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# How disease constrains the evolution of social systems

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Animal populations are occasionally shocked by epidemics of contagious diseases. The ability of social systems to withstand epidemic shocks and mitigate disruptions could shape the evolution of complex animal societies. We present a mathematical model to explore the potential impact of disease on the evolutionary fitness of different organizational strategies for populations of social species whose survival depends on collaborative efficiency. We show that infectious diseases select for a specific feature in the organization of collaborative roles—cohort stability—and that this feature is costly, and therefore unlikely to be maintained in environments where infection risks are absent. Our study provides evidence for an often-stated (but rarely supported) claim that pathogens have been the dominant force shaping the complexity of division of labour in eusocial societies of honeybees and termites and establishes a general theoretical approach for assessing evolutionary constraints on social organization from disease risk in other collaborative taxa.

## 1. Introduction

An outstanding challenge in social evolution research is to resolve how the interplay between demographic and behavioural processes generates variation in social structure across time, populations and species [1]. Animal societies have evolved under a myriad of ecological and environmental pressures, which probably contribute to the structure of their social systems. Social behaviours form the basis of emergent social organization, which itself governs the ability to accomplish collaborative tasks efficiently [2–4]. Moreover, there are fundamental questions about how populations maintain stability in social structure with ongoing membership turnover due to natural mortality or catastrophic events [5–8].

In addition to shaping the workforce for the performance of collaborative tasks, another potential consequence of animal social structures is to immediate the spread of infections [9–11]. Pathogens pose a significant threat to group-living species, especially when their populations are composed of closely related (and therefore genetically/immunologically similar) individuals. Theoretical models have been used to explore how and when animal populations are vulnerable to infections, and how social grouping can help to reduce transmission risk [12–14]. Recent findings suggest that social organization patterns (resulting from individual-level interaction rules) can lower disease risk without associated disease avoidance or immunity [15]. This raises questions: to what extent does disease regulation in extant animal societies rely upon emergent features of their social organization [16]? And to what extent does individual behaviour play a role in generating organizational social immunity [10]?

Of course, disease is not the only evolutionary constraint on a population. It is also critical to consider the fitness consequences of adapting social structures to minimize infection risk in view of other life-history considerations [17]. To increase the probability of individual survival of participants, not only should collaborative social groups favour increased efficiency in total productivity (thereby ensuring sufficient provision and safeguarding of the group),

they should also favour robust strategies that prevent the total population size from dropping below thresholds that expose the group to risk of extinction (i.e. the 'small population paradigm' [12,18]). Trading these fitness constraints against each other can lead to concrete evolutionary questions. For instance, how does selection for disease resistance impact populations of a species that live in naturally variable habitats? Is there a point at which lowering disease risk will maximize fitness despite any incurred costs to organizational efficiency? It has been demonstrated that individuals play different roles in animal social networks [19], altogether maintaining connectivity and social cohesion in populations. These functions will have several implications for a population's vulnerability to epidemics [20] as well as its capacity to survive other forms of disruptions [3,21,22].

We explore the evolutionary significance of risk from infectious diseases as a selective pressure that may have shaped the social organization of group-living species that must achieve sufficient productivity and population size to persist. To do this, we develop and analyse a computational model. This model focuses on individual contribution to population-level productivity, assuming that participating individuals share directly in the success of the group (i.e. indirect fitness benefits are accrued by the individual via multilevel selection on group efficiency and persistence; see [23,24]). We employ this model to consider whether the patterns of social organization that maximize fitness in the face of infectious disease are concordant or conflicting with patterns that would maximize fitness in the absence of disease pressures.

## 2. Methods

We evaluated a stochastic population model by Monte Carlo simulation describing a population of group-living species with individual birth-death processes. In our population, individuals are born/recruited at a fixed rate. While alive, individuals associate in various collaborative contexts, which generate a collective fitness benefit. Deaths occur as a stochastic process due both to aging and external causes (e.g. predation or infections). In particular, we assume individuals die randomly (i.e. due to a stochastic process sampled from a uniform distribution) while alive or deterministically after they have reached a maximum lifespan (senescence).

