggett, 1990).. The population size affects the course of infection by determining the density of individuals and therefore the frequency of interactions. Fig. 1 shows that if the colony is below the critical size the infection goes extinct, and as the population size (N) increases the infection begins to persist for a longer time and above a critical N it persists indefinitely (becomes endemic). While the fate of the infection is clear if N is well above or below the critical size, the infection takes a long time to reach its equilibrium level in colonies which are close to critical size. This critical size of the colony are therefore points of phase transitions and a slight change in the colony size can either result in an eruption of a disease or make it go extinct.

INITIAL NUMBER OF INFECTIVES

Fig. 2 shows that the simulations are stable with respect to the initial number of infectives

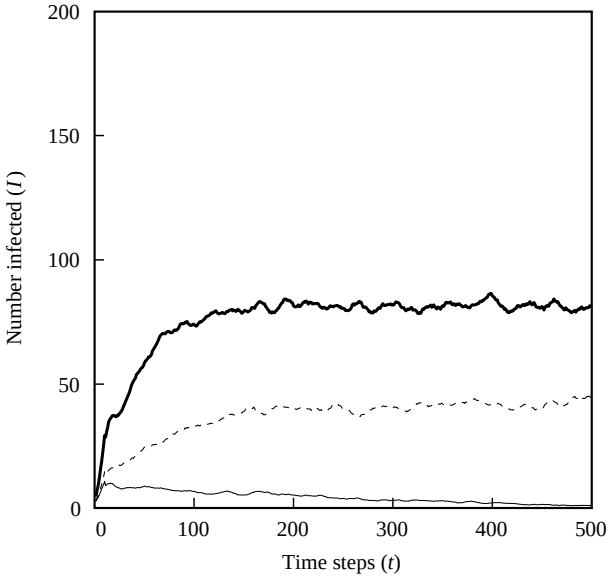

FIG. 1. Influence of colony size (N) on the number of individuals infected over time. The infection becomes extinct in colonies that are below the critical population size and reaches an endemic state in colonies larger than the critical population size. (——) Small colony refers to $N = 200$, a colony below the critical population size; (- - - -) intermediate colony refers to $N = 250$, a colony close to the critical population size; and (——) large colony refers to $N = 300$, a colony above the critical population size.

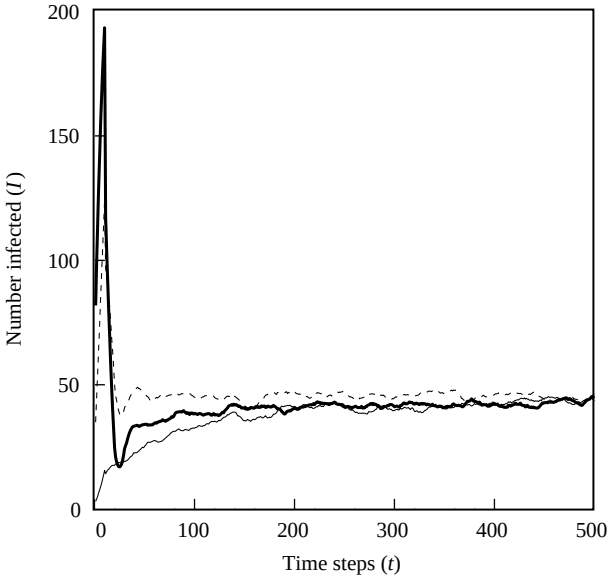

FIG. 2. Influence of the initial number of infectives $I(0)$ on the number of individuals infected over time. The endemic point of infection is not influenced by $I(0)$ while the routes to the endemic point differ. (——) Low initial infection refers to $I(0) = 0.01N$; (- - - -)intermediate initial infection refers to $I(0) = 0.1N$; and (——) high initial infection refers to $I(0) = 0.25N$.

$I(0)$ and the final number of individuals infected (the endemic fixed point) does not differ with changes in $I(0)$. However, the route to the endemic fixed point differs depending on $I(0)$, the system showing initial oscillatory behavior if $I(0)$ is large while reaching the endemic point smoothly when $I(0)$ is small. Such oscillatory behavior resulting from high $I(0)$ could lead to an extinction of the infection from the colony and may select for a pathogen that initiates an infection in low numbers.

RATE OF MOVEMENT

If individuals can move more than one neighboring sites in one time step, the infection dissipates faster and the infection is more likely to persist in the population. For a specific population size, a higher movement rate (m) can elevate the endemic point of a disease while a lower m can result in the extinction of a disease. A higher m also introduces oscillations in the initial time course of the infection (Fig. 3). Such changes in movement rates can occur in social insect colonies due to shifts in activity levels resulting from changes in temperature or resource flows in the environment.

