

## **2. Wildlife population structure and parasite transmission: implications for disease management.**

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## **Abstract**

The beginning stages of research often involve describing patterns (e.g. males tend to more heavily parasitized than females for a given subset of species). From those initial patterns, researchers then test hypothesised mechanisms that may create those patterns. As an emerging discipline, much of wildlife disease management is about detecting trends and associations, and currently there are few guiding principles that both explain the trends and are applicable across a broad range hosts and parasites. This chapter focuses on the associations between prevalence/transmission and host social structure, where host social structure includes within group factors (eg. sex, age, dominance) and among group factors (eg. group size and movement among groups). Although there are few general theories of how host social structure affects disease dynamics it is clear that a narrow focus on either the host or the pathogen is not as productive as an integrated approach that considers the host-parasite interaction, which is itself affected by the abiotic and biotic factors in the surrounding environment.

## **2.1 Introduction**

Emerging infectious diseases have become an important challenge for wildlife ecologists and managers. Management actions to control these diseases are usually directed at the parasite, the host population, or a key component of the environment with a goal of reducing disease exposure and transmission (Wobeser 2006). Control methods directed at the host population, however, remain limited in approach (e.g. vaccination, population reduction, test-and-remove) and scope by financial, logistical, and political constraints. Furthermore, these control methods are often implemented without considering how host ecology and behaviour affect disease dynamics. This chapter highlights how host population structure and social organisation affect parasite transmission and prevalence. Traditionally, variation in disease prevalence among

species, genders, and ages may have been explained by immunological differences in susceptibility. However, ecological and behavioural factors can also affect the rates and routes of parasite transmission and potential control options. Using this information, future control efforts may be improved by focusing on subsets of individuals, areas, environmental factors, or times of year that are most important in the propagation and persistence of a pathogen.

The social systems of mammalian populations exhibit structure at several levels. Individuals vary by age, sex, reproductive state, relatedness, position in a dominance hierarchy, social interactions and patterns of space-use. Group sizes can vary within and among species, from solitary individuals that only interact during mating, to monogamous pairs, socially complex groups or aggregations of over a million individuals. Within a group, the sex, age and social status of an individual, as well as the season, will often affect the number and type of contacts a parasite experiences, thus affecting exposure and transmission rates. Meanwhile, the transmission of a parasite among groups may depend on group size, composition, territoriality and levels of inter-group movement or contact. This chapter explores how the characteristics of host social systems may interact with parasite life-history characteristics to affect parasite transmission, prevalence and dynamics, and hence the effectiveness of disease management strategies.

## **2.2 Intra-group factors**

The gender, age, dominance and reproductive status of hosts are some of the characteristics that affect parasite prevalence and transmission within a group of individuals. Most studies of these host characteristics have focused on differences in

prevalence, while only a few studies have compared incidence rates (but see Begon et al. 1999, Caley and Hone 2002, Heisey, Joly and Messier 2006). Disease prevalence depends on the transmission rate, disease-induced mortality, duration of infection (or duration of antibodies for seroprevalence), and the length of time a disease has been present in the population. On the other hand, incidence, or the rate of infection per unit time, is a function of prevalence. The distinction between prevalence and incidence is important because differences in prevalence are often assumed to correspond to differences in incidence. In some cases, however, differences in prevalence may instead be driven by disease-induced mortality or infectious periods that vary by sex, age and dominance.

### **2.2.1 Sex**

Several recent species-specific studies suggest male-biased parasitism for bovine tuberculosis (*Mycobacterium bovis* infection; bTB) and chronic wasting disease (CWD) in deer (Shang, Xiao and Yin 2002, Miller and Conner 2005), cowpox in rodents (Burthe et al. 2006), and nematodes in chamois (*Rupicapra r. rupicapra*) (Citterio et al. 2006). Analyses using data collated from studies on a range of mammal species also report male-biased prevalence and intensity of parasitism (Poulin 1996, Schalk and Forbes 1997, Moore and Wilson 2002). Several studies have identified positive correlations between host body weight and the intensity of parasite infection (Poulin 1995, Arneberg, Skorping, Grenfell and Read 1998, Burthe et al. 2006, Ezenwa et al. 2006). These findings have produced a variety of hypotheses to explain male-biased parasitism. Larger hosts may provide more space or a greater diversity of niches for parasites. They may also present a larger target for vectors (Davies, Ayres, Dye and Deane 1991), and the

greater nutritional requirements of larger hosts could increase their exposure to parasites that can be transmitted by ingestion (Poulin 1995, Vitone, Altizer and Nunn 2004). In many species males have larger home ranges, which may also lead to increased exposure (Miller and Conner 2005). Sex-related differences in physiology and behaviour may also produce differences in exposure and susceptibility to disease. During the breeding season, male mammals often experience increased stress levels, which may be linked to immunosuppression and increased susceptibility to disease (Zuk and McKean 1996). In addition, testosterone has a suppressive effect on the immune system, further increasing male susceptibility (Zuk and McKean 1996).

Mating behaviour is also likely to have important implications for parasite exposure, particularly when considering sexually transmitted diseases (STDs). For STDs transmission rates are likely to depend more on the prevalence, or frequency, of the infectious individuals rather than the overall density, because the number of sexual contacts each individual has is likely to be constant across a wide range of population densities. Mating systems range from monogamy (one male mates with one female) to polygynandry (both sexes mate with multiple partners). Polygamy (one male mates with several females) is the most common mating system among mammals (Alcock 1998). This system tends to increase the variance in mating success amongst males; some males mate with many females whilst others fail to mate with any. Theoretical investigations suggest that this reproductive variation may increase the prevalence of disease amongst females and reduce prevalence in males, as the few reproductive males are more likely to acquire and transmit infection to their partners, while non-reproductive males remain uninfected (Thrall, Antonovics and Dobson 2000). Although few empirical studies have

been conducted, the prevalence of STDs was significantly higher amongst adult females in studies of STDs in primates (Nunn and Altizer 2004) and koalas (*Phascolarctos cinereus*) (Jackson, White, Giffard and Timms 1999).

### **2.2.2 Age**

The relationship between age and parasite prevalence is related to host characteristics and parasite life histories. Assuming that hosts do not recover from infection and parasite-induced mortality is low, parasite prevalence often increases with age because older individuals have been exposed for longer (Figures 1 and 5, Heisey, Joly and Messier 2006). This has been demonstrated for bTB in bison (*Bison bison*) (Joly and Messier 2004) and African buffalo (*Syncerus caffer*, Jolles, Cooper and Levin 2005), and for CWD in deer (Miller and Conner 2005). When antibody titers persist, seroprevalence (i.e. prevalence based on serological test results) is also likely to increase with age. In these cases, seroprevalence reflects past exposure rather than current infection. The form of the relationship between age and prevalence is also influenced by changes in immunity, age-dependent exposure, and both host and parasite mortality (Heisey, Joly and Messier 2006). For example, if parasite-induced mortality increases with time since infection then prevalence may be lower in older age categories than in juveniles because older individuals are likely to have had the disease for longer and as a result die at a faster rate (Figure 1).

### **Figure. 1**

When hosts can recover from infection and become immune, juveniles may have

a higher prevalence than adults because many adults may have already been exposed and recovered (e.g. Cattadori et al. 2005). Age-dependent changes in immunity may also influence host susceptibility to disease. Infants may initially be protected by maternal antibodies, but once passive immunity wanes they may become susceptible, as in the case of rabbit haemorrhagic disease (Cooke 2002) and tapeworm infestation in mice (Theis and Schwab 1992). Furthermore, senescent individuals may be more susceptible to disease due to declining immune function (Lloyd 1995). Parasite-induced immunity may also affect age-prevalence patterns by either suppressing the immune system or priming the host for a stronger response to subsequent exposure (Duerr, Dietz and Eichner 2003). The latter seems to be the case for *Nematodirus gazellae* infections in saiga antelope (*Saiga tatarica tatarica*), in which parasite intensity peaked in 2-3 year olds but declined thereafter (Morgan et al. 2005).