We assume that social living is advantageous for all population members. Individuals work together in groups to perform different tasks (e.g. foraging, care of offspring, defence against predation, etc.). The set of potential tasks is given by $X = \{1, 2, \ldots, m\}$. Groups of individuals who perform the same task are assumed to be in close spatial proximity with each other. Changes in membership of a task group can occur as a result of individual behaviour (e.g. dispersal) or mortality. The emergence of collaborative subpopulations (i.e. task groups) thus represents a form of social structure, which can impact the population's capacity to adapt to demographic fluctuations from different sources while remaining sufficiently productive.

We consider how populations of individuals using a particular strategy for task selection during their lifetime can drive both emergent social structure and organizational efficiency (figure 1). These strategies are drawn from the empirical literature (see electronic supplementary material, appendix G). Under the fixed strategy, individuals perform the same task throughout life, creating groups with permanent specialists. Under the discrete strategy, individuals perform a single task during a set age interval and move to a next new task (as a cohort) according to a pre-determined time schedule. (This strategy of life stage specialization may occur due to developmental constraints or learned social norms.) Under the repertoire strategy, individuals expand their performance of different tasks as they age (e.g. due to accumulation of knowledge or experience). Under the random strategy, individuals may switch among different tasks throughout life, creating groups with flexible generalists.

The evolutionary fitness of each strategy depends on the individual lifetime benefits it generates as a result of the distributed share of population productivity achieved (see electronic supplementary material, appendix F for an empirical argument). For each task $x$, we therefore define a productivity level, $p_{x,t}$, which describes its expected contribution to population fitness at time $t$. The total amount of benefit generated in a single time step depends on the effectiveness of population deployment among various tasks. We assess a time penalty for task switching: individuals that are chosen to switch tasks enter an inactive state for a short-time period before they can contribute to their new task (reflecting an efficiency penalty in task switching itself from, e.g. spatial distance between task locations, time required to acquire skills for a new task, etc.).

Let $n_{x,t}$ denote the number of individuals in task $x$ at time $t$ and let $\bar{n}_{x,t}$ denote the subset of active participants within the group. The amount of benefit generated in time $t$ is determined by the size of $\bar{n}_{x,t}$. To reflect the nature of collaborative task performance, we incorporate both a minimum number of individuals required to be in collaboration on each task before benefits can be generated, $W_{\min}$, and also a maximum number, $W_{\max}$, beyond which there is no additional benefit produced by the addition of another individual. Therefore, for each individual in $\bar{n}_{x,t}$, such that $W_{\min} < \bar{n}_{x,t} < W_{\max}$, each active participant contributes a benefit $B_x$, to the group's performance. Moreover, each task has an associated mortality risk $M_x$ associated with its operation and individuals who have died stop contributing work (see electronic supplementary material, appendix A for details).

For each strategy, we define an expected fitness $\pi_{\text{strategy}}$, which is the cumulative size of benefits generated from different tasks over a sequence of time periods $t = 0, 1, 2, \ldots, T$,

$$\pi_{\text{strategy}} = \sum_{t}^{T} \sum_{x}^{m} p_{x,t}(\bar{n}),$$

where

$$p_{x,t}(\bar{n}) = \begin{cases} \boldsymbol{W}_{\max} \cdot B_x & \bar{n}_{\boldsymbol{x,t}} > \boldsymbol{W}_{\max} \\ \bar{n}_{\boldsymbol{x,t}} \cdot B_x & \boldsymbol{W_{min}} \leq \bar{n}_{\boldsymbol{x,t}} \leq \boldsymbol{W}_{\max} \\ 0 & \bar{n}_{\boldsymbol{x,t}} < \boldsymbol{W}_{\min} \end{cases}.$$

We also define the expected survival $N_{\text{strategy}}$, which is the total number of individuals left alive in the group at the end of period $T$:

$$N_{\text{strategy}} = \sum_{x=1}^{m} n_{x,T}.$$

We use Monte Carlo simulations to determine whether certain task performance strategies lead to populations that are efficient and robust to diseases with catastrophic epidemic potential (electronic supplementary material, appendix B). We initialize a population by randomly generating individuals with ages drawn from a uniform distribution and assigning them to tasks according to the corresponding strategy (figure 1). For simulations with disease risk, a single individual is chosen from the population and designated as infectious (i.e. colonized with a pathogen). The spread of disease within the population is driven by frequency-dependent contact and a probabilistic transmission risk (see 'Incorporating risk from pathogens' in electronic supplementary material, appendix A). The force of infection is assumed to be greater within tasks than between tasks. Infected individuals in task x suffer an additional per-period mortality risk $M_x^*$ due to burden of carrying the disease. Once infected, an individual remains infectious until