TRANSMISSION PROBABILITY

An increase in the disease transmission probability, p, tends to decrease the critical population size for the persistence of an infection. For a specific population size, a higher p can result in the disease becoming endemic in the population while a lower p can result in the disease dying out (Fig. 4). The transmission probability is, therefore, the most fundamental parameter that determines the course of an infection in a population. The probability of transmission may depend on the intensity of contact between individuals and members of social insect colonies are known to differ in the manner in which they interact with different individuals in the colony.

BIRTH AND DEATH RATES

The birth rate in this model is an inverse of t_{reins}, the time taken by the recovered (R) to be reinstated as new susceptible (S) individuals. A

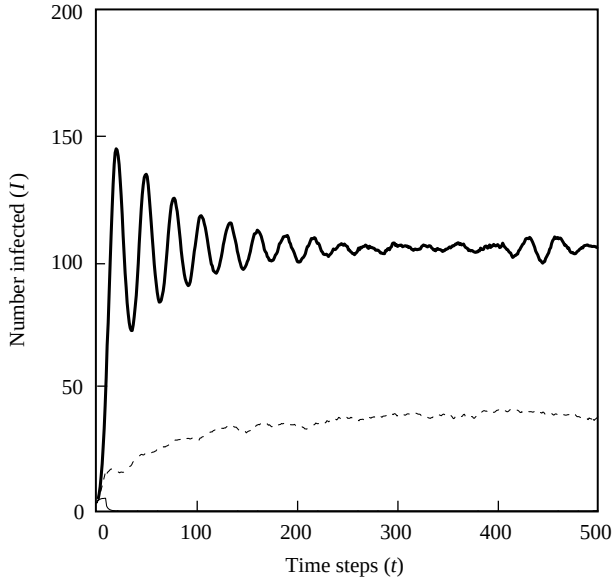

FIG. 3. Influence of the movement rate, m on the number of individuals infected over time. (——) Low movement referring to $m = 1/2$ (movement in every alternate time step) results in the extinction of the infection; (- - - -) intermediate movement referring to $m = 1$ results in a smooth approach to the endemic point; and (——) high movement referring to $m = 3$ results in an oscillatory path to the endemic point.

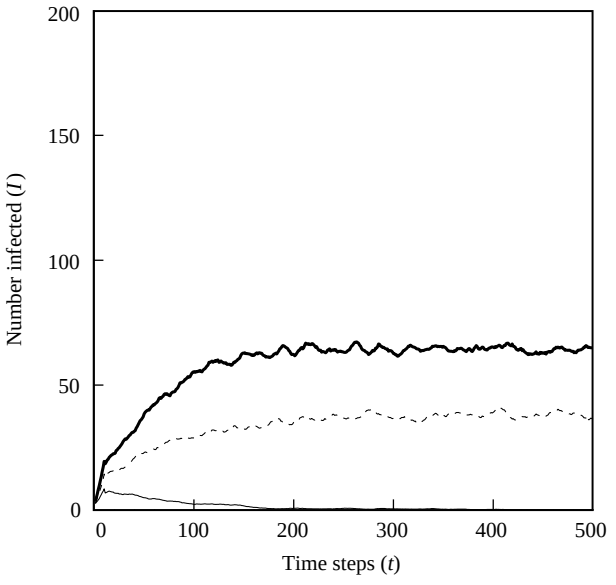

FIG. 4. Influence of transmission probability, p on the number of individuals infected over time. An increase in the value of p is seen to raise the endemic point of an infection. (——) Low transmission referring to $p = 0.25$ causes the infection to become extinct; (- - - -)intermediate transmission referring to $p = 0.5$ and (——) high transmission referring to $p = 0.75$ cause the infection to reach an endemic point.

large t_{reins} means that individuals spend a long time in the recovered (R) class before being reinstated to the susceptible (S) class or that the rate at which new susceptible individuals are born is low. Similarly, the death rate of infected individuals is modeled as an inverse of t_{rec}, the time taken by the infected individuals to move to the recovered (R) class and thereby lose the potential to infect other individuals. The larger the value of t_{rec}, greater is the time the infected individuals remain in the infective (I) class and lower is the rate at which they are removed from the population. A steady-state population is represented by $t_{rec}=t_{reins}$, a scenario where the rates at which new susceptible individuals are born and that at which infected individuals are removed are the same. Fig. 5 shows that when the population goes below a steady-state level ($t_{re}<t_{reins}$), the disease cannot maintain itself in the population. This is because the high death rate of infected individuals rapidly removes them from the population lowering their probability of infecting other individuals while on the other hand new susceptible individuals are not born rapidly enough to produce new infectives.

