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How behavioural ageing affects infectious disease

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ABSTRACT

Ageing is associated with profound changes in behaviour that could influence exposure and susceptibility to infectious disease. As well as determining emergent patterns of infection across individuals of different ages, behavioural ageing could interact with, confound, or counteract age-related changes in other traits. Here, we examine how behavioural ageing can manifest and influence patterns of infection in wild animals. We discuss a range of age-related changes that involve interactions between behaviour and components of exposure and susceptibility to infection, including social ageing and immunosenescence, acquisition of novel parasites and pathogens with age, changes in spatial behaviours, and age-related hygiene and sickness behaviours. Overall, most behavioural changes are expected to result in a reduced exposure rate, but there is relatively little evidence for this phenomenon, emerging largely from a rarity of explicit tests of exposure changes over the lifespan. This review offers a framework for understanding how ageing, behaviour, immunity, and infection interact, providing a series of hypotheses and testable predictions to improve our understanding of health in ageing societies.

1. Introduction

Ageing is a near-universal phenomenon in the animal kingdom. As animals age, they generally experience increased mortality rates, reduced fertility, and reduced function in terms of a suite of physiological characteristics (Ricklefs, 2008). This process is known as "senescence". Although initially thought to be uncommon in the wild, in the last few decades it has been demonstrated that many wild animals experience substantial age-related declines in a variety of traits before they die (Elliott et al., 2015; Froy et al., 2015; Nussey et al., 2009, 2006). These include a wide range of physiological traits (Elliott et al., 2015), and with underlying declines in molecular and cellular functioning (i.e., "cellular senescence"), particularly concerning aspects of immunity (i.e., "immunosenescence" (Nikolich-Žugich, 2018; Pawelec, 2018; Peters et al., 2019). Because one of the immune system's main roles is to defend against infection, such age-related changes in immunity may have important ramifications for infection status. However, despite these well-appreciated impacts of age on susceptibility to infection, we have a much shallower appreciation of age-related changes in behaviour, their effects on exposure to pathogens, and their joint implications for infection.

Alongside immunity, an individual's behaviour is a central determinant of its infection status (Albery et al., 2021a; Bansal et al., 2007; Ezenwa et al., 2022; Sweeny and Albery, 2022; VanderWaal and Ezenwa, 2016). Because individuals that make more contacts are generally more often exposed to parasites, greater sociality is often expected to correspond to higher parasite count or increased infection probability (Altizer et al., 2003; Cote and Poulin, 1995). Increasingly, there has been a developing interest in age-related changes in behaviour, and in particular in the altered social environments of senescent individuals (Albery et al., 2022a; Rosati et al., 2020; Siracusa et al., 2022a). This "social ageing" is likely to have implications for older individuals' parasite counts by altering exposure patterns (Siracusa et al., 2022a). Reciprocally, many animals have evolved behavioural mechanisms to minimise infection risk (Ezenwa et al., 2016a; Kappeler et al., 2015; Loehle, 1995) – for example, by avoiding infection (Curtis, 2014; Stockmaier et al., 2021), by grooming or caring for infected individuals (Akinyi et al., 2013; Stewart and Macdonald, 2003), or by acquiring resources at greater rates, allowing a more effective immune response (Almberg et al., 2015a). Changes in such behaviours with age could have knock-on effects on infection status over the lifespan. Further, because exposure and susceptibility often interact and counteract one another,

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both on ecological and evolutionary timescales (Hawley et al., 2021, 2011; Poulin and Filion, 2021; Sweeny and Albery, 2022), age-related changes that alter either exposure or susceptibility can be expected to have downstream or compensatory changes that affect the other. For example, if age-related changes in immunity reduce resistance and therefore increase parasite count, behaviour may change to reduce exposure as a result to compensate (Hawley et al., 2021). Links between behaviour and susceptibility across an animal's lifespan could therefore be vital in determining disease dynamics in many systems. Understanding these processes is important for many reasons, not least because the age structuring of infection has important implications for emergent disease dynamics (Clark et al., 2017).

Here, we discuss how behaviour changes with age ("behavioural ageing"), and how these changes could alter infection status, with a focus on wild animals and with varying levels of mechanistic complexity. Given that this is a relatively nascent field with an early evidence base to draw from, for each concept we make sure to outline how empirical evidence could be attained through observation, experiments, and simulations. Ultimately, we weave together what is known or hypothesised about age-related changes in behaviour in wild animals with other known aspects of ageing, providing a synthetic framework for understanding the age structuring of infection (Fig. 1).