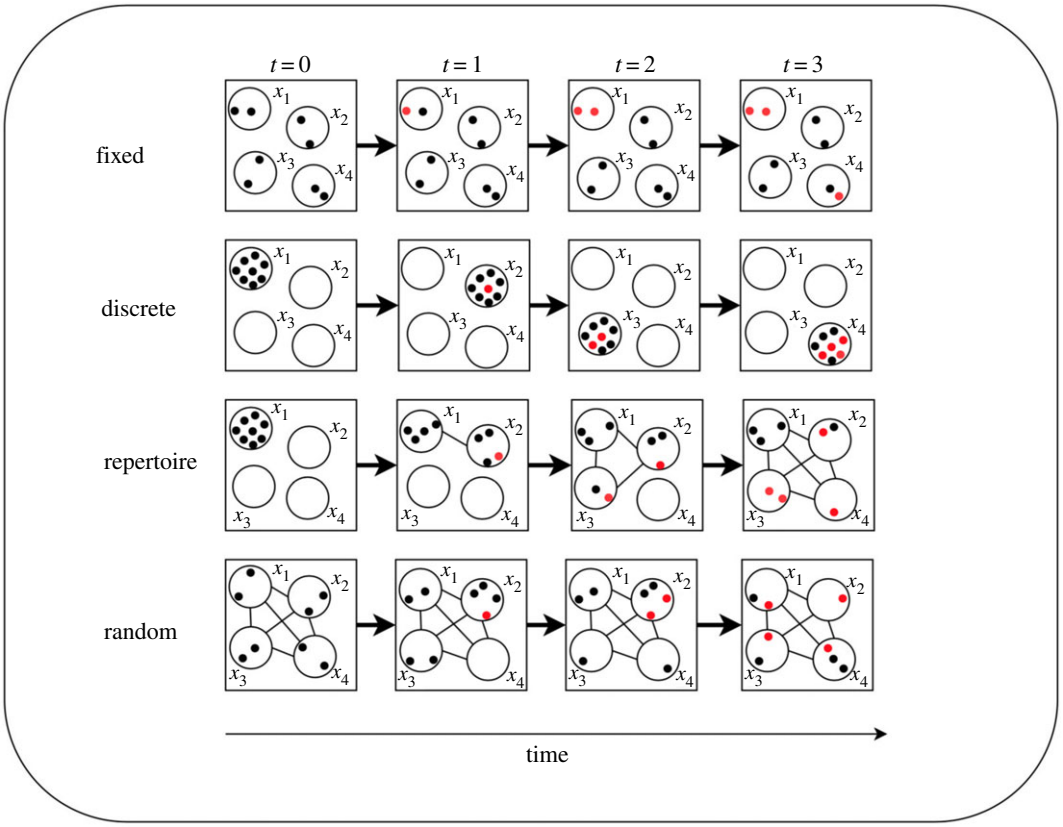


**Figure 1.** The dynamics of task allocation and individual movement with different strategies. Individuals enter the population at time $t = 0$ and assigned to one of four tasks $X = (x_1, x_2, x_3, x_4)$ either uniformly (fixed, random) or based on their age (discrete, repertoire). The duration of time spent in a task varies with different strategies. Explanatory example. Fixed: individuals remain in their natal task until death. Discrete: individuals spend a fixed amount of time in their natal task before progressing to a specific next task. Task switching occurs at the same time for all cohort members (e.g. age 0 → $x_1$, age 1 → $x_2$, and so on). Repertoire: individuals gain access to new tasks as they become older and switch among these tasks (uniformly) at stochastically chosen intervals during their lifetime. The ability to switch tasks is indicated by a line connecting the subpopulations. Random: individuals can change tasks at any point in life regardless of their age. The potential effects of social organization on pathogen transmission risks are also shown. At time $t = 1$, a single individual is chosen from the population and designated as infectious (red). The infection spreads with frequency-dependent transmission probabilities specified from an S-I epidemic model. The transmission risk is greater within tasks versus between tasks. The population-level impacts of disease will depend on the mobility of infected individuals as well as task-specific demographic rates. Births and deaths are not included in the figure. (Online version in colour.)

its death. Deaths occur as a stochastic process due to combined impacts of task-specific mortality risk, disease or aging (senescence).