### **2.2.3 Dominance**

The influence of social dominance on parasitism is complicated by breeding behaviour, rank stability, and coping mechanisms for subordinates. Dominance is likely to affect exposure rates as well as stress. In general, mild and transient stressors enhance immunity, particularly innate immunity. Chronic stress, however, can suppress the immune system, but it remains unclear whether these changes are sufficient to increase the risk of infection (Dhabhar and McEwen 1999, Sapolsky 2005). Furthermore, those individuals that experience the most stress may be at either the top or the bottom of the dominance hierarchy, depending on the stability of the hierarchy and potential coping mechanisms (Sapolsky 2005). A study of captive cynomolgus monkeys (*Macaca fascicularis*) showed that low ranking individuals had higher rates of adenovirus infection

(Cohen et al. 1997), whereas subordinate males in a koala population had lower levels of STDs than dominant individuals (Jackson, White, Giffard and Timms 1999). At this point it is difficult to determine whether these differences are driven by contact patterns, routes of transmission, stress, and/or susceptibility. Further research is necessary before information on dominance hierarchies can be used by managers to help control disease.

#### **2.2.4 Superspreaders**

Researchers, managers and disease modellers have in the past often assumed that all hosts are equally susceptible and infectious for microparasites. However, studies of some human diseases have shown that the distribution of the number of infections caused by an individual is also strongly skewed, whereby most individuals do not infect anyone, while a few infect a large number of people. As a result, focusing half of all control effort on the most infectious 20% of cases may be up to threefold more effective than random control (Lloyd-Smith, Schreiber, Kopp and Getz 2005). Such heterogeneities are also likely to apply to wildlife populations (Cross, Johnson, Lloyd-Smith and Getz 2007) offering the potential for more effective management strategies if these so-called ‘superspreaders’ can be effectively targeted. Unfortunately, there are significant logistical and diagnostic difficulties in identifying superspreaders in wildlife populations which will require the development of new theoretical and diagnostic tools. In addition, it is not clear whether managers could focus control efforts on ‘superspreader groups’ and achieve similar improvements in effectiveness of control. Intuitively however, it seems reasonable to focus attention on individuals (or classes of animals) that either have long infectious periods and/or have high rates of contact with susceptibles, as these animals are likely to be significant in the spread of disease.

## **2.3 Inter-group factors**

### **2.3.1 Territoriality**

Territorial defence often involves aggressive encounters that may increase exposure to parasites (Loehle 1995). Defensive behaviours are energetically costly and may increase stress and testosterone levels, which can then suppress immune function (Zuk and McKean 1996). Acts of aggression may also enhance transmission by biting or scratching (Hawkins et al. 2006). Territorial species may also encounter a high rate of contact with infectious pathogens within their territory through environmental contamination with parasite-laden faeces. A study of strongyle nematodes in African bovids found higher levels of infection in territorial than in non-territorial species, most likely as a result of environmental contamination with faeces (Ezenwa 2004). On the other hand, territoriality may also serve to reduce parasite transmission by reducing the overall level of direct contact between individuals or groups. This may be particularly pronounced amongst species that use indirect communication (e.g. scent marking and vocalisations) to minimise the need for direct contact. Individuals that occupy territories may also have access to more desirable resources making them less susceptible to parasitism. Meanwhile individuals that are unable to control a territory may “float” from one occupied territory to another, increasing their own exposure rates and facilitating the spread of disease across territories.

### **2.3.2 Group Size and Population Density**

Hosts living in large aggregations are likely to have more direct contacts than those in small groups. When parasite transmission is a function of direct contacts, then

prevalence is likely to increase with group size or population density (McCallum, Barlow and Hone 2001). The relationship between transmission rate and host density has profound implications for disease management. If transmission rates increase with density then reducing population size or density may be an effective management option (Lloyd-Smith et al. 2005). The distinction between population size and density is important (de Jong, Diekmann and Heesterbeek 1995). In many cases, host population size may be strongly correlated with the extent of area occupied, such that as population size increases so does the area occupied, resulting in minimal changes to density and contact rates (Begon et al. 2002). Although it is logical to assume that contact and transmission rates increase with density, the relationship may be confounded by host behaviour (e.g. territoriality vs hosts seeking contacts at low densities) and it is seldom clear how to estimate the area occupied (i.e. the denominator). Even in the simple case of a fenced park, not all habitats may be accessible or usable by a given host species. For species living in groups, contact rates are more likely to be related to local group size than overall population density.

The aggregation of animals at experimental feeding sites has been associated with significant increases in the prevalence of endoparasites in racoons (*Procyon lotor*) (Wright and Gompper 2005), *M. bovis* in white-tailed deer (*Odocoileus virginianus*) (Chaddock 1998), and brucellosis (*Brucella abortus*) in elk (*Cervus elaphus*, Cross et al. 2007). A population size of 200 susceptible animals in an area of 220km<sup>2</sup> has been suggested as the threshold density necessary for the maintenance of classical swine fever virus in populations of free living wild boar (*Sus scrofa*) (Artois et al. 2002). However, population size rather than density, was important in determining whether cowpox would

invade and persist in a field study of wood mice (*Apodemus sylvaticus*) and bank voles (*Clethrionomys glareolus*) (Begon et al. 2003). Meta-analyses have shown nematode richness, abundance and prevalence to be positively associated with population density in mammals (Arneberg 2002). Group size has also been implicated in promoting parasitism. A meta-analysis covering diverse taxa showed a positive association between group size, prevalence and intensity of contagious parasites (Côté and Poulin 1995). The relationship between parasite species richness and group size, however appears highly variable, with studies showing positive, negative and an absence of association between the two factors (Poulin 1991, Watve and Sukumar 1995, Nunn, Altizer, Jones and Sechrest 2003, Vitone, Altizer and Nunn 2004, Ezenwa et al. 2006).

For directly-transmitted parasites in a single-host system, the relationship between population density and parasite transmission may be complicated by several factors. One theoretical study showed that the probability of a pandemic occurring depended on rates of host movement among groups, group size and the duration of infectiousness (Cross, Lloyd-Smith, Johnson and Getz 2005). Chronic infections with long infectious periods (e.g. bTB) required less movement among groups to create a pandemic (i.e. an epidemic that propagates across a large region and hence many groups) than those causing acute conditions, because they were able to persist for longer within the local group. Longer persistence within a group increases the likelihood that an infectious individual moves to another group. Larger group sizes and higher movement rates amongst groups facilitated the invasion of acute infections (e.g. rabies and rinderpest). This suggests that group sizes and movement rates are likely to affect the spread of acute diseases, such as rabies, more than chronic infections, such as tuberculosis. However, parasites causing acute disease

often have alternative means of persisting, either in the environment or alternative hosts, or by causing latent infections in some individuals.

Transmission rates that vary seasonally or annually are also likely to affect the relationship between host population size and parasite prevalence. Seasonal variation in host social behaviour, such as breeding or wintering aggregations of deer and migrations of wildebeest in East Africa, may introduce temporal patterns in disease transmission. For example, brucellosis induces abortions in elk and bison prior to and during the calving season (Cheville, McCullough and Paulson 1998). Other individuals become infected by licking or consuming the contaminated foetus. In northwestern Wyoming, USA, brucellosis seroprevalence was higher at sites where elk were provided with supplementary feed later into spring, because the timing and duration of host aggregation coincided with peak transmission (Cross et al. 2007). This sort of complexity in the relationship between host population size or density and parasite transmission may be common to many wildlife disease systems.

The effects of group size and population density appear to vary widely for indirectly transmitted parasites. Studies of malaria in primates have shown a higher prevalence of infection in larger groups, possibly because more hosts increase the strength of olfactory cue to mosquito vectors (Davies, Ayres, Dye and Deane 1991, Nunn and Heymann 2005). A meta-analysis of multiple taxa of social animals, however, found that parasite prevalence in the host was negatively associated with host group size when the parasite had a mobile vector (Côté and Poulin 1995).

Many parasites are neither specific to one host species nor directly transmitted amongst individual hosts. In primates, 68% of recorded parasites infected more than one

host species and 43% were transmitted indirectly (eg. fomites or contaminated soil or water), 32% by arthropod vectors, 15% by intermediate hosts and 34% could be transmitted through multiple routes (Pedersen et al. 2005). When parasites are transmitted by vectors or have intermediate or alternate hosts, this adds further complexity to the relationships between host social structure and parasite dynamics. Consequently, in the many cases where multiple hosts share a parasite, the relationship between group size and prevalence in each host may be weak.