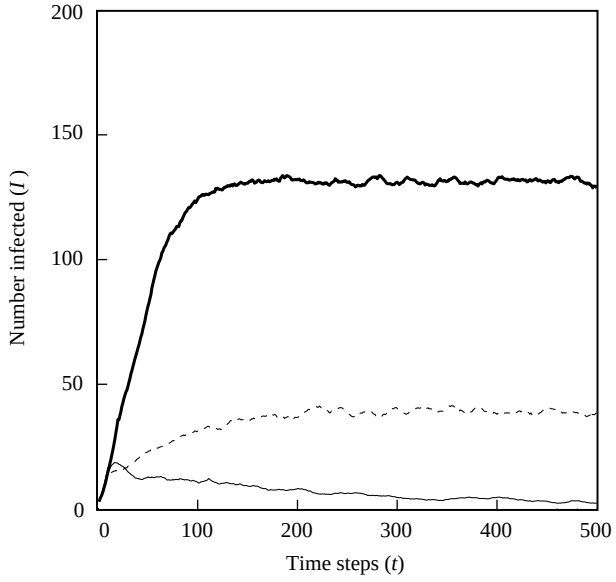

FIG. 5. Influence of birth and death rates on the number of individuals infected over time. (——) Birth rate < death rate refers to $t_{reins}=20>t_{rec}=10$ and leads to the extinction of an infection; (- - - -) birth rate = death rate refers to $t_{rec}=t_{reins}=10$ leads to the maintenance of an infection; (——) birth rate > death rate refers to $t_{reins}=10<t_{rec}=20$ and leads to the eruption of an infection.

Conversely, the disease erupts in the population when $t_{rec}>t_{reins}$ because individuals remain infective longer and newly born susceptible individuals are infused into the population rapidly. Therefore, a colony is highly prone to an epidemic during its growing phase when the number of susceptibles is high. The influence of the death rate on an epidemic is somewhat more counterintuitive. If an infection is highly lethal, it will kill an infected individual rapidly and thereby limit its ability to transmit the infection to other susceptible individuals. On the other hand, a sub-lethal infection will result in a chronic persistence of the disease in a colony leading to the well-known prediction that a pathogen may be selected to limit its virulence.

THE INFLUENCE OF SOCIAL ORGANIZATION ON PATHOGEN TRANSMISSION

Now, we modify the basic model to incorporate a social organization in the population in terms of the three factors that we discussed as being important to pathogen transmission in a social insect colony, (a) division of labor, (b) interaction network and (c) colony demography. The model is then simulated to determine how such heterogeneity among the individuals influences the transmission of a pathogen in a social group. Following the idea of division of labor as discussed earlier, the colony population N is equally divided into two types of individuals, *Type* I and *Type* II. Beginning with a scenario where the value of each parameter is the same for the two types of individuals, thereby representing the homogeneous population as modeled above, we increase the complexity of the social group in three steps by taking one parameter at a time and assigning it type-specific values.‡ Type-specific values for each parameter were assigned in such a way that the mean value of the parameter for the entire colony is kept constant throughout this set of simulations. This is to ensure that the observed effect on the

‡ Please note that the heterogeneity introduced into the model is not specific to the situation in any particular social insect group. We keep the model sufficiently general so that it applies to the broad scenario in all social insects and can be extended to other non-insect social groups.

spread of the infection is solely due to colony heterogeneity and not due to absolute changes in the parameter values for the entire colony.

Step 1: Division of Labor

As a result of division of labor, the initial number of infectives $I(0)$ could be different in the two types of individuals if, for example, the probability of contacting a pathogen is determined by the activity of that type. As a first step in adding heterogeneity to the previously homogeneous population, we assume a lower number of initial infectives among the *Type* I individuals, or $I_1(0) < I_2(0)$. Other than this, there is no further heterogeneity among the two types of individuals and they are distributed randomly across the lattice. Such a division of labor, however, does not alter the final endemic state of the infection in the colony (Fig. 6). This suggests that the mere presence of division of labor in the colony is not sufficient to impede the transmission of a pathogen in the colony.