2. Age-related changes in behaviour and their effects on infection

2.1. Social ageing drives changes in exposure

Recent years have seen a rapidly growing interest in the ageing of social behaviour specifically ("social ageing"; (Siracusa et al., 2022a)). Generally, individuals alter their social behaviour for several reasons: through direct effects of senescence, changes to compensate for the direct effects of senescence, social benefits of ageing, or demographic changes (Siracusa et al., 2022a). Because social behaviour is an important driver of exposure risk, these changes are expected to influence infection status (Albery et al., 2022a, 2022b; Siracusa et al., 2022a).

Older individuals most often exhibit reduced sociality – for example, in Barbary macaques (*Macaca sylvanus*) (Rathke and Fischer, 2021), red deer (Albery et al., 2022a), and ibex (*Capra ibex*) (Brambilla et al., 2022). This process could convey reduced exposure risk as individuals age, resulting in a lower burden of infection in older individuals. In contrast, some species become *more* social as they age: for example, bonobos (*Pan paniscus*) exhibit increased grooming rates (Franz, 1999; Tokuyama and Furuichi, 2017), which could drive exposure to endoparasites (Poirotte et al., 2017). In scenarios where individuals increase interaction rates in this way as they age, they may greater exposure and infection rates. There is also some evidence of mid-life peaks in sociality, as in Alpine ibex (Brambilla et al., 2022); these are likewise expected to produce complex, non-linear changes in exposure rates with age. Only

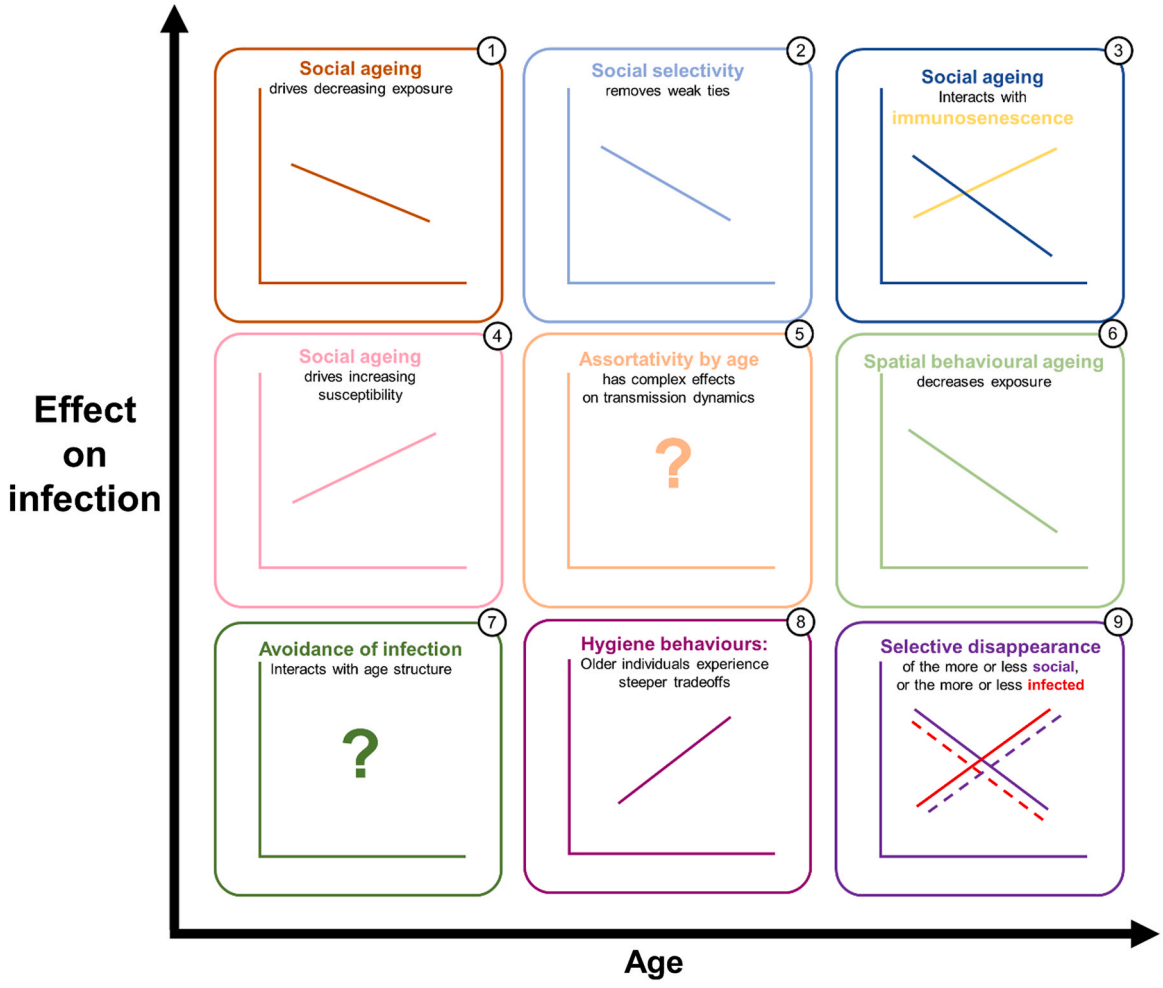

Fig. 1. Schematic presenting our framework for understanding how age-related changes in behaviour influence infection. In all panels, age is on the x axis and the proposed mechanism's generalisable effect on infection prevalence or intensity is on the y axis.