### (a) Experimental design

We ask whether individual-level strategies for task performance could have differential consequences for the productivity and survival of a population under normal environmental risks (i.e. without infectious disease; see electronic supplementary material, appendix B for details). We perform Monte Carlo simulations to evaluate the effects of different strategies with a specified distribution of task-specific mortality hazards (that alter natural demographic progression). We compute the expected productivity ($\pi_{\text{strategy}}$) and survival ($N_{\text{strategy}}$) of populations in which individuals all use the same task selection strategy. These predictions are compared across the populations using the four different strategies to generate predictions for evolutionary fitness. We then perform an analogous set of simulations, but now also incorporating the introduction of infection (see above) and ask whether the prediction for evolutionarily favourable strategies (based on expected population fitness) changes with the introduction of an infectious disease.

## 3. Results

We analyse results of Monte Carlo simulations in which we explore how the uniform behavioural choices of a population according to each strategy could influence its robustness against environmental hazards and outbreaks of infectious diseases. Overall, our findings show that a strategy for social organization can drastically alter (i) a population's robustness with normal environmental risks and (ii) how well a population can handle disease threats.

### (a) Task switching maximizes demographic robustness in the absence of infections

We compare the model predictions for productivity (cumulative output of task groups, $\pi_{\text{strategy}}$) and survival (total number of alive individuals, $N_{\text{strategy}}$). Assessing the results for productivity, we find that the amount of benefits generated with generalist strategies is on the same order of magnitude as strategies with specialist strategies (table 1). However, there is a remarkable difference in the mean size of surviving populations under each strategy (figure 1). In general, we find smaller population sizes with strategies that restricted individuals' task roles at any age (fixed or discrete) relative to strategies that allowed for overlapping roles at different ages (random or repertoire). Overall, our analysis indicates that randomized task switching is by far the most effective strategy for ensuring productivity and survival under normal environmental risk (see electronic supplementary material, appendix C).

**Table 1.** Summary statistics of Monte Carlo simulations that include mortality from task-related hazards only. Entries show the mean, standard deviation (s.d.) and coefficients of variation (CV) of productivity ($\log_{10}$ amount of benefits generated during simulations) and final population size (total number of alive individuals at the end of simulations). Productivity outcomes are less variable than survival with each strategy. Coefficients of variation are lower for the age-independent strategies (fixed and random) than the age-based strategies (discrete and repertoire).

| strategy | productivity ($\pi$) $\log_{10}$ mean ± s.d. | *CV* | final pop. size ($N$) mean ± s.d. | *CV* |
|---|---|---|---|---|
| fixed | 5.5 ± *0.003* | 0.02 | 8.6 ± *4.13* | 0.47 |
| discrete | 5.3 ± *0.01* | 0.11 | 27.7 ± *30.0* | 1.08 |
| repertoire | 5.3 ± *0.09* | 0.09 | 179.1 ± *137.5* | 0.77 |
| random | 5.4 ± *0.004* | 0.04 | 72.8 ± *28.4* | 0.39 |

### (b) Infectious diseases change expectations for population success with task-switching strategies

We examine how population success with task-switching strategies might change with the introduction of infectious diseases (figure 3). Predictably, there is a significant reduction in productivity and survival under epidemic conditions. Survival outcomes under infectious exposure are lowest with strategies that either allow for unbiased movement between tasks or restrict individuals to a single task (figure 4). By contrast, survival outcomes improve with strategies that modulate individuals' task behaviours according to their age (discrete, repertoire).