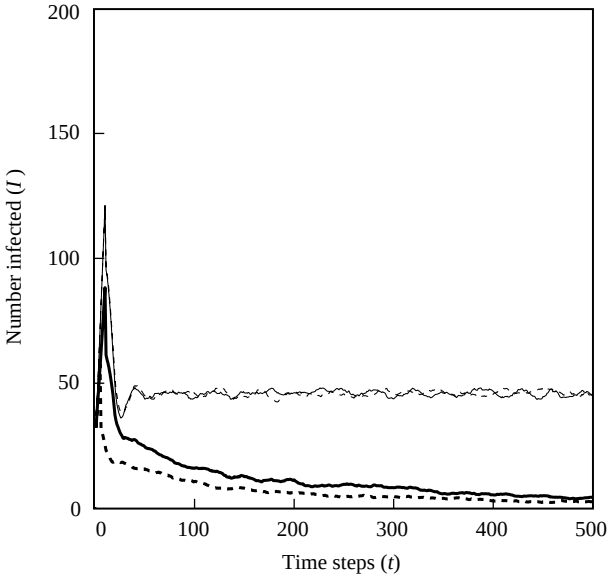

FIG. 6. Influence of colony heterogeneity on the number of individuals infected over time. Homogeneous social group referring to no division of labor (DOL) [$I_1(0) = I_2(0) = 0.1N$], no interaction heterogeneity (IH) [$p_1 = p_2 = 0.5$] and no demographic heterogeneity (DH) [$t_{rec1} = t_{reins1} = t_{rec2} = t_{reins2} = 10$] leads to the infection reaching an endemic point. Introducing only DOL [$I_1(0) = 0.01N < I_2(0) = 0.19N$] continues to lead the infection to the endemic point. Addition of IH [$p_2 = 0.05 < p_1 = 0.95$] to DOL leads to a decrease and a gradual extinction of the infection. A further introduction of DH [$t_{reins1} = 5 < t_{rec} = 10$, $t_{rec2} = 5 < t_{reins2} = 10$] to the already existing DOL and IH leads to a more rapid extinction of infection. (——) No DOL; (- - - -) DOL; (——) DOL + IH; (- - - -) DOL + IH + DH.

Step 2: Heterogeneity in the Interaction Network

In a social insect colony there could be differences in the frequency of interactions between different types of individuals and a particular type of individuals may interact more among themselves than with individuals of the other type. For example, the foragers may interact more among themselves to exchange information about foraging locations and may not interact as much with other individuals in the colony. Since the transmission probability p is likely to have a strong positive correlation with the frequency of interactions, we use p as a proxy to model differences in the frequency of interactions. Adding a second feature of heterogeneity in the colony, we now separate p into two components, the probability being p_1 for interactions between individuals of similar types (a *Type* I with another *Type* I or a *Type* II with another *Type* II), and the probability being p_2 for interactions between individuals of dissimilar types (a *Type* I with a *Type* II and vice versa). We assume $p_2 < p_1$, a scenario, where the probability of interactions between dissimilar types of individuals is lower than that between similar types. The resulting decrease in the endemic point of the infection (Fig. 6) suggests that division of labor coupled with heterogeneity in the interaction network could reduce the spread of an infection within the colony.

Step 3: Demographic Heterogeneity

As a final step, we now incorporate the third important feature of social organization in the form of demographic heterogeneity among the individuals. The two types of individuals are assumed to have different demographic schedules, e.g. foragers working outside the colony might suffer a higher mortality compared to individuals working inside the colony. Here, we assume that while the birth rate of Type I

individuals is higher than their death rate ($t_{reins1} < t_{rec1}$), the death rate of Type 2 individuals is higher than their birth rate ($t_{rec2} < t_{reins2}$). This results in a further decrease in the endemic fixed point in the colony due to a lower availability of the type that is susceptible to the infection (Fig. 6).

Using the general model to illustrate how various factors influence the spread of a pathogen in a social group, the model incorporating heterogeneity in various forms shows how certain features of social organization, especially pertinent to an insect colony, could change the transmission dynamics of a pathogen. From this model, we conclude that the heterogeneity within a social insect colony can impede the spread of an infection, lending support to our speculation that the social organization of an insect colony has evolved partly as a result of selection pressure from pathogens. The idea that colony organization could indeed present an effective barrier against pathogens is further supported when we look at how changes in colony organization due to environmental influences changes the pattern of an infection.