## **2.4 Mathematical modelling of host population structure**

### ***2.4.1 Contacts, transmission and host density***

The discussion above on the relationship between population size or group size and parasite transmission and prevalence plays a critical role in efforts to mathematically model host-parasite systems and to develop effective disease management strategies (Lloyd-Smith et al. 2005). If contact rates among hosts increase with population size, then the transmission and prevalence of directly transmitted parasites are also likely to increase. This density-dependent relationship implies a threshold host population size below which the disease is unable to persist (Kermack and McKendrick 1927, Bartlett 1957). This is the logic that underpins management strategies aimed at reducing the density of susceptible individuals below some threshold by culling, sterilisation or vaccination (Lloyd-Smith et al. 2005). However, few studies have evaluated the functional relationship between contact rates and density (but see, Caley, Spencer, Cole and Efford 1998) and the evidence for host population thresholds in wildlife disease

systems remains limited (Lloyd-Smith et al. 2005).

The paucity of evidence supporting density-dependent transmission and population thresholds is however not surprising considering the difficulties in collecting contact and transmission data at a range of densities and over the seasonal fluctuations common in many wildlife populations. Furthermore, for many parasites, it is not clear what constitutes an infectious contact (with the possible exception of STDs) nor is it simple to determine the probability of a contact resulting in infection of a susceptible host. Many species have home ranges that limit contact between infected animals and the remainder of the population. As a result, factors that drive parasite transmission such as contact, density and environmental sources of infection are likely to operate only at the local scale that affects the rate of infection across the population. For directly transmitted pathogens, contact rates are probably related to local group sizes, the spatial scale of transmission (i.e. aerosol transmission vs direct contact) and the amount of movement among groups.

The relationship between group size and total population size can also suggest how contact rates are likely to change in the face of management actions that reduce the number of hosts. For many species, the distribution of group sizes is strongly right-skewed. For example, many ungulate populations in the Kruger National Park (KNP) in South Africa, contain many small groups with a few much larger groups (Figure 2). Aerial surveys are likely to miss small groups more often than large groups, which can further contribute to the right-skew of these group size distributions. Means are often used in studies relating parasitism to group size but the expected group size of a randomly chosen individual (Krause and Ruxton 2002) may be a more relevant measure

for disease studies. This parameter ( $\sum_i n_i^2 / \sum_i n_i$ , where  $n_i$  is the  $i$ th group size) is essentially a weighted group size, which represents the average group size experienced by each individual and more closely relates to the average per-capita risk of infection.

## Figure. 2

For species with right-skewed distributions, the weighted mean will generally be much larger than either the median or mean group size (Figure 3), indicating that, although average group sizes may be small, most individuals experience groups of intermediate size. For many ungulate species studied in the KNP little or no association between the total population size and any measure of group size was observed (Figure 3). Group sizes for African buffalo and kudu (*Tragelaphus strepsiceros*) were weakly correlated with total population size, such that a doubling of the population was only associated with an increase in the weighted group size of about 25% (Figure 3). Because the perimeter of KNP was entirely fenced during this study, recorded population size was correlated with density. Hence, in this system, increases in population size are accompanied by an increase in the number of groups, while group size generally remains constant. Thus, one would not expect per capita contact rates to increase with population size, and disease management efforts focused at reducing the population size as a whole may not be effective in this case. However, contact among groups may increase with the number of groups, facilitating the invasion of a disease even if group size remains constant (Cross, Lloyd-Smith, Johnson and Getz 2005).

Similar patterns may also occur for other species where social behaviour limits the frequency or intensity of contact. For example, adult female white-tailed deer are more

likely to contact other females within their matrilineal social group (Schauber, Storm and Neilson 2007), therefore, increases in population density may not substantially alter the number of other females contacted and direct pathogen transmission may be limited.

Eurasian badgers (*Meles meles*) in the UK live in medium to large social groups where population density may be driven by changes in group size, whilst the number of groups remains relatively constant (Cheeseman, Wilesmith, Ryan and Mallinson 1987, Rogers et al. 1999). Interestingly, badger population density does not appear to correlate with levels of bTB infection, suggesting that other factors may drive transmission (see text box).

## Figure 3

### ***Group structure***

Early disease models often assumed that the host population was homogeneously mixed (Anderson and May 1991) so that each individual was equally likely to contact every other individual per unit time. Because these conditions do not hold for many human or wildlife situations, alternative methods have been developed to account for the effects of spatial heterogeneity or social structure on contact rates (e.g. Hess 1996b, Swinton, Harwood, Grenfell and Gilligan 1998). One approach is to combine individuals within categories, which may be based on sex, age, dominance or core risk groups (e.g. drug addicts sharing needles), and then incorporate data on contact rates within and among classes of individuals using a mixing matrix (Blower and McLean 1991) to scale transmission rates within and among categories or subpopulations of individuals.

Researchers have also used network models as a flexible method of capturing the socio-spatial structure of populations (Keeling 1999, Watts 1999, Ferrari, Bansal, Meyers

and Bjornstad 2006). While traditional transmission models assume that the risk of infection depends on the prevalence or density of infectious individuals in the local (or global) population, network models explicitly incorporate information about relationships among individuals and calculate infection risk as a function of known contacts with infectious individuals. These models have been used primarily to describe the dynamics of sexually-transmitted infections where contacts among individuals may be limited and variable. One strength of network modelling is its inherent flexibility to represent a wide range of social or spatial structures. In fact, metapopulation or patch models of disease can be thought of as a subset of network models where everyone within a group is connected and between group connections are infrequent. To date, most network models have been static due to the lack of empirical data on temporal changes in network structure. However, these models can be used to illustrate how the contact network evolves over time as individuals become infected and die. Individuals with the most connections are likely to be infected first, leaving a more sparsely connected network of susceptibles and hence making disease persistence more difficult (Ferrari, Bansal, Meyers and Bjornstad 2006).

Network models are often under-pinned by a matrix of pairwise contact probabilities (Figure 4), where the element in row  $i$  and column  $j$  of the matrix describes the connection (or lack thereof) between individuals  $i$  and  $j$ . These connections are often assumed to be binary in that contact either does or does not occur. Alternatively, values of the matrix may reflect the relative strength of the connections between individuals or populations. In a study of African buffalo, the proportion of time that pairs of individuals spent in the same herd was estimated from radio-tracking data (Cross et al. 2004). These

contact indices were multiplied by infection rates or probabilities, to simulate disease transmission dynamics. Properties of the contact network may be particularly important for acute infections where the disease can become extinct within a local group prior to any connections forming between groups. For chronic diseases (e.g. CWD, bTB) the network structure connecting different groups may be less important because disease persistence is long relative to the rate of new connections between groups. However, the importance of network structure for intra-group transmission of TB in meerkats (see Text Box) suggests important species-specific differences in this relationship.

Network and metapopulation models can also be used to understand the roles of connectivity and group sizes in disease dynamics. Metapopulation models assume that populations are distributed over a number of patches, or areas, which are connected by dispersal (Hanski 1999). This approach has been adapted to diseases where host groups or host individuals represent suitable habitat patches (Hess 1996a). These models can be used to ask questions about the spread of disease between populations and the likely effectiveness of implementing different management strategies, such as quarantine in some subpopulations and not others.

Early work using metapopulation models showed that host movement may facilitate recolonisation of unoccupied habitat (Hanski 1999). However, host movement may also facilitate parasite invasion (Hess 1996a). Metapopulation models show that the probability of a disease pandemic (i.e. parasite spread among many groups) may not be a simple function of host or parasite characteristics, but a more complex interaction between the two (Cross, Lloyd-Smith, Johnson and Getz 2005, Cross, Johnson, Lloyd-Smith and Getz 2007). Consequently, acute diseases may require more frequent host

movement compared to chronic diseases, in order to create a pandemic. Assuming that all individuals in a group become infected, then the movement rate, recovery rate and group size determine the expected number of infectious dispersers, which must be greater than one for a pandemic to occur (Cross, Lloyd-Smith, Johnson and Getz 2005, Cross, Johnson, Lloyd-Smith and Getz 2007)

Despite the flexibility of network models to accurately represent complex host social structures, their utility in investigations of wildlife disease systems is currently limited. A particular problem is that it is not clear how to scale-up the network from a sub-sample of the population such that it represents the entire population of interest. Rare linkages among groups that allow a parasite to move from one group to another may be absent from the sampled population. Raccoons hitchhiking on refuse trucks is one example of potentially rare but important long-distance movements that may have a significant impact on disease spread (Real et al. 2005). Theoretical work has shown that just a few such connections can radically alter the structure of a network and may be critical to understanding disease dynamics (Watts 1999). It is however empirically challenging to document these potentially rare but influential connections at spatial and temporal scales that are relevant to many management problems (but see Text Box).