### (c) Fitness costs of infections are lowest in populations with age-based social organization

We also find a remarkable effect of age-based social organization under infectious exposure that suggests enhanced demographic robustness (see electronic supplementary material, appendix D). These results show that the cohort structures created with age-determined task assignments are equally productive even when the population experiences larger, more severe, disease outbreaks. Hence, the fitness costs of infection should be systematically lower for populations where tasks are strictly divided (or assorted) according to age compared to populations without such demographic subdivisions.

## 4. Discussion

Our computational model provides the first explicit study of how social species may have evolved demographic organizational strategies as a way to balance the need for collaborative productivity against the need to mitigate the costs of socially transmitted diseases. We show that infectious diseases select for a specific regulatory feature in the organization of collaborative roles: cohort stability. We show that this feature is costly, and therefore unlikely to be maintained in environments where infection risks are absent.

### (a) Major implications

Previous studies have explored the potential influences disease might have on the evolution of social behaviour and population organization in various animal societies [25–28]. Our study advances this literature by characterizing the evolutionary fitness of populations that use different organizational strategies in the face of environmental hazards and infectious diseases. In the absence of infections, we see the four considered strategies yield different outcomes for work production and population survival (figure 2). On average, we find that populations composed of generalists (individuals with stochastically changing task roles) are equally as productive as populations composed of specialists (individuals with fixed or age-determined roles). Moreover, generalist populations were more robust in terms of survival (i.e. persistence in large numbers) compared to specialist populations (see electronic supplementary material, figure C1 in appendix C). Conversely, when we consider the impacts of infectious exposure in a standardized environment (i.e. considering only the case in which the discrete strategy is consistent with evolution under senescence theory), we see a change in the relative advantage of generalist populations over specialists (figure 3). In particular, we find that under epidemic conditions, populations where individuals performed distinct roles, independent of age (fixed), are less productive and had fewer surviving members compared to populations where individual roles changed with age (discrete or repertoire).

Our results show that age-based social organization can buffer populations from impacts of both small and large-scale epidemics. This finding can be explained by a general mechanism. In a society where individuals associate in social groups, forming stable subpopulations, the likelihood of a locally transmitted infection spreading globally is low [14,29,30]. This is because when there are few between-group contacts for the duration of a disease event, transmission becomes localized to subpopulations (i.e. increased modularity in contact structure) [9]. Under the discrete strategy, the population is stratified into age classes and temporally re-structured through the scheduled progression of cohorts into new tasks. During an epidemic event, the stability of social connections among individuals of similar ages provides a form of organizational immunity [16] in which some groups remain protected simply because contact processes does not provide a direct chain of transmission linking them to infectious individuals [9].

An important finding of our study is the limited protective effect of compartmentalization from a socially transmitted disease. This result mirror known factors in infectious disease epidemiology—the impact of link switching, for example, and household size, clustering and community size, all have well-known impacts on disease transmission [31–36]. The effectiveness of the discrete strategy in buffering infection risk comes from the cohort structure and the fact that population members always enter an uninfected social environment. With a marginally lower force of infection in the natal environment, the duration of insulation (i.e. the

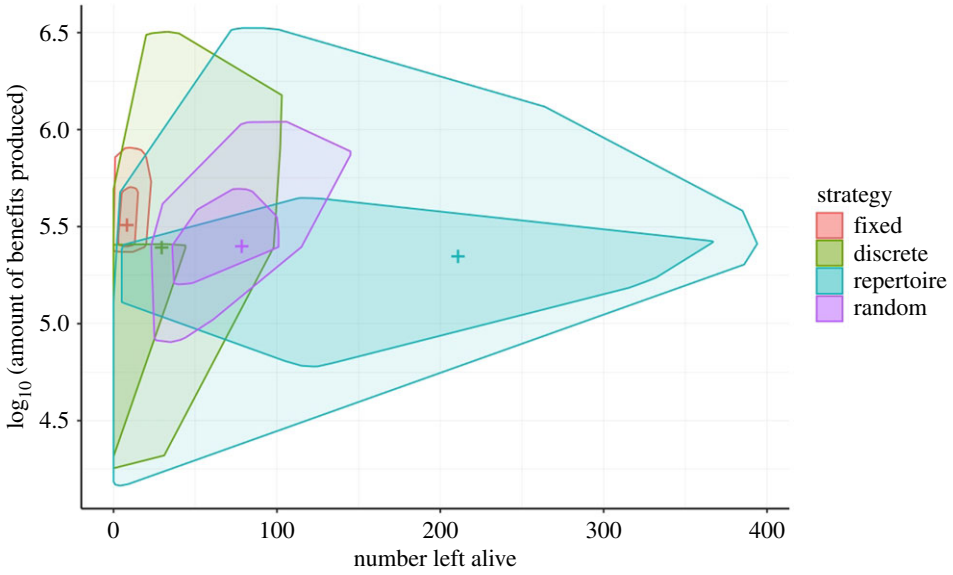


**Figure 2.** Task switching maximizes demographic robustness in the absence of infections. We use Monte Carlo methods to evaluate how well different strategies perform under environmental exposure (i.e. non-infectious mortality risk). We ask whether a population's ability to produce collaborative benefits over an expected lifespan is differentially altered by strategies that constrain individuals' task roles (fixed, discrete) compared to strategies that allow for more flexible roles (random, repertoire). An evolutionarily favourable strategy should safeguard populations against stochastic extinction (i.e. avoidance of small population sizes). The plots show the median amount of benefits generated during simulations (*y*-axis) against the median size of surviving populations at the end of simulations (*x*-axis). The depth median for each strategy (indicated by crosses) is centred on a shaded 'bag' region that contains 50% of the distribution. The outer polygon denotes convex hull that is 3 times the size of the bag region. Considering the location, spread and skew of the output distributions, we conclude that a strategy of randomized task switching can maintain populations within viable bounds without sacrificing productivity. (Online version in colour.)

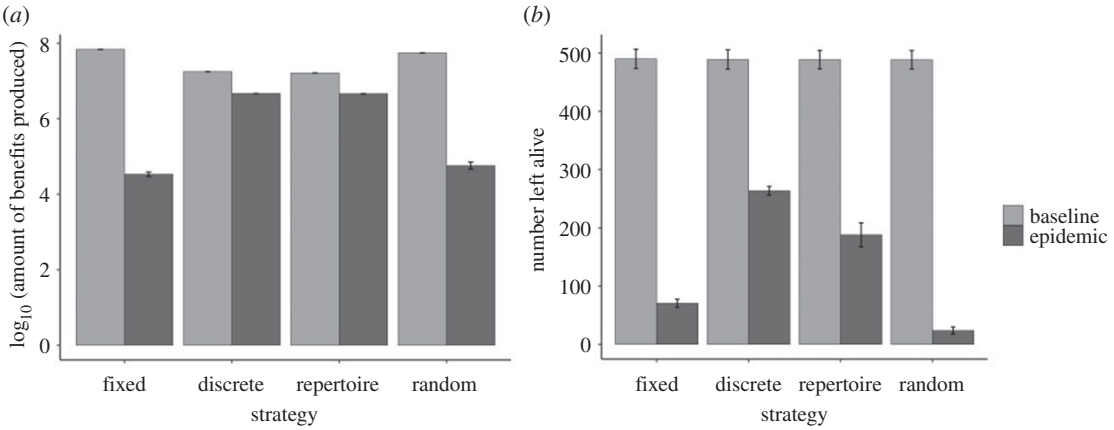


**Figure 3.** Task switching increases population vulnerability to stochastic epidemics. We test the robustness of different organizational strategies to outbreaks of a frequency-transmitted disease. Barplots summarize outputs of simulations with a disease outbreak (epidemic) compared simulations without an outbreak (baseline). Differences seen across strategies from baseline to infectious conditions are not the same for productivity (*a*) as for survival (*b*). (Note: this figure shows the subset of cases consistent with senescence theory in which older individuals undertake the riskier, but more beneficial tasks.)

length of time between when a cohort enters the system and when any one member is likely to be colonized a pathogen) is significantly increased, as has been shown in theoretical network studies (e.g. [14]). Ultimately, the perpetual flow of cohorts through task roles with age creates a temporally persistent social structure that protects subsequent cohorts from an actively circulating pathogen.