Environmental Influences on Colony Organization and the Resulting Changes in Pathogen Transmission

The social organization in an insect colony dynamically interacts with the environment as a result of the varying demand inside the colony and the fluctuating supplies and contingencies outside. For example, there could be an increase in the number of foragers due to an increased demand for food by growing larvae or, in contrast, there could be a sudden decrease in the foraging force as a result of a number of foragers dying from a storm. Such changes could lead to alterations in the size of various task groups as well as interaction patterns in the colony, leading to possible changes in the course of an infection process.

In honeybees, sudden outbreaks of chalkbrood disease are known to occur from low, background levels of the disease as a result of poor weather or inadequate pollen supplies (Heath, 1982). Similarly, outbreaks of *Nosema* disease in honeybees have been related to disruptions in resource flow or colony organization (Bailey, 1953, 1959; Doull, 1961) and prolonged periods of dearth sometimes result in eruptions of sacbrood virus in honeybee colonies (Bailey, 1969).

Bailey (1981) suggested that an increase in the spread of sacbrood disease in a honeybee colony during the early part of the year or a period of drought could be a result of excessive crowding or weak division of labor during such times. However, our model suggests that weak division of labor is not sufficient *per se* and must be accompanied by changes in interaction patterns to result in an increase in the spread of an infection. Bailey (1960) also suggested that the cyclic pattern in the natural outbreaks of European Foulbrood (EFB) disease could be regulated by periodic changes in nectar flow. An increase in the resource flow possibly leads to an overabundance of larval food that allows the survival of even the diseased larvae, which die *en masse* when the flow subsides and food availability becomes low, leading to a disease outbreak. In contrast, Reinhardt (1947) suggested that a good nectar flow results in a cure from American Foulbrood (AFB) disease due to a dilution of the pathogen and an increased removal of diseased larvae ensuing from a need for increased storage space. Such observations suggest that the exact functional pathways by which environmental factors influence disease transmission remain largely unknown.

Thus, the dynamics of pathogen transmission in social insect colonies are likely to be highly complex due to the elaborate social organization of the colony furthered by its dynamic interactions with the environment. Although a large number of empirical and theoretical work have shown that epidemic phenomena follow complex, nonlinear patterns (Grassberger, 1983; Anderson & May, 1986; Mollison, 1991; Mollison & Levin, 1995; Rand *et al.*, 1995; Johansen, 1996; Rhodes *et al.*, 1997), there have been few such explorations in social insects. The few models dealing with social insects (Royce & Rossignol, 1990; Royce *et al.*, 1991; Fries *et al.*, 1994; Schmid-Hempel, 1998) recognize that population dynamics of the colony would have a strong influence on transmission dynamics and that rates of transmission will differ across

different types of individuals. In our model, we first consider the basic variables that influence transmission dynamics in any homogeneous social group and then build in the essential features of colony organization of social insects, one step at a time. The merit of this explicit, bottom-up approach makes it easier (1) to understand how each aspect of a developing social organization will influence transmission dynamics and (2) to expand the model if one needs to account for any other feature of social organization that is specific to a particular group.

An Epidemiological Framework for a Social Insect Colony

The results of our model and scattered observations about disease phenomena in social insects strongly suggest that various colony and environmental factors play an important role in determining the course of an epidemic process in a social insect colony. However, the lack of rigorous empirical studies on the population biology of epidemic phenomena in social insects makes it difficult to trace the exact functional mechanisms by which such factors influence disease transmission. An empirical approach to social insect epidemiology will require studies that monitor the course of an epidemic under controlled scenarios of division of labor, interaction network and colony demography. Such studies will help us understand how various parameters influence the potential routes of pathogen flow and how environmental factors could affect these parameters to control the course of an epidemic.

Such proximate studies about the mechanisms through which a disease erupts in the colony as a result of changes in its organizational structure could use ideas from Control theory and help us devise practices that insulate critical points in the social organization and reduce the spread of an epidemic. In terms of evolutionary or ultimate viewpoint, it will be interesting to ask if parasitic pressure, in addition to ergonomic considerations, has played a role in shaping the organization of insect colonies. Such studies deserve considerable merit since the arguments presented in this paper and the scattered empirical data certainly suggest that social organization of insect colonies and pathogens could have been involved in an evolutionary arms race.

We also suggest that the study of social insect epidemiology could profit significantly from using honeybees as a model system since their colonies are host to a large number of well-known diseases. The complex social organization of honeybee colonies also provides us the scope for exploring the rich dynamics of epidemic processes that are likely to result from such organizations. Honeybees, which are highly accessible to experimental manipulations, could even be a useful model system for understanding the epidemiology of other social groups such as human societies, where there is an understandable lack of opportunities to perform manipulative studies.

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