### ***2.4.2 Estimating host social structure***

Understanding disease transmission in most wild populations is difficult because it usually involves three steps: exit from the host, passage across an external environment to a new host and infection of the new host (Wobeser 2006). Determining when and where these events occur in cryptic wild animals and with parasites that are difficult to detect can be demanding. As a result, understanding the additional complexity of host

social structure on wildlife disease dynamics has proved extremely challenging. Logistic and financial limitations in research studies often require a trade-off between the collection of detailed data on local movements, contact rates and host infection rates on a limited spatial and temporal scale, and coarse data on dispersal and migration at a broader scale.

Where the target species is conspicuous and diurnally active (e.g. meerkats), direct observation may be possible. However, this is seldom the case, and more often researchers have relied on traditional ecological methods such as mark-recapture and radio-tracking to provide information on movements and population structure. Live trapping has been widely employed to generate demographic data using capture-mark-recapture models (see Thompson, White and Gowan 1998), although this can be labour intensive. For some species (e.g. small mammals, badgers) live trapping can provide useful information on individual movements with relatively large sample sizes (see Vicente *et al.*, 2007), albeit at a lower resolution to that obtained from radio-tracking. Mark-recapture studies that incorporate disease infection status of captured animals can also be used to estimate transmission rates and evaluate the impact of parasites on host demographics (Lachish, Jones and McCallum 2007).

Researchers have traditionally monitored wildlife movements using very high frequency (VHF) radiotelemetry or more recently global positioning systems (GPS). VHF transmitters are cheaper but more labour-intensive and may result in data that is spatially coarse and temporally sparse. GPS collars, on the other hand, yield very fine-resolution spatio-temporal data but the costs can be prohibitive. Proximity collars or dataloggers that record when tagged individuals are within a certain range present new

opportunities to investigate how wildlife associate and contact one another (Ji, White and Clout 2005).

Another approach to monitoring mammal movements is to use bait laced with a persistent physical or chemical tag to mark the excretory products. This approach has been usefully employed in small mammals (e.g. Randolph 1973) and is commonly used to delineate the social group territories of the Eurasian badger (Delahay et al. 2000a). This bait-marking technique has potential applications for monitoring the spatial organisation of other mammals, particularly where the faeces are used to mark territories.

Whatever method is used, a sufficient number of individuals are required to maximise the chances of recording relatively rare long-range dispersal events and transient short-distance movements. Once the data are collected, a further significant challenge remains in terms of interpreting how contact data relate to transmission risks.

## **Figure 4**

In many cases, unambiguous determination of group membership may be difficult, particularly as levels of inter-group movement increase. In this case, cluster analysis can provide an alternative approach to describing host structure (Cross et al. 2004).

Association indices based on the proportion of time or observations where pairs of individuals are together can be used to construct an association matrix where each row and column represents an individual. Cluster analyses can then be used to objectively group individuals (Figure 4) according to their levels of association. Otherwise, the association matrix can be used directly to create a network model. The time interval used to construct the association values is critical to the resulting structure. Investigations of

disease dynamics should use time intervals similar to the infectious period of the parasite (Cross, Lloyd-Smith, Johnson and Getz 2005).

The application of population genetics provides an alternative means of estimating the potential connectivity between subpopulations across a larger spatial and temporal scale (e.g. Epps et al. 2005). These patterns of host gene flow may reflect historical movement patterns or translocation rather than contemporary movement and will therefore be expected to be of limited use in investigating disease dynamics. Biek et al. (2006) used feline immunodeficiency virus (FIV) to investigate the genetic structure of mountain lion (*Puma concolor*) populations across the northern Rocky Mountains. Because RNA viruses evolve rapidly compared to the host their phylogeography can reveal more recent host demographic and movement patterns. Thus, FIV was used as a marker for the host. In Wisconsin, gene flow in white-tailed deer populations has been used to evaluate potential barriers to deer dispersal and gene flow has been correlated with the spatial spread of CWD from a focus of infection (Blanchong et al. In press). As it may be possible to collect genetic samples (either from trapped animals, carcasses or faeces) over a wider area than it is usually possible to radio-track known individuals, this approach may allow researchers to investigate host connectivity and predict direction of disease spread on a much larger scale.

## **2.5 Conclusion**

The integration of wildlife ecology, behaviour and disease dynamics is a relatively new area of research. As a result, although this chapter presents many patterns, the observations often apply to only a limited number of situations and there are few general

principles that relate to a wide range of hosts or parasites. In many cases, it is the interaction of host and parasite life-histories that will drive disease dynamics and hence determine management options. In this chapter, we have highlighted several factors that are likely to be important with respect to host behaviour and social organisation (e.g. sex, age, group structure, and dispersal). However, their importance will depend on the parasite in question, and whether it has an intermediate host or is directly-transmitted, and whether the host recovers from infection. For example, small group sizes may help to exclude some directly-transmitted diseases like measles (Bjornstad, Finkenstadt and Grenfell 2002), but have little affect on the persistence of a parasite that has a mosquito vector and multiple alternative hosts. Thus, the management of wildlife diseases must consider not only the particular parasite and host, but also their interaction.

## **Text Boxes**

### ***Chronic Wasting Disease (CWD) in deer***

CWD belongs to a family of diseases known as transmissible spongiform encephalopathies (TSEs) which affect a wide range of mammals including humans (Williams *et al.*, 2002). The causative agent of TSEs is most likely an abnormal prion protein that is consistently associated with the disease (Prusiner 1991). CWD is the only TSE which affects free-ranging cervids (Miller *et al.* 2000). The origins of the disease are unknown, but in North America it was first recognised in the 1960s in captive cervids, and since 1981 in free-ranging deer. Clinical signs of illness develop about 1.5 years after infection, and no captive or wild cervid has subsequently recovered (Williams *et al.* 2002).

Studies of CWD in captive animals indicate that direct contact (Miller and Williams 2003), and contact with prion contamination of the environment (Miller, Williams, Hobbs and Wolfe 2004) are important routes of transmission. However, the relative importance of direct and indirect transmission routes is not well understood. The relative importance of direct and environmental transmission, the role of social groups, and the scale over which CWD transmission occurs are factors that will affect whether CWD behaves like a frequency or density-dependent disease (Gross and Miller 2001, Schaubert and Woolf 2003). For example, female social groups may overlap spatially, but have limited direct contact with other groups (Schauber, Storm and Neilson 2007). Thus, indirect transmission of CWD may be an important route of between group infections, but direct contact and indirect transmission may be important routes within social groups.

Adult male mule deer (*Odocoileus hemionus*) and white-tailed deer tend to have a higher prevalence of CWD than adult females and this increases with age (Figure 5., Grear, Samuel, Langenberg and Keane 2006). Because there are no indications that adult males are more susceptible or harbour the disease for longer, this suggests that differences in social structure and behaviour of males and females may influence disease transmission. Several hypotheses have been suggested to explain the increased risk of CWD infection in males compared to females. First, males are typically more social than females, especially outside the breeding season when they form single sex groups, in which unrelated males readily groom each other. In contrast, female grooming usually takes place between mother-daughter pairs or among individuals from the same matrilineal group. Second, transmission to susceptible males may increase during the breeding season when they either contact infected females or visit scent stations used by

infected males. These behaviours may expose breeding males to prions, which are shed through the alimentary tract. In addition, males may be at greater risk of contact with prions in the environment than females owing to their larger home range size and breeding season movements.

## **Fig. 5**

Differences in movement and dispersal between male and female white-tailed deer may also be a significant component of CWD distribution across the landscape, especially in areas where animals do not show seasonal migration. Between 50 to 80% of yearling males disperse distances of 10 to 30 km, depending on habitat characteristics (Long et al. 2005), whereas less than 20% of females disperse (e.g. Rosenberry, Lancia and Conner 1999). Infected yearling males are therefore more likely to move CWD to new areas. Prevalence of CWD in yearling males and females is similar and considerably lower than in adult males. If environmental transmission is an important route of infection in free-ranging deer, then adult males have the potential to contaminate a wider area than females because of their larger home ranges and increased movements during breeding.

To a limited extent, movement and dispersal information have been used to establish CWD surveillance zones and assess local disease prevalence. In addition, movement distances and estimated scales for disease transmission have been used to identify areas for intensive culling or disease detection around new CWD positive deer or in areas of high infection risk (e.g., infected game farms). However, more extensive evaluation of culling strategies to reduce numbers of adult males (which have higher rates of infection) or yearling males (which have higher rates of dispersal) may deserve further

consideration. Whether strategies that focus on these higher risk components of the deer population could reduce transmission or spread of CWD is currently unknown, as is the geographical scale over which control should be implemented. In many cases, implementation of such male--biased strategies to control CWD will conflict with goals for trophy deer management and make public support for this approach challenging. Because of the long-term chronic nature and slow transmission of CWD in deer, epizootics are likely to last for decades making control a long-term problem, and emphasising the need for prevention or early detection and eradication.

### ***Bovine tuberculosis and the social structure of badger populations***

The Eurasian badger (*Meles meles*) is implicated in the transmission of bTB to cattle in the UK and Ireland. However, the extent to which badgers contribute to the persistent reservoir of infection in cattle herds is still disputed. Badgers are social animals, and in the UK they live in groups of typically three to ten individuals (Neal and Cheeseman 1996), although this varies widely with population density. Each social group defends a territory, within which will be several burrow systems (setts), one of which is likely to be their principal residence (the main sett). In medium to high density badger populations social group territories may be largely contiguous, and boundaries are characterised by latrine sites where faeces and other scent marks are deposited. This structured system of social organisation determines patterns of movement, contact rates and hence the distribution of infection in the population.

The dynamics of bTB infection in badgers has been the subject of a long-term study at Woodchester Park, Gloucestershire in southwest England. In this 11km<sup>2</sup> study

area of lowland pastoral farmland and mixed woodland, the resident badgers have been regularly captured, marked, examined and released. In addition, bait-marking (see main text) was carried out each year to determine the territorial configuration of the resident social groups. In this high density population, fluctuations in badger numbers were driven largely by changes in social group size, whilst the number of groups and their territorial configuration has remained relatively stable (Cheeseman, Wilesmith, Ryan and Mallinson 1987, Rogers, Cheeseman, Mallinson and Clifton-Hadley 1997). Initially during the study, the badger population increased in size, but this was followed by a leveling-off. These changes in host density did not however correlate with the incidence of infection detected in the population (Rogers et al. 1999). Furthermore, as group size was also not related to the incidence of infection (Delahay et al. 2000b) it appeared that host density did not drive TB dynamics either at the scale of the population or the social group.

## **Fig. 6**

The territorial behaviour of badger social groups inhibits the free movement of individuals, encouraging them to remain within clearly defined ranges and limiting levels of inter-group contact. Such a highly structured system of social organisation is likely to have a profound influence on the dynamics of disease distribution. As a consequence, in the Woodchester Park badger population, infection remained spatially restricted for many years, with only limited spread beyond a cluster of persistently affected groups (Figure 6, Delahay et al. 2000b). Nevertheless, there was some movement of individuals between social groups (Rogers et al. 1998), probably largely stimulated by the pursuit of breeding opportunities (Carpenter et al. 2005). Interestingly, these movements (detected from

trapping records) were highly correlated with the incidence of infection, such that years with high rates of movement between social groups were followed by years with an increase in the number of new cases of disease detected (Rogers et al. 1998). Although the presence of other infected individuals in a group was the most important predictor of further infections, this relationship became less important as the level of immigration and/or emigration in a group increased (Vicente, Delahay, Walker and Cheeseman 2007). Hence even individuals in groups that were diminishing in size experienced an enhanced risk of infection. This may be related to the observation that movement of an individual badger may be a protracted affair, during which it may divide its time between two groups for several days or weeks before settling (Roper, Ostler and Conradt 2003). Clearly, this repeated movement back and forth represents a window of enhanced association between two groups that may increase transmission risks.

The limited movement of badgers among social groups in relatively high density populations probably limits disease spread. This is an important consideration for developing strategies for managing bTB in badgers, as some interventions (e.g. culling) may have counter-productive effects if they disrupt this social system and enhance movement rates.

### ***Bovine tuberculosis and social networks in meerkats***

Meerkats (*Suricata suricatta*) are desert-adapted social mongooses living in groups of 3-40 animals in southern Africa. In the South African Kalahari, 300 individually-identifiable wild meerkats living in 14 social groups were habituated to researchers investigating social interactions between individuals and groups in a long-term study (Clutton-Brock, Russell and Sharpe 2004). In the late 1990s, a pathogenic mycobacterial

infection was diagnosed in the study population (Alexander et al. 2002) which was subsequently confirmed to be *M. bovis*, the causal agent of bTB). Infection in meerkats is invariably fatal following the onset of clinical signs, and appears to have been responsible for the extinction of four social groups in the study population between 1995 and 2005.

Investigation of infectious disease transmission in wild animals is often constrained by lack of empirical data on social interactions. As the meerkat study had a long history of behavioural research, it offered a unique opportunity to examine the role of social interaction in the transmission of bTB in a wild mammal. As part of a longitudinal study, meerkats were routinely caught and tested for bTB using serology and culture techniques. Social Network Analysis (SNA) was used to determine the role of host population structure in the transmission of bTB. This approach is used to describe the social position of each individual using both direct and indirect interactions, and so offers a way of determining which individuals within an at-risk population are more likely to be involved in disease transmission (Corner, Pfeiffer and Morris 2003). A set of precise formal definitions for SNA measures has been produced (Wasserman and Faust 1994) and the techniques can be applied to quantify interactions both between and within groups of animals.

Adult male meerkats frequently visit other groups in search of mates before returning to their original group (Young, Carlson and Clutton-Brock 2005) and this behaviour may be important in inter-group transmission of bTB. The temporary intergroup movements of male meerkats and corresponding bTB transmission dynamics can be illustrated using network diagrams (Figure 7). Meerkats are often aggressive towards other members of their resident social group and the outcome of these

interactions determines host population structure. Common causes of aggression include competition to become the dominant male or female, and dominance assertion between any two group members. Subordinate females are often forcibly evicted by the dominant female when she is about to give birth (Stephens et al. 2005). Data from a group of meerkats in the Kalahari study population showed that the incidence of aggressive encounters temporarily increased following the death of a dominant meerkat as others competed for the vacancy (Figure 8). Disease appears to disproportionately affect dominant individuals, although this does not appear to be simply age-related, as younger and similar aged subordinates are also susceptible. Stress-induced immunosuppression of dominant meerkats is one possible explanation. In one group a single female (F43) was involved in the greatest number of aggressive encounters (Figure 7), shown as the highest number of lines connecting her to other group members. If intra-group aggression were responsible for bTB transmission between meerkats, incidence within this group should have fallen in the months following the death of F43. However, subsequently high levels of infection suggested that transmission may have already occurred but was not detected before this female died, or that an interplay of several social interactions (e.g. aggression, grooming, feeding) determined bTB transmission in wild meerkats.

SNA has rarely been applied to the study of wildlife diseases although it has potential to significantly improve current understanding and aid in the development of effective management strategies. The examples shown here illustrate how SNA may be used to elucidate the role of specific behaviours in generating spatial and temporal variation in bTB transmission within and between meerkat social groups. Differences in bTB transmission patterns within meerkat groups are beginning to be quantified by

epidemiological modelling of social behaviour data. These data are being used in the development of a predictive model for quantifying the risk of bTB transmission, which is likely to be useful in informing policy for the management of bTB in other social mammals.

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## Figure Legends

**Figure 1.** Prevalence generally increases with age for many pathogens when individuals are born susceptible and do not recover. Higher transmission rates correspond to higher prevalence (cf. solid line and dashed line). Disease recovery or disease-induced mortality may reduce prevalence in older ages (dot-dashed line).

**Figure 2.** Histograms of group size for ungulates in Kruger National Park, South Africa. Buffalo data originate from annual helicopter surveys from 1985-2003, and other data is from annual fixed-wing surveys from 1980-1993.