one system we know of has yet demonstrated age-dependent changes in behaviour that could influence emergent infection status: in female red deer, age-dependent decreases in social connectedness (Albery et al., 2022a) occur in opposition to age-related increases in strongyle helminth infection (Albery et al., 2022b). Nevertheless, the two phenomena have not yet been mechanistically linked, and there are no other examples in which social ageing and their emergent effects on infection have been investigated. As such, this remains a priority area to provide more case studies and examples of social ageing effects in disease ecology.

Given the possibility of multidimensional changes in different behaviours, an underexplored aspect of behavioural ageing concerns divergent changes in different contact types: for example, if older individuals experience fewer aggressive interactions (Rosati et al., 2020) but greater grooming rates (González et al., 2021), it could drive a change in parasite community from those spread through fights (e.g. tuberculosis (Drewe, 2010)) to gastrointestinally transmitted ones (e.g. some protozoa (Poirotte et al., 2017)). Given a relative scarcity of multi-pathogen studies spread over the length of the lifespan, there is as yet unsurprisingly little evidence for these shifting communities, even before considering the involvement of behaviour.

2.2. Social selectivity and the removal of weak ties

In many cases, social ageing does not manifest as a simple increase or decrease in sociality, even when considering only one contact type. Often, older individuals have fewer, but stronger, social connections, and with a preference for positive rather than negative interactions – i.e., they exhibit increased "social selectivity" (Rosati et al., 2020; Siracusa et al., 2022c, 2022a). This trend is relatively common in humans, and in some primate societies (González et al., 2021; Machanda and Rosati, 2020; Rosati et al., 2020; Siracusa et al., 2022b). Depending on the specific patterns of behavioural change, social selectivity may decrease older individuals' overall risk due to lower connectedness, but the removal of weak social connections could have further complex effects on infection dynamics (Sah et al., 2017; Vanderwaal et al., 2016). For example, in giraffes (*Giraffa camelopardalis*), weak ties – represented by an individual's tendency to connect disparate cliques of other individuals – are stronger determinants of that individual's infection with helminths than overall connectedness (Vanderwaal et al., 2016). Losing these ties with age could therefore have a disproportionately strong effect on reducing exposure risk (and therefore infection), allowing selective older individuals to optimise the ratio of exposure to social benefits that they experience.

Social selectivity should specifically result in a lower rate of *novel* exposure events. Because meeting new individuals (especially those from new social groups (Sah et al., 2017)) is more likely to result in exposure to new parasites than meeting well-known individuals, increasing social selectivity with age could function to minimise novel pathogen exposure. In social systems where the risk of novel pathogen exposure is high when connecting remote groups (as in giraffes (Vanderwaal et al., 2016)), it is likely that social selectivity will be more strongly favoured with age. Nevertheless, although these patterns of overall social selectivity have been identified broadly, it has yet to be shown how the rate of entirely novel encounters changes over age. To address both questions might require researchers looking at long-term ageing systems to identify how individuals' acquisition of novel partners – or reacquisition of very old ones – corresponds to acquisition of novel parasites. Alternatively, simulations could be used to identify how the costs of repeated exposure to well-known pathogens compares to occasional high-risk exposure to unknown pathogens when selecting for social selectivity behaviour.

Focusing on few strong relationships as one ages could have further benefits beyond reducing exposure rates. Notably, an important component of an individual's behaviour is the ability to recognise and respond to infection in others (Gibson and Amoroso, 2022; Lopes, 2022; Weinstein et al., 2018). Having relatively few (but stronger) relationships may allow older individuals to more effectively anticipate infection risk or recognise infections in the individuals they know well, allowing them to behave appropriately – e.g. by avoiding them. Social selectivity could therefore be an adaptive response to the declining perceptive ability of senescent individuals (Siracusa et al., 2022a), given that some sensory acuity is required to accurately identify infection risks. More widely assessing how older individuals differ in the strength of their hygiene behaviours (see below) and using specific age-related metrics of sensory ability like macular degeneration (Fernandes et al., 2023), particularly in the context of multiple social environments, will help to test this possible role of social selectivity.

2.3. Interactions between social ageing and immunosenescence

Rather than manifesting in isolation, behavioural ageing could interact with immunosenescence: by reducing exposure to parasites, reducing one's contact rates over the lifespan could compensate for weaker resistance and thereby allow older individuals to circumvent a greater parasite load (Albery et al., 2022a; Siracusa et al., 2022a). Behavioural compensation for a weak immune response such as this is relatively common (Hawley et al., 2021, 2011): for example, guppies (*Poecilia reticulata*) show stronger conspecific avoidance when they are more susceptible to infection (Stephenson, 2019). Nevertheless, empirical evidence directly linking immunosenescence and social ageing remains scarce, and there is a need for more empirical tests in longitudinal study populations. Specifically, these studies would have to examine covariance between social behaviour and immunity across individuals' lifespans, and potentially then link them to infection, to show that metrics taken to represent susceptibility and exposure are negatively associated (Albery et al., 2021a; Sweeny and Albery, 2022). There is substantial evidence for immunosenescence in wild animals: for example, ageing Soay sheep experience declines in immune function that predict decreased survival probability independently of changes in parasite burden (Froy et al., 2019), and a recent meta-analysis uncovered a number of studies showing similar trends (Peters et al., 2019). However, such studies are rarely carried out longitudinally and in a fashion that would allow extrication of a role of behaviour. Given that many long-term studies are well-placed to identify changes in both ageing and behaviour (Clutton-Brock and Sheldon, 2010; Sheldon et al., 2022), the time is ripe that concurrent changes in these processes be linked with their changes in immunity and infection, allowing us to investigate this possibility. In a cross-system context, if these processes are indeed linked, we expect that systems in which the immune system senesces more strongly will also exhibit stronger patterns of social ageing, allowing older individuals to ameliorate this cost of senescence.

In practice, these processes may be difficult to extricate (or at least confirm) observationally: if immunosenescence and social ageing are linked, they could obfuscate ageing patterns in the wild by weakening the relationship between age and infection status within individuals, even where immunosenescence is in fact occurring. That is, where age modifies both exposure and susceptibility in opposite patterns, the emergent trend might be no change in infection prevalence or intensity over the lifespan. As such, a possible route to uncovering this process could involve eco-evolutionary simulations that model individual age trajectories in behaviour and immunity, and then to examine the emergent outcomes for age structuring of infection patterns. These models could identify – and attempt to overcome – the possible problem of a weakening strength of selection with age when evolving adaptive traits in this context (see below).

2.4. Social ageing may drive changes in susceptibility

On the other hand, reduced sociality could *cause* age-related increases in parasite count via effects on immunity and health. An individual's social environment can influence physiological parameters

like stress levels (Hermes et al., 2009; Razzoli et al., 2018; Sapolsky, 2004) and nutritional state (Almberg et al., 2015b), while correlating directly with immune functions like wound healing (Archie et al., 2012) and markers of ageing like telomere length (Lewin et al., 2015). Consequently, sociality has implications for immune function and susceptibility to infection (Albery et al., 2021a; Hawley et al., 2021; Snyder-Mackler et al., 2020; Sweeny and Albery, 2022). Conversely, animals could lose social benefits such as cooperative defence or hunting as they age (Silk, 2007a; Siracusa et al., 2022a). If age-related changes in sociality influence physiological traits downstream, individuals' infection status may change as a result. For example, because older individuals are often less competitive, they may receive more aggressive interactions from their younger, stronger counterparts (Siracusa et al., 2022a). Receiving aggression often causes stress, which may reduce immune resistance and therefore drive greater parasite count in older individuals (Hing et al., 2016; Martin, 2009; Romero, 2004). In some circumstances, infected individuals may receive greater levels of aggression (McFarland et al., 2021), so these processes could form a positive feedback cycle. This example accentuates that the interrelationships between ageing, behaviour, immunity, and infection can be complex, and teasing apart possible driving mechanisms may be difficult – particularly in fully observational systems (i.e., those that do not allow experimental manipulations).

Providing evidence for social ageing-driven increases in susceptibility will require teasing apart the process from endogenous immune changes (i.e. immunosenescence) to demonstrate that age-specific social interactions themselves are causing the reduction in resistance (or similar). Practically, this could involve experiments that manipulate individuals' social environments in an age-dependent manner, allowing researchers to control for other within-individual immune changes. In strictly observational systems, time-structured models coupled with behavioural and immunoparasitological sampling may help researchers to infer mechanistic pathways that link ageing with sociality, and then with changes in immunity and infection. Alternatively, physiological measures of ageing such as telomere lengths could be combined with calendar age to control for "biological age" when analysing the data (e.g. (Watowich et al., 2022)).

2.5. Age assortativity and modularity in socio-spatial networks

In some circumstances, age-specific behaviours may drive assortative age patterns – that is, where older individuals tend to associate with older individuals (and younger with younger). For example, great tits (*Parus major*) tend to more often socialise with individuals of a similar age than expected by chance (Farine et al., 2015). Such structuring could come about through a variety of mechanisms: through social behaviours such as age preference or the competitive exclusion of older individuals, spatial processes like habitat selection or landscape structure, or demographic processes like selective disappearance of individuals in certain areas.

Age assortativity could have a variety of effects on disease dynamics by clustering together older individuals in the contact network. When accompanied by age-dependent declines in immune resistance, these clusters could provide fertile ground for disease outbreaks because an invading pathogen will be highly likely to find susceptible hosts in contact with its focal host. Reciprocally, if older individuals are generally resistant to a given pathogen by the time they reach old age – e.g. as with many childhood pathogens such as measles in humans – these areas of the contact network will be difficult to invade. Similar differences could emerge with age-dependent mortality: for example, when a pathogen reproduces well in young individuals but only causes high mortality in older individuals, highly mixed age structures could lead to a disproportionate death toll in ageing individuals. Reciprocally, when older individuals are able to transmit well but younger individuals are not, social structures that involve high levels of age assortativity could produce higher mortality. These two variables (age skew in transmission and age skew in mortality) therefore produce an interesting landscape of possible source-sink-like outcomes in disease dynamics.

These source-sink dynamics may prove particularly important in the case of novel pathogens. Most saliently, age-dependent social structuring was a crucial mediator of the impact of the SARS-CoV-2 pandemic on older people: due to the virus's strong age skew in pathogenicity, older individuals were hit particularly hard, with high morbidity and mortality both in tight-knit age-structured communities like assisted living facilities (Davidson and Szanton, 2020; Hashan et al., 2021; Thompson et al., 2020) and in communities in which elder individuals often live with younger family members in single households (D'Onofrio et al., 2021; Esteve et al., 2020; Giorgi and Boertien, 2021), effectively representing opposite ends of a spectrum in terms of age structuring and assortativity. Understanding how different age structures conspire to determine the burden of infection in the elderly is an important future research direction, both in humans and in animals.

These sorts of dynamics could also come about where risky behaviours for pathogen transmission are themselves assortatively arranged by age: for example, older humans often have low rates of condom usage, which predisposes clusters of older individuals to outbreaks of sexually transmitted infections (Macdonald et al., 2016); this trend has an analogy in wild animals, where older individuals are often less picky in terms of their reproductive behaviour (Han and Dingemanse, 2023). Although age-dependent mortality and age-structured outbreaks are common, the behavioural mechanisms underlying these age structuring processes are relatively opaque in wild animals.

More generally, correlations can emerge between age and infection through a variety of non-random structuring processes, without necessitating behavioural changes through individuals' lifespans. For example, landscape features may result in spatial clustering of individuals of a similar age or life stage (Devan-song et al., 2022), which will introduce spatial autocorrelation between contact network structuring and age. Given that epidemics generally begin at one point and expand in space, even given no age-dependent structuring of susceptibility and mortality, the course of an outbreak can likewise be age-structured (e.g. shifting from mainly infecting young to mainly older individuals) when it occurs in an age-structured population.

2.6. Age-related changes in spatial behaviour

Older individuals often behave differently in space compared to their younger counterparts; for example, albatross, deer, and sheep all alter their foraging behaviour as they age (Albery et al., 2022a; Froy et al., 2018, 2015). These changes can emerge for a range of reasons, linked to e.g. the exploitation of different food sources, escaping competition from younger conspecifics, or reduced movement or navigation capacities. Specifically, older individuals could alter their habitat use, preferred local density, or population structure (Albery et al., 2022a), all of which may have to be accounted for when investigating the drivers of infection. Because contact networks are often spatially structured, these effects could have complex implications for exposure (Albery et al., 2021a): for example, movements to areas of lower population density over the lifespan could reduce contact rates (Albery et al., 2021b; Albery et al., 2022a), with resultant reductions in exposure and parasite prevalence. Older individuals are generally thought to be physiologically weaker, which leads them to have reduced ranging capacities (Froy et al., 2018); because spatial activity and social connection numbers generally correlate positively (Webber et al., 2023), we therefore predict that age-related changes in spatial behaviour will generally function to reduce exposure rates. As weak ties generally connect relatively distant spatial groups (Centola and Macy, 2007) this is particularly likely to be true of the changes in social selectivity outlined above.

Because infection risk is often spatially heterogeneous, even on small scales (Albery et al., 2022), behavioural ageing may be important because it determines *where* the animal lives as well as *how much* it moves. As they age, individuals could move to or from areas of high

infection risk – e.g. those with microclimatic conditions that favour transmission, or areas with host other host species that maintain generalist pathogens. As with changes in social behaviour, multidimensional changes in spatial behaviour could have divergent effects on different parasites: for example, where older individuals reduce their forage intake they may ingest fewer infective stages of gastrointestinal parasites, or where they move to less-preferred areas they could experience exacerbated exposure to biting insects and (therefore) vector-borne parasites. Alternatively, where a given area is particularly favourable to transmission of a parasite with heavy geriatric mortality, the spatial age structuring of the population may be skewed to younger individuals in this area due to local mortality of older individuals. More commonly mapping the spatial distributions of infections and of environmental parasite stages, alongside spatial age structure of hosts through time, may help to identify the role these phenomena play in determining the age distribution of infection.

Finally, spatial behaviour could likewise influence elements of susceptibility via resource use: older individuals could be less competitive, and thereby forced to inhabit areas with poorer-quality nutrition, which could reduce their immune resistance (Becker et al., 2018; Calder and Jackson, 2000). As with social ageing, the balance of these two components (exposure and susceptibility) will determine the observed age patterns of infection across the population.

2.7. Age structuring and the avoidance of infection in space

Populations often arrange themselves in space according to the distribution of parasite exposure risk in the same way they arrange themselves according to predator threat, forming "landscapes of disgust" (Buck et al., 2018; Hutchings et al., 2006; Weinstein et al., 2018). All else being equal, individuals will avoid areas that they can identify as being high-risk for pathogen exposure. This "push" factor is weighed against other "pull" factors like graze availability in determining each individual's space use, which magnifies at the population level to produce a population distribution that is negatively correlated with the spatial distribution of infection risk (Albery et al., 2020; Hutchings et al., 2006). In systems where older individuals are less competitive, they may suffer from a reduced set of options regarding where and when to forage or spend their time, thereby reducing their ability to avoid areas of high infection. Broadly, this will intensify tradeoffs between avoiding infection risk and other priorities such as resource acquisition or predator avoidance (Hutchings et al., 2006). This latter tradeoff could be especially potent because older individuals are also often more vulnerable to predators; in fact, predators often specifically target old, weak or sickly individuals, which creates substantial positive correlation among these components. Alternatively, in systems where older individuals are generally dominant (as in some primate systems (Machanda and Rosati, 2020)), older individuals may have access to preferred, disease-free areas, such that the landscape of disease provides a balancing force selecting against younger individuals and facilitating the survival of older individuals. As yet, despite substantial evidence for parasite avoidance in experimental contexts (Gibson and Amoroso, 2022; Poirotte et al., 2017; Stroeymeyt et al., 2018) there remain relatively few putative examples of landscapes of disgust in wild populations (though see (Albery et al., 2020) for a putative example). Further investigating age effects on the intensity of parasite avoidance behaviours (see below), and magnifying it to the landscape level, will prove helpful in examining this possibility – particularly if these processes can be examined and compared in systems with opposing patterns of dominance across the lifespan.

2.8. Age-related hygiene and sickness behaviours

Animals exhibit a range of behavioural responses to disease, which generally involve some combination of avoiding parasites, increasing resistance to infection, or ameliorating the cost of infection (Gibson and Amoroso, 2022; Stockmaier et al., 2023). These responses can occur either in the infected individual (e.g. fungus-infected ants tend to inhabit areas further from the colony and make fewer contacts (Stroeymeyt et al., 2018)), or in their uninfected conspecifics (e.g. mandrills (*Mandrillus sphinx*) avoid grooming infected conspecifics (Poirotte et al., 2017)). "Sickness behaviours" are characterised by increased lethargy and reduced sociality, and may function to reduce onward transmission of parasites and/or to conserve energy for the immune system (Lopes et al., 2021; Shakhar and Shakhar, 2015). It is highly likely that the expression of hygiene and sickness behaviours depends on age – particularly because of their strong link to physiology (Lopes et al., 2021). For example, because older individuals are often physically weaker as well as being less resistant, they may exhibit stronger sickness responses to conserve energy more effectively when sick. Similarly, given weaker immune resistance, older individuals may be motivated to exhibit stronger hygiene behaviours where they can. However, hygienic behaviours like parasite avoidance can often result in important tradeoffs with other priorities, such as resource acquisition or predator avoidance (Hutchings et al., 2006). As such, anti-disease behaviours may clash with the other resource intake needs of older individuals. Importantly, age-related changes in hygiene behaviours is a "second-order" relationship with infection. That is, it represents an interaction effect where age alters the relationship between infection and behaviour, rather than age influencing infection through altering behaviour.

As outlined above, there is some evidence for changes in connectedness over the lifespan (Siracusa et al., 2022a); however, this is distinct from evidence for age-dependent expression of hygiene behaviours themselves. The latter requires demonstrating that there is age-dependent variation in the slope of a temporary behavioural response to perceived or actual infection, rather than a gradual decline in the behaviour over the lifespan. In practical terms, this would represent an interaction effect between age and infection in a statistical model examining behaviour as the response. Although protozoan infection reduces grooming received in mandrills, alongside an effect of age itself (Poirotte et al., 2017), the same study did not show an interaction between age and infection status. Additionally, age was found to have no effect on hygienic personalities in mouse lemurs (Poirotte and Kappeler, 2019). The evidence for age-dependent modification of hygienic or sickness behaviours is therefore scarce, and it remains unclear whether these behaviours should most often be intensified (due to increased susceptibility and greater need to avoid infection) or lessened (due to intensified tradeoffs with other resource demands) with age.

2.9. Selective disappearance of the more or less social, or the more or less infected

Where a given trait drives greater mortality, individuals expressing this trait will be more likely to die at each unit of time, removing the individual from the population. Over time, this "selective disappearance" can drive an observed age-related change in the trait at the population level rather than within individuals; as this trend often removes the weakest individuals, it often masks age-related declines, making senescence harder to detect (Nussey et al., 2008; van de Pol and Verhulst, 2006). In longitudinal systems, selective disappearance and within-individual declines can be extricated and estimated by fitting a combination of individual identity and longevity (known age at death) alongside continuous age (van de Pol and Verhulst, 2006). That is, being able to account for each individual's identity and age at the time of sampling, and mathematically weighting it against the age that they will eventually attain, can statistically differentiate within- and between-individual declines. Recognising and deciphering these processes – and how to unpack them – was an important stage in identifying patterns of ageing and senescence in wild animals, and identifying within-individual declines specifically is a crucial part of many senescence studies (Albery et al., 2022a; Froy et al., 2019, 2018; Siracusa et al., 2022b).

Selective disappearance is particularly important where it comes to sociality and infection because both can reasonably be expected to produce both positive and negative selective disappearance trends. With regards to infection, a number of patterns could be produced by selective disappearance. Many parasites exact a substantial mortality toll (Tompkins and Begon, 1999), particularly on young individuals (Ashby and Bruns, 2018). In these cases, all else being equal, individuals with higher parasite counts will die sooner, producing an apparent age-related decrease in infection. In contrast, particularly if parasites are relatively mild, resisting them may be costly, such that the fittest individuals are those that tolerate infection and maintain relatively high parasite counts (Graham et al., 2011; Råberg et al., 2009; Viney et al., 2005). In this case, individuals that resist infection and have lower counts may be more likely to die, which will produce an apparent *increase* in parasite count across the population. As yet, such contrasting age-related trends have not been demonstrated.

Further complicating matters, in some cases, the fitness costs of resistance, tolerance, and infection can vary between life stages. For example, in red deer, strongyle nematodes exact substantial fitness costs for both calves (Acerini et al., 2022) and adults (Albery et al., 2021c); however, the costs of infection are far higher for calves, with much steeper relationships between parasite count and survival probability compared to adults (Acerini et al., 2022). Similarly, in black grouse (*Lyrurus tetrix*), stronger immune responses are favoured in younger individuals, while the reverse is true in adults (Soulsbury et al., 2017). In such cases complex, nonlinear age-infection relationships could emerge. For example, if individuals gain fitness benefits from resisting infection when young but by tolerating it when old, it is unlikely that the emergent age-infection trend will be linear. Nevertheless, no study has yet investigated an interaction between continuous measures of age and mortality in the context of infection and immunity; using models capable of identifying non-linear relationships (e.g. (Jones et al., 2008)) may be highly informative here. The central role that selective disappearance likely plays in determining age patterns is therefore important to investigate in the near future, especially given the current lack of longitudinal studies of immunosenescence in animals (Peters et al., 2019).

For sociality, there are obvious benefits at the individual level including improved hunting, defence, and mating opportunities (Ezenwa et al., 2016b; Silk, 2007b; Snyder-Mackler et al., 2020), all of which could produce selective disappearance of less-social individuals. However, there are also disadvantages to sociality: for example, greater sociality can correspond to greater competition, which could result in selective mortality of more-social individuals. Moreover, crucially, one of the most-often-cited costs of increased sociality is an increased risk of exposure to infectious disease (Altizer et al., 2003; Cote and Poulin, 1995; Ezenwa et al., 2016b); as such, these two phenomena form two simultaneous and intertwined selective processes that could interact across the lifespan. Further, social structure itself is heavily dependent on the demography of the population (Shizuka and Johnson, 2019), and therefore any skew in mortality patterns could inherently determine emergent properties of the network that influence observed age-behaviour-infection patterns. The ability to track both sociality and infection and detect positive and negative survival effects from both causes, while detecting within-individual trends in both, will be one of the foremost challenges in identifying the behavioural components of age-infection trajectories.

3. Concluding remarks

We therefore recognise a wide range of possible outcomes concerning behavioural ageing's effects on infection. Nevertheless, the resounding impression is that most straightforward "first-order" age-related changes in behaviour (reduced ranging and sociality) should function to reduce exposure, while more complex interactions with physiology (greater stress, intensified behavioural tradeoffs, and reduced competitive ability) could work to produce greater burdens of disease in older individuals. The balance of these two processes is still unclear. Building on the rising interest in social ageing (Machanda and Rosati, 2020; Siracusa et al., 2022a), this area of research stands ready to be elucidated further – particularly by more widely explicitly investigating age-infection slopes in behavioural study systems of individuals with known age and infection status. The number of case studies of behavioural ageing has grown substantially in recent years, and incorporating infection into these same systems is a logical next step that could be highly revealing.

Ultimately, we hope that deepening our understanding of the myriad ways behavioural ageing could influence infection may help to identify and counteract the ecological and evolutionary drivers of disease in ageing humans. However, there are precious few between-species comparative studies in this area, hampering our ability to generalise and draw conclusions concerning these processes across non-human and human animals. A notable exception is a meta-analysis of immunosenescence studies (Peters et al., 2019), which was highly informative concerning age-related changes in immunity but did not incorporate changes in infection. Furthermore, despite a growing literature on age-related changes in (social) behaviour (Siracusa et al., 2022a), these studies have likewise not been meta-analysed or connected with infection. As such, there remains a gap in our understanding of the role of age-related changes in exposure, interactions with immunosenescence, and their emergent effects on infection status. Future studies in this vein could examine how ageing covaries with the evolution of social structure (Korb and Heinze, 2021) and how social structure coevolves with infection (Altizer et al., 2003; Hawley et al., 2021; Poulin and Filion, 2021), although they may have to deal with a range of between-species and between-system confounders (e.g. where age and body size covary; (Watkins and Blouin-Demers, 2019)). Such studies could be parameterised using novel data sources, including databases of age structuring in humans (Mistry et al., 2021) and open repositories of animal social networks, some of which have associated individual metadata including age (Sah et al., 2019; Strauss et al., 2022). Cross-system approaches such as these could help to close the gap between animal and human systems of infection and ageing, as well as facilitating a richer view of the many roles of age-related changes in disease ecology and epidemiology.

Importantly, behavioural ageing can come about through a number of mechanisms, often either related to the direct effects of senescence or changes to compensate for these effects (Siracusa et al., 2022a), benefitting from behaviour's ability to respond plasticly to a wide range of internal and external conditions. These behavioural changes could serve an adaptive function, allowing an individual to survive for longer than it otherwise would in the process of senescence; indeed, a wide range such advantages have been outlined (Siracusa et al., 2022a; Thompson González et al., 2023). These explanations come into play specifically in the area of infection: for example, because older individuals often have weaker immune systems, social ageing could have evolved specifically to counteract this waning immunity and reduce the burden of infection in late life (as outlined above). However, fundamental evolutionary theory states that – for a variety of reasons – the strength of selection is expected to weaken with age (Hamilton, 1966; Williams, 1957), such that senescent traits are rarely evolved for the purpose observed during senescence. Rather, they often represent mechanistic links that are difficult to lose in late life, or an ageing individual's ability to deal with physiological deterioration. This fact could limit the extent of behavioural ageing's influence on ecology and evolution, and vice versa. As such, without considering the waning of selection, adaptationist explanations of social ageing could be overstated. On the other hand, advantageous effects of behavioural ageing on infection could nevertheless come about despite the weakening of selection: for example, reductions in social connectedness with age could emerge because contact reduction when immunocompromised is selected for in early life (Stephenson, 2019; Zylberberg et al., 2013) and

remains mechanistically linked (e.g. through pleiotropy) into later life. Alternatively, older individuals could be selected to reduce their contacts because of indirect benefits to kin (Albery, 2022; Frank, 1998). In the future, evolutionary simulations and adaptive dynamics approaches could be used to examine how severe this weakening of selection should be, and how strong the mechanistic links across the lifespan, in order for behavioural ageing to evolve in response to infection.

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