One way to appreciate the value of cohort stability in populations under infectious exposure is by comparing outcomes for the discrete strategy (wherein population members had a single task at any given age) with the repertoire strategy (wherein population members were allowed more flexibility at older ages). Our results show that difference in population vulnerability was minimal between the two strategies, despite differentially higher rates of task switching under the repertoire strategy (figure 4). Further evidence of this value can be found by comparing outcomes of the fixed strategy (wherein population members remained in their natal task until death) and the random strategy (wherein population members could move freely among different tasks at any age). Our results show population vulnerability was similar between the two strategies, thus showing that compartmentalization alone is not sufficient to protect populations against disease threats (see electronic supplementary material, appendices D and E).

Our findings have important implications about the evolutionary pressures that pathogens place on host societies. Several empirical studies have shown that social structure can have major impacts on resilience and functioning of social populations, as well as their susceptibility to infectious agents [27,37,38]. However, studies have not necessarily considered evolutionary trade-offs between the two components and strategies populations might evolve to mitigate these simultaneous, and potentially conflicting, constraints. In

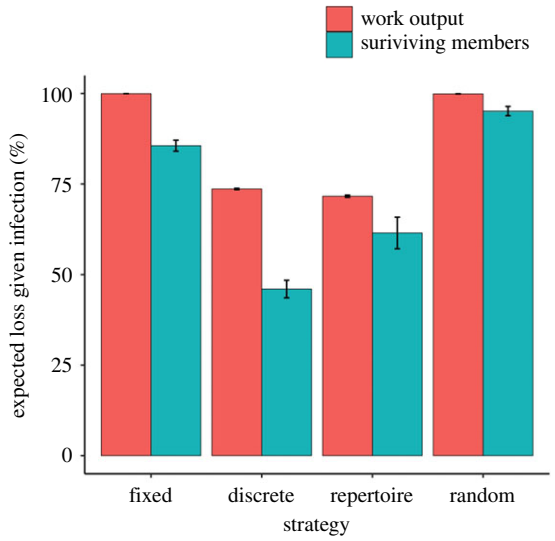


**Figure 4.** Risk exposure from epidemics is lowest in populations with age-based social organization. The expected cost of an epidemic is defined as the share of population assets (work output and population numbers) lost when an outbreak occurs (i.e. percentage difference from baseline conditions, in figure 3). Our model predicts a lower risk exposure for populations with age-determined task roles (discrete) compared to populations where individuals have lifetime task assignments (fixed). (Online version in colour.)

general, social structure can emerge from individual decisions about whom to associate with in a population, and this structure can modulate evolutionary consequences of social interactions [39]. We show that increasing the stability of social connections according to age (or any other mechanism that leads to stable demographic variation among relatively closed subgroups) provides a reliable pathway for evolving population-level robustness in the face of infectious disease. More importantly, our findings suggest there is an efficiency (performance) cost associated with evolving this robustness. In the absence of disease pressures, populations with open social structures have a higher evolutionary fitness (in terms of productivity and survival) under an unknown distribution of task-specific mortality risk (see electronic supplementary material, appendix C).

Our findings suggest types of patterns in social structure that support the theory that pathogens have been a critical selective pressure driving the evolution of complex animal societies (table 2). The evolution of social complexity depends on selective pressures acting on the behavioural traits that generate social structure within a population [40]. Obviously, social structure will be influenced by natural demographic processes (birth, deaths and dispersal), which drive changes in social connections and behaviour [1]. By showing that the productivity and persistence of social host populations under normal demographic progression varies with different levels of exposure to infectious diseases, our study provides compelling evidence in support of the hypothesis that pathogens have been a significant force shaping the evolution of animal sociality [13].

Our model allows us to make general, testable predictions. For instance, in populations where individuals lack effective immunity against pathogens, we expect that selection should favour decreases in social mobility to mitigate infection risks. Such adaptations have direct implications for levels of behavioural (cognitive) complexity required to navigate group living within structured populations [41].

**Table 2.** Evolutionary constraints on social organization from disease risk. Here we summarize model predictions about how environmental and infection risk factors could influence selection for different organizational strategies. Our model predicts that in species facing significant pressures from pathogens, age-based strategies (discrete, repertoire) should arise more frequently than age-independent strategies (fixed, random) despite costs of decreased social flexibility. This result has implications for the evolution of social complexity since behavioural rules that underlie variation in social structure and group organization will change under selective pressures from disease.

| | selective factors in social organization | | |
|---|---|---|---|
| strategy | ergonomic productivity | population survival | disease mitigation |
| fixed | ✓ | ✘ | ✘ |
| discrete | ✓ | — | ✓ |
| repertoire | ✓ | — | ✓ |
| random | ✓ | ✓ | ✘ |

Of course, as we showed in our model, a complete restriction of social connections (e.g. by remaining in natal task group) may be insufficient to guarantee population benefits and disease protection (figure 3). Thus, we should expect to see differences in social complexity among species that face disease pressures where individuals deploy different tactics for distributing social effort by age, or where whole populations have evolved integrated strategies for organizational social immunity [16]. While still preliminary, in electronic supplementary material, appendix G, we show some evidence that cross-taxa comparison of social organization in social insects is consistent with our findings. However, these are simply isolated examples, rather than a compelling, full exploration and further empirical investigation across the broad diversity of social insect taxa would be needed to see if there is true consistency in our predictions, or if these are just isolated coincidental occurrences.

## (b) Study limitations

Of course, relative success of the different organizational strategies under the two different constraints (disease robustness and efficiency) are the mechanisms by which populations with different genetic underpinnings for these strategies would be selected for/against in competition with each other over time. Therefore, these studies give us insight into the types of selective pressures that may have shaped the emergence of observed patterns in nature. In general, the intensity of selection from disease risk over evolutionary history will also depend on additional important factors that we have explicitly omitted from these initial models. For instance, while we have explored the severity of loss due to an outbreak of infectious disease, we do not consider the frequency with which the introduction of infection into populations might occur [26]. The expected intensity of selective pressure from infection should depend on the compound probability of both exposure and subsequent loss. If exposure is sufficiently unlikely in natural settings over evolutionary time, then even if the result of disease introduction is catastrophic, we should not expect that infection would play a strong role in the evolution of

social organization since the vast majority of instances would never face such pressures at all [42]. Further, the intensity of selection from infection may also be mitigated by evolved traits that are not directly related to population organizational structure (e.g. physiological immunity, allogrooming, etc.).

The logic of our study requires a comparative assessment of social organization strategies under variable environmental conditions (i.e. the key challenges confronting social populations). We therefore formulate our model as a simulation to capture the impact of stochasticity in a system with thresholds for sufficiency in payoffs, rather than exploring average expected outcomes. Similarly, while it would have been possible to derive closed-form analytical expressions for labour outputs with different organizational strategies (e.g. [43,44]), such models would be insufficient to understand the impacts of societal tasks that require a minimum degree of cooperation among group members over time [45]. Lastly, the current study focuses only on the selective pressures from disease risks while assuming that other features of social organization are held constant. A future study that incorporates the additional features of social interaction networks could be useful to further elucidate how different sources of heterogeneity within a population can limit exposure from diseases [46].

## (c) Concluding remarks

Our study explores logical foundations for an often-stated (but rarely supported) claim that pathogens have been a significant selective force shaping the organization of collaborative animal societies. Our findings are unequivocal: infectious disease can select for patterns of social organization in which individuals associate with others of similar ages and roles within the population, as this allows for compartmentalization of infection process within locally cohesive subpopulations. Moreover, we show that in the absence of disease pressures, selection for robustness and efficiency in a population (i.e. productivity and persistence under risk from extrinsic mortality) should lead to opposite patterns of social organization (i.e. one in which individuals adopt flexible social roles), as this allows individuals greater opportunities to avoid natural mortality risks and, as a result, stabilize long-term population size. While a few comparative case studies from social insects seem to support these conclusions, testing these predictions across a broader diversity of social taxa will help discern the extent to which these theoretical truths have been real-world influences on the evolution of organizational behaviours in all social species.

Data accessibility. This article has no additional data.

Authors' contributions. N.H.F. proposed and designed the study; O.U. and N.H.F. both contributed to the model/statistical implementation; O.U. analysed the data and prepared the figures; O.U. and N.H.F. both contributed to the writing of the manuscript.

Competing interests. We declare we have no competing interests.

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