**Figure 3.** Mean (circles), weighted mean (squares), and median (triangles) group size as a function of total population size for ungulates in the Kruger National Park, South Africa. Weighted means equal

$\sum_i n_{ijk}^2 / \sum_i n_{ijk}$ , where  $n_{ij}$  is the  $i$ th group size observation of species  $j$  in year  $k$ , and represent the expected

group size experienced by a randomly chosen individual. Buffalo and elephant data come from annual helicopter surveys from 1985-2003, while data for other species come from annual fixed-wing surveys from 1980-1993. Statistically significant correlations ( $p < 0.05$ ) are depicted with lines.

**Figure 4.** Visualization methods for the social structure of African buffalo using data collected over different time periods. The two top figures are dendrograms constructed by cluster analyses using the unpaired group averaging method based on the association matrices shown on the bottom row. Individuals that spent more time in the same herd are joined at lower linkage distances. The association matrices (bottom row) represent the proportion of time each individual spent with all other individuals. Within each association matrix there is a row and column for every individual with a radio-collar, and those pairs that spent most of their time together are shown in black. Individuals' rows and columns are ordered according to the cluster analyses above them.

**Figure 5.** Prevalence of Chronic Wasting Disease (CWD) in male and female white-tailed deer harvested in south central Wisconsin, USA, during 2002-2004 (after Gear *et al.*, 2006).

**Figure 6.** The spatial distribution of bovine TB infection in the Woodchester Park badger population in 1996. Polygons represent social group territories and pie charts are scaled relative to group size, and show the proportion of residents falling into different disease status categories (exposed = seropositive, excretors and superexcretors = infectious). During a 15 year period, infection in the study population remained spatially clustered.

**Figure 7.** Intergroup movements of meerkats and the spread of bovine tuberculosis (bTB) between eight social groups over a two-year period (open circle = uninfected group, grey diamond = group seropositive for bTB (indicating exposure to the pathogen), black diamond = group with clinical bTB). Line thickness is proportional to the number of intergroup movements. For visual clarity, only eight of the fourteen meerkat groups studied are shown. During the study period individuals transferred from negative (disease-free) status to seropositive (indicating exposure to bTB), to clinically positive and ultimately death. Harsh environmental conditions during 2007 markedly reduced the frequency of intergroup movements and this is reflected in network diagram (d) by fewer lines connecting meerkat groups compared with the beginning of the study.

**Figure 8.** Intragroup aggression networks and transmission of bTB within a meerkat group (M = male, F = female, open circles = uninfected individuals, grey diamonds = individuals seropositive for bTB (indicating exposure to the disease), black diamonds = individuals with clinical bTB, dashed diamonds = deceased individuals). Line thickness is proportional to the number of aggressive interactions between individuals. Male 80 (M80) immigrated from another group in August 2006.

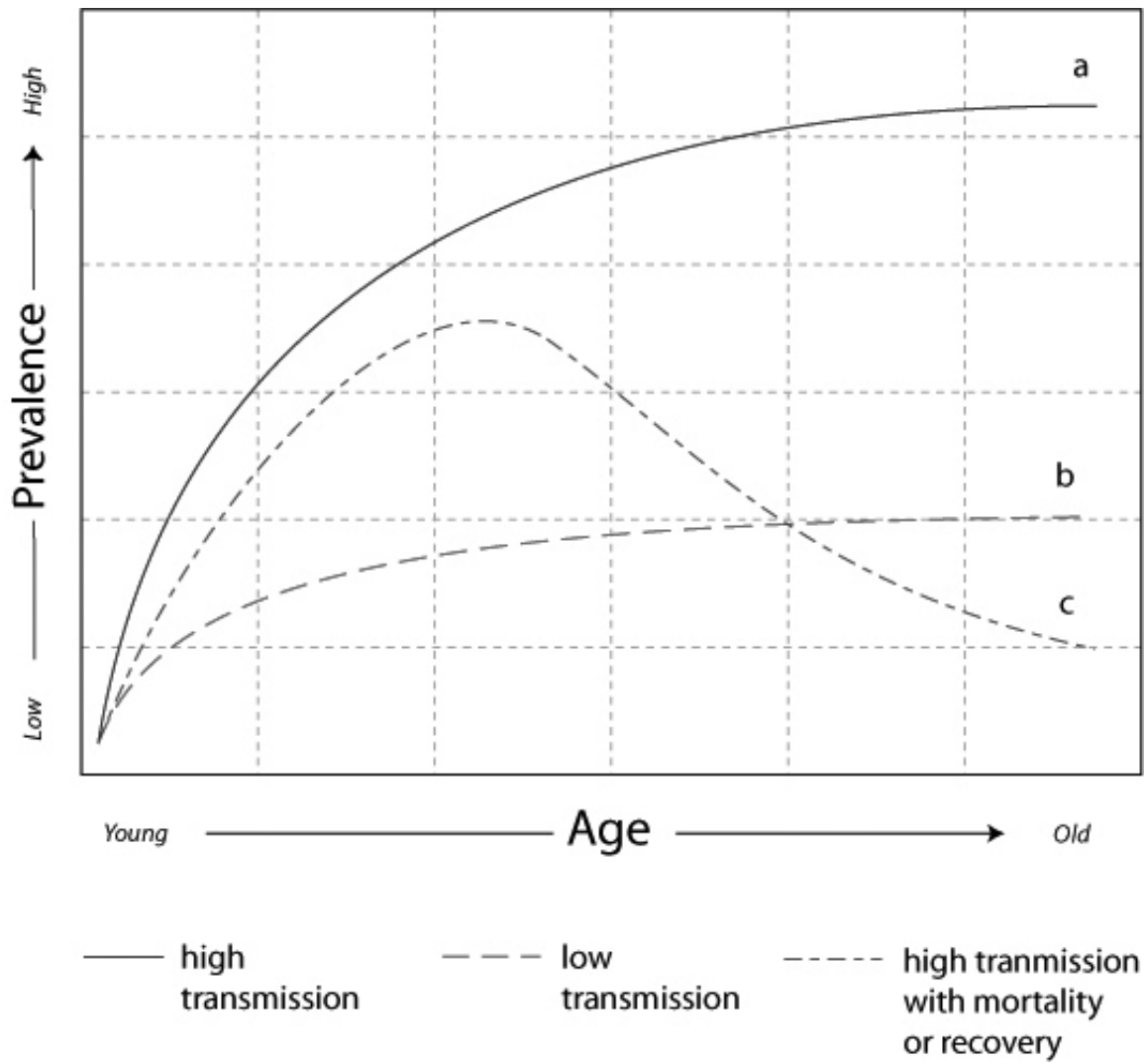


Figure 1.

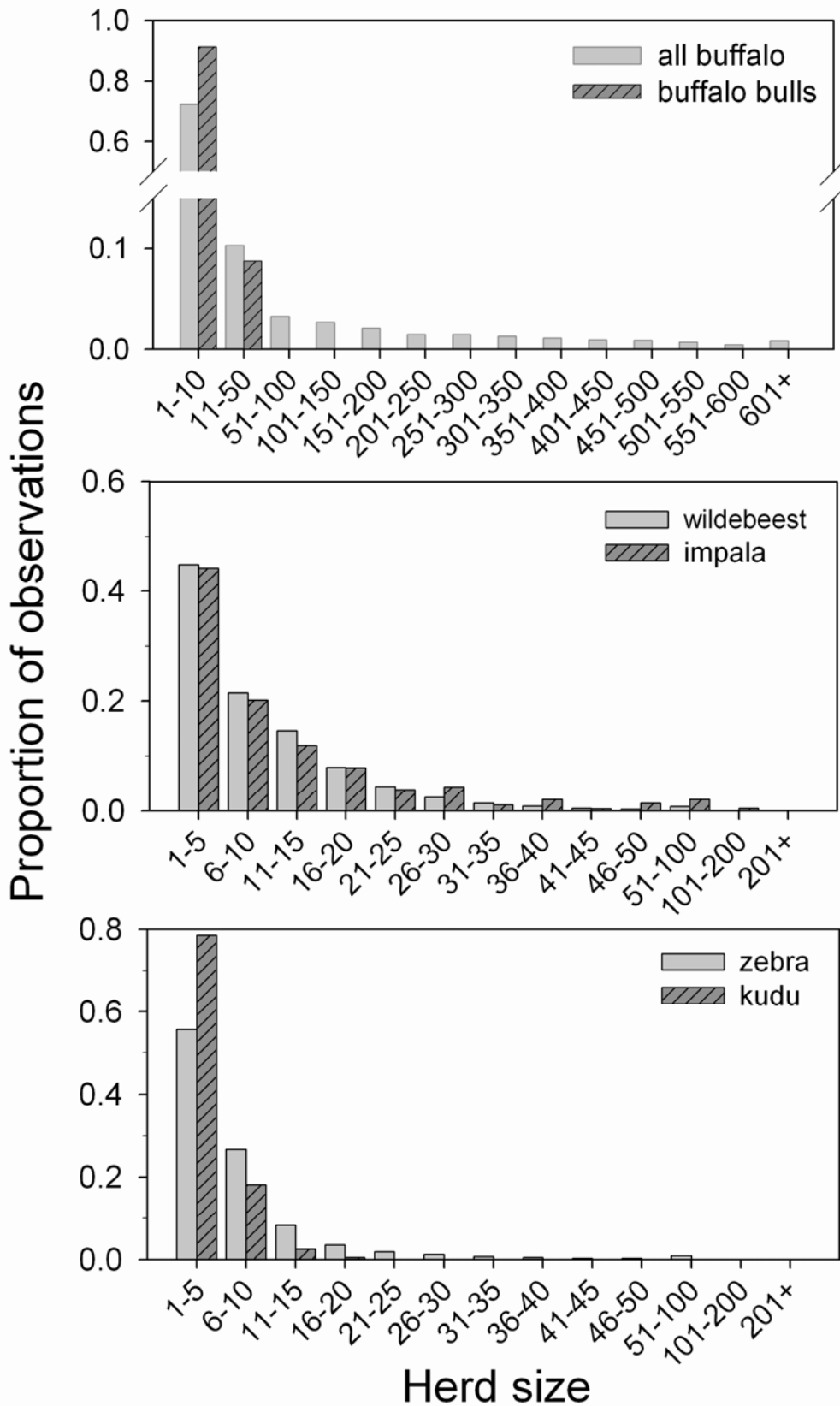


Fig. 2.

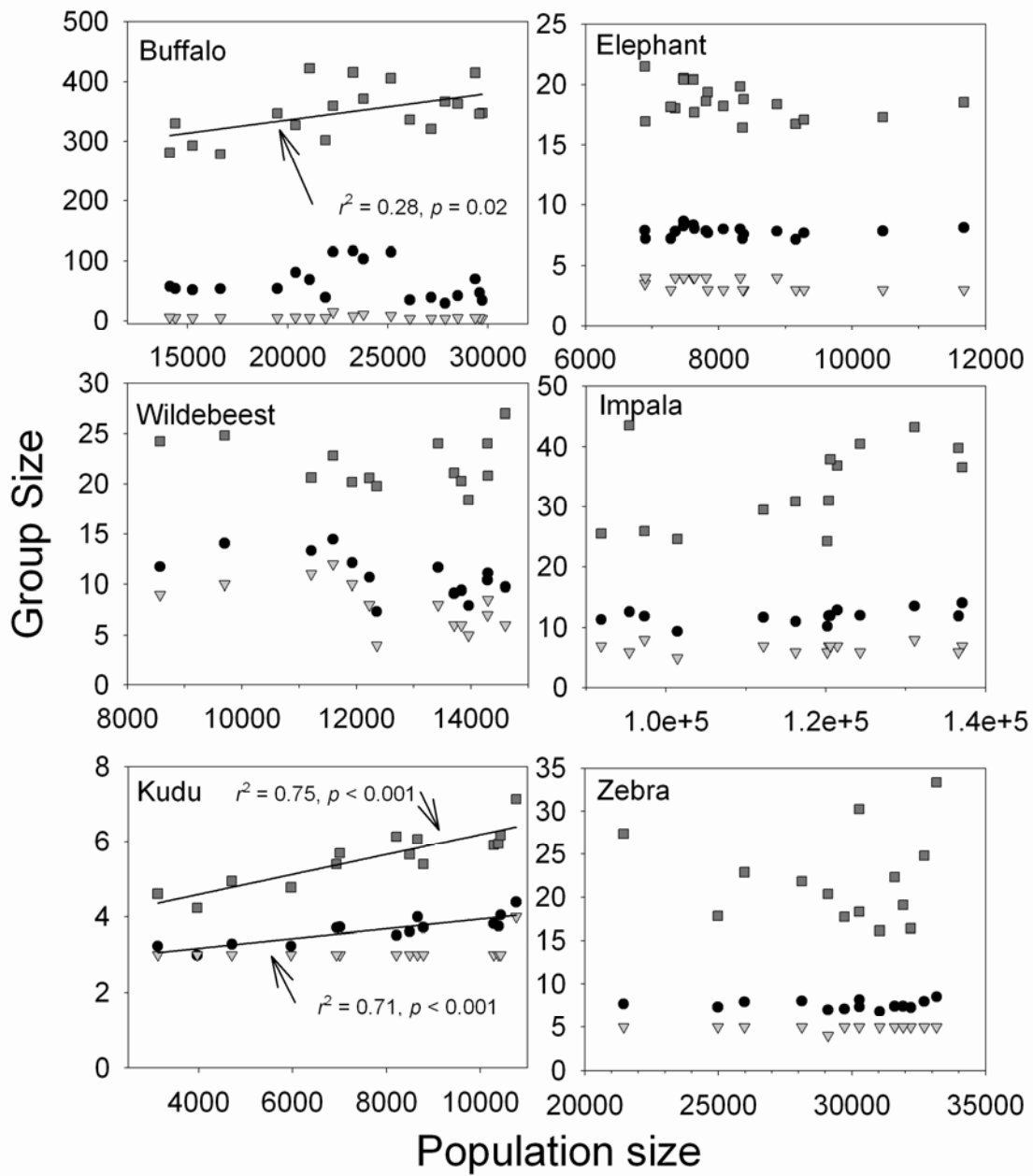


Fig. 3

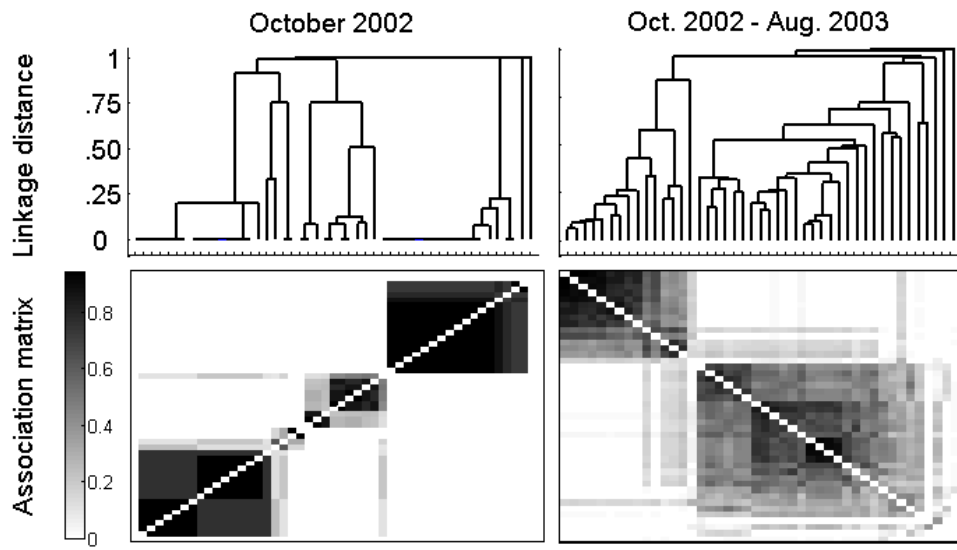


Fig. 4



Fig. 5.

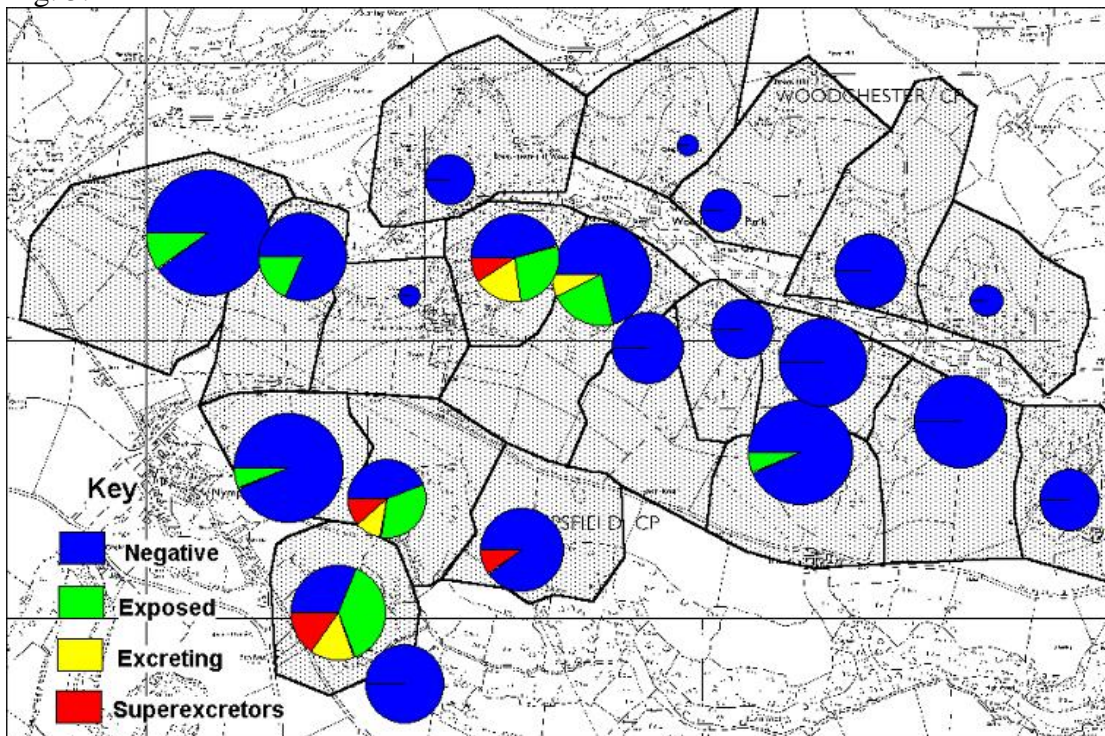


Fig. 6

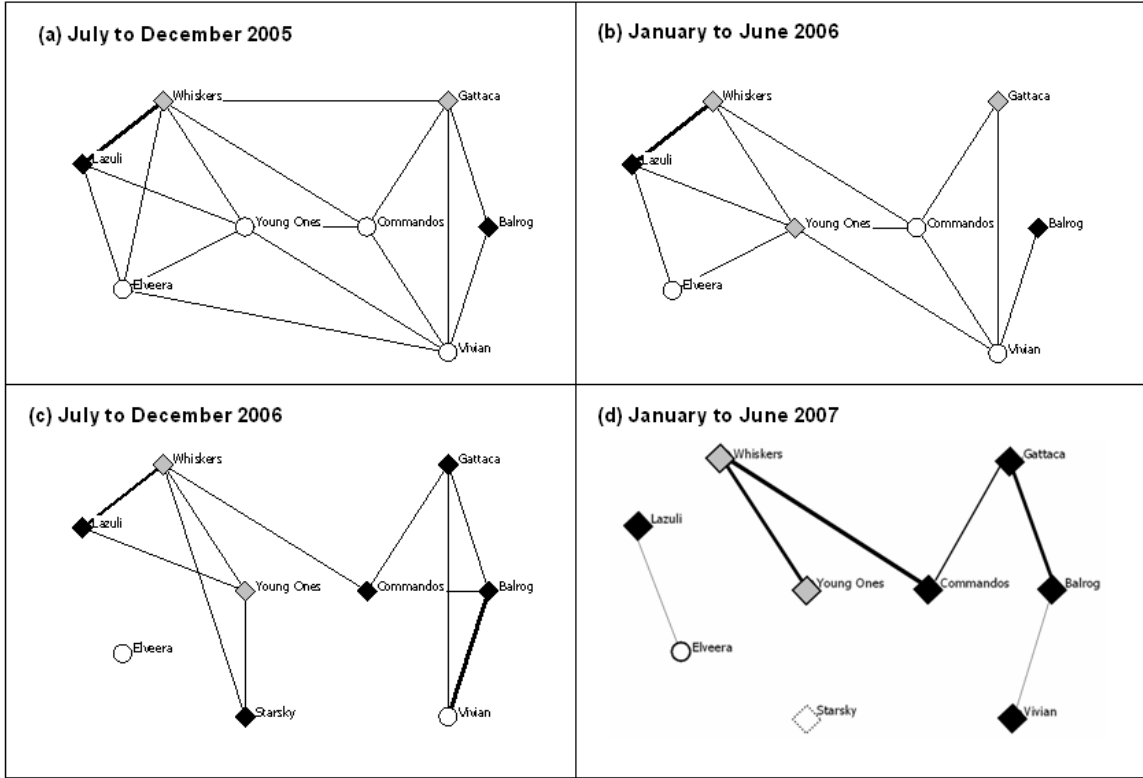


Fig. 7.

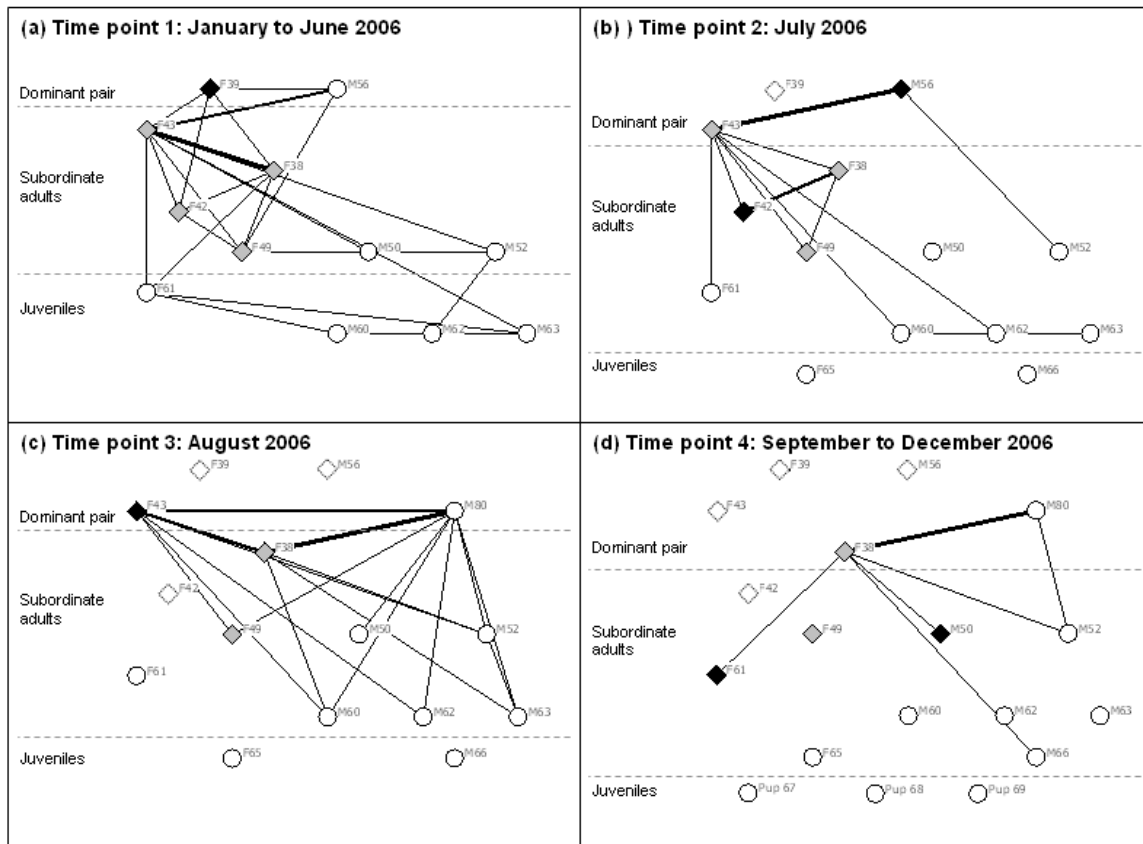


Fig. 8.