Applied Animal Behaviour Science 285 (2025) 106573

Contents lists available at ScienceDirect

Applied Animal Behaviour Science

journal homepage: www.elsevier.com/locate/applanim

Behavioural indicators of infectious disease in managed animals

Christine Janet Nicol

Royal Veterinary College, Hawkshead Lane, Brookmans Park, Hatfield AL9 7TA, UK

ARTICLE INFO

Keywords:
Animal welfare
Sickness behaviour
Behavioural indicator
Core behaviour
Infection

ABSTRACT

Identifying reliable and valid behavioural indicators of infection in managed animals is increasingly important as the risks of emergent diseases increase alongside concomitant concerns over pathogen resistance and the environmental and safety impacts of traditional therapeutic treatments. Early behavioural detection of disease could help to curb transmission, assist in selecting resilient animals and guide facility design to help animals avoid infection and to support their recovery. This review explores the adaptability and flexibility of animal responses to pathogens, including behaviours that (i) favour disease avoidance (ii) are associated with immune activation (iii) directly resist pathogens (iv) are dysregulated leading to hypersensitivity and (v) are associated with pathogen tolerance. A key theme is that all of these behavioural responses are strongly modulated by contextual factors such as pain, hunger and social priorities. Efforts to develop and validate, and increasingly automate, behavioural indicators of infection have so far primarily focused on infection-induced changes in core behaviours such as feeding and general activity or on hypothesis-free machine-learning comparison. However, such approaches have limited specificity, sensitivity and may be hard to generalise across contexts. The current review suggests ways in which specificity could be improved by monitoring changes in behaviours that are more closely linked to immune activation e.g. sleep, attention and motor function, by a more granular focus, and by integration with clinical symptoms. It also proposes that sensitivity could be improved by monitoring pliant ("luxury") behaviours and by intentional challenges or tests. Improved knowledge of how animals behave when infected could be used to design environments where the costs of resistance or tolerance are reduced and where recovery is promoted.

1. Introduction

Applied animal behaviour scientists have long striven to identify reliable and valid behavioural indicators of good or poor welfare. Effective welfare indicators include activities such as play, self-grooming or stereotypies as well as altered levels of activity that can produceinactive, over-active,diverse or fragmented behavioural profiles (e.g. Dawkins, 2008; Held and Spinka, 2011; Miller et al., 2020; Fureix and Meagher, 2015). Confidence in the validity of behavioural indicators is improved if they correlate well with other measures of welfare such as physical health, and the animal's own preferences and cognitive biases (Nicol et al., 2009; Paul et al., 2022). General welfare indicators are useful for tracking, comparing and benchmarking the well-being of animals in farms, zoos and research facilities and for setting and enforcing regulations. However, behaviour may also be able to identify more specific components of overall welfare. One such component is infectious disease, which has the potential to debilitate, harm or kill large numbers of animals.

Identifying specific behavioural indicators of infection is timely in a world where the growing risk of a new pandemics sits alongside calls to limit many traditional therapeutics due to concerns about antimicrobial resistance, environmental impact and food safety. Behaviour-based indicators could assist in the earliest possible detection of infection, curbing transmission and in supporting the identification of disease-resilient animals for breeding programmes. Increasing knowledge of how immune responses to pathogens interact with the nervous system could help to guide the design of facilities that prevent infection or promote recovery with minimal intervention.

Previous reviews have explored how host immune responses elicit behavioural changes ('sickness behaviour', Hart, 1988) and have outlined progress in the development of automated tools to pinpoint behavioural changes as disease markers for individuals or groups of managed animals (Stachowicz and Umstatter, 2021). The aim of the current (non-systematic) review was to paint a broader picture, relevant to the animals that are managed by humans, and thus the readership of Applied Animal Behaviour Science. Literature was selected by

---

E-mail address: cnicol@rvc.ac.uk.

https://doi.org/10.1016/j.applanim.2025.106573
Received 6 November 2024; Received in revised form 16 February 2025; Accepted 19 February 2025

pre-existing knowledge of seminal papers in the field and through searching citations of these. Keywords were also used for searches, using Web of Science. Another intention of the review was to place my own work in this broad area, which has been conducted alongside many colleagues over many years, within a logical narrative structure.

The review begins with a (non-systematic) account outlining how animals can counter threats from pathogens (fungi, protists, multicellular parasites, bacteria and viruses) through a variety of immune and behavioural strategies, not only by immune resistance (Section 2). It highlights the adaptive and flexible nature of behavioural responses to infection, showing how they can be modulated by hunger, pain and other competing priorities (Section 3).

Suggestions to improve the specificity and sensitivity of behavioural indicators of disease are made in Section 4, while Section 5 suggests how this could strengthen the hand of applied animal behaviour scientists in monitoring the quality of life of infected animals.

2. Strategies to counter infection

The threat posed by pathogens can be met through numerous strategies. The first considered is the ability of animals to avoid exposure to pathogens in the first place (Section 2.1). However, if and when infection takes hold then immune resistance strategies are rapidly deployed (Section 2.2). Separately, responses such as self-grooming or self-medication can contribute directly to a reduction in pathogen load, and can be regarded as examples of **behavioural resistance** (Section 2.3). In some circumstances, immune and behavioural responses to infections can become dysregulated leading to over-activation and **hypersensitivity** (Section 2.4). rResistance (immune or behavioural) is not the only way to counter pathogenic threat. Animals may instead exhibit **tolerance** by mounting responses that protect their bodies from the damaging effects of pathogens without necessarily reducing pathogen numbers (Section 2.5)

The concept of a ‘behavioural immune system’ has gained currency as a way of thinking about the cognitive and behavioural strategies employed by animals to avoid, resist or tolerate pathogens (Schaller and Park, 2011). The behavioural immune system can even be conceived as incorporating both innate and acquired adaptive features, analogous to the physiological immune system (Amoroso, 2021). The current review adopts this adaptive perspective and acknowledges bi-directional routes between behaviour and immune function (Lopes, 2017). Behaviour can clearly influence immune function (e.g. more social or exploratory animals may encounter more antigens leading to immune system upregulation, Lopes et al., 2020), but immune function can also influence behaviour (e.g. neonatal immune challenge can reduce exploratory behaviour in adult rodents (Spencer et al., 2005) and impair cognitive ability in adolescents (Custódio et al., 2018).

In the following sections the role of behaviour is explored in more detail in relation to each strategy.

2.1. Avoiding Infection

The threat of infection is effectively neutralised if animals are able to **avoid** exposure in the first place. A number of familiar behaviours are employed by animals to avoid exposure to pathogens, including avoidance of faeces, (Judge et al., 2005) or contaminated carcasses (Gonzalvez et al., 2021) and thecleaning or fumigating of. nesting sites (Bush and Clayton, 2018), and many animals will swat away insect vectors (Hart, 2011). Carnivores also avoid feeding on the carcasses of the same, or closely-related species e.g. reducing the risk of ingesting meat-borne parasites. Infected and potentially-infected individuals may be avoided by conspecifics because they emit detectable olfactory and other sensory cues (Renault et al., 2008; Kavaliers and Choleris, 2018; Kavaliers et al., 2014). The bodily secretions of individuals treated with LPS are also avoided to a greater extent than those of controls, in species as diverse as snails (Kavaliers et al., 2023) and humans (Gordon et al., 2023). In mammals, avoidance is modulated by altered receptor activity for the hormones which are strongly implicated in many forms of subtle social discrimination (Arakawa et al., 2010). For example, expression of oxytocin receptor mRNA in the amygdala was increased when rats encountered the odour of healthy conspecifics, while no expression of mRNA for the arginine vasopressin receptor was detected when rats encountered the odour of sick conspecifics. Female rodents avoid the urinary odours of males infected with a wide range of pathogens, including influenza, Salmonella, nematode and protozoan internal parasites, and ectoparasites such as lice (Kavaliers and Choleris, 2018). The whole style of social interaction can be altered. For example, in the presence of infected individuals, rodents generalise social avoidance towards all unfamiliar animals (Kavaliers et al., 2014).

Generally, behavioural avoidance is less likely evolve in close-knit groups where social interactions are important (Amoroso and Antonovics, 2020). The extent to which humans will generalise avoidance strategy towards unfamiliar outgroups is controversial (van Leeuwen and Petersen 2018), although some studies have reported reduced extraversion in human societies that have experienced high disease burdens, suggesting that infection may influence personality through selection or culture (Schaller and Murray, 2008).

An innate emotional disgust response (Ekman and Cordaro, 2011) underpins many of these avoidance strategies (Sarabian et al., 2023). Disgust itself triggers pro-inflammatory immune responses (Schaller et al., 2010) and a range of behaviours such as gaping, avoidance of moist substances, and washing or wiping of body parts (Sarabian et al., 2023). General avoidance could reduce contact with multiple pathogens similar to the broad protections offered by the innate immune system (Amoroso, 2021). These underpinning strategies may have a largely genetic basis. For example, in a study of the behaviour of two strains of sheep, individuals genetically selected for physiological resistance to parasites also showed greater behavioural avoidance of grass types prone to harbouring parasites than sheep selected for susceptibility (Hutchings et al., 2007).

Even if most behavioural avoidance is innate, learning may play a role in protecting animals from certain pathogens associated with food ingestion. Conditioned aversions are formed to novel flavours (or other characteristics of feed) that subsequently, sometimes after delays of many hours, cause nausea or vomiting. Rodents can form relative aversions to flavours paired directly with the administration of pathogens (e.g. nematode larvae, Keymer et al., 1983), or with compounds associated with infection (e.g. interleukin-1, Bauer et al., 1995; LPS, Nilsson et al., 2017). However, the specific learnt aversion is relatively weak in comparison to the overall anorexic effect of these pathogenic cues and the immune-neural signalling routes differ (Nilsson et al., 2017). In addition, the typical incubation periods of most parasites are too long for learnt avoidance of this kind to be effective (Amoroso, 2021).

Social learning is another potential route by which animals may learn to avoid exposure to pathogens, but the situation is complex. Social interactions provide opportunities for gaining information (e.g. about food or predators) but they also increase the direct risk of infection. The optimal balance will vary between species and may be reflected in the different social structures that have evolved (Evans et al., 2021). Social learning about shifting risks of *within-group* infection could be adaptive for some species, particularly those that are social but without very close physical contact e.g. group-living birds. However, there are very few studies in this area and the evidence that social learning is used for disease detection is not strong. Chicks (Johnston et al., 1998) but not adult hens (Sherwin et al., 2002) learned to avoid novel feeds after observing the disgust reaction of a conspecific, suggesting that social learning might be more useful to naïve animals. However, Galef (1996) concluded there was no evidence that the food preferences of observer rats were influenced by the state of health of demonstrators. The smell of a novel food on the breath of a demonstrator encouraged naïve rats to accept rather than reject that same food, even when the demonstrator itself was apparently moribund (sedated) (Galef, 1996). In humans, social learning clearly influences responses to infection risk and to infected individuals (e.g. Carrignon et al., 2022). Animal studies would help to establish the extent to which any social learning of infection-avoidance is driven by biological predisposition or by cognitive information processing.

2.2. *Behaviour associated with immune resistance*

2.2.1. *General pattern of immune response*

Avoidance is not always entirely successful and once an individual animal becomes infected a cascade of physiological **immune resistance** responses may be deployed. The immune system is exquisitely evolved to disable and kill pathogens via an immediate (acute) innate response and a subsequent antibody-mediated response. The presence of a pathogen is initially detected by a group of molecular receptors that recognise generic structural components of pathogens such as bacterial cell wall components or virus RNA that are distinct from any host structures (pathogen associated molecular patterns, PAMPs). The innate response involves the release of pro-inflammatory chemokines and cytokines, including interleukin-1 (IL-1), interleukin 6 (IL-6) Interleukin-8 (IL8) and tumour necrosis factor alpha (TNFa), which recruit more immune cells to the site of infection and cause inflammation. In experimental studies examining the downstream effects of innate immune activation, a PAMP, usually bacterial lipopolysaccharide (LPS), is administered instead of live pathogens. This 'immune challenge' procedure ensures that the effects of host response are studied rather than changes due to pathogen presence. The appearance of sickness behaviour in association with the acute phase of immune resistance is thought to be an adaptation to conserve energy, allowing resources to be diverted to disabling or killing invasive pathogens. The second (adaptive) phase of the immune response is mediated by antibody producing B- and T-cells that target molecules unique to each individual pathogen. This process takes several days to become activated on the first encounter with a pathogen but will respond rapidly in the event of a second encounter. This phase is generally characterised by a reduction in sickness behaviour and signs of recovery as inflammation reduces.

The general pattern of immune response to infection outlined above is common to all vertebrates (with certain features also present in many invertebrates) but the details differ according to taxonomy. In endothermic (warm-blooded) mammals and birds the release of pro-inflammatory cytokines as part of the acute phase response triggers physiological fever, activation of the HPA axis and sickness behaviours (Dantzer and Kelley, 2007) and sensitisation of nociceptors, leading to somatic or visceral pain (Watkins et al., 1994; Meseguer et al., 2014). Because hosts experience direct harms from the activation of the immune system, immune resistance can have evolved only in situations where the pathogens pay an even higher price, a situation that has been described as 'immune brinkmanship' (LeGrand and Alcock, 2012). This brinkmanship is constantly in the balance as shown by the regulation of the anti-inflammatory cytokine IL-10. Normally, IL-10 functions to inhibit pro-inflammatory cytokines, bringing the immune response back under control after infection has been countered. However, its immunosuppressive effects are exploited by some pathogens (including the protozoan *Eimeria* which causes coccidiosis in chickens) so that the immune response is curtailed too early, aiding the survival of the pathogen (Cyktor and Turner, 2011).

2.2.2. *Immune influences on behaviour*

The mechanisms by which the immune system influences behaviour are multi-faceted and complex. Relatively large cytokine molecules do not readily cross the blood-brain barrier but they can pass through certain 'leaky' regions, activate endothelial cells which release secondary messengers, or transmit signals through peripheral nerves, such as the vagus nerve, to reach the brain (Balabanov and Dore-Duffy, 1998). Within the brain a comprehensive neural substrate regulates these cytokine signals producing effects on behaviour via multiple mechanisms, including cytokine modulation of serotonin, dopamine and glutamate metabolism. Cytokines also activate the HPA-axis (Turnbull and Rivier, 1999), stimulating the release of CRH, ACTH and cortisol during acute stages of infection, but flattening the cortisol response during chronic stages and altering host behaviours related to fear, motivation and social interaction. Cytokines can also sensitise pain pathways (Dantzer et al., 2008; Capuron and Miller, 2011).

Typically, across species and pathogen types, infected animals experience a combination of lethargy, fatigue, depression and anorexia, manifested by sickness behaviour in the form of increased rest, postural changes and reduced feedingOriginally, sickness behaviour was regarded as an involuntary response to infection, indicating host weakness or vulnerability. However, from as early as the 1960s some scientists argued that sickness behaviour was a motivational state rather than an inevitable sign of debilitation. In an influential review, Hart (1988) proposed that sickness behaviours could be regarded as adaptations that aid host resistance. This view has gained widespread support and responses to pathogen infection are now generally viewed as part of a larger category of adaptive responses to perturbation (Wingfield, 2003). It seems intuitively plausible that some features of sickness behaviour, such as reductions in activity (together with postural changes such as curling up to reduce heat loss), might conserve energy and support the reallocation of resources to counter threat posed by pathogens, or might help sick animals avoid detection by predators (Dantzer and Kelley, 2007).

2.2.3. *Closer scrutiny of the adaptive hypothesis of sickness behaviour*

Despite its plausibility, all aspects of the adaptive hypothesis of sickness behaviour require ongoing scrutiny and evaluation in the light of emerging empirical evidence.

The view that immune resistance is energetically demanding has not been universally accepted. Klasing (1998) calculated that the energetic costs of maintaining immunity in the chicken were very small relative to growth or reproduction, a view countered by Lochmiller and Deerenberg (2000) who felt that insufficient account had been taken of the indirect consequences of mounting an immune response (e.g. the inflammatory response). It is difficult to calculate the costs of immune response separately from other intertwined physiological process but Lochmiller and Deerenberg (2000) presented a range of evidence suggestive of high costs. They citedstudies that have shown that mild immune responses to vaccination can raise metabolic rate (MR) by 15–30 % and increase the breakdown of proteins by 30 %, while fever elicits a 10–15 % elevation in MR for every 1°C rise in body temperature. High costs are also suggested by trade-offs whereby infection depresses growth. Conversely, selection for particularly rapid growth can interfere with immunity, as seen in broiler chickens which have a lower ability to generate fever and lower cytokine expression than laying hens (Leshchinsky and Klasing, 2001). Specific genes that enhance growth at the expense of immunity have been identified (Zou et al., 2020).

Despite this suggestive evidence, experimental studies that directly measure the MR of infected animals relative to controls have produced mixed results. Positive (e.g. rodents, Scantlebury et al., 2007; Garrido et al., 2016), non-existent (rodents, Kam et al., 2011; chickens, Murillo et al., 2016) or negative (e.g. rodents, Devevey et al., 2008; Tu et al., 2008) associations exist between MR and parasite load, probably because hosts compensate for the costs of infection through diverse plasticity e.g. re-allocating resources from growth to maintenance, decreasing active behaviour or increasing feed intake (Garrido et al., 2016). Murillo et al. (2016), for example, found that hens infested with Northern fowl mites ate more without this leading to an increased MR. Overall, immune resistance is likely to be energetically demanding but this demand can be met in numerous ways, including reductions in the most energy-expensive behaviours.

A more difficult question is whether cytokine-induced anorexia can be regarded as a host adaptation. Cytokine-induced anorexia can also occur in association with many non-infectious clinical conditions, including cancer (PlataSalaman, 1996; Ezeoke and Morley, 2015) and it is not always strongly correlated with other aspects of immune resistance (e.g. fever, Larson et al., 1996). Infection-induced anorexia can be selective with energy intake maintained but protein or fat intake disproportionately reduced (Adamo et al., 2010; Murdock et al., 2017; Taylor et al., 2022), possibly as a strategy to reduce the costs associated with digestion (Adamo et al., 2010) or to deprive pathogens of nutrients (e.g. iron) they require for growth. These potential advantages to the host are nonetheless accompanied by the direct cost of reducing feed intake at a time when nutritional demands have increased and by multiple indirect costs e.g. depriving pathogens of nutrients requires complex physiological adjustments to protect beneficial gut microbiota (Pickard et al., 2014). Anorexia is thus a paradoxical and potentially counter-productive response. While a few studies have linked pre-infection starvation with enhanced survival (Wing and Young, 1980), many other studies have found that starved animals are more susceptible to infection, and malnutrition is the leading cause of secondary immunodeficiency in humans (Sinha and Guerrant, 2023). Although there are associations between anorexia and enhanced immune function (Klasing, 1988) direct evidence of a survival benefit is seemingly limited to a widely-cited study showing that severely feed-restricted mice were more likely to survive a bacterial infection (*Listeria monocyogenes*) than mice subject to force-feeding (Murray and Murray, 1979). However, forced starvation and forced feeding do not mimic naturally-induced anorexia. The survival benefit may also be limited to food-borne pathogens (such as *Listeria* or *Salmonella*) where anorexia could prevent further pathogen intake. Indeed, *Salmonella* appears able to inhibit host anorexia in mice and thereby promote its own transmission (Rao et al., 2017). To counter such pathogenic influences, the cytokine response to food-borne pathogens often results in nausea which is not a feature of all infections. Overall, a tentative conclusion is that partial, selective or short-term anorexia may be a beneficial host strategy during the initial stages of immune resistance, but prolonged anorexia is likely to be deleterious (PlataSalaman, 1996; Langhans, 1996; Kyriazakis et al., 1998).

Social withdrawal is another common feature of sickness behaviour (Hart, 1988; Dantzer and Kelley, 2007) and one that could indicate either host debility or an adaptive response to reduce unwanted attention. Epidemiological studies have shown associations between diseases such as egg peritonitis or infectious bronchitis and injurious pecking in laying hens (Poetzsch et al., 2001; Green et al., 2000) and between respiratory disease and tail biting in pigs (Moinard et al., 2003). Recent work has started to uncover the directional effects that may underpin such associations. Immune-challenged pigs were more likely to engage in tail-biting and ear-manipulation than controls, possibly due to cytokine-induced irritability (Munsterhjelm et al., 2019; Veit et al., 2021) but pigs showing social withdrawal were more, not less, likely to receive unwanted attention from healthy group members. However, it should be noted that under commercial farming conditions full social avoidance is almost impossible. Interestingly, an emerging body of research in humans suggests that under some circumstances inflammation *increases* sociality, directly challenging the classic sickness-behaviour narrative. For example, Reiber et al. (2010) found that in the 48 h after a ‘flu vaccination people were more socially interactive than in the preceding 48 h.

In vertebrates that cannot produce a physiological fever (ectotherms such as reptiles, amphibians, fish), infected individuals may move to a warmer locations to elevate their core body temperature, exhibiting ‘behavioural fever’ (e.g. tortoises, Goessling et al., 2017; fish, Huntingford et al., 2020). But ectotherm responses are variable and sometimes involve a shift to a lower body temperature (Cabanzo-Olarte et al., 2024) or highly variable inter-individual thermal preferences as a mechanism to counter threats from temperature-sensitive pathogens (Sauer et al., 2019). This variation may explain why intentional increases in environmental temperature for infected reptiles sometimes assist, but often accelerate, disease processes (Warwick, 1991). Variable behavioural fever and sickness behaviours are also found in some invertebrates (Sullivan et al., 2016). Anorexia, lethargy and predator avoidance are not always observed (Sullivan et al., 2016; Kelly and Leroux, 2020).

2.3. *Direct behavioural resistance*

Pathogens can be killed not only by immune activation but also by direct behavioural interventions that show greater diversity across species and contexts. Grooming, for example, has an important role in limiting ectoparasite numbers. In many mammalian species grooming is regulated by internal motivational cues, so that it occurs regularly as a prophylactic measure. However, the presence of ectoparasites and the itching (pruritis) they induce (Wilson, 2014) over-rides this baseline motivation. IL– 31 (the “itchy cytokine”) plays a significant role. It binds to a receptor found on sensory neurons in the dorsal root ganglia. Hence IL-31 directly links the immune system and the nervous system (Olomski et al., 2020). The sensory neurons then transmit the itch signals via a specialized population of C-fibres, distinct from those that transmit pain. These signals lead to increased rubbing, self-licking and scratching.

Calves infected with the cattle louse, *Linognathus vituli*, showed a 3-fold increase in rubbing behaviour relative to controls (Weeks et al., 1995). However, the extent to which rubbing is effective in removing parasites rather than just relieving the sensation of itching is not clear. Berriatura et al. (2001) suggested that rubbing might either directly kill the mites that cause sheep scab, or trigger the release of antigens from interior parts of the mite to strengthen the host’s immune response but were not able to test these proposals.

There is stronger evidence that preening, scratching and dust-bathing by birds can reduce parasite numbers. Many studies in poultry have found relationships between infestation level and preening intensity (mites, Murillo et al., 2020; lice, Murillo et al., 2024) or dustbathing behaviour (Martin and Mullens, 2012). Indirect evidence that dust-bathing can lower parasite burden is the increase in Northern fowl mite numbers observed when dust-bathing facilities are removed (Murillo and Mullens, 2016). The effectiveness of preening is also shown by the fact that beak-trimmed hens had substantially higher numbers of mites and of lice after experimental infection than hens with intact beaks (Vezzoli et al., 2015a). The potentially beneficial effects of sun-bathing in birds include the exposure of ectoparasites to uv light or desiccation, or the possibility that sunlight might encourage parasites to move to parts of the plumage more available for preening but the effects of this behaviour on parasite numbers have not yet been studied (Bush and Clayton, 2018). Animals such as reptiles and fish that are unable to directly groom themselves may actively seek out “cleaning stations” where they can be groomed by species that ingest the ectoparasites that are removed (e.g. turtles, Schofield et al., 2017).

Grooming and preening behaviours are not effective under all circumstances. In one experiment, when caged hens were provided with a variety of in-cage substrates, and experimentally infested with northern fowl mites, dustbathing behaviour was not effective in controlling mite numbers and there was no relation between time spent dustbathing and infestation level. The addition of feed particles to the dustbathing substrate even promoted mite growth and survival (Vezzoli et al., 2015b). And in some wild bird species, other pathogens have co-opted ectoparasites to increase their own transmission. Lice, for example, can harbour parasitic helminths which can infect hosts if ectoparasites are ingested during preening (Bush and Clayton, 2018).

Self-medication is another form of behavioural resistance. Disease can alter taste perception and increase intake of normally aversive but medicinal substances (insects, Leung et al., 2024). Free-living primates have been observed to select plants with medicinal properties (e.g. high proportion of alkaloids) when infected with gastro-intestinal parasites (bonobos, Fruth et al., 2014; Ghai et al., 2015) or when wounded and at risk of infection (orang utan, Laumer et al., 2024).

Just as pathogens can sometimes subvert host immune responses (see IL-10 in section above) and the sickness behaviours that accompany them, they can also induce and subvert host behaviour. Coughing and sneezing favours the transmission of respiratory viral or bacterial agents far more than it helps hosts to reduce pathogenic load, as these micropathogens thrive inside numerous host cells. The best known example of pathogen manipulation of host behaviour relates to the increased activity and risk-taking (including sexual risk taking and loss of aversion to predatory odours) that occurs in mammals infected with the protozoan, *Toxoplasma gondii* (Webster et al., 2013). This parasite induces an elevation of host testosterone level, altering behaviour and personality, and facilitating its own transmission (reviewed by Heil, 2016). Other potential examples are less clear. Berriatura et al. (2001) found no evidence that the rubbing behaviour of sheep enhanced the transmission of mites, even though sheep rubbed in the same locations. The authors proposed that rubbing could benefit the mites indirectly for example if increased inflammation improved the micro-environment for mite survival and reproduction but this was not tested.

2.4. Hypersensitivity

In some circumstances, immune and behavioural responses to infections can become dysregulated resulting in hypersensitivity. Type I hypersensitivity is an exaggerated response to pathogenic antigens and a widespread and well-studied form of dysregulation. It is primarily driven by IgE antibodies which bond to mast cells and basophils. On reexposure to pathogenic antigens, these IgE-coated cells release inflammatory mediators, such as histamine, cytokines, and prostaglandins, producing symptoms such as swelling and sometimes respiratory or other disturbances. IgG antibodies are also involved in some Type I hypersensitivity reactions (Wilson, 2014). Genetic and environmental factors strongly influence the development of Type 1 hypersensitivity which is often seen only in some individuals within an exposed population. The behavioural consequences are usually excessive grooming and signs of irritation. In horses with insect-bite hypersensitivity to salivary antigens from the midge, *Culicoides*, reduced activity and increased signs of irritation (tail-swishing, head shaking and stamping) were observed even during winter months when ectoparasites were inactive (Daw, 2024). These behavioural changes can not be regarded as adaptive. They result in lethargy, denudation of the coat, lesions and pain and sometimes profound reductions in overall quality of life. In these cases, monitoring behaviour is an important adjunct in the quest for effective therapeutic strategies to induce tolerance (Marti et al., 2021).

Other forms of hypersensitivity can occur in response to persistent infection by bacterial pathogens, leading to inflammation and tissue damage (e.g. Purpura haemorrhagica can arise in some horses following strangles infections caused by *Streptococcus equi*).

2.5. Behaviour associated with immune tolerance

The immune and behavioural responses that accompany tolerance (the protection of cells and tissues from dysfunction or damage without necessarily reducing pathogen numbers) are still relatively understudied and the triggers that promote a path towards either resistance or tolerance are not yet clear. Rivas et al. (2014) suggested that resistance and tolerance are best conceived as a set of interlinked mechanisms aimed at damage control but in practice these two strategies are most often studied separately and are associated with different physiological profiles.

Inducing tolerance to harmful pathogenic antigens is clearly preferable to the consequences of chronic allergy described above but there are other examples where tolerance might be beneficial. However, just as mounting an immune resistance response is costly, mounting a tolerance response will also require a diversion of resources from other functions, leading to trade-offs. Hosts may have to re-organise metabolic pathways to promote cellular regeneration and tissue repair (Soares et al., 2017; Seal et al., 2021). The precise mechanisms of immune tolerance remain relatively unexplored, but are likely to involve anti-inflammatory cytokines and a dampening of pro-inflammatory pathways. Glucocorticoids appear to play an important role reducing the normal pro-inflammatory response to infection. Virally infected but glucocorticoid-deficient mice showed significantly higher levels of IL-6, TNF and sickness behaviour than mice with functioning adrenal glands (Silverman et al., 2007). At equivalent parasite loads, wild blackbirds with higher concentrations of glucocorticoids were more tolerant of malaria infection (Schoenle et al., 2018).

Highly tolerant individuals can pose an existential risk to those who are more susceptible, either between or within species. *Salmonella* is tolerated relatively well by chickens but causes illness in humans. Coronavirus (SARS-CoV-2) and mpox (MPXV) are caused by viruses that are well tolerated by many small mammals, but can be lethal to susceptible human hosts. Within species, the adaptive value of tolerance can sometimes be so strong that a rapid fixation of tolerance-related genes is favoured, giving rise to a homogeneous population (e.g. tolerance for intestinal worms in Soay sheep, Hayward et al., 2014). But when populations remain heterogeneous the most tolerant and asymptomatic individuals can act as “super-shedders” or “super-spreaders”, harbouring very high levels of pathogen (e.g. Salmonella in pigs, Kempf et al., 2023; bovine paratuberculosis, Alonso-Hearn et al., 2022). Applied goals therefore focus either on the identification and removal of tolerant “super-spreader” individuals or on longer-term selective breeding to produce fully homogenous and tolerant populations. Selective breeding for disease tolerance is a realistic possibility in livestock (e.g. Johne’s disease in cattle, Zanella et al., 2011; or trypanosome infection in cattle, Hanotte et al., 2003) and has potential advantages in comparison with selection for disease resistance, particularly if implemented at a herd level (Doeschl-Wilson et al., 2021). Tolerance mechanisms are thought to generalise across multiple pathogens, and therefore should exert lower selection pressure for pathogen escape. However, identifying disease-tolerant phenotypes is a significant challenge (Lough et al., 2018; Hu et al., 2020).

Adelman and Hawley (2017) suggest that physiological tolerance should be accompanied by behavioural tolerance, perhaps in the form of reduced sickness behaviour. This raises the possibility that behavioural signatures of pathogen tolerance may be more subtle than those of resistance. If tolerance is accompanied by a reduction in sickness behaviour across the board then it will be harder to detect than if it is accompanied by differential changes e.g. lethargy (allowing diversion of resources to tissue protection) but not by huddling or anorexia (given there is no requirement to maintain an elevated body temperature). Some have suggested that disease-tolerant animals should express *all* normal behaviours (e.g. Nakov et al., 2019) but this ignores the substantial costs likely to be incurred in mounting the tolerance response. Many badgers, for example, tolerate high levels of bTB without showing clinical symptoms (Swift et al., 2021) but, under pressure, the behaviour of infected individuals differs from that of non-infected or vaccinated (Woodroffe et al., 2017) animals. bTB infected badgers have larger home ranges, forage further away from their main sett (Garnett et al., 2005) and are more likely to sustain bite wounds (Jenkins et al., 2012). Although infected badgers do not have higher energy requirements (Barbour et al., 2019) they appear less able to compete for food and maintain social status. When food and other resources are freely available the costs of tolerance may be less obvious. Sakkas et al. (2018) found no difference in tolerance to *Eimeria* infection in chickens growing at fast or slower rates, suggesting no trade-off between growth and physiological effort directed towards tissue protection. No alterations in feeding, drinking or activity could be detected in chickens pre- and post Salmonella infection or between infected and non-infected chickens in the home pen (Toscano et al., 2010) but under the higher-pressure of a resource competition test, chickens harbouring *Salmonella* competed less effectively than *Salmonella*-negative birds (Toscano et al., 2010).

Identifying tolerant animals might therefore require a degree of challenge to provoke responses that reveal the costs of tolerance. Over the longer term the prolonged costs of tolerance may result in chronic fatigue.

Pro-social actions directed towards infected individuals, such as food provision (Loehle, 1995) or territory defence (wolves, Almberg et al., 2015) may promote disease tolerance by maintaining the fitness of individuals who withstand high pathogen loads. However, reports of such behaviours are largely anecdotal (Hart, 2011) with few, if any examples, from livestock species.

2.6. *Trade-offs between strategies*

Trade-offs between strategies, particularly between avoidance and either resistance or tolerance are likely to exist. Intuitively, it makes sense that an animal investing strongly in one approach may have reduced resources available for an alternative. However, a direct test with zebra finches *Taeniopygia guttata,* found no support for this hypothesis (Schreier and Grindstaff, 2020). Associations between avoidance of a sick conspecific and immune response to an LPS challenge were either neutral or positive, with some individuals mounting stronger defences across the board. A stronger influence on the nature of the strategy deployed may be the social context in which an individual lives. Individuals within close-knit, stable social groups may be less likely to avoid infected conspecifics, especially those with whom they have mutual or dependent relationships, than individuals living within more transient herds or flocks (Stockmaier et al., 2023).

3. Behavioural responses to infection are modulated

Although the behavioural responses to infection are widely shared across species and contexts they are not fixed or uniquely protected. They are influenced by contextual factors including sex hormones (Cai et al., 2016) and cognitive expectations (Lasselin et al., 2018), indicating that they are not solely under the control of the immune system (Aubert, 1999). Pain is a powerful modulating influence on sickness behaviour and on forms of behavioural resistance. Rubbing behaviour in mite-infected sheep was suppressed when lesions were particularly large and painful (Berriatua et al., 2001). Similarly, pain due to swollen udders reduced lying time in infected cows (Siivonen et al., 2011). Conversely, the pain of oral lesions in foot-and-mouth disease reduced feed intake further than expected from sickness-induced anorexia (Somagond et al., 2023). Hunger is another modulator, and one that may underpin some apparent species differences in response, such as the findings that ducks exposed to PAMPs experienced anorexia but not lethargy (Marais et al., 2013) and that immune-challenged rats developed anorexia but continued to hoard food in anticipation of future needs (Aubert et al., 1997a). Blersch et al. (2021) argued that sickness behaviours may be more obvious in captive animals where food is provided than in wild animals living in conditions of food scarcity. However, in captive animals, large individual variation has been detected in the feeding behaviour of pigs with a range of health issues (Bus et al., 2024). In addition, anorexia associated with diarrhoea was observed only in calves on a high milk allowance not hungrier calves on lower allowance (Lowe et al., 2021)

Sickness behaviours can also be adjusted according to social priorities. Zebra finches kept in isolation showed predicted reductions in activity in response to an immune challenge whereas socially-kept zebra finches did not, despite similar levels of a pro-inflammatory cytokine (IL-6) (Lopes et al., 2012), and similar effects of have been found in rats where social contact of any kind reduced the tendency to show sickness behaviour (Russell et al., 2022). Sickness behaviour can also be suppressed to avoid loss of mating opportunities (Lopes, 2014). Male vertebrates have been shown to suppress sickness behaviour in the presence of females (Japanese quail *Cortunix japonica*, Gormally et al., 2022; guppies *Poecilia reticulata*, Jog et al., 2022). Sickness behaviours can also be suppressed to ensure continued parental care (Aubert et al., 1997b). Immune-challenged mice housed in normal temperatures retrieved pups from outside the nest but did not engage in nest-building behaviour, but at low temperatures they also engaged in nest-building as effectively as controls (Aubert et al., 1997b). A recent study showed that a cow's motivation to groom her calf was stronger than her motivation to express sickness behaviours in the first day after giving birth such that the only difference in behaviour between healthy and infected mothers was that the sick cows took longer to lie down near their calves (Perier et al., 2019). Morris et al. (2022) found that the activity levels of lambs infected with the parasitic nematode *Teladorsagia* were reduced to a greater extent if they were housed with other infected individuals than if they were housed in mixed groups. Millman (2007) suggested that sickness behaviours in farmed animals were more likely to be expressed in familiar environments and amongst familiar conspecifics.

These examples demonstrate that behavioural response to infectious disease vary according to context, potentially complicating the search for reliable early indicators. On the other hand, Weary et al. (2009) saw "*much potential for future research exploring how the expression of sickness behaviour varies in response to changing motivation for food and other important resources*" as a way of assessing the impact of disease on overall animal welfare.

4. Using behaviour to detect disease

4.1. *Current approaches*

There is huge interest in identifying behaviours that can be detected easily or automatically as a route to control disease spread or to cull animals at a point where recovery seems unlikely (Matthews et al., 2016). Most of this work is focused on the detection of changes in the core behaviours characteristic of sickness behaviour, particularly reductions in activity and feed-intake (see Table 1 for examples published in Applied Animal Behaviour Science in recent years).

Conventional techniques, whereby human observers classify behaviours into categories and count their occurrence, are still widely used. However, the rapid rise of precision livestock farming (PLF) and automated methods of recording behaviour cannot be ignored (García et al., 2020; Oliveira et al., 2021; Wang et al., 2022). These developments mean that changes in core behaviours associated with disease can be detected more rapidly and at scale. Automatic registration of reductions in feeding time or the frequency of visits to feeding locations show associations with bacterial or viral infections in pigs (reviewed by Bus et al., 2021), bovine respiratory disease and diarrhoea in calves (Mee et al., 2024) and metritis in dairy cows (Huzzey et al., 2007). Accelerometers fitted to individuals show that calves with diarrhoea lie down more frequently but for longer than healthy calves (Swartz et al., 2020) and identify ewes (Williams et al., 2022) and young grazing cattle (Högberg et al., 2021) infected with gut nematodes, even at low infection levels and in animals that showed normal bodyweight gain (Högberg et al., 2021). Similar devices also successfully detected a drop in activity of 10–20 % in free-ranging wild boar with African Swine Fever (Morelle et al., 2023), suggesting a role for this technology not only in captive populations but in the monitoring of sentinel animals in wildlife health surveillance.

Some studies have recorded posture or facial expression alongside activity. For example, pigs positive for PRRSV spent more time lying in a ventral than a lateral position, and more time in contact with a pen-mate (both behaviours that might help to reduce heat loss, Escobar et al., 2007). Whereas observers trained to examine facial expression were able to identify sheep suffering from foot-rot or mastitis with a relatively high accuracy (McLennan et al., 2016).

Computer vision systems have been developed to track changes in overall flock movements associated with campylobacter infection (Colles et al., 2016); Monitoring the health of individuals within groups using vision-based systems is even more challenging and requires

Table 1
Studies on sickness behaviours in farm animals published in Applied Animal Behaviour Science.

Authors	Species	Disease: induced or spontaneous		Behaviours Increased by Disease	Behaviours Decreased by Disease	Comments
Healy et al. (2002)	Sheep	Scrapie (spontaneous)	N	Time eating concentrate; abnormal posture; lie; rub; self-bite.	Time eating hay; masticate;stand; ruminate; social interaction; aggression	
Escobar et al. (2007)	Pigs 4–6 weeks	Pneumonia (PRRSV challenge)	I	Lie; lie ventral; lie in contact with pen-mate.	Eat	
Reiner et al. (2009)	Pigs	Sarcocystis (challenge)	P	Lie inactive.	Lie with activity: eat; drink; root	
Siivonen et al. (2011)	Cows	Acute mastitis (E.coli challenge)	I	Step.	Lie; ruminate; drink	Pain of mastitis interferes with lying.
Sepulveda-Varas et al. (2016)	Cattle (cows)	Mastitis (spontaneous)	I		Feed intake; competition at feeder	
Toaff-Rosenstein et al. (2016)	Cattle (steers)	Bovine respiratory disease (viral and bacterial challenge)	I	Lie total time; Lie bout length.	Groom	
Des Roches et al. (2018)	Cattle (cows)	Mastitis (E.coli challenge)	I	QBA characterised behaviour as “suffering, dejected, lethargic”.		
Lowe et al. (2021)	Cattle (calves)	Neonatal calf diarrhoea (spontaneous)	I	Lie total time.	Visits to feeder	
Thomas et al. (2021)	Cattle (beef heifers)	Digital dermatitis (spontaneous)	I	Inactive.	Ruminate	
Lewis et al. (2023)	Sheep (ewes and lambs)	Lameness (spontaneous)	M	Inactive.	Graze; stand	
Lopez-Colom et al. 2023	Pigs	Post-weaning diarrhoea (E.coli challenge)	I	Inactive.	At feeder; stand	
Reeves et al. (2024)	Sheep (lambs)	Gut parasite (Teladorsagia challenge)	P	Inactive; QBA characterised “fearfulness”.	Feeding.	

background work that can locate and track individual animals against complex backgrounds (e.g. pigs, Cowton et al., 2019; Gan et al., 2021; van der Zande, 2021).

4.2. *Limited specificity and sensitivity*

Despite the immense promise of new technologies in this area, Stachowicz and Umstatter (2021) concluded that PLF technologies with sufficient accuracy to achieve the goal of disease detection were still very rare.

One problem is a lack of specificity in relation to the core activities that are normally monitored as indicators of disease (described in Section 4). Indeed, remarkably similar changes are often seen in association with non-infectious conditions. For example, increased time spent lying down is observed both in younger cows with clinical infectious disease and in older cows that are lame for other reasons (Sepulveda-Varas et al., 2014). Postural changes are seen in chickens with avian influenza and Newcastle disease (He et al., 2022) but also in association with leg weakness related to rapid growth (Abeyesinghe et al., 2021). Similarly, altered usage of feeders is observed in pigs in association with infection but also with non-infectious lameness and tail wounds (reviewed by Bus et al., 2021). In dairy cows, reductions in visits to the feeder occur with metabolic conditions such as ketosis (Goldhawk et al., 2009). Stivanin et al. (2021) found that, although dry matter intake was lower for 3 breeds of cows with infections such as mastitis and metritis, these cows did not make fewer visits to the feed trough or spend less time there. Rather, feed trough behaviour was reduced in cows with a host of non-infectious conditions including dystocia, subclinical hypocalcaemia, clinical and subclinical ketosis. Even decreases in rumination time, which might be expected to be characteristic for infection-induced anorexia, were most strongly associated with subclinical ketosis and hypocalcaemia (systematic review, Sadiq et al., 2024). And automated sensors not only measure changing patterns in location or activity associated with infectious disease but also with metabolic conditions such as ruminal acidosis (Wagner et al., 2020) or general lameness (Lewis et al., 2023). Some of these overlaps may occur because inflammation is a shared feature of some infectious and non-infectious diseases.

However, it remains the case that core behaviours that are often described as intimately-linked with the acute immune response (Hart, 1988) show similar changes in association with many other conditions where acute immune activation is not a feature. Sometimes infectious and non-infectious diseases are considered together within a broader category of “ill health” (Weary et al., 2009). Indeed, a lack of specificity may not be a problem at all if the aim is to develop a generic system to detect welfare problems of any kind. This is often the intention of current advances in PLF e.g. the design of a system to detect ‘anomalous’ changes in chicken flock movements (Chen et al., 2023) or to detect generic changes in welfare (Bus et al., 2021). But the detection of infectious disease at its acute stage is important if the aim is to avoid onward transmission or to minimise the use of anti-microbials, and PLF approaches can play a role here too. It would seem useful to identify behaviours that are as specifically as possible linked with acute immune activation even if they are eventually incorporated into broader welfare-monitoring tools and protocols.

The second problem is that changes in core behaviours may not be particularly sensitive markers of infection, despite their wide occurrence across all husbandry systems and the fact that they are relatively easy to identify and interpret (Stachowicz and Umstatter, 2021). There are many situations where it would be valuable to develop more sensitive behavioural indicators. First, where the classic sickness behaviours of reduced feeding or activity are minimised due to partial or full pathogen tolerance. Second, where individuals have developed only mild infections. Third, during periods preceding the acute immune response, to allow the early detection of infection when there is still time for effective action to be taken to aid recovery or prevent disease spread, or during periods following acute infection to monitor recovery.

The next section suggests how improvements in specificity and sensitivity could be achieved.

4.2.1. *Improving specificity*

Granularity. Specificity may be improved by increasing granularity in the measurement of core behavioural changes. Bus et al. (2021) considered that more work was needed to establish detailed effects of disease on feeding patterns taking account factors such as time of day, age and breed and measuring more detailed parameters e.g. feeding frequency, visit duration, feeding rate and simultaneous feeding to enable sensitive and specific automated detection of disease. Another route to increased granularity is to focus on more than just frequency and duration of core behaviours but to their patterning over time. The measurement of behavioural complexity, assessed by transitions between different behavioural states, is a promising way of detecting subtle alterations in activity associated with disease (Rutherford et al., 2004; Asher et al., 2009). Some studies of free-living primates have confirmed that fractal analysis detects changes in the behavioural complexity of foraging behaviour associated with gut parasite or respiratory diseases (Alados and Huffman, 2000; MacIntosh et al., 2011). Fractal analysis also identified reduced complexity in the activity sequences of sheep infected with gut nematodes (Burgunder et al., 2018). However, it is essential to move beyond simple association studies and to evaluate which measures might be the most specific and predictive indicators of acute immune activation and infection.

Focus on other aspects of 'sickness behaviour'. Improved specificity could be achieved by monitoring changes in behaviours that are more specifically linked to immune activation than feeding and activity. For example, pronounced increases in sleep duration characterise mild and moderate infection (Krueger and Opps, 2016) and differentiate it from other forms of stress where sleeping time is generally reduced (Abou-Ismail et al., 2008). More work is needed to establish the effects of infection and other stressors on sleep quality (latency, fragmentation, proportion of REM sleep) as results from animal studies have not always supported human findings. In dogs, for example, negative emotional experience was associated with increased REM duration and shortened sleep latency (Kis et al., 2017). Another example is that cytokine-provoked changes to the basal ganglia can result in motor slowing (Capuron and Miller, 2011), which might be characterised by delays in motor initiation, impaired motor coordination and slower movement.

Not just immune resistance. An explicit recognition that animals respond in various ways to pathogenic threat could encourage the development of systems to detect upregulation of animals' disease avoidance strategies e.g. avoidance of other conspecifics, and systems that directly measure behavioural resistance to ectoparasites e.g. increased preening or grooming (Li et al., 2020; Nasiri et al., 2024)

Integration with Clinical symptoms. Core behaviour changes could be measured alongside clinical symptoms. Machine learning (ML) can detect specific clinical symptoms (e.g. coughs, sneezes or other abnormal sounds) of disease arising within groups or herds (chickens, Liu et al., 2020; pigs, Hong et al., 2020) and, since changes in health status can affect vocal parameters (Coutant et al., 2024), there is also potential for automated systems to detect altered vocalisation patterns. The development of artificial olfactory sensors that can analyse biomarkers of disease (e.g. in exhaled breath, Kim et al., 2022) provides yet another potential route for detection. Any one measure is likely to be subject to considerable error when taken from groups of animals living in challenging environmental farming conditions, so the future may lie in integrating multiple measures to build diagnostic profiles. He et al. (2022) advocate the integration of specific disease features and behavioural changes in the development of effective automated early warning of disease in poultry production.

4.2.2. Improving sensitivity

Pliant Behaviours. To detect subtle signs of infection e.g. under conditions of mild immune resistance, during periods that precede or follow full immune resistance, or under conditions of tolerance, it may be necessary to shift focus away from core behaviours. McFarland (1993) postulated that the brain prioritizes competing behavioural motivations based on the current situation and the individual's state. For instance, if an animal is very hungry, the motivation to eat will take precedence over other needs but, eventually hunger is satisfied and another motivation will take precedence. He also specified that some behaviours are more "resilient" than others. Core behaviours that are essential for immediate survival such as eating and drinking are maintained as far as possible even in the face of pressures such as limited energy or time resources, pain or discomfort. Other behaviours, including prophylactic grooming, exploration, play, (some) social activities and cognitively-demanding tasks can be postponed with less catastrophic effects and reinstated when pressures ease. These behaviours are sometimes called "less-resilient" or "luxury" behaviours. Neither term is particularly useful here. Behavioural resilience might be confused with disease resilience, whilst luxury implies something that is nice to possess, but not particularly essential, which is not the case. An alternative is to describe behaviours that are readily re-scheduled in response to varying pressures as "pliant".

Littin et al. (2008) and Weary et al. (2009) argued from slightly different starting points that pliant behaviours should be the most sensitive indicators of disease. When animals have urgent and competing motivational priorities (hunger, fear, pain, social demands) the most pliant behaviours will be rescheduled to allow core activities to continue. Stachowicz and Umstatter (2021) also argued that pliant behaviours should change earliest in the disease process and therefore be prime candidates for sensitive early indication of disease.

A few studies have demonstrated this almost incidentally. Immune-challenged mice built less complex nests (Gaskill et al., 2016). Immune-challenged rats were slower to learn a new operant task compared with controls, despite no differences in performance once the skill had been acquired (Aubert et al., 1995) and *Campylobacter*-infected crows were less likely to solve a string-pulling problem to obtain a food reward than healthy conspecifics (Townsend et al., 2022). Calves diagnosed with diarrhoea, respiratory disease or general debility reduced exploratory (unrewarded) visits to their feeder whilst continuing to make normal (rewarded) feeding visits (Svensson and Jensen, 2007) and goats infected with unspecified clinical conditions reduced their agonistic behaviour and social feeding but not their feed intake (Wolf et al., 2020).

These observations support the general hypothesis but these predictions have also been tested within a theoretical framework. Working in the context of disease detection in commercial dairy herds, Mandel et al. (2013) first identified a candidate pliant behaviour that could be monitored alongside the core behaviours of activity, rumination and lying. Rubbing against an automated brush showed the required pliancy under a disparate range of pressures. For example, brush use decreased by up to 50 % in response to an increased distance of the brush from food, higher temperature and humidity levels, and on days when intrusive artificial insemination was conducted (Mandel et al., 2013). Data on the daily brush use of individual cows was then recorded for 28 days postpartum using an automated system, alongside data on core behaviours. A proportion of cows developed metritis during the post-partum period and their behaviour was compared with that of healthy cows. Although no differences in brush usage were found before clinical diagnosis (and subsequent treatment) of metritis, a lower proportion of cows with metritis used the brushes between 8 and 21 days postpartum, and their daily duration of brush usage was 50 % lower than that of healthy individuals. These behavioural changes were apparent at early stages of the disease process before any elevation of body temperature. The unexpectedly slow return of this pliant behaviour also demonstrated that full recovery from infection had not occurred even after 2 weeks of medical treatment (Mandel et al., 2017). Reduced brush-use was also observed in a separate study in cows with moderate or severe lameness (Mandel et al., 2018). Despite such positive results, Stachowicz and Umstatter (2021) pointed out that, to their knowledge there was currently not one commercially available PLF system which uses pliant behaviour as an indicator.

Postponing grooming, social interaction or exploration may have few negative consequences in the short-term but will ultimately compromise survival or reproduction. To ensure that pliant behaviours are not suppressed for too long the motivation to perform them tends to increase with time since they were last performed. This has been shown for comfort, grooming and bathing behaviours (Nicol, 1987; Jones et al., 2009; Dickson et al., 2024), lying down (Metz, 1985; Schutz et al., 2024) and some, but not all, aspects of play (Bertelsen and Jensen, 2019). The implication is that reductions in pliant behaviours should be most apparent at the *earliest stages* of infection, allowing particularly sensitive detection at this point. If infection persists and these behaviours continue to be suppressed this indicates that the disease is having a more severe effect on the animals. A good indicator of full recovery might be the appearance of rebound in pliant behaviour where an animal makes up for lost time with more than a usual amount of play, exploration or grooming.

4.2.3. Integration

Even if monitoring pliant rather than core behaviour provides greater sensitivity it is unlikely to be particularly specific. By definition, pliant behaviours are affected by a wide range of pressures. As Mandel et al. (2013) showed, brush use in cattle is reduced by increasing energy demands, high temperatures, stress and discomfort. Brush-use is also minimal when cattle are newly arrived in feedlots until they have acclimatised to the new environment (Toaff-Rosenstein and Tucker, 2018). And changes in pliant behaviours are seen with non-infectious progressive diseases, including Huntington's disease (HD) (Littin et al., 2008).

The best approach may therefore be to deliberately monitor changes in pliant and core behaviours side by side, with appropriate prior hypotheses. Caplen and Held (2021) took this approach in a study aimed at detecting changes associated with subclinical mastitis in dairy cows. Matched pairs of healthy and infected cows (identified using an increase in somatic cell count) were observed for a 24 h period during the early infection stage. No differences between healthy and infected cows were detected for the core behaviours of time spent feeding, drinking or lying. However, infected cows showed fewer behavioural transitions and moved over a smaller area, indicating reduced activity, and there was also a negative correlation between feeding duration and specific marker of inflammation (serum amyloid alpha). Behaviours classified in advance as pliant (luxury) included social agonistic and social non-agonistic interactions, including social grooming, all of which were reduced in infected cows relative to controls although, unexpectedly not in brush use, possibly due to the central and accessible position of the brush in this barn. This thorough and detailed investigation (Caplen and Held, 2021) detected peripheral changes in core behaviours but, overall, it provides good support for the general principle that pliant behaviours offer the greatest potential for early disease detection.

As a general principle, an integrated approach will depend on animals having sufficient resources to express pliant behaviours in the first place. If animals are housed in barren conditions then it will be more difficult to detect subtle changes. In a direct test of this principle, Littin et al. (2008) housed mice in either conventional laboratory cages, or cages containing additional ropes, beams, ladders and exploratory chambers. Mice carrying a transgene for Huntington's disease (HD) were initially more active and exploratory than healthy controls but their activity declined with age. Declines in the pliant behaviour of climbing occurred up to 71 days before the HD mice showed obvious clinical symptoms. Thus, in addition to the direct value of environmental enrichment in improving animal welfare (e.g. Mandel et al., 2016), environments that promote a more diverse behavioural repertoire may have benefits for disease detection.

Studies to date have relied either on labour-intensive coding of behaviours from videos or on the automatic registration of the proximity of animals to resources that support pliant behaviours in specific locations. Developments and refinements in the discriminative performance of inertial sensors, algorithms and image data analysis techniques (see Rohan et al., 2024) to enable continuous monitoring of pliant behaviours will be required to reach the full potential of this approach (Stachowicz and Umstatter, 2021).

5. Promoting recovery, humane endpoints and developing therapies

The acute phase of infection-associated sickness behaviour typically lasts 1–3 days as cytokine levels rise quickly and return to base levels once the infection is under control (Dantzer and Kelley, 2007). However, even after peripheral inflammation subsides, CNS inflammation can sustain sickness behaviour for several days (Dantzer et al., 2008) and behavioural fever can also persist for 6–7 days in reptiles (Bernheim and Kluger, 1976). Under some circumstances, chronic sickness behaviour (with features of depression) can become associated with long-term cytokine dysregulation (Miller et al., 2009). The ways in which behaviour might change at these different stages have been insufficiently considered, with only some studies monitoring behaviour explicitly at different stages of infection (Mandel et al., 2017; Taylor et al., 2022), often finding that behavioural changes persist for some weeks after clinical signs have resolved (Szyszka et al., 2012; Lennon et al., 2023).

Many farm animals will find it hard to show sickness behaviours, due to high stocking densities and lack of facilities to hide or rest (Proudfoot et al., 2012). Millman (2007) argued that it was important to consider the needs of ill animals and those expressing sickness behaviour and yet found that many farms do not provide such facilities. This is a neglected area of research, including for animals that become chronically sick (Sundman et al., 2024). Knowledge of how animals behave when infected could be used to design environments where the costs of immune or behavioural resistance or tolerance are reduced and where recovery is promoted. The provision of appropriate sick pens would also enable closer behavioural monitoring to support decisions around humane endpoints if recovery is unlikely. Often sick animals are not euthanased until disease has progressed to a point of debilitation and collapse. Yet proof of concept exists that behavioural monitoring can predict recovery or non-recovery at a far earlier stage (Littin et al., 2008) and there are promising signs that this area of work is being taken forwards. Recently, a novel video-based system was developed identify changes in fish positioning within tanks indicative of infection, and with the aim of optimising the timing of humane endpoints (Bonnichsen et al., 2025). Developing these methods for application in farming has the potential to greatly reduce animal suffering.

6. Conclusions

The strategies that animals employ to avoid, resist or tolerate infection are more flexible than once assumed. A general hygienic avoidance of certain sites, secretions or individuals can be upregulated if an elevated risk of infection is perceived. The sickness behaviours that accompany immune resistance must also compete with other priorities and may be overshadowed in very hungry animals or those that are attempting to avoid pain or maintain social bonds. When the costs of immune or behavioural resistance are very high, selection may favour animals that are able to tolerate rather than eliminate a range of pathogens. There is a research need to develop reliable behavioural markers of tolerance.

Most attempts to use behaviour to detect infection have so far monitored changes in core behaviours like feeding or activity which are not particularly accurate, or have taken an hypothesis-free approach to detect differences between groups of animals known to be infected and those not using machine-learning, which may not generalise well to untrained situations. By incorporating new insights into immune system effects on behaviour, more targeted indicators – such as changes in sleep patterning, motor slowing and attention – can be proposed. Prospective studies are needed to confirm these behaviours as reliable indicators, while also accounting for contextual factors. For instance, infected animals in crowded, barren or hot environments may not exhibit classic sickness behaviours at all due to physical constraints and competing demands.

Under relatively benign conditions e.g. when monitoring animals experiencing mild infections, during recovery periods, or under conditions of high pathogen tolerance, core behaviours may be unaffected for a different reason - a lack of competing pressures. Here, infection may be detected by imposing behavioural challenges (e.g. social competition, restricted access to resources), although this may only be feasible for specific purposes such as selecting breeding animals or identifying super-spreaders. Pliant behaviours, like exploration, play, grooming and certain social interactions, could more easily serve as sensitive indicators of mild infection, but only if animals are housed in varied and enriched environments and with advances in monitoring technology. Pliant behaviours may also be the most sensitive way to monitor the effectiveness of new therapies for chronic conditions that persist beyond the acute phase, or of allergen-specific immunotherapies designed to reduce hypersensitivity reactions (e.g. Marsella et al., 2023) such as those described in Section 2.4.

Investment in enriched environments and in associated behavioural monitoring could ultimately be offset by earlier disease detection, improved therapies, faster recovery and reduced reliance on costly and potentially harmful treatments.

CRediT authorship contribution statement

Christine Nicol: Writing – original draft, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

Acknowledgements

I would like to thank Dr Andrew Douglas Wilson and Dr Siobhan Abeyesinghe for their insightful comments on an earlier draft as well as the three reviewers whose comments have helped to improve the review. I would especially like the thank the many colleagues who have worked with me over the years on the issues and concepts discussed.

References

Abeyesinghe, S., Chancellor, N., Moore, D., Chang, Y., Pearce, J., Demmers, T., Nicol, C., 2021. Associations between behaviour and health outcomes in conventional and slow-growing breeds of broiler chicken. Animal 15.

Abou-Ismail, U.A., Burman, O.H.P., Nicol, C.J., Mendl, M., 2008. Let sleeping rats lie: Does the timing of husbandry procedures affect laboratory rat behaviour, physiology and welfare? Appl.Anim. Behav. Sci. 111, 329–341.

Adamo, S., Bartlett, A., Le, J., Spencer, N., Sullivan, K., 2010. Illness-induced anorexia may reduce trade-offs between digestion and immune function. Anim. Behav. 79, 3–10.

Adelman, J., Hawley, D., 2017. Tolerance of infection: a role for animal behavior, potential immune mechanisms, and consequences for parasite transmission. Horm. Behav. 88, 79–86.

Alados, C., Huffman, M., 2000. Fractal long-range correlations in behavioural sequences of wild chimpanzees: a non-invasive analytical tool for the evaluation of health. Ethology 106, 105–116.

Almberg, E., Cross, P., Dobson, A., Smith, D., Metz, M., Stahler, D., Hudson, P., 2015. Social living mitigates the costs of a chronic illness in a cooperative carnivore. Ecol. Lett. 18, 660–667.

Alonso-Hearn, M., Badia-Bringue, G., Canive, M., 2022. Genome-wide association studies for the identification of cattle susceptible and resilient to paratuberculosis. Front. Vet. Sci. 9.

Amoroso, C., 2021. Integrating concepts of physiological and behavioral resistance to parasites. Front. Ecol. Evol. 9.

Amoroso, C., Antonovics, J., 2020. Evolution of behavioural resistance in host-pathogen systems. Biol. Lett. 16.

Arakawa, H., Arakawa, K., Deak, T., 2010. Oxytocin and vasopressin in the medial amygdala differentially modulate approach and avoidance behavior toward illness-related social odor. Neuroscience 171, 1141–1151.

Asher, L., Collins, L., Ortiz-Pelaez, A., Drewe, J., Nicol, C., Pfeiffer, D., 2009. Recent advances in the analysis of behavioural organization and interpretation as indicators of animal welfare. J. R. Soc. Interface 6, 1103–1119.

Aubert, A., 1999. Sickness and behaviour in animals: a motivational perspective. Neurosci. Biobehav. Rev. 23, 1029–1036.

Aubert, A., Goodall, G., Dantzer, R., Gheusi, G., 1997a. Differential effects of lipopolysaccharide on pup retrieving and nest building in lactating mice. Brain Behav. Immun. 11, 107–118.

Aubert, A., Kelley, K., Dantzer, R., 1997b. Differential effect of lipopolysaccharide on food hoarding behavior and food consumption in rats. Brain Behav. Immun. 11, 229–238.

Aubert, A., Vega, C., Dantzer, R., Goodall, G., 1995. Pyrogens specifically disrupt the acquisition of a task involving cognitive processing in the rat. Brain Behav. Immun. 9, 129–148.

Balabanov, R., Dore-Duffy, P., 1998. Cytokines and the blood-brain barriers. In: Pardridge, W.M. (Ed.), In: Introduction to the Blood-Brain Barrier: Methodology, Biology and Pathology. Cambridge University Press, pp. 354–361.

Barbour, K., McClune, D., Delahay, R., Speakman, J., McGowan, N., Kostka, B., Montgomery, W., Marks, N., Scantlebury, D., 2019. No energetic cost of tuberculosis infection in European badgers (*Meles meles*). J. Anim. Ecol. 88, 1973–1985.

Bauer, C., Weingarten, S., Senn, M., Langhans, W., 1995. Limited importance of a learned aversion in the hypophagic effect of interleukin-1-beta. Physiol. Behav. 57, 1145–1153.

Bernheim, H., Kluger, M., 1976. Fever - effect of drug-induced antipyresis on survival. Science 193, 237–239.

Berriatua, E., French, N., Broster, C., Morgan, K., Wall, R., 2001. Effect of infestation with *Psoroptes ovis* on the nocturnal rubbing and lying behaviour of housed sheep. Appl. Anim. Behav. Sci. 71, 43–55.

Bertelsen, M., Jensen, M., 2019. Does dairy calves' motivation for social play behaviour build up over time? Appl. Anim. Behav. Sci. 214, 18–24.

Blersch, R., Bonnell, T., Ganswindt, A., Young, C., Barrett, L., Henzi, S., 2021. Sick and tired: sickness behaviour, polyparasitism and food stress in a gregarious mammal. Behav. Ecol. Sociobiol. 75.

Bonnichsen, R., Nielsen, G.G.B., Dam, J.S., Schroder-Petersen, D., Buchmann, K., 2025. AI-driven realtime monitoring of early indicators for Ichthyophthirius multifiliis infection of rainbow trout. J. Fish. Dis. 48, e14027.

Burgunder, J., Petrzelková, K., Modry, D., Kato, A., MacIntosh, A., 2018. Fractal measures in activity patterns: do gastrointestinal parasites affect the complexity of sheep behaviour? Appl. Anