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The impact of the built environment on health behaviours and disease transmission in social systems

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The environment plays an important role in disease dynamics and in determining the health of individuals. Specifically, the built environment has a large impact on the prevention and containment of both chronic and infectious disease in humans and in non-human animals. The effects of the built environment on health can be direct, for example, by influencing environmental quality, or indirect by influencing behaviours that impact disease transmission and health. Furthermore, these impacts can happen at many scales, from the individual to the society, and from the design of the plates we eat from to the design of cities. In this paper, we review the ways that the built environment affects both the prevention and the containment of chronic and infectious disease. We bring examples from both human and animal societies and attempt to identify parallels and gaps between the study of humans and animals that can be capitalized on to advance the scope and perspective of research in each respective field. By consolidating this literature, we hope to highlight the importance of built structures in determining the complex dynamics of disease and in impacting the health behaviours of both humans and animals.

This article is part of the theme issue 'Interdisciplinary approaches for uncovering the impacts of architecture on collective behaviour'.

1. Introduction

The health of individuals and populations is affected by the environment in which they live. Some environments harbour more pathogens than others and population densities vary across environment, which influences disease transmission dynamics. Moreover, variation in resource distribution across environments can determine movement patterns, which can expose individuals to new pathogens, but also contribute to their health by increasing activity. The built environment can be modified to promote healthy behaviours and reduce the risk of contracting a disease.

Perhaps the most striking illustration of how the built environment can affect both health behaviour and disease comes from the history of urban planning over the past century [1–4]. Disease was the raison d'être for the advent of urban planning in Europe and the USA, and one of the central motifs that shaped architecture of modernism. Throughout the nineteenth and early twentieth century, urban environments such as London, Paris, New York City and Chicago were densely populated and characterized by residences in proximity to factories, animal yards, slaughter houses and crowded tenement houses with little airflow or light. The cities were plagued with epidemics of infectious disease. Waves of cholera, tuberculosis and typhoid swept through these cities, wiping out significant portions of the population. Disease was not well understood at the time and models such as 'miasma theory'—that 'bad air' vapours transmitted pathogens—prevailed. However, there was a sense that the

congestion, pollution, lack of sunshine and poor airflow contributed to illness. In response, the mid-nineteenth century public health movements [5] and the extensive rebuilding of European and North American cities ensued, with the aim of improving the overcrowded and unsanitary urban living conditions [6,7]. Zoning, i.e. separation of uses, was introduced to spatially segregate residential, commercial and industrial uses, and housing regulations required light and air flow. Remarkably, these efforts to configure the built environment to control infectious disease in the late 1800s and early 1900s ultimately contributed to chronic diseases in the twenty-first century.

The separation of uses through zoning and development of suburbs, along with the advent of the automobile, led, 100 years later, to environments that discourage walking and promote movement in the private automobile. We now have a physically inactive population with rising rates of obesity and related chronic diseases such as diabetes, cancer and coronary heart disease. The field of urban planning and, to some extent, architecture have now—since the early 2000s—renewed partnership with public health to respond to the new health crises: physical inactivity, poor diet and obesity. In an attempt to curb the obesity epidemic, urban planning efforts have begun to target both sides of the energy balance equation—diet and physical activity [8]—by considering the accessibility, availability and affordability of healthy foods and aspects of the built environment (e.g. density, mixed use and design features) to encourage physical activity. In addition, there is growing recognition that low-income and minority neighbourhoods are often 'food deserts' characterized by the abundance of liquor stores and fast food restaurants but with a dearth of grocery stores. On a parallel front, the relationship between mental health and the built environment, in particular in urban centres, is becoming an equally important concern. Chronic disease, such as depression, has been linked to both social and physical aspects of the built environment—from factors such as social isolation and poverty in the neighbourhood to housing quality, crowding and urban design of streets and green spaces [9–11]. Current designers' proposals for addressing these mental health issues include the creation of spaces supporting physical activity, social interaction and high-quality access to nature, and are thus coinciding with the design strategies for improving the physical health of the population.

The history of urban planning in the past century highlights the effects that the built environment can have on both the prevention and containment of chronic and infectious diseases. Chronic disease is defined as a non-communicable disease that persists for a long time and that cannot be prevented by vaccination or cured by medication. Infectious disease is caused by pathogenic microorganisms and can spread among individuals. Strategies for battling both types of disease includes pre-emptive preventions, such as hand washing and vaccinations for infectious diseases, and health-promoting behaviours, such as an active lifestyle and healthy food habits for chronic diseases. Once a disease becomes prevalent in a population, containment becomes the main strategy for defence. For example, quarantine of diseased individuals in the case of infections and caring for sick individuals and improving their environment in the case of chronic disease. Non-human animals are also prone to both chronic and infectious diseases and they too engage in prevention and containment behaviours. Ways that the built environment can facilitate the prevention and containment of disease in non-human animals include the type of building materials that are used and the way built structures organize the society and promote or prevent certain interactions. Thus, the built environment can promote both the containment and prevention of chronic and infectious disease in human and non-human animals.

The built environment can affect health directly and indirectly either through immediate, passive impact (e.g. effects of indoor environmental quality) or by influencing behaviours that can affect health, which can involve individuals' active participation (e.g. encouraging walking to increase physical activity). It is worth noting that the definitions of human health and disease are products of history, politics, economics and culture [2]. In this sense, the notion of what it means to be healthy or sick is guided not only by the available medical knowledge, but also by broader social and cultural factors. For most of the nineteenth and twentieth centuries, medicine was concentrated on pathology and finding ways of treating/curing disease. However, as societies experienced an epidemiological transition [12]—diminishing infectious disease and increasing the prevalence of chronic diseases—the idea of health-related quality of life has emerged as an instrument to cope with the new situation [13]. Accordingly, the current understanding of health is not only as an absence of disease but also as a state of complete physical, mental and social well-being, which holds prevention as important as cure and looks for long-term solutions [14,15]. This idea is reflected in the current design approaches to health problems and what is considered as a problem; the aim of architectural and urban designs and behaviour interventions is to enhance overall well-being through mental and physical health.

Prevention and containment of disease can happen at many social and biological scales, given the multilayered physical, social and socio-economic context of the built environment. For example, at the society level, governments can establish policy, which impacts states, counties, schools and individuals. Individuals, in turn, may take actions to impact their immediate environment, regardless of global policy. Modelling approaches in biology scale from agent-based [16], to population, to evolutionary models, and each level provides different insights on disease dynamics. The scale at which actions take place can impact what proportion of the population is affected and how quickly remediation can occur. Considering scales of action is important when discussing the design of the physical environment. In this sense, three scales are of particular relevance: the urban, architectural (or building) and behavioural design. Urban design and planning can impact population-level processes by affecting the proximity of individuals to one another, while at the architectural scale, with the help of behaviour and product design, spatial structures and targeted interventions can impact individuals' behaviour, thus promoting local changes.

In this paper, we review the impact of the built environment on both chronic and infectious disease. For each, we detail ways that the built environment has been and can be used for prevention or containment through examples from both human and animal societies. Through this review of the literature, we attempt to identify gaps between the study of humans and animals that can be capitalized on to advance the scope and perspective of research in each respective field. For example, the scale at which containment

action is taken in human societies might inform the conservation of animal populations, and the evolutionary perspective that often characterizes studies of animal behaviours might inform prevention strategies for human disease. Our goal with this review of the literature is to set the groundwork for further, more in-depth studies of each of the various ways that the built environment affects health behaviours and disease dynamics in humans and non-human animals.

2. Chronic disease

(a) Prevention in humans

In this section, we consider how the architecture of the human environment can contribute to the *prevention of chronic disease—related to both physical health and mental health*. We consider three themes to illustrate health-promoting qualities of physical environment. First, we consider how the physical environment (at the urban, architectural and behavioural scales) promotes physical activity, which helps to prevent obesity and the associated chronic diseases including diabetes, heart disease and cancer. Second, we describe how the built environment affects dietary habits, another important factor in obesity. Third, we consider the effects of housing and urban design on mental health. These three themes are representative of the main research topics in current health-related design, and the possibilities available for promoting health and preventing chronic disease through the built environment.

(i) Physical activity

In recent decades, recognition that the built environment can affect physical activity or inactivity has led to efforts that leverage the environment to promote physical activity and thereby reduce the prevalence of associated chronic disease. This realization has resulted in a reconnection of urban planning and public health, two fields that united in the early 1900s to combat infectious disease and then had little association for many decades. Planners summarize the features of the environment influencing physical activity at the urban or neighbourhood scale by referring to the '3 Ds': density, diversity and design [4,17,18]. Density refers to the compactness of physical infrastructure (i.e. distances between buildings and functions). With more proximate destinations, residents are more likely to walk rather than drive a vehicle. Diversity refers to 'mixed use'—in other words, combining residential and retail within the community. This notion is a reversal of the segregation of uses that occurred in the early twentieth century in response to infectious diseases. Diversity means that there will be walkable destinations near the places where people live. The third D, Design, is relevant on various levels. Neighbourhood design has been revisited via neotraditional or new urbanist neighbourhoods that are pedestrian-, rather than car-focused. Such pedestrian-oriented designs have small lots, short setback distances (i.e. distance from the street to the front of the building), porches and sidewalks, in contrast to car-oriented suburbs that typically have 1 acre (or larger) lots, large setbacks and no sidewalks. The features of neotraditional communities promote social interaction, sense of community and walking [19]. People who live in neighbourhoods with a grid-like street network pattern also tend to drive less than those living in other kinds (e.g. suburban 'loops and lollipops') of street networks [17]. Design further includes smaller-scale design elements, such as street lights and benches, that make a place pleasant and comfortable for walking. In recent years, a fourth and fifth D have been added: Destination accessibility (i.e. ease of travel to a central business district) and Distance to transit (i.e. the average distance from the residence to the workplace or to the nearest train station or bus stop) [18].

Building design can also be employed for its potential to encourage physical activity. For example, placing a stairway in a salient location and making it inviting and aesthetically pleasing, while locating elevators in a less obvious, less central position, may encourage stair use [20]. Colour, music and artwork have been used to encourage the use of stairs [21]. These efforts to design buildings to promote physical activity are ironic in light of research a century ago aimed at essentially the opposite goal: 'saving steps' by improving the efficiency of daily tasks [22]. In 2010, New York City published 'Active Design Guidelines' encouraging design decisions to help promote physical activity [18]. The guidelines address building design and urban design strategies. On the building scale, four key themes are identified as most critical to promoting physical activity [18]:

— *Building circulation system*. The design of the 'connecting spaces' such as corridors, stairways, elevators and lobbies can play a critical role in encouraging physical activity within a building.
— *Building elements*. The availability, safety and comfort of spaces such as stairs, shower rooms and bicycle storage as well as smaller details such as the presence and location of drinking fountains and benches can promote movement.
— *Organization of the building programme*. Configuration of the activities within the building can help to ensure that physical activity is 'built in' to daily activities. For example, daily tasks that require physical activity include going to a central location to retrieve mail, get coffee, or pick up lunch. These strategies employing intentional distance or inefficiencies are referred to as 'functional inconvenience' [23].
— *Activity spaces*. Building areas specifically programmed for physical activity can also contribute to occupants' total physical activity. These spaces include swimming pools, running tracks and exercise rooms.

Research has also begun to examine the effect of small-scale environmental changes on increasing physical activity or reducing sedentary behaviour, particularly within the workplace. Neuhaus *et al.* [24] reviewed the evidence regarding the influence of 'activity-permissive' workstations, including fixed standing desks, height-adjustable desks, treadmill desks, cycle ergometers and pedal devices fitted under the desk. Of the 14 studies that examined effects on sedentary behaviour, 11 found a significant effect of the intervention with an average reduction in workplace sedentary time of 90 min per 8-h workday. Other researchers have begun to study the influence of architectural design in combination with activity-promoting furniture within the school environment [25]. Dutch architects RAAAF (Rietveld Architecture-Art Affordances) have responded to society's epidemic of sedentary behaviour with the 'End of Sitting', an art–architecture–philosophy installation that questions

the inclusion of desks and chairs as default components of the workplace and imagines what a space that affords supported standing and varied postures might look like [26].

Finally, the field of behavioural economics [27], a cousin to environmental psychology, offers additional insights regarding the possible influence of context on physical activity. For example, social norms can be used as 'anchors' to influence behaviour. By intervening in people's perceptions regarding what are 'normative' or typical levels of physical activity, people might become more active. Framing physical activity as fun, rather than obligatory, could also affect people's likelihood to engage in physical activity [28]. Related to these themes, both policies and physical infrastructure can, together, affect physical activity norms. For example, making public transportation affordable, providing biking lanes and making automobile parking expensive can encourage walking and biking, and discourage driving.

(ii) Diet

In parallel with studies examining the association of neighbourhood design characteristics with physical activity, other studies have been examining the association of neighbourhood features with dietary intake or obesity. A study of the New York City food environment found that access to healthy food stores was inversely associated with body mass index and obesity prevalence [29]. Another study of more than 3000 New Orleans residents found that after adjusting for individual characteristics, each additional supermarket in a respondent's neighbourhood was linked to a reduced likelihood of obesity, while fast food restaurants and convenience stores were associated with greater obesity odds [30]. Research also indicates that disparities in access correspond to disparities in dietary intake. For example, in a study of African American boys, greater availability of vegetables and juice at local restaurants was associated with greater juice and vegetable consumption [31]. In a rare natural experiment, Wrigley *et al.* [32] found that when a new grocery store was constructed within a 'retail-poor' area, consumption of fruits and vegetables (FV) increased significantly among those with the most FV-deficient diets.

On the building scale, research has begun to examine how design features affect dietary intake. In the grocery store, Cheadle *et al.* [33] found that the proportion of shelf space dedicated to healthy foods, such as low-fat milk and dark bread, was associated with individual dietary practices. The effects may be similar within the home environment. Open layouts, which provide visual access between the kitchen and the living room, may encourage trips to the kitchen and increase food intake [34]. In addition, research indicates that smaller-scale environmental and product design features also affect dietary intake. Larger plates, portions and packages influence people to consume more [35–37]. In addition, people tend to eat in 'units'; in other words, typically, a person eats the entire item, regardless of the size of the muffin or cookie [38]. Fortunately, small-scale environmental features such as plate size can be modified to mitigate over-consumption [39].

(iii) Mental health

In addition to its effects on health-related behaviours and physical health, the built environment can affect mental health, both positively and negatively. In the light of current urbanization rates and evidence suggesting that city dwellers have higher risks of mental health problems, such as depression and anxiety, compared to inhabitants of rural areas [10,40], the relationship between the urban mental health and design has recently gained importance. The physical and social environments of urban life can influence the mind and the body at the neurophysiological and psychological levels, and thus affect mental well-being [41,42].

Environmental properties such as spatial layout, architectural features, traffic intensity, noise and pollution can have a direct impact on physiological and psychological stress mechanisms. For example, at the urban scale, the spatial configuration of the city and, more specifically, environmental properties such as typology of open public spaces (e.g. park, square and street), building density and local integration of street segments (i.e. how well a street segment is integrated in the wider city network and traffic patterns) can be used as predictors of urban stress [43]. Researchers found that high values of local street integration, which is associated with good walkability, are associated with low stress, while large streetscapes and squares with low detailing and complexity in building facades are more likely to be perceived as stressful.

Although only depression is currently considered a chronic mental disease, stress and anxiety cannot be excluded as factors affecting the well-being of people in cities because prolonged and cumulative exposure to cortisol can lead to physical chronic diseases like stress-induced hypertension [9,44]. Indirect effects of urban environments have been associated with psycho-social processes, such as personal control, crowding and presence of social networks and support [41,45,46]. Thus, physiological and psychological stressors have the capacity to influence mental health both at the individual level (e.g. individuals' perceptions of the environment) and through neighbourhood effects (e.g. the experience of neighbourhood walkability and state of maintenance and upkeep).

Because both the physical and social aspects of the urban environment impact mental health and well-being, design strategies aimed at preventing or diminishing the negative effects and emphasizing the beneficial ones typically rely on the interplay between these two dimensions. Specifically, a recent report on the 'Five Ways to Well-being' [47] illustrates how the social–physical interdependency can be used in the design of architectural and urban spaces [14,48]. Three of the five points are relevant here. First, the 'connect' idea correlates the quantity and quality of social connections with reported well-being and physical health. In the built environment, this is translated in the emphasis on designing everyday public spaces, especially at the neighbourhood scale, to create opportunities for people to see, hear and connect with others [49–51]. However, social interactions are also tightly connected with density and crowding, which have been linked with increased stress and anxiety [52,53]. Second, the 'keep active' point emphasizes the link between physical activity and well-being, which, in addition to effects on physical health, as detailed above, is associated with beneficial effects on mental health problems, like depression, and thus requires designing more walkable and pedestrian-friendly neighbourhoods [54,55]. Third, 'take notice' considers the benefits of mindfulness and paying attention to the present as a way to reduce the symptoms of stress, anxiety and depression. In the urban environment, 'taking

notice' can be achieved through art, landscaping, wildlife features and seating [14,56]. Overall, the availability of diverse open public spaces, the high density of mixed-use development that encourages walking and cycling, and access to high-quality green spaces in the city can be linked to the positive effects of the physical and social urban environment on mental health. While some of these aspects have been better investigated, many mechanisms, e.g. how the physical environment impacts the mind–body at the neurophysiological level and how this, in turn, might modify behaviours, are still unknown.

Housing quality, housing type and floor level are three aspects of housing that have been associated with mental health outcomes [57]. The relation between housing quality and mental health may be mediated by social withdrawal. In other words, poor housing quality can lead to increased social withdrawal which, in turn, leads to poor mental health [58]. One aspect of housing is interior density, i.e. the number of people per room. Density, a physical, objectively measureable phenomenon, affects crowding, a psychological phenomenon, which in turn negatively affects psychological well-being or mental health. The linkage between crowding and mental health is explained by a disruption of socially supportive relationships among residents of a crowded home. To cope with chronically crowded conditions that provide limited ability to regulate social interaction, occupants often socially withdraw. However, by allowing opportunities to control social interaction, architectural interior design can help to reduce the need to socially withdraw and thereby dampen the effect of crowding on mental health. Evans *et al.* [59] found that homes with greater architectural depth—the number of spaces one must pass through to reach rooms of the home [60]—buffer the impact of density of mental health by reducing social withdrawal. Complete social isolation can also impact mental health negatively [61–63], and so, the built environment should balance the ability to avoid crowding without risking the isolation of its occupants.

It is important to note that these effects of architecture on the prevention of chronic disease in humans occur within a larger, complex ecological system [64] and thus are not simple, direct effects. Rather, a variety of moderators or 'effect modifiers' influence the valence and strength of the impact of the environment on human health and health-related behaviours. This notion is illustrated by Evans *et al.*'s findings [59] that architectural depth moderates the effect of crowding on mental health. Similarly, Fich *et al.* [65] showed that when exposed to a strong social stressor (simulated job interview), the features of the built environment—presence or absence of openings in the room—influence how fast participants recover from stress (measured as cortisol levels). Thus, architecture might modulate people's physiological response in the case of acute stress events, including social situations. Further research is necessary to understand the role of built spaces in the case of acute as well as prolonged or chronic stress, especially when their causes are found in a complex socio-economic network.

Overall, it should be emphasized that the scale of effects resulting from interactions between social and physical environmental factors is still an open question. The aim of this paper is to highlight the myriad ways that the built environment shapes social relations and behaviour in space, and in turn affects human health.

(b) Prevention in animals

Chronic diseases in animals are most commonly found in domesticated and zoo animals. However, some chronic conditions, such as long-term stress and nutritional deficiencies, can impact wild animal populations. Specifically, chronic stress can decrease animals' survival in the wild [66] and increase their susceptibility to infectious diseases [67]. In this section, we detail how stress, diet and physical activity may be impacted by the built environment in animals.

(i) Stress

Built structures can prevent chronic stressful conditions if they provide an enriched physical and social environment. Many industries have been impacted by the interaction between the built environment and chronic stress, including zoos, biomedical research and agriculture. Zoos have been increasingly considering enclosure designs that provide animals with enriched environments to reduce stereotypical behaviours, such as pacing and other repetitive movements, which can lead to chronic heightened physiological stress, i.e. high cortisol levels [68,69]. Built structures that facilitate social interaction reduce stress because grooming in primates and ungulates alleviates stress through the release of β-endorphins [70–72]. Housing conditions of research animals may impact their physiology, thus biasing the results of scientific studies. For example, housing conditions of rhesus macaques can influence their social environment, elevating their stress levels if they are housed alone, which can bias the results of biomedical research [73]. Housing conditions that lead to stereotypical behaviour of rodents used for research may affect the validity, replicability and reliability of studies through changes to animals' brain function [74]. In agricultural settings, the structure of rearing enclosures can influence long-term chronic social stress. For example, piglets raised in an enriched environment do not develop social stress later in life, but piglets reared in a featureless environment (simple farrowing crates) develop chronic social stress [75]. Finally, the chronic stress of wild animal populations can be impacted by built structures. For example, great tits in urban environments express more genes related to stress responses than rural birds [76]. Thus, the built structures that humans construct to hold animals, whether in zoos, laboratories, farms or cities, can have a great impact on the chronic physiological conditions of the animals, which affect their fitness, welfare, utility for scientific research and economic output.

(ii) Diet

Structures built by the animals themselves (rather than by humans) that allow for food storage or acquisition can buffer nutritional deficiencies that compromise animals' health. For example, social insect nests often include chambers that are dedicated to the storage of seeds [77]. Honeybees store nectar in the form of honey, and pollen for protein, at specific locations in their hive [78]. These food stores can ensure colony survival during the winter months, when there are no flowers [79]. Spider webs and beaver dams are structures that assist animals in collecting food [80], thus potentially reducing long-term nutritional deficiencies. Finally, bird nests and carnivores' dens provide both protection from predators and reduce the amount of energy spent by parents caring for offspring by restricting

their movements in search of food [81] and by reducing the energetic costs associated with carrying offspring [82].

(iii) Physical activity

In contrast with humans, physical activity might not necessarily promote long or healthy lives in non-human animals. For example, in social insects, queens that are long-lived (more than 30 years in some species) are extremely sedentary, compared to workers who are very mobile, yet short-lived (mostly up to 1 year) [83]. These differences between queens and workers likely stem from differences in metabolic rates [84], genetics [85,86] and exposure to dangers. The high activity of workers leads them outside the safety of their nest, exposing them to dangers such as predation and desiccation. Thus, the built environment, i.e. the ants' nest, provides shelter that may promote longevity. In mammals too, captivity can increase longevity, especially for species with a fast pace of life, for whom captive conditions, such as zoos, provide protection from predators, intraspecific competition and disease [87].

(c) Containment in humans

Despite the efforts to prevent chronic diseases, like obesity and depression, through the built environment, some illnesses—especially those that are age-related—can only be prevented and postponed to a certain point. For this reason, an important part of design interventions in the physical environment is aimed at the *management of chronic conditions*, i.e. developing and maintaining the systems of care. In this section, the issue of care is considered in three ways. First, we discuss how the social and material environment can serve as the support system in the context of diseases that follow the ageing process, and what kinds of transformations at the urban, neighbourhood and architectural scales can be implemented as strategies for 'caring through design'. Second, we explore the potential of the built environment as a therapeutic tool to alleviate or diminish the effects of everyday stress and anxiety. We further discuss the topics of biophilic design and cognitive restoration as elements of passive design care, i.e. treating lifestyle consequences by directly affecting individuals without requiring active participation or behavioural changes. Finally, we raise the question of care in the context of geographical disparities in health and the issues stemming from the lack of care in the state of the built environment.

(i) Lifetime care through design

The global increase in ageing populations and corresponding age-related physical and mental illnesses such as cardiovascular conditions and dementia, coupled with sensory impairments and reduced mobility, present a public health challenge that can be partially answered through the design of built environment. Over the past decade, different age- and dementia-friendly design strategies for urban and architectural spaces have been developed under the common theme of 'ageing in place' or 'lifetime neighbourhoods'. The guiding principle behind these strategies is supporting active and independent involvement in local communities to maintain health and manage existing long-term conditions in older individuals. For example, at the urban and neighbourhood levels, dementia-friendly designs target the critical issues such as ease of wayfinding by proposing environments that are familiar, legible, distinctive, accessible, comfortable and safe [88]. Some of the key design features include the presence of small, open public spaces with a variety of activities and features, walkable neighbourhoods, architecture with distinctive local character and identity, public seating and ground-level building access as measures of accessibility—in short, all environmental characteristics that encourage physical activity and social interaction as beneficial for physical and mental health in older people. This is in accordance with recent studies indicating the links between social deprivation and depression in high-density cities like Hong Kong [89] and negative effects of deprived and deteriorated neighbourhoods on physical activity [90,91]. These health-related urban design interventions are effective for most age groups. However, it should be acknowledged that some policies, such as active design guidelines, can lead to segregation of various user groups (e.g. young, mobile individuals versus individuals with reduced or no mobility [92]) that call for the development of inclusive approaches.

The possibilities of caring through design for individuals with chronic disease such as cancer have been explored at the architectural scale of healthcare institutions. A well-known example are Maggie's Centres, which were established with the idea that psycho-social interventions increases patients' chances of living longer [92,93]. These buildings are designed to offer cancer patients a place to interact with doctors and families outside of the stressful setting of a traditional hospital and provide a sense of home, through architectural design.

(ii) Therapeutic design and nature

Although we are only starting to understand how architectural and urban environments can act therapeutically on human minds and bodies, designers have been intuitively exploring these capacities for their restorative effects, in particular for the purposes of managing stress and stress-related diseases. Recently, the idea of biophilic design has linked the extensive body of research on the health and stress-relieving benefits of nature and the innate human inclination to seek connections with nature, life and life-like processes; essentially, biophilic design emphasizes the necessity of maintaining, enhancing and restoring the beneficial experience of nature in the built environment [94].

Views of and access to nature have been linked to a wide variety of health outcomes (see reviews, [95,96]). Nature can contribute to the management of stress and stress-related diseases. For example, recent studies by Japanese researchers examine the practice of 'Shinrin-yoku' or 'taking in the forest atmosphere'. In a series of studies, male college students were randomly assigned to walk in the city and then in the forest, or vice versa. Results indicated lower levels of blood pressure, pulse rate and the stress hormone cortisol along with increased parasympathetic nerve activity and lower sympathetic nerve activity following the forest walks compared to the urban walks [97].

For people with disease diagnoses, nature can enhance their capacity to cope effectively. Cimprich [98,99] studied women recently diagnosed with breast cancer and found that patients randomly assigned to a nature intervention showed significant improvements in attentional capacity in the weeks following surgery, compared to those in the

non-intervention group. Underlying this work is Attention Restoration Theory [100], which suggests that we have two types of attention: effortful 'directed' attention and 'involuntary' attention that is captured easily and effortlessly. With use, directed attention becomes fatigued, resulting in difficulty focusing, distractibility and irritability. The natural environment engages involuntary attention and allows the mechanism underlying directed attention to rest and recover. Thereby, nature enhances attentional capacity and the ability to cope and manage life's demands, including coping with illness.

Thus, design intentions are focused on fostering beneficial contact between people and nature in both architectural and urban spaces, by giving importance to features such as natural light, water, vegetation, views of nature, sensory/spatial variability and establishing place-based relationships. In brief, these architecture–nature principles can be summarized in three broad experience categories: (i) nature in the space—which refers to the presence and diversity of natural elements and environmental conditions within the built environment; (ii) natural analogues—which refers to objects, materials and shapes that evoke nature; and (iii) nature of the space—which refers to the spatial configurations resonating with evolutionary human preferences for exploration, mystery and prospect/refuge [94,101].

(iii) Caring disparities

A final aspect of containment of chronic disease concerns the uneven distribution of health, particularly in the USA. Low-income and ethnic minority populations are more likely than wealthy groups or than 'Whites' to experience a variety of adverse health outcomes, from coronary heart disease to diabetes to chronic bronchitis [102]. The physical places where people live—their houses, their neighbourhood and their workplaces—contribute to the uneven and unequal geographical distribution of health. For example, in the USA, researchers have documented that health-promoting and health-deterring neighbourhood features such as supermarkets, liquor stores and fast food outlets are correlated with race and socio-economic status of communities. Wealthier neighbourhoods are more likely to have supermarkets and gas stations with convenience stores compared to poor neighbourhoods; the same is true of White compared to Black neighbourhoods [103]. Powell *et al.* [104] found that in Black neighbourhoods, the availability of chain supermarkets was 52% of what it was in White neighbourhoods; differences existed even after controlling for neighbourhood level income [104] (for review, see [105]). A similar pattern is evident with respect to the natural environment, which has well-documented beneficial effects on human health and well-being [95,96]. Nature is often unequally distributed, with disenfranchised populations having less access to natural amenities [106,107]. In New York City, playgrounds in low-income neighbourhoods are more likely, compared to playgrounds in high-income neighbourhoods, to have a variety of hazards including paint chips, trash, rot, rust, splinters and vandalism [108]. Similarly, in Baltimore, Maryland, while Blacks are more likely to live within walking distance of a park, those parks are more likely to be hazardous or polluted, and are typically smaller than those to which Whites have access. So, what do these geographical patterns of health disparities suggest with respect to containment? To most effectively contain the epidemics of chronic disease that disproportionately affect low-income and minority populations, it is essential to tackle the underlying environmental justice issues, and to distribute healthy, safe, nurturing environments across the population to promote equitable public health.

(d) Containment in animals

Just as space may be used by humans to care for individuals who are at risk of chronic disease, animals too designate locations within their built environments for sensitive individuals. For example, social insects, such as ants and bees, dedicate specific locations within the nest or hive for brood (eggs, larvae and pupae) [77]. Brood can further be moved around the nest to expedite development, for example, by bringing larvae from deep inside the nest to near the soil surface, where it is warm during the day [109–111]. Whether or not animals modify their built spaces to create healthy environments, or to create spaces to care for chronically sick individuals, as humans do, is an open question.

(i) Spatial disparities

Disparity in habitat quality is key in determining population structure and competition in animals. Animals regularly compete over high-quality habitats and defend their territories [112]. Low population densities result in lower competition and better access to resources [113], thus potentially creating more healthy environments in which animals may be less likely to suffer from malnourishment that could lead to chronic stress. The need for shelter can create socially facultative structures in animals that would not be social otherwise. For example, yellow-bellied marmots rely on burrows for wintering and for escaping from predators, thus forming facultative social structures [114]. Interestingly, individuals in larger groups express higher levels of faecal glucocorticoid metabolites, an indicator of stress [115]. Thus, living in a built structure can, in some cases, lead to chronic stress, and dispersing to find a less crowded burrow system might be the best way to contain such chronic stress. Similarly, harvester ant colonies will relocate to new nest sites more frequently in environments with fewer resources compared with areas that have high primary productivity [116]. Thus, changing the built environment, i.e. the nest, by relocating to a new one (instead of restructuring) can potentially help avoid or contain stress induced by low resource availability. Some animals prefer locations that are near conspecifics, for example, to gain better access to mates, and potentially because conspecifics can indicate high habitat quality and be used as cues. Such attraction to high-density areas is known as Alee effects [117,118] and they may facilitate social interactions that can reduce chronic stress, as detailed above.

3. Infectious disease

(a) Prevention in humans

In this section, we consider how the built environment can prevent epidemics and the flow of infectious disease. As noted above, contagious diseases have been the direct cause for changes in the fields of urban planning and architecture since the mid-nineteenth century in the efforts to eradicate

the unhealthy living conditions that were believed to support various epidemics. Specifically, the hygienist agenda was embraced in the early twentieth century as one of the postulates of modernism [1,92]. Closely linked with tuberculosis as a medical obsession of the time, modern architecture has produced a specific set of spatial typologies with assumed therapeutic and prevention effects, such as large windows, flat roofs and terraces open to sunlight, air, nature and physical exercise [6,119]. In this sense, modern architecture developed around two kinds of symbolic figures: the 'fragile tuberculosis patient seeking a cure' and the 'athletic figure seeking prevention from the diseases of modernity' [119]. As in the case of chronic diseases, architects and urban designers have historically applied similar strategies for dealing with infectious diseases, whether through prevention or finding ways to contain the epidemics' spread and help alleviate the symptoms once they appear. As with our consideration of chronic disease, we examine the relationship between infectious disease and the built environment and design strategies at several levels, including urban, architectural and small-scale design features within buildings.

(i) Health, indoor environmental control and building materials

The legacy of modernist hygienist ideas for prevention of epidemics can be seen in contemporary sanitary approaches to designing indoor environmental climate and in regulations regarding the health effects of various building materials. Environmental factors such as indoor air quality (e.g. air pollution, odours, fresh air supply and ventilation), lighting quality (e.g. view and illuminance), thermal comfort (e.g. moisture and temperature) and acoustical quality (e.g. noise from outside and indoors) are measured and controlled for their effects on the three systems of the human body—the nervous, immune and endocrine systems—through which they influence physical and mental health [120]. To prevent, or reduce, the spread of infectious diseases, contemporary building standards take into account the different modes of disease transmission, including indirect contact with airborne pathogens and contaminated objects, direct person-to-person contact and droplet spread. For example, in the case of airborne viruses, such as influenza, engineering control methods include the careful design of hospital building air cleaning and ventilation (both natural and mechanical). Such measures help dilute airborne pathogens and control their movement between spaces [121]. The role of physical structures in preventing disease spread was highlighted in the 2003 outbreak of SARS (severe acute respiratory syndrome) in a private residential apartment complex in Hong Kong, where the ventilation system and sanitary plumbing expedited the spread of viral aerosols [122,123]. Furthermore, disease can spread through contaminated objects, and the choice of building materials and coatings of indoor surfaces, such as walls, floors and furniture, can decrease the survival of pathogens and ease cleaning and sterilization.

Pollutants originating from toxic substances in building materials, such as heavy metals and asbestos, cause various neurological, cognitive and behavioural disorders and diseases like cancer [46]. Besides 'sick-building' syndrome, there is now a movement toward transparency regarding the chemicals and potentially harmful substances employed in building materials, furniture and finishes within the interior environment. Led by the architecture firm Perkins and Will, the 'Transparency Project' (www.transparency.perkinswill.com) documents substances such as arsenic, phthalates and volatile organic components, and their associated health risks. Moreover, with respect to urban outdoor environments, a recent study of citizens in Barcelona has found a link between urban air pollution and an increase in cases of depression and anxiety [124]. One way to control the pollution of urban air has been through the development of new 'living façade' systems that use plants and other organisms to absorb the pollutants of the city and purify the air [2]. Similarly, with the attentiveness to which species are planted in green areas, urban spaces are being transformed into allergy- and asthma-free environments.

(ii) Small-scale design interventions

In addition to influences on infectious disease at the urban and building scales, in recent years, researchers have begun to consider how smaller design features of buildings, particularly healthcare environments, might deter the spread of infectious disease. Approximately 5–10% of patients in US hospitals acquire an infection while in the hospital, resulting in 99 000 deaths each year [125]. Handwashing is a proven strategy to reduce infection rates and yet medical staff compliance has been elusive. Birnbach *et al.* [126] found that if the hand sanitizer dispenser was directly in the line of vision, in comparison to when the dispenser was adjacent to the doorway (as is quite typical), nearly 55% of physicians sanitized their hands. When the sanitizer was near the doorway, just 11.5% of physicians used it.

(b) Prevention in animals

Various aspects of the built environment can facilitate the prevention of spreading infectious agents. Here, we discuss a number of prevention measures observed in animals: antibacterial or antifungal materials embedded within structures; removing vectors of infection from the built environment; avoiding locations that have been previously exposed to pathogens, or show evidence of harmful consequences to its occupants, and structuring the built environment in a way that reduces interactions that may facilitate disease transmission among individuals.

(i) Building materials

Certain building materials, such as plant parts with antibacterial or antifungal properties, are integrated into animal nests to protect the inhabitants from disease [127]. Wood ants use resin from coniferous trees as nesting material. This resin inhibits the growth of bacteria and fungi and enhances the survival of the nest's inhabitants [128]. Honeybees incorporate resin from plants into the wax that forms their hive, thereby reducing the bees' investment in the expression of immune function genes [129]. Several bird species include green aromatic vegetation in their nest materials to reduce parasite load [130–133] and wood rats place California bay foliage in their nests to reduce the abundance of ectoparasites [134]. Termites line their nest walls with faecal pallets that decrease the germination of fungus spores [135], and certain ant species secrete antimicrobial compounds onto their nest walls to prevent the growth of harmful microbes in the nest [136]. Finally, dry nesting

material used by termites has lower loads of microorganisms compared with damp nesting material [137].

(ii) Removing and avoiding infectious agents

Removing vectors of infection from the nest, such as waste, excretions and dead or sick individuals, is common in animals, especially in social insects. Honeybees and many ant species remove dead individuals from their hive or nest [138–140], a behaviour that extends the lives of the remaining colony members [141]. Ants and bees can detect diseased individuals and behave aggressively towards them [142] until they leave the nest [143]. However, aggression is not always required and, in some ant species, sick individuals will remove themselves from the nest, without interacting with nest-mates [144]. Studies of the mechanisms underlying the 'undertaking' hygienic behaviour in honeybees have revealed complex gene regulation [145] and uncovered which neurotransmitters are associated with this task [146]. Waste is removed from the nest by specific ant workers that do not perform other tasks [147,148] and will not go on to perform other tasks before they die [149]. Interestingly, leaf cutter ant species that live in wet environments will dig special waste chambers inside their nest, while leaf cutter ant species from arid environments will dispose of their waste outside the nest [150]. One potential explanation for this difference is that in wet environments, microorganisms in the waste are more likely to spread and so confining waste in chambers that can be closed off reduces the risk of spreading pathogens. Other sanitary behaviours in animals include the use of latrines to concentrate excretions in one or a few locations inside or outside the nest. For example, all individuals in a colony of social spider mites defaecate in one location, usually near the exit of the nest [151], some ant species concentrate their faeces in certain locations inside the nest [152], and birds remove faecal matter from their nest, especially when there are offspring present [153]. Many mammal species create faecal latrines; however, these are mostly used for communication, rather than for sanitation [154–158].

In addition to removing infectious agents, animals can avoid locations that have either been exposed to pathogens or show evidence of disease. For example, mole crickets change where they dig tunnels to avoid areas where fungi are present [159]. Pathogens may linger in the environment and lead to the spread of disease. Non-synchronous crevice use in the Gidgee skink results in more frequent transmission of pathogens than direct social interactions [160]. Furthermore, the behaviours and habitat preference of the parasites may play a critical role in where they are found and how likely they are to persist inside a host's burrow [161]. Thus, the spatial behaviour of both hosts and parasites can impact the spread of infectious disease. For example, pygmy bluetongue lizards occupy burrows built by spiders and their choice of which burrow to occupy and how frequently to move between burrows can impact their parasite load. Individuals that move frequently between burrows are more likely to encounter and transmit a parasitic nematode [162]. Some animals avoid locations that have signs of infections. For example, great tits avoid nest-boxes with fleas and preferentially select clean nest-boxes [163]. Some ant species avoid areas in a nest with microbes [164] or avoid moving into nests with dead ants when selecting a new nest site [165]. However, other ant species preferentially choose nest sites with fungi [166], or with dead ants that are visibly infected with fungi [167], over clean, empty nests. It is possible that a low-dose exposure to such pathogens results in immunity during later encounters with it (like a vaccination) [168,169] or that the pathogen is attracting the ants and manipulating them behaviourally to facilitate its spread. Thus, nest selection does not always lead to the avoidance of disease.

(iii) Structure design

Animals may create structures that influence direct interactions that facilitate disease transmission between individuals. For example, creating compartmentalized spaces can segregate the society and allow only subsets of individuals to interact at any given time. Models comparing disease spread in various structures predict that if an infection begins at a single location, it will take longer to reach everyone in a group housed in a compartmentalized structure, compared with a compartment-less structure, in which individuals interact with one another uniformly [16]. However, other models show that spatial structures have only a small impact on disease transmission [170]. Empirical studies that examine the relationship between the built environment, interaction patterns and disease transmission are still lacking. Studies of how population densities influence disease prevalence provide some insights into how built structures may affect disease transmission. For example, ecto-parasite loads decrease with nest density in colonies of bee-eaters [171]. Furthermore, a common argument in the social insect literature is that the high density of social insects inside their nests puts them at risk of rapidly transmitting infectious diseases within the nest. However, such disease spread is seldom seen, leading to the development of many hypotheses about how social insects achieve 'social immunity' [172,173] or 'organizational immunity' [174], including through structuring their nests to regulate interaction rates [174]. For example, small nest entrances protected by guard workers may prevent pathogens from entering the nest [175]. Finally, wildlife managers may take action to prevent the spread of disease, for example through vaccination. However, such management actions can, in fact, expedite the spread of disease by creating unnatural spatial clustering of animals. For example, the use of feeding stations to distribute vaccinations for disease prevention spatially clusters animals and increases the risks of disease transmission [176].

(c) Containment in humans

While epidemic outbreaks of many communicable diseases, like measles and poliomyelitis, have been largely reduced thanks to vaccination and immunization, the complete eradication of infectious pathogens has been limited [177]. One reason for this includes the changes in epidemiological characteristics of infectious diseases due to increasing urbanization. According to Alirol *et al*. [178], higher population density affects the transmission speed of diseases, such as influenza and tuberculosis, that rely on direct contact and proximity. The rural-to-urban migrations and worldwide travel have also led to an increased risk of epidemics—whether by introducing new pathogens to the urban environment from adjacent rural areas or because newcomers lack the immunity to certain endemic diseases. The physical environment of cities has either provided or eliminated

favourable conditions for many infectious diseases. Unplanned urban expansion, such as slums, has brought about problems of inadequate sanitation, allowing the spread of water-borne diseases and creating breeding sites for various disease vectors, while improved housing conditions and destruction of vector habitats resulted in a decline in infectious diseases in other parts of cities [178]. For example, dengue fever (a mosquito-borne disease) is now found mainly in tropical urban environments. This disease has re-emerged recently due to high population densities, low herd immunity and increased mobility of people, including viremic individuals, leading to broader spatial propagation of the disease within the city [179]. In this section, we discuss the current models for understanding the flow of infectious diseases and strategies for containment of epidemics in urban environments and within buildings.

(i) Disease transmission and spatial configuration

Modelling the dynamics of infectious diseases in human social networks requires looking at three interacting components: the transmission of disease, the flow of information regarding the disease and the spread of human preventive behaviours against the disease [180]. The built environment can have a significant impact on two of these components of epidemics. First, because the diffusion of many infectious diseases is closely linked to the patterns of human mobility and social interaction, it is also directly influenced by the properties of the built environment such as spatial configuration of spaces within the buildings. Second, contemporary disease outbreaks cause a disturbance in the usual everyday functioning of public spaces and city infrastructures. Such changes occur especially when preventive measures (both planned and spontaneous) take place and include emptying streets, fever checkpoints at transportation hubs, forced closures of hospitals and voluntary quarantines [123]. Hence, a major challenge for epidemiology models lies in identifying and mapping the overlap between the social, behavioural and spatial factors that enable the transmission of disease.

Existing models have uncovered several important aspects related to the effects of social interactions and mobility patterns on disease dispersion. Modes of social interaction and of disease spread both impact the number of infected individuals. The nature of social contacts can be close, e.g. individual contacts that happen at home, in workplaces and in social situations with friends, or casual, e.g. occasional contacts at service places. Modes of disease transmission include airborne droplets, contaminated surfaces or direct transmission [181,182]. Importantly, contact networks are heterogeneous, i.e. opportunities for transmission are not equal for all individuals [183,184], but will depend on their spatial and temporal patterns of use and mobility. For example, the daily mobility patterns in developed high-density urban societies (e.g. journeys to work) are highly predictable. Therefore, public transportation and transfer points are considered 'transmission highways'. City-level models based on integrated traffic information, geo-spatial data and infection dynamics and spreading characteristics allow for developing preventive strategies for particular diseases, like airborne pandemic influenza A (H1N1) [185] and vector-borne malaria influenced both by infected mosquitoes and daily commuters [186]. When the daily routines of inhabitants are irregular, for example in resource-poor neighbourhoods in Peru, geographical space, economic and social context structure all influence transmission dynamics of an influenza-like pathogen. Less predictable movement patterns corresponded with increased epidemic size [179]. While current epidemiological models generally consider the spatial dimension of disease dispersion [187,188] typically by using new technologies, such as geo-spatial mapping (GIS), Bluetooth, mobile phone tracking and social networks, the exact spatial configuration of the physical environment is not taken into account. As illustrated in previous sections, spatial structures can directly affect social interactions within cities and buildings. A promising approach for incorporating architectural and organizational data into large-scale epidemic forecasting models was proposed by Potter *et al.* [189]. In their model, they used architectural distances measured between workstations to model contact networks between members of a research institute as directly dependent on the spatial layout of the building.

(ii) Containment strategies through isolation and quarantine

The main aim of disease dynamics models is to identify critical infection points and propose effective mitigation strategies either to prevent disease outbreaks (e.g. through targeted immunization) or to contain epidemics. Control measures that are directly related to the built environment typically involve social distancing and include separation of ill individuals from the rest of the population through spatial clustering, i.e. isolation or quarantine, and closing public places such as schools [181,190,191]. Historically, isolating sick individuals began in the first hospitals, as early as the twelfth century. Similarly, between the mid-nineteenth and mid-twentieth century, there was mass building of sanatoriums, especially for tuberculosis. The architecture of these sanatorium buildings was envisaged with the ease of care and sanitization in mind, with specifically designed furniture and materials. It further included open terraces and large windows as therapy involving exposure to sunlight and air [6,119].

(d) Containment in animals

Containing infectious disease in animals can be achieved by altering social interactions, for example, to facilitate grooming behaviour and remove or avoid diseased individuals. Furthermore, human intervention, for example, in the case of wildlife management and conservation, may impact the containment of infectious disease in animals.

(i) Social interactions

Grooming behaviour is one common method for containing infectious diseases. In addition to reducing stress levels, as discussed above, grooming is commonly used by animals to clean themselves and others in their group of ectoparasites [192–195]. Although grooming behaviour may prevent the spread of ectoparasites, it can facilitate fomite transmission and spread certain infectious diseases, such as tuberculosis [196]. Therefore, self-grooming is more likely to contain a disease and allogrooming is more likely to facilitate disease spread [197]. Furthermore, grooming can be associated with energetic costs, because individuals who are grooming are not resting, eating or watching out for predators [198]. Thus, built structures that can reduce these costs of grooming, for example, by creating food stores and protecting from predators, may promote animal health.

As discussed above, removing diseased or dead individuals from built structures is common in the animal world, especially in social insects. This is similar to quarantine in human societies, where sick individuals are spatially isolated from healthy individuals to contain a disease. Relocating to a new nest site, similar to human evacuations, is another way to contain the spread of infectious agents [199].

More broadly, altering social interaction patterns through modifications to the spaces that animals occupy can change disease dynamics [200]. Theoretical work linking social interactions and disease transmission reveals which interaction patterns expedite disease transfer [201–203]. For example, highly compartmentalized social structures, which can be achieved by living in compartmentalized structures such as nests with chambers, may slow the transmission of disease [204–206]. Experimental work in honeybees provides some information on how spatial organization may affect disease transmission throughout a society. When colonies are exposed to a pathogen for a short time, the disease remains on the outskirts of the nest, but when the colony is exposed to a disease for long periods, the infectious agents can reach the centre of the hive and potentially affect the entire colony [207]. Thus, the structure of the hive or nest can impact the rate at which infectious agents spread and modifications to this structure may aid in the containment of a disease, once it has been introduced. Whether or not animals modify the structures they live in to contain the spread of infectious disease is an open question.

(ii) Human intervention

Containing infectious disease is a special concern for wildlife management and conservation. For example, bat populations have declined substantially due to a fungus causing 'white nose syndrome' [208]. Models for containing the disease take into account the spatial distribution of the caves in which bats sleep to determine the best course of intervention that will have the largest positive impact on the entire population [209]. A large-scale, long-term, containment effort to reduce tuberculosis in cattle in the UK has been to cull badgers, which are a vector for the disease. However, the spatial arrangement of badger populations and the dispersal of healthy individuals into areas where badgers had been culled led to faster spread of the disease instead of its containment [210,211]. Thus, spatial behaviour, such as dispersal, den structure and occupation patterns, should be carefully considered in wildlife management plans aimed at containing infectious diseases [212].

4. Conclusion: the effects of the built environment on disease and health behaviours in both humans and animals

In our review of the literature to identify how the built environment might impact disease and health behaviour in both humans and animals, we identified parallels and differences between human and non-human animal societies that may provide a basis for expanding our knowledge of both.

Many chronic diseases in both animals and humans emerge from heightened stress. The built environment may facilitate the reduction of stress by changing social interactions. However, not all animals require the same amount of social interaction to reduce stress. Crowding in humans can induce stress and depression, but so can complete isolation. Animal species differ in the amount of social interactions they require: highly social species require frequent interactions, whereas many social interactions increase the stress in facultatively social species. Thus, the amount of social interactions facilitated by the built environment should fit the social structure and preferences of the species occupying the built structures. Feedback between social processes and built structures can further influence their effect on health behaviours. These social processes differ between humans and animals and among social situations, thus raising the importance of considering social processes and built structures in tandem. Future theoretical work on the amount of social interactions that various structures facilitate may help prevent and contain chronic diseases that stem from heightened stress in a wide range of species, including humans, highly social non-human animal species and solitary species.

In some cases, we found opposite impacts of built structures on human and animal health. For example, physical activity promotes health and longevity in humans, but in animals, we see the largest within-species longevity differences between individuals that are completely sedentary, protected by their built environment and living to old age (social insect queens), and those that are extremely active and die relatively young (social insect workers). Similarly, built environments that create easy access to energy-rich foods (such as sugars and fats) benefit animals but harm humans. This difference likely stems from the agricultural and industrial revolutions that have enabled humans to produce food in excess and escape the 'Malthusian trap'.

Hygienic behaviours are used by both humans and animals to prevent the transmission of infectious agents. Both humans and animals use certain building materials that promote health. Humans may be inspired by some of the materials that animals use and incorporate those into their buildings, or cleaning supplies, using biomimicry to prevent the growth of microorganisms where they are not wanted. Both humans and animals engage in sanitary behaviour—whether it is hand washing in humans or removing infectious agents in animals. Perhaps studies on where hand sanitizers are positioned in hospitals can inform studies of sanitation behaviour in animals. For example, these studies may guide researchers seeking locations that promote sanitary behaviours in wild animals and inform the configuration of animal enclosures for captive animals in zoos and biomedical research facilities, to facilitate sanitary behaviours, such as grooming.

Modifying how spaces are used can prevent and contain infectious disease in both humans and animals. Isolation and quarantine are common in human societies, similar to the removal of infectious agents, dead or diseased individuals, in animals. Evacuation of areas where epidemics are spreading rapidly can be a way to contain infectious disease in humans, similar to nest evacuations in animals. Furthermore, animals may use spaces infected with low doses of microbes to gain immunity, similar to vaccination in humans. Modelling how individuals move in different spaces and how these movements influence interactions that may lead to disease transmission, using social network analysis, can improve our understanding of the effects of the built environment on disease transmission in both humans and animals.

In both animals and humans, restructuring the built environment can reduce or prevent disease transmission. However, little is known about whether such changes are, in fact, made. The built structures we discuss can be constructed by the individuals who occupy them, by other individuals from the same species or by other species. Regarding animals, we discuss both the structures that the animals build themselves and structures that humans build for them—for example, in zoos, agriculture and the laboratory. Humans often occupy spaces that others have designed and built for them. Considering who designs and constructs the built environment is fundamental for understanding whether and how it can be modified in response to various conditions, such as chronic and infectious diseases. For example, in humans, many agencies may be involved in permitting the re-modelling of spaces, which may slow down the changes. Whether animals modify the structures they build and occupy in response to disease is still an open question. This open question can be examined in animal systems using experimental manipulations that might not be feasible to conduct in humans, but may inform both human building designs and our understanding of animal behaviours.

The impact of global climate change (GCC) will have a variety of effects on the health of both humans and animals [213–215]. While some of these impacts are predictable, others cannot yet be forecast. As sea levels and temperatures rise, humans will be increasingly faced with drought, floods, natural disasters and consequent relocation and migration to new regions. As temperatures rise, physical activity may be less possible in some places but more viable in others [216]. In addition, the distribution of animal and human disease vectors will likely be affected by rising temperatures. For example, increase in mosquito populations and changes in their spatial distribution may increase rates of malaria and affect unprepared populations [217]. Human migration in response to sea-level rise and natural disasters could further change global disease transmission dynamics. Animal health will also be impacted by GCC, for example, through changes in distribution ranges that will expose animals to new areas with potentially different pathogens or increased temperatures that will increase the range of pathogens and/or their persistence in the environment.

New digital and communication technologies that are increasingly infusing the built environment, such as the 'Internet of Things', virtual reality, mobile communication devices and cloud servers, are becoming indispensable in understanding and monitoring health issues in both humans and animals. For example, individual health tracking devices are increasingly used to gather physiological and psychological data to monitor individuals' general health or specific chronic conditions [218,219]. Various smart sensors are currently used to improve the quality of indoor environments by gathering data on people's comfort needs and behaviour, both at the individual and at the social scales [183,220,221]. These tracking systems will likely be used in the future to develop personalized treatments and can contribute to the investigation of the effects of physical and social environments on health outcomes. Similarly, epidemiological models could benefit from combining information on spatial, social and behavioural factors when modelling disease transmission within human and/or animal populations separately, or in cases of zoonosis outbreaks [222]. Novel technologies like virtual reality are also being explored for their possible application for therapeutic purposes, such as restorative effects of being virtually immersed in natural settings [223]. The cybersphere may have multiple beneficial contributions for understanding the underlying causes of health conditions in humans and non-human animals.

We have discussed many ways in which humans and animals interact through built structures: for example, humans build structures to keep animals in zoos, farms and laboratories; cities have become part of the habitat of many animals and humans modify animal spaces as part of conservation actions. One important interaction between humans and animals that can be mediated by the built environment is the propagation of zoonotic disease. Such interactions have led to zoning of cities, as we detailed at the beginning of the paper. However, zoonotic diseases are still prevalent around the world and present an ongoing public health concern because their emergence is tightly connected to urbanization processes, global travel and trade routes, and changes to ecosystems and biodiversity [178,224]. All these changes have opened new disease transmission pathways between humans, domestic animals and wildlife and are fuelling multi-disciplinary approaches to control and prevent infectious diseases. Therefore, epidemiological models would benefit from insights into the impact of the physical environment on the prevention and containment of both infectious and chronic diseases. For example, understanding the effects of urbanization on human and non-human animal systems can offer new ways to predict the emergence of novel diseases and new methods to control endemic zoonoses in developing countries and unplanned fast-growing city areas. Interestingly, the implementation of health-promoting design strategies to reduce chronic disease in humans, such as providing green spaces in cities, has led to an increase in biodiversity and in controlling potential zoonotic diseases. One way to examine the links between human and animal activities and how they affect disease dynamics has been through the development of human and animal health information systems, which collect spatial disease data at regional and national scales [225]. Further work is needed to develop models of disease epidemics, especially for crossing boundaries between human and animals and across geographical space. Such models will require information on zoonotic disease, host and vector-borne transmission, and movement patterns of both humans and animals. Thus, in-depth understanding of how interaction patterns depend on the built environment would greatly contribute to developing spatial models of infectious disease transmission through social mixing networks, both within and across species.

Overall, infectious and chronic diseases in humans and non-human animals need to be understood as a worldwide public health concern, given the largely anthropogenic drivers such as the built environment behind many diseases. Accordingly, securing the health and well-being of all living social systems requires holistic and mutually informed understanding and development of prevention strategies at local, regional and global levels.

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References

1. Schrank S, Ekici D. 2016 *Healing spaces, modern architecture, and the body*. Oxford, UK: Routledge.
2. Borasi G, Zardini M. 2012 *Imperfect health: the medicalization of architecture*. Montreal, Canada: Canadian Centre for Architecture.
3. Sloane DC. 2006 From congestion to sprawl—planning and health in historical context. *J. Am. Plann. Assoc.* **72**, 10–18. (doi:10.1080/01944360608976720)
4. Frumkin H, Frank LD, Jackson R. 2004 *Urban sprawl and public health: designing, planning, and building for healthy communities*. Washington, DC: Island Press.
5. Hamlin C, Sheard S. 1998 Revolutions in public health: 1848, and 1998? *Br. Med. J.* **317**, 587–591. (doi:10.1136/bmj.317.7158.587)
6. Campbell M. 2012 Strange bedfellows: modernism and tuberculosis. In *Imperfect health: the medicalization of architecture* (eds G Borasi, M Zardini), pp. 133–151. Montreal, Canada: Canadian Centre for Architecture.
7. Murphy M. 2015 In search of the water pump: architecture and cholera. *Harvard Des. Mag.* **40**, 148–153.
8. Hill JO, Peters JC, Wyatt HR. 2009 Using the energy gap to address obesity: a commentary. *J. Am. Diet Assoc.* **109**, 1848–1853. (doi:10.1016/j.jada.2009.08.007)
9. Cooper R, Boyko CT, Cooper C. 2011 Design for health: the relationship between design and noncommunicable diseases. *J. Health Commun.* **16**, 134–157. (doi:10.1080/10810730.2011.601396)
10. Gruebner O, Rapp MA, Adli M, Kluge U, Galea S, Heinz A. 2017 Cities and mental health. *Deutsches Arzteblatt Inter.* **114**, 121–127. (doi:10.3238/arztebl.2017.0121)
11. Kim D. 2008 Blues from the neighborhood? Neighborhood characteristics and depression. *Epidemiol Rev.* **30**, 101–117. (doi:10.1093/epirev/mxn009)
12. Omran AR. 1971 The epidemiologic transition: a theory of the epidemiology of population change. *Milbank Mem. Fund Q.* **49**, 509–538. (doi:10.2307/3349375)
13. Gimmler A, Lenk C, Aumüller G. 2002 *Health and quality of life: philosophical, medical, and cultural aspects*. Münster, Germany: Lit.
14. Steemers K. 2015 Architecture for well-being and health. *Daylight Architecture* **Spring 2015**, Issue 23, pp. 6–27.
15. Tsekleves E, Cooper R. 2017 *Design for health*. Oxford, UK: Routledge.
16. Pie MR, Rosengaus RB, Traniello JFA. 2004 Nest architecture, activity pattern, worker density and the dynamics of disease transmission in social insects. *J. Theor. Biol.* **226**, 45–51. (doi:10.1016/j.jtbi.2003.08.002)
17. Cervero R, Kockelman K. 1997 Travel demand and the 3Ds: density, diversity, and design. *Transport Res. D. Transp. Environ.* **2**, 199–219. (doi:10.1016/S1361-9209(97)00009-6)
18. City of New York. 2010. *Active design guidelines: promoting physical activity and health in design*. New York, NY. See https://centerforactivedesign.org/dl/guidelines.pdf.
19. Kim J, Kaplan R. 2004 Physical and psychological factors in sense of community—new urbanist Kentlands and nearby orchard village. *Environ. Behav.* **36**, 313–340. (doi:10.1177/0013916503260236)
20. Wells NM, Ashdown SP, Davies EHS, Cowett FD, Yang YZ. 2007 Environment, design and obesity—opportunities for interdisciplinary collaborative research. *Environ. Behav.* **39**, 6–33. (doi:10.1177/0013916506295570)
21. Kerr NA, Yore MM, Ham SA, Dietz WH. 2004 Increasing stair use in a worksite through environmental changes. *Am. J. Health Promot.* **18**, 312–315. (doi:10.4278/0890-1171-18.4.312)
22. van Rensselaer M. 1901 *Saving steps (Cornell reading course for farmers' wives, supplement No. 1)*. Ithaca, NY: Cornell University, College of Agriculture.
23. Becker FD. 1990 *The total workplace: facilities management and the elastic organization*. New York, NY: Van Nostrand Reinhold.
24. Neuhaus M, Eakin EG, Straker L, Owen N, Dunstan DW, Reid N, Healy GN. 2014 Reducing occupational sedentary time: a systematic review and meta-analysis of evidence on activity-permissive workstations. *Obes. Rev.* **15**, 822–838. (doi:10.1111/obr.12201)
25. Brittin J, Sorensen D, Trowbridge M, Lee KK, Breithecker D, Frerichs L, Huang T. 2015 Physical activity design guidelines for school architecture. *PLoS ONE* **10**, e0132597. (doi:10.1371/journal.pone.0132597)
26. Rietveld E, Rietveld R, Mackic A, Waalwijk Van Doorn E, Bervoets B. 2015 The end of sitting. *Harvard Des. Mag.* **40**, 180–181.
27. Thaler RH, Sunstein CR. 2008 *Nudge: improving decisions about health, wealth, and happiness*. New Haven, CT: Yale University Press.
28. Zimmerman FJ. 2009 Using behavioral economics to promote physical activity. *Prev. Med.* **49**, 289–291. (doi:10.1016/j.ypmed.2009.07.008)
29. Rundle A, Neckerman KM, Freeman L, Lovasi GS, Purciel M, Quinn J, Richards C, Sircar N, Weiss C. 2009 Neighborhood food environment and walkability predict obesity in New York City. *Environ. Health Perspect.* **117**, 442–447. (doi:10.1289/ehp.11590)
30. Bodor JN, Rice JC, Farley TA, Swalm CM, Rose D. 2010 The association between obesity and urban food environments. *J. Urban Health* **87**, 771–781. (doi:10.1007/s11524-010-9460-6)
31. Edmonds J, Baranowski T, Baranowski J, Cullen KW, Myres D. 2001 Ecological and socioeconomic correlates of fruit, juice, and vegetable consumption among African-American boys. *Prev. Med.* **32**, 476–481. (doi:10.1006/pmed.2001.0831)
32. Wrigley N, Warm D, Margetts B. 2003 Deprivation, diet, and food-retail access: findings from the Leeds 'food deserts' study. *Environ. Plann. A* **35**, 151–188. (doi:10.1068/a35150)
33. Cheadle A, Psaty BM, Curry S, Wagner E, Diehr P, Koepsell T, Kristal A. 1991 Community-level comparisons between the grocery store environment and individual dietary practices. *Prev. Med.* **20**, 250–261. (doi:10.1016/0091-7435(91)90024-X)
34. Rollings KR, Wells NM. 2017 Effects of residential kitchen floor plan openness on eating behaviors. *Environ. Behav.* **49**, 663–684. (doi:10.1177/0013916516661822)
35. Rolls BJ, Morris EL, Roe LS. 2002 Portion size of food affects energy intake in normal-weight and overweight men and women. *Am. J. Clin. Nutr.* **76**, 1207–1213. (doi:10.1093/ajcn/76.6.1207)
36. Wansink B. 2004 Environmental factors that unknowingly increase food intake and consumption volume of unknowing consumers. *Annu. Rev. Nutr.* **24**, 455–479. (doi:10.1146/annurev.nutr.24.012003.132140)
37. Wansink B, Painter JE, North J. 2005 Bottomless bowls: why visual cues of portion size may influence intake. *Obes. Res.* **13**, 93–100. (doi:10.1038/oby.2005.12)
38. Rolls BJ. 2003 The supersizing of America: portion size and the obesity epidemic. *Nutr. Today* **38**, 42–53. (doi:10.1097/00017285-200303000-00004)
39. Wansink B. 2006 *Mindless eating: why we eat more than we think*. New York, NY: Bantam Books.
40. Clark C, Myron R, Stansfeld S, Candy B. 2007 A systematic review of the evidence on the effect of the built and physical environment on mental health. *J. Public Mental Health* **6**, 14–27. (doi:10.1108/17465729200700011)
41. Gong Y, Palmer S, Gallacher J, Marsden T, Fone D. 2016 A systematic review of the relationship between objective measurements of the urban environment and psychological distress. *Environ. Int.* **96**, 48–57. (doi:10.1016/j.envint.2016.08.019)

42. Tost H, Champagne FA, Meyer-Lindenberg A. 2015 Environmental influence in the brain, human welfare and mental health. *Nat. Neurosci.* **18**, 1421–1431. (doi:10.1038/nn.4108)
43. Knöll M, Neuheuser K, Cleff T, Rudolph-Cleff A. 2017 A tool to predict perceived urban stress in open public spaces. *Environ. Plann. B: Urban Anal. City Sci.* **38**, 026581351668697. (doi:10.1177/0265813516686971)
44. Corburn J. 2015 City planning as preventive medicine. *Prev. Med.* **77**, 48–51. (doi:10.1016/j.ypmed.2015.04.022)
45. Cooper R, Burton E, Cooper C. 2014 *Wellbeing and the environment*. Chichester, UK: John Wiley & Sons, Ltd.
46. Evans GW. 2003 The built environment and mental health. *J. Urban Health Bull. N. Y. Acad. Med.* **80**, 536–555. (doi:10.1093/jurban/jtg063)
47. Aked J, Marks N, Cordon C, Thompson S. 2008 *Five ways to well-being: the evidence*. London, UK: New Economics Foundation.
48. Sarkar C, Webster C. 2017 Healthy cities of tomorrow: the case for large scale built environment–health studies. *J. Urban Health* **94**, 4–19. (doi:10.1007/s11524-016-0122-1)
49. Bates C, Imrie R, Kullman K. 2016 Configuring the caring city: ownership, healing, openness. In *Care and design: bodies, buildings, cities* (eds C Bates, R Imrie, K Kullman), pp. 95–115. Chichester, UK: John Wiley & Sons.
50. Gehl J, Koch J. 1987 *Life between buildings: using public space*. New York, NY: Van Nostrand Reinhold Company.
51. Barnfield A. 2016 Affect and public health—choreographing atmospheres of movement and participation. *Emotion Space Soc.* **20**, 1–9. (doi:10.1016/j.emospa.2016.04.003)
52. Boyko CT, Cooper R. 2014 Density and mental wellbeing. In *Wellbeing: a complete reference guide: wellbeing and the environment* (eds R Cooper, E Burton, C Cooper), pp. 69–90. Chichester, UK: John Wiley & Sons.
53. Brown SC, Lombard J. 2014 Neighborhoods and social interaction. In *Wellbeing and the environment* (eds R Cooper, E Burton, C Cooper), pp. 91–118. Chichester, UK: John Wiley & Sons.
54. Ettema D, Smajic I. 2015 Walking, places and wellbeing. *Geogr. J.* **181**, 102–109. (doi:10.1111/geoj.12065)
55. Melis G, Gelormino E, Marra G, Ferracin E, Costa G. 2015 The effects of the urban built environment on mental health: a cohort study in a large northern Italian city. *Int. J. Environ. Res. Public Health* **12**, 14898. (doi:10.3390/ijerph121114898)
56. Salvo D, Banda JA, Sheats JL, Winter SJ, Santos DLD, King AC. 2017 Impacts of a temporary urban pop-up park on physical activity and other individual- and community-level outcomes. *J. Urban Health* **94**, 470–481. (doi:10.1007/s11524-017-0167-9)
57. Evans GW, Wells NM, Moch A. 2003 Housing and mental health: a review of the evidence and a methodological and conceptual critique. *J. Soc. Issues* **59**, 475–500. (doi:10.1111/1540-4560.00074)
58. Wells NM, Harris JD. 2007 Housing quality, psychological distress, and the mediating role of social withdrawal: a longitudinal study of low-income women. *J. Environ. Psychol.* **27**, 69–78. (doi:10.1016/j.jenvp.2006.11.002)
59. Evans GW, Lepore SJ, Schroeder A. 1996 The role of interior design elements in human responses to crowding. *J. Pers. Soc. Psychol.* **70**, 41–46. (doi:10.1037/0022-3514.70.1.41)
60. Hillier B, Hanson J. 1984 *The social logic of space*. Cambridge, NY: Cambridge University Press.
61. Dean A, Kolody B, Wood P, Matt GE. 1992 The influence of living alone on depression in elderly persons. *J. Aging Health* **4**, 3–18. (doi:10.1177/089826439200400101)
62. Umberson D, Montez JK. 2010 Social relationships and health: a flashpoint for health policy. *J. Health Soc. Behav.* **51**, S54–S66. (doi:10.1177/0022146510383501)
63. Cornwell EY, Waite LJ. 2009 Social disconnectedness, perceived isolation, and health among older adults. *J. Health Soc. Behav.* **50**, 31–48. (doi:10.1177/002214650905000103)
64. Stokols D. 1996 Translating social ecological theory into guidelines for community health promotion. *Am. J. Health Promot.* **10**, 282–298. (doi:10.4278/0890-1171-10.4.282)
65. Fich LB, Jönsson P, Kirkegaard PH, Wallergård M, Garde AH, Hansen Å. 2014 Can architectural design alter the physiological reaction to psychosocial stress? A virtual TSST experiment. *Physiol. Behav.* **135**, 91–97. (doi:10.1016/j.physbeh.2014.05.034)
66. Wey TW, Lin L, Patton ML, Blumstein DT. 2015 Stress hormone metabolites predict overwinter survival in yellow-bellied marmots. *Acta Ethol.* **18**, 181–185. (doi:10.1007/s10211-014-0204-6)
67. Molnar B, Fattebert J, Palme R, Ciucci P, Betschart B, Smith DW, Diehl PA. 2015 Environmental and intrinsic correlates of stress in free-ranging wolves. *PLoS ONE* **10**, ARTN e0137378. (doi:10.1371/journal.pone.0137378)
68. Carlstead K, Brown JL, Seidensticker J. 1993 Behavioral and adrenocortical responses to environmental changes in leopard cats (*Felis bengalensis*). *Zoo. Biol.* **12**, 321–331. (doi:10.1002/zoo.1430120403)
69. Vaz J, Narayan EJ, Kumar RD, Thenmozhi K, Thiyagesan K, Baskaran N. 2017 Prevalence and determinants of stereotypic behaviours and physiological stress among tigers and leopards in Indian zoos. *PLoS ONE* **12**, e0174711. (doi:10.1371/journal.pone.0174711)
70. VanDierendonck MC, Spruijt BM. 2012 Coping in groups of domestic horses—review from a social and neurobiological perspective. *Appl. Anim. Behav. Sci.* **138**, 194–202. (doi:10.1016/j.applanim.2012.02.007)
71. Keverne EB, Martensz ND, Tuite B. 1989 Beta-endorphin concentrations in cerebrospinal fluid of monkeys are influenced by grooming relationships. *Psychoneuroendocrino* **14**, 155–161. (doi:10.1016/0306-4530(89)90065-6)
72. Curley JP, Keverne EB. 2005 Genes, brains and mammalian social bonds. *Trends Ecol. Evol.* **20**, 561–567. (doi:10.1016/j.tree.2005.05.018)
73. Hannibal DL, Bliss-Moreau E, Vandeleest J, McCowan B, Capitanio J. 2017 Laboratory rhesus macaque social housing and social changes: implications for research. *Am. J. Primatol.* **79**, e22528. (doi:10.1002/ajp.22528)
74. Garner JP. 2005 Stereotypies and other abnormal repetitive behaviors: potential impact on validity, reliability, and replicability of scientific outcomes. *ILAR J.* **46**, 106–117. (doi:10.1093/ilar.46.2.106)
75. deJonge FH, Bokkers EAM, Schouten WGP, Helmond FA. 1996 Rearing piglets in a poor environment: developmental aspects of social stress in pigs. *Physiol. Behav.* **60**, 389–396. (doi:10.1016/S0031-9384(96)80009-6)
76. Watson H, Videvall E, Andersson MN, Isaksson C. 2017 Transcriptome analysis of a wild bird reveals physiological responses to the urban environment. *Sci. Rep.* **7**, 44180. (doi:10.1038/srep44180)
77. Tschinkel WR. 1999 Sociometry and sociogenesis of colonies of the harvester ant, *Pogonomyrmex badius*: distribution of workers, brood and seeds within the nest in relation to colony size and season. *Ecol. Entomol.* **24**, 222–237. (doi:10.1046/j.1365-2311.1999.00184.x)
78. Seeley TD, Morse RA. 1976 The nest of the honey bee (*Apis mellifera* L.). *Insectes Soc.* **23**, 495–512. (doi:10.1007/bf02223477)
79. DeGrandi-Hoffman G, Chen YP, Rivera R, Carroll M, Chambers M, Hidalgo G, de Jong EW. 2016 Honey bee colonies provided with natural forage have lower pathogen loads and higher overwinter survival than those fed protein supplements. *Apidologie* **47**, 186–196. (doi:10.1007/s13592-015-0386-6)
80. Hansell MH. 2007 *Built by animals: the natural history of animal architecture*. Oxford, NY: Oxford University Press.
81. Ferguson SH, Taylor MK, Rosing-Asvid A, Born EW, Messier F. 2000 Relationships between denning of polar bears and conditions of sea ice. *J. Mammal.* **81**, 1118–1127. (doi:10.1644/1545-1542(2000)081<1118:RBDOPB>2.0.CO;2)
82. Gittleman JL, Thompson SD. 1988 Energy allocation in mammalian reproduction. *Am. Zool.* **28**, 863–875. (doi:10.1093/icb/28.3.863)
83. Giraldo YM, Traniello JFA. 2014 Worker senescence and the sociobiology of aging in ants. *Behav. Ecol. Sociobiol.* **68**, 1901–1919. (doi:10.1007/s00265-014-1826-4)
84. Grozinger CM, Fan YL, Hoover SER, Winston ML. 2007 Genome-wide analysis reveals differences in brain gene expression patterns associated with caste and reproductive status in honey bees (*Apis mellifera*). *Mol. Ecol.* **16**, 4837–4848. (doi:10.1111/j.1365-294X.2007.03545.x)
85. Bonasio R *et al.* 2010 Genomic comparison of the ants *Camponotus floridanus* and *Harpegnathos*

saltator. *Science* **329**, 1068–1071. (doi:10.1126/science.1192428).

86. Wurm Y *et al.* 2011 The genome of the fire ant *Solenopsis invicta*. *Proc. Natl Acad. Sci. USA* **108**, 5679–5684. (doi:10.1073/pnas.1009690108)
87. Tidiere M, Gaillard JM, Berger V, Muller DWH, Lackey LB, Gimenez O, Clauss M, Lemaitre JF. 2016 Comparative analyses of longevity and senescence reveal variable survival benefits of living in zoos across mammals. *Sci. Rep.* **6**, 36361. (doi:10.1038/srep36361)
88. Mitchell L. 2014 A step too far? Designing dementia-friendly neighborhoods. In *Wellbeing and the environment* (eds R Cooper, E Burton, C Cooper), pp. 185–218. Chichester, UK: John Wiley & Sons.
89. Ho H, Lau K, Yu R, Wang D, Woo J, Kwok T, Ng E. 2017 Spatial variability of geriatric depression risk in a high-density city: a data-driven socio-environmental vulnerability mapping approach. *Int. J. Environ. Res. Public Health* **14**, 994. (doi:10.3390/ijerph14090994)
90. Chaudhury H, Campo M, Michael Y, Mahmood A. 2016 Neighbourhood environment and physical activity in older adults. *Soc. Sci. Med.* **149**, 104–113. (doi:10.1016/j.socscimed.2015.12.011)
91. Mooney SJ, Joshi S, Cerdá M, Kennedy GJ, Beard JR, Rundle AG. 2017 Neighborhood disorder and physical activity among older adults: a longitudinal study. *J. Urban Health* **94**, 30–42. (doi:10.1007/s11524-016-0125-y)
92. Borasi G, Zardini M. 2012 Demedicalize architecture. In *Imperfect health: the medicalization of architecture* (eds G Borasi, M Zardini), pp. 15–37. Montreal, Canada: Canadian Centre for Architecture.
93. Martin D. 2016 Curating space, choreographing care: the efficacy of the everyday. In *Care and design: bodies, buidings, cities* (eds C Bates, R Imrie, K Kullman), pp. 37–55. Chichester, UK: John Wiley & Sons.
94. Kellert SR. 2008 Dimensions, elements, and attributes of biophilic design. In *Biophilic design: the theory, science, and practice of bringing buildings to life* (eds SR Kellert, J Heerwagen, M Mador), pp. 3–19. Hoboken, NJ: John Wiley & Sons.
95. Frumkin P. 2001 Beyond toxicity—human health and the natural environment. *Am. J. Prev. Med.* **20**, 234–240. (doi:10.1016/S0749-3797(00)00317-2)
96. Wells NM, Phalen KB. In press. Everyday and nearby natural environments. In *The environment and human behavior: effects of built and natural settings on well-being* (ed. AS Devlin). New York, NY: Elsevier.
97. Park BJ, Tsunetsugu Y, Ishii H, Furuhashi S, Hirano H, Kagawa T, Miyazaki Y. 2008 Physiological effects of Shinrin-yoku (taking in the atmosphere of the forest) in a mixed forest in Shinano Town, Japan. *Scand J. Forest Res.* **23**, 278–283. (doi:10.1080/02827580802055978)
98. Cimprich B. 1992 Attentional fatigue following breast cancer surgery. *Res. Nurs. Health* **15**, 199–207. (doi:10.1002/nur.4770150306)
99. Cimprich B. 1993 Development of an intervention to restore attention in cancer patients. *Cancer Nurs.* **16**, 83–92. (doi:10.1097/00002820-199304000-00001)
100. Kaplan S. 1995 The restorative benefits of nature—toward an integrative framework. *J. Environ. Psychol.* **15**, 169–182. (doi:10.1016/0272-4944(95)90001-2)
101. Ryan CO, Browning WD, Clancy JO, Andrews SL, Kallianpurkar NB. 2014 Biophilic design patterns: emerging nature-based parameters for health and well-being in the built environment. *J. Archit. Res.* **8**, 62–76. (doi:10.26687/archnet-ijar.v8i2.436)
102. Woolf SH, Aron L, Dubay L, Simon S, Zimmerman E, Luk K. 2015 How are income and wealth linked to health and longevity? Retrieved from http://webarchive.urban.org/publications/2000178.html
103. Morland K, Wing S, Roux AD, Poole C. 2002 Neighborhood characteristics associated with the location of food stores and food service places. *Am. J. Prev. Med.* **22**, 23–29. (doi:10.1016/S0749-3797(01)00403-2)
104. Powell LM, Slater S, Mirtcheva D, Bao YJ, Chaloupka FJ. 2007 Food store availability and neighborhood characteristics in the United States. *Prev. Med.* **44**, 189–195. (doi:10.1016/j.ypmed.2006.08.008)
105. Walker RE, Keane CR, Burke JG. 2010 Disparities and access to healthy food in the United States: a review of food deserts literature. *Health Place* **16**, 876–884. (doi:10.1016/j.healthplace.2010.04.013)
106. Evans GW, Kantrowitz E. 2002 Socioeconomic status and health: the potential role of environmental risk exposure. *Annu. Rev. Publ. Health* **23**, 303–331. (doi:10.1146/annurev.publhealth.23.112001.112349)
107. Strife S, Downey L. 2009 Childhood development and access to nature: a new direction for environmental inequality research. *Organ. Environ.* **22**, 99–122. (doi:10.1177/1086026609333340)
108. Suecoff SA, Avner JR, Chou KJ, Crain EF. 1999 A comparison of New York City playground hazards in high- and low-income areas. *Arch. Pediatr. Adolesc. Med.* **153**, 363–366. (doi:10.1001/archpedi.153.4.363)
109. Penick CA, Tschinkel WR. 2008 Thermoregulatory brood transport in the fire ant, *Solenopsis invicta*. *Insectes Soc.* **55**, 176–182. (doi:10.1007/s00040-008-0987-4)
110. Bollazzi M, Roces E. 2002 Thermal preference for fungus culturing and brood location by workers of the thatching grass-cutting ant *Acromyrmex heyeri*. *Insectes Soc.* **49**, 153–157. (doi:10.1007/s00040-002-8295-x)
111. Porter SD, Tschinkel WR. 1993 Fire ant thermal preferences—behavioral control of growth and metabolism. *Behav. Ecol. Sociobiol.* **32**, 321–329. (doi:10.1007/BF00183787)
112. Kaufmann JH. 1983 On the definitions and functions of dominance and territoriality. *Biol. Rev. Camb. Philos. Soc.* **58**, 1–20. (doi:10.1111/j.1469-185X.1983.tb00379.x)
113. Maher CR, Lott DF. 2000 A review of ecological determinants of territoriality within vertebrate species. *Am. Midl. Nat.* **143**, 1–29. (doi:10.1674/0003-0031(2000)143[0001:AROEDO]2.0.CO;2)
114. Armitage KB. 1977 Social variety in yellow-bellied marmot—population-behavioral system. *Anim. Behav.* **25**, 585–593. (doi:10.1016/0003-3472(77)90108-7)
115. Blumstein DT, Keeley KN, Smith JE. 2016 Fitness and hormonal correlates of social and ecological stressors of female yellow-bellied marmots. *Anim. Behav.* **112**, 1–11. (doi:10.1016/j.anbehav.2015.11.002)
116. Pinter-Wollman N, Brown MJF. 2015 Variation in nest relocation of harvester ants is affected by population density and food abundance. *Behav. Ecol.* **26**, 1569–1576. (doi:10.1093/beheco/arv108)
117. Alee WC. 1931 *Animal aggregations, a study in general sociology*. Chicago, IL: University of Chicago Press.
118. Stephens PA, Sutherland WJ, Freckleton RP. 1999 What is the Allee effect? *Oikos* **87**, 185–190. (doi:10.2307/3547011)
119. Colomina, B. 2015 X-ray architecture: the tuberculosis effect. *Harvard Des. Mag.* **40**, 70–91.
120. Bluyssen PM. 2009 *The indoor environment handbook: how to make buildings healthy and comfortable*. Sterling, VA: Earthscan.
121. Eames I, Tang JW, Li Y, Wilson P. 2009 Airborne transmission of disease in hospitals. *J. R. Soc. Interface* **6**, S697–S702. (doi:10.1098/rsif.2009.0407.focus)
122. McKinney KR, Gong YY, Lewis TG. 2006 Environmental transmission of SARS at amoy gardens. *J. Environ. Health Denver.* **68**, 2.
123. Sample H. 2012 Emergency urbanism and preventive architecture. In *Imperfect health: the medicalization of architecture* (eds G Borasi, M Zardini), pp. 231–250. Montreal, Canada: Canadian Centre for Architecture.
124. Vert C *et al.* 2017 Effect of long-term exposure to air pollution on anxiety and depression in adults: a cross-sectional study. *Int. J. Hyg. Environ. Health* **220**, 1074–1080. (doi:10.1016/j.ijheh.2017.06.009)
125. Centers for Disease Control and Prevention 2018 CDC at Work: Preventing healthcare-associated infections. See https://www.cdc.gov/washington/~cdcatwork/pdf/infections.pdf (Retrieved 18 May 2018.)
126. Birnbach DJ, Nevo I, Scheinman SR, Fitzpatrick M, Shekhter I, Lombard JL. 2010 Patient safety begins with proper planning: a quantitative method to improve hospital design. *Qual. Saf. Health Care* **19**, 462–465. (doi:10.1136/qshc.2008.031013)
127. Hart BL. 2005 The evolution of herbal medicine: behavioural perspectives. *Anim. Behav.* **70**, 975–989. (doi:10.1016/j.anbehav.2005.03.005)
128. Chapuisat M, Oppliger A, Magliano P, Christe P. 2007 Wood ants use resin to protect themselves against pathogens. *Proc. R. Soc. B* **274**, 2013–2017. (doi:10.1098/rspb.2007.0531)
129. Simone M, Evans JD, Spivak M. 2009 Resin collection and social immunity in honey bees. *Evol. Int. J. Org. Evol.* **63**, 3016–3022. (doi:10.1111/j.1558-5646.2009.00772.x)
130. Clark L, Mason JR. 1985 Use of nest material as insecticidal and anti-pathogenic agents by the

European Starling. *Oecologia* **67**, 169–176. (doi:10.1007/BF00384280)

131. Lafuma L, Lambrechts MM, Raymond M. 2001 Aromatic plants in bird nests as a protection against blood-sucking flying insects? *Behav. Process.* **56**, 113–120. (doi:10.1016/S0376-6357(01)00191-7)
132. Petit C, Hossaert-McKey M, Perret P, Blondel J, Lambrechts MM. 2002 Blue tits use selected plants and olfaction to maintain an aromatic environment for nestlings. *Ecol. Lett.* **5**, 585–589. (doi:10.1046/j.1461-0248.2002.00361.x)
133. Scott-Baumann JF, Morgan ER. 2015 A review of the nest protection hypothesis: does inclusion of fresh green plant material in birds' nests reduce parasite infestation? *Parasitology* **142**, 1016–1023. (doi:10.1017/S0031182015000189)
134. Hemmes RB, Alvarado A, Hart BL. 2002 Use of California bay foliage by wood rats for possible fumigation of nest-borne ectoparasites. *Behav. Ecol.* **13**, 381–385. (doi:10.1093/beheco/13.3.381)
135. Rosengaus RB, Guldin MR, Traniello JFA. 1998 Inhibitory effect of termite fecal pellets on fungal spore germination. *J. Chem. Ecol.* **24**, 1697–1706. (doi:10.1023/A:1020872729671)
136. Tranter C, Graystock P, Shaw C, Lopes JFS, Hughes WOH. 2014 Sanitizing the fortress: protection of ant brood and nest material by worker antibiotics. *Behav. Ecol. Sociobiol.* **68**, 499–507. (doi:10.1007/s00265-013-1664-9)
137. Rosengaus RB, Moustakas JE, Calleri DV, Traniello JFA. 2003 Nesting ecology and cuticular microbial loads in dampwood (*Zootermopsis angusticollis*) and drywood termites (*Incisitermes minor, I. schwarzi, Cryptotermes cavifrons*). *J. Insect Sci.* **3**, 31. (doi:10.1093/jis/3.1.31)
138. Diez L, Deneubourg J-L, Detrain C. 2012 Social prophylaxis through distant corpse removal in ants. *Naturwissenschaften* **99**, 833–842. (doi:10.1007/s00114-012-0965-6)
139. Diez L, Le Borgne H, Lejeune P, Detrain C. 2013 Who brings out the dead? Necrophoresis in the red ant, *Myrmica rubra*. *Anim. Behav.* **86**, 1259–1264. (doi:10.1016/j.anbehav.2013.09.030)
140. Trumbo ST, Huang ZY, Robinson GE. 1997 Division of labor between undertaker specialists and other middle-aged workers in honey bee colonies. *Behav. Ecol. Sociobiol.* **41**, 151–163. (doi:10.1007/s002650050374)
141. Diez L, Lejeune P, Detrain C. 2014 Keep the nest clean: survival advantages of corpse removal in ants. *Biol. Lett.* **10**, 20140306. (doi:10.1098/rsbl.2014.0306)
142. Richard FJ, Aubert A, Grozinger CM. 2008 Modulation of social interactions by immune stimulation in honey bee, *Apis mellifera*, workers. *BMC Biol.* **6**, 50. (doi:10.1186/1741-7007-6-50)
143. Leclerc JB, Detrain C. 2016 Ants detect but do not discriminate diseased workers within their nest. *Sci. Nat.* **103**, 70. (doi:10.1007/s00114-016-1394-8)
144. Bos N, Lefevre T, Jensen AB, d'Ettorre P. 2012 Sick ants become unsociable. *J. Evol. Biol.* **25**, 342–351. (doi:10.1111/j.1420-9101.2011.02425.x)
145. Lapidge KL, Oldroyd BP, Spivak M. 2002 Seven suggestive quantitative trait loci influence hygienic behavior of honey bees. *Naturwissenschaften* **89**, 565–568. (doi:10.1007/s00114-002-0371-6)
146. Spivak M, Masterman R, Ross R, Mesce KA. 2003 Hygienic behavior in the honey bee (*Apis mellifera* L.) and the modulatory role of octopamine. *J. Neurobiol.* **55**, 341–354. (doi:10.1002/neu.10219)
147. Hart AG, Ratnieks FLW. 2001 Task partitioning, division of labour and nest compartmentalisation collectively isolate hazardous waste in the leafcutting ant *Atta cephalotes*. *Behav. Ecol. Sociobiol.* **49**, 387–392. (doi:10.1007/s002650000312)
148. Ballari S, Farji-Brener AG, Tadey M. 2007 Waste management in the leaf-cutting ant *Acromyrmex lobicornis*: division of labour, aggressive behaviour, and location of external refuse dumps. *J. Insect Behav.* **20**, 87–98. (doi:10.1007/s10905-006-9065-9)
149. Gordon DM, Chu J, Lillie A, Tissot M, Pinter N. 2005 Variation in the transition from inside to outside work in the red harvester ant *Pogonomyrmex barbatus*. *Insectes Soc.* **52**, 212–217. (doi:10.1007/s00040-004-0796-3)
150. Farji-Brener AG, Elizalde L, Fernandez-Marin H, Amador-Vargas S. 2016 Social life and sanitary risks: evolutionary and current ecological conditions determine waste management in leaf-cutting ants. *Proc. R. Soc. B* **283**, 20160625. (doi:10.1098/rspb.2016.0625)
151. Sato Y, Saito Y, Sakagami T. 2003 Rules for nest sanitation in a social spider mite, *Schizotetranychus miscanthi* Saito (Acari: Tetranychidae). *Ethology* **109**, 713–724. (doi:10.1046/j.1439-0310.2003.00905.x)
152. Czaczkes TJ, Heinze J, Ruther J. 2015 Nest etiquette—where ants go when nature calls. *PLoS ONE* **10**, e0118376. (doi:10.1371/journal.pone.0118376)
153. Gow EA, Wiebe KL, Musgrove A. 2015 Nest sanitation in response to short- and long-term changes of brood size: males clean more in a sex-role-reversed species. *Anim. Behav.* **104**, 137–143. (doi:10.1016/j.anbehav.2015.03.014)
154. Rostain RR, Ben-David M, Groves P, Randall JA. 2004 Why do river otters scent-mark? An experimental test of several hypotheses. *Anim. Behav.* **68**, 703–711. (doi:10.1016/j.anbehav.2003.10.027)
155. Green M, Monick K, Manjerovic MB, Novakofski J, Mateus-Pinilla N. 2015 Communication stations: cameras reveal river otter (*Lontra canadensis*) behavior and activity patterns at latrines. *J. Ethol.* **33**, 225–234. (doi:10.1007/s10164-015-0435-7)
156. Irwin MT, Samonds KE, Raharison JL, Wright PC. 2004 Lemur latrines: observations of latrine behavior in wild primates and possible ecological significance. *J. Mammal.* **85**, 420–427. (doi:10.1644/1545-1542(2004)085<0420:LLOOLB>2.0.CO;2)
157. Roper TJ, Shepherdson DJ, Davies JM. 1986 Scent marking with feces and anal secretion in the European badger (*Meles meles*)—seasonal and spatial characteristics of latrine use in relation to territoriality. *Behaviour* **97**, 94–117. (doi:10.1163/156853986X00333)
158. Freeman EW, Meyer JM, Adendorff J, Schulte BA, Santymire RM. 2014 Scraping behavior of black rhinoceros is related to age and fecal gonadal metabolite concentrations. *J. Mammal.* **95**, 340–348. (doi:10.1644/13-MAMM-A-059)
159. Thompson SR, Brandenburg RL. 2005 Tunneling responses of mole crickets (Orthoptera: Gryllotalpidae) to the entomopathogenic fungus, *Beauveria bassiana*. *Environ. Entomol.* **34**, 140–147. (doi:10.1603/0046-225X-34.1.140)
160. Godfrey SS, Bull CM, James R, Murray K. 2009 Network structure and parasite transmission in a group living lizard, the Gidgee skink, *Egernia stokesii*. *Behav. Ecol. Sociobiol.* **63**, 1045–1056. (doi:10.1007/s00265-009-0730-9)
161. Godfrey SS, Nelson NJ, Bull CM. 2011 Microhabitat choice and host-seeking behaviour of the tuatara tick, *Amblyomma sphenodonti* (Acari: Ixodidae). *New Zeal J. Ecol.* **35**, 52–60.
162. Fenner AL, Godfrey SS, Bull CM. 2011 Using social networks to deduce whether residents or dispersers spread parasites in a lizard population. *J. Anim. Ecol.* **80**, 835–843. (doi:10.1111/j.1365-2656.2011.01825.x)
163. Oppliger A, Richner H, Christe P. 1994 Effect of an ectoparasite on lay date, nest-site choice, desertion, and hatching success in the Great Tit (*Parus major*). *Behav. Ecol.* **5**, 130–134. (doi:10.1093/beheco/5.2.130)
164. Karlik J, Epps MJ, Dunn RR, Penick CA. 2016 Life inside an acorn: how microclimate and microbes influence nest organization in *Temnothorax* ants. *Ethology* **122**, 790–797. (doi:10.1111/eth.12525)
165. Franks NR, Hooper J, Webb C, Dornhaus A. 2005 Tomb evaders: house-hunting hygiene in ants. *Biol. Lett.* **1**, 190–192. (doi:10.1098/rsbl.2005.0302)
166. Brutsch T, Felden A, Reber A, Chapuisat M. 2014 Ant queens (Hymenoptera: Formicidae) are attracted to fungal pathogens during the initial stage of colony founding. *Myrmecol. News* **20**, 71–76.
167. Pontieri L, Vojvodic S, Graham R, Pedersen JS, Linksvayer TA. 2014 Ant colonies prefer infected over uninfected nest sites. *PLoS ONE* **9**, e111961. (doi:10.1371/journal.pone.0111961)
168. Konrad M *et al.* 2012 Social transfer of pathogenic fungus promotes active immunisation in ant colonies. *PLoS Biol.* **10**, e1001300. (doi:10.1371/journal.pbio.1001300)
169. Ugelvig LV, Cremer S. 2007 Social prophylaxis: group interaction promotes collective immunity in ant colonies. *Curr. Biol.* **17**, 1967–1971. (doi:10.1016/j.cub.2007.10.029)
170. Fefferman NH, Traniello JFA, Rosengaus RB, Calleri DV. 2007 Disease prevention and resistance in social insects: modeling the survival consequences of immunity, hygienic behavior, and colony organization. *Behav. Ecol. Sociobiol.* **61**, 565–577. (doi:10.1007/s00265-006-0285-y)
171. Hoi H, Darolova A, Konig C, Kristofik J. 1998 The relation between colony size, breeding density and ectoparasite loads of adult European bee-eaters

(*Merops apiaster*). *Ecoscience* **5**, 156–163. (doi:10.1080/11956860.1998.11682455)

172. Cremer S, Armitage SAO, Schmid-Hempel P. 2007 Social immunity. *Curr. Biol.* **17**, R693–R702. (doi:10.1016/j.cub.2007.06.008)
173. Meunier J. 2015 Social immunity and the evolution of group living in insects. *Phil. Trans. R. Soc. B* **370**, 20140102. (doi:10.1098/rstb.2014.0102)
174. Stroeymeyt N, Casillas-Perez B, Cremer S. 2014 Organisational immunity in social insects. *Curr. Opin. Insect Sci.* **5**, 1–15. (doi:10.1016/j.cois.2014.09.001)
175. Drum NH, Rothenbuhler WC. 1985 Differences in non-stinging aggressive responses of worker honeybees to diseased and healthy bees in May and July. *J. Apicult. Res.* **24**, 184–187. (doi:10.1080/00218839.1985.11100669)
176. Sorensen A, van Beest FM, Brook RK. 2014 Impacts of wildlife baiting and supplemental feeding on infectious disease transmission risk: a synthesis of knowledge. *Prev. Vet. Med.* **113**, 356–363. (doi:10.1016/j.prevetmed.2013.11.010)
177. Klepac P, Metcalf CJE, McLean AR, Hampson K. 2013 Towards the endgame and beyond: complexities and challenges for the elimination of infectious diseases. *Phil. Trans. R. Soc. B* **368**, 20120137. (doi:10.1098/rstb.2012.0137)
178. Alirol E, Getaz L, Stoll B, Chappuis F, Loutan L. 2011 Urbanisation and infectious diseases in a globalised world. *Lancet Infect. Dis.* **11**, 131–141. (doi:10.1016/S1473-3099(10)70223-1)
179. Vazquez-Prokopec GM, Kitron U, Montgomery B, Horne P, Ritchie SA. 2010 Quantifying the spatial dimension of dengue virus epidemic spread within a tropical urban environment. *PLoS Neglect. Trop. Dis.* **4**, e920. (doi:10.1371/journal.pntd.0000920)
180. Mao L. 2014 Modeling triple-diffusions of infectious diseases, information, and preventive behaviors through a metropolitan social network—an agent-based simulation. *Appl. Geogr.* **50**, 31–39. (doi:10.1016/j.apgeog.2014.02.005)
181. Read JM, Edmunds WJ, Riley S, Lessler J, Cummings DAT. 2012 Close encounters of the infectious kind: methods to measure social mixing behaviour. *Epidemiol. Infect.* **140**, 2117–2130. (doi:10.1017/S0950268812000842)
182. Read JM, Eames KTD, Edmunds WJ. 2008 Dynamic social networks and the implications for the spread of infectious disease. *J. R Soc. Interface* **5**, 1001–1007. (doi:10.1098/rsif.2008.0013)
183. Salathé M, Jones JH. 2010 Dynamics and control of diseases in networks with community structure. *PLoS Comput. Biol.* **6**, e1000736. (doi:10.1371/journal.pcbi.1000736)
184. Lloyd-Smith JO, Schreiber SJ, Kopp PE, Getz WM. 2005 Superspreading and the effect of individual variation on disease emergence. *Nature* **438**, 355–359. (doi:10.1038/nature04153)
185. Mei S, Chen B, Zhu Y, Lees MH, Boukhanovsky AV, Sloot PMA. 2015 Simulating city-level airborne infectious diseases. *Comp. Environ. Urban Syst.* **51**, 97–105. (doi:10.1016/j.compenvurbsys.2014.12.002)
186. Mpolya EA, Yashima K, Ohtsuki H, Sasaki A. 2014 Epidemic dynamics of a vector-borne disease on a villages-and-city star network with commuters. *J. Theor. Biol.* **343**, 120–126. (doi:10.1016/j.jtbi.2013.11.024)
187. Bian L. 2013 Spatial approaches to modeling dispersion of communicable diseases—a review. *Trans. GIS* **17**, 1–17. (doi:10.1111/j.1467-9671.2012.01329.x)
188. Qi F, Du F. 2013 Tracking and visualization of space–time activities for a micro-scale flu transmission study. *Int. J. Health Geogr.* **12**, 6. (doi:10.1186/1476-072X-12-6)
189. Potter GE, Smieszek T, Sailer K. 2015 Modeling workplace contact networks: the effects of organizational structure, architecture, and reporting errors on epidemic predictions. *Netw. Sci.* **3**, 298–325. (doi:10.1017/nws.2015.22)
190. Milne GJ, Kelso JK, Kelly HA, Huband ST, McVernon J. 2008 A small community model for the transmission of infectious diseases: comparison of school closure as an intervention in individual-based models of an influenza pandemic. *PLoS ONE* **3**, e4005. (doi:10.1371/journal.pone.0004005)
191. Yang Y, Atkinson PM, Ettema D. 2011 Analysis of CDC social control measures using an agent-based simulation of an influenza epidemic in a city. *BMC Infect. Dis.* **11**, 199. (doi:10.1186/1471-2334-11-199)
192. Mooring MS, McKenzie AA, Hart BL. 1996 Grooming in impala: role of oral grooming in removal of ticks and effects of ticks in increasing grooming rate. *Physiol. Behav.* **59**, 965–971. (doi:10.1016/0031-9384(95)02186-8)
193. Eckstein RA, Hart BL. 2000 Grooming and control of fleas in cats. *Appl. Anim. Behav. Sci.* **68**, 141–150. (doi:10.1016/S0168-1591(00)00095-2)
194. Rosengaus RB, Maxmen AB, Coates LE, Traniello JFA. 1998 Disease resistance: a benefit of sociality in the dampwood termite *Zootermopsis angusticollis* (*Isoptera: Termopsidae*). *Behav. Ecol. Sociobiol.* **44**, 125–134. (doi:10.1007/s002650050523)
195. Reber A, Purcell J, Buechel SD, Buri P, Chapuisat M. 2011 The expression and impact of antifungal grooming in ants. *J. Evol. Biol.* **24**, 954–964. (doi:10.1111/j.1420-9101.2011.02230.x)
196. Drewe JA. 2010 Who infects whom? Social networks and tuberculosis transmission in wild meerkats. *Proc. R. Soc. B* **277**, 633–642. (doi:10.1098/rspb.2009.1775)
197. Theis FJ, Ugelvig LV, Marr C, Cremer S. 2015 Opposing effects of allogrooming on disease transmission in ant societies. *Phil. Trans. R. Soc. B* **370**, 20140108. (doi:10.1098/rstb.2014.0108)
198. Giorgi MS, Arlettaz R, Christe P, Vogel P. 2001 The energetic grooming costs imposed by a parasitic mite (*Spinturnix myoti*) upon its bat host (*Myotis myotis*). *Proc. R. Soc. B* **268**, 2071–2075. (doi:10.1098/rspb.2001.1686)
199. McGlynn TP. 2012 The ecology of nest movement in social insects. *Annu. Rev. Entomol.* **57**, 291–308. (doi:10.1146/annurev-ento-120710-100708)
200. Pinter-Wollman N, Fiore SM, Theraulaz G. 2017 The impact of architecture on collective behaviour. *Nat. Ecol. Evol.* **1**, 0111. (doi:10.1038/s41559-017-0111)
201. Pinter-Wollman N, Keiser CN, Wollman R, Pruitt JN. 2016 The effect of keystone individuals on collective outcomes can be mediated through interactions or behavioral persistence. *Am. Nat.* **188**, 240–252. (doi:10.1086/687235)
202. Naug D, Camazine S. 2002 The role of colony organization on pathogen transmission in social insects. *J. Theor. Biol.* **215**, 427–439. (doi:10.1006/jtbi.2001.2524)
203. Read JM, Keeling MJ. 2003 Disease evolution on networks: the role of contact structure. *Proc. R. Soc. B* **270**, 699–708. (doi:10.1098/rspb.2002.2305)
204. Baracchi D, Cini A. 2014 A socio-spatial combined approach confirms a highly compartmentalised structure in honeybees. *Ethology* **120**, 1167–1176. (doi:10.1111/eth.12290)
205. Mersch DP, Crespi A, Keller L. 2013 Tracking individuals shows spatial fidelity is a key regulator of ant social organization. *Science* **340**, 1090–1093. (doi:10.1126/science.1234316)
206. Pinter-Wollman N, Wollman R, Guetz A, Holmes S, Gordon DM. 2011 The effect of individual variation on the structure and function of interaction networks in harvester ants. *J. R. Soc. Interface* **8**, 1562–1573. (doi:10.1098/rsif.2011.0059)
207. Naug D, Smith B. 2007 Experimentally induced change in infectious period affects transmission dynamics in a social group. *Proc. R. Soc. B* **274**, 61–65. (doi:10.1098/rspb.2006.3695)
208. Frick WF, Cheng TL, Langwig KE, Hoyt JR, Janicki AF, Parise KL, Foster JT, Kilpatrick AM. 2017 Pathogen dynamics during invasion and establishment of white-nose syndrome explain mechanisms of host persistence. *Ecology* **98**, 624–631. (doi:10.1002/ecy.1706)
209. Foley J, Clifford D, Castle K, Cryan P, Ostfeld RS. 2011 Investigating and managing the rapid emergence of white-nose syndrome, a novel, fatal, infectious disease of hibernating bats. *Conserv. Biol.* **25**, 223–231. (doi:10.1111/j.1523-1739.2010.01638.x)
210. Woodroffe R *et al.* 2006 Culling and cattle controls influence tuberculosis risk for badgers. *Proc. Natl Acad. Sci. USA* **103**, 14 713–14 717. (doi:10.1073/pnas.0606251103).
211. Carter SP, Delahay RJ, Smith GC, Macdonald DW, Riordan P, Etherington TR, Pimley ER, Walker NJ, Cheeseman CL. 2007 Culling-induced social perturbation in Eurasian badgers *Meles meles* and the management of TB in cattle: an analysis of a critical problem in applied ecology. *Proc. R. Soc. B* **274**, 2769–2777. (doi:10.1098/rspb.2007.0998)
212. Delahay RJ, Langton S, Smith GC, Clifton-Hadley RS, Cheeseman CL. 2000 The spatio-temporal distribution of *Mycobacterium bovis* (bovine tuberculosis) infection in a high-density badger population. *J. Anim. Ecol.* **69**, 428–441. (doi:10.1046/j.1365-2656.2000.00406.x)
213. Levy BS, Patz J. 2015 *Climate change and public health*. Oxford, NY: Oxford University Press.

214. Luber G, Lemery J. 2015 *Global climate change and human health: from science to practice*. San Francisco, CA: Jossey-Bass APHA Press, an imprint of American Public Health Association.
215. Watts N *et al.* 2015 Health and climate change: policy responses to protect public health. *Lancet* **386**, 1861–1914. (doi:10.1016/S0140-6736(15)60854-6)
216. Evans GW. Projected behavioral impacts of global climate change. *Annu. Rev. Psychol.* In press.
217. Brilliant L. 2007 Climate, poverty and health: time for preventive medicine. In *Seventh annual John H. Chafee memorial lecture on science and the environment*, presented at the 7th National Conference on Science, Policy and the Environment, Washington, DC, 1 February 2007. Washington, DC: National Council for Science and the Environment.
218. Chan M, Estève D, Fourniols J-Y, Escriba C, Campo E. 2012 Smart wearable systems: current status and future challenges. *Artif. Intell. Med.* **56**, 137–156. (doi:10.1016/j.artmed.2012.09.003)
219. Majumder S, Mondal T, Deen MJ. 2017 Wearable sensors for remote health monitoring. *Sensors* **17**, 130. (doi:10.3390/s17010130)
220. van der Valk S, Myers T, Atkinson I, Mohring K. 2015 Sensor networks in workplaces: correlating comfort and productivity. In *2015 IEEE Tenth International Conference on Intelligent Sensors, Sensor Networks and Information Processing (Issnip)*, *Singapore, 7–9 April 2015*, pp. 43–48. Piscataway, NJ: Institute of Electrical and Electronics Engineers (IEEE).
221. Bluyssen PM, van Zeist F, Kurvers S, Tenpierik M, Pont S, Wolters B, van Hulst L, Meertins D. 2018 The creation of SenseLab: a laboratory for testing and experiencing single and combinations of indoor environmental conditions. *Intell. Buildings Int.* **10**, 5–18. (doi:10.1080/17508975.2017.1330187)
222. Salathé M, Kazandjieva M, Lee JW, Levis P, Feldman MW, Jones JH. 2010 A high-resolution human contact network for infectious disease transmission. *Proc. Natl Acad. Sci. USA* **107**, 22 020–22 025. (doi:10.1073/pnas.1009094108)
223. Calogiuri G, Litleskare S, Fagerheim KA, Rydgren TL, Brambilla E, Thurston M. 2018 Experiencing nature through immersive virtual environments: environmental perceptions, physical engagement, and affective responses during a simulated nature walk. *Front. Psychol.* **8**, 2321. (doi:10.3389/fpsyg.2017.02321
224. Cunningham AA, Daszak P, Wood JLN. 2017 One Health, emerging infectious diseases and wildlife: two decades of progress? *Phil. Trans. R. Soc. B* **372**, 20160167. (doi:10.1098/rstb.2016.0167)
225. Stevens KB, Pfeiffer DU. 2015 Sources of spatial animal and human health data: casting the net wide to deal more effectively with increasingly complex disease problems. *Spat. Spatio-temporal Epidemiol.* **13**, 15–29. (doi:10.1016/j.sste.2015.04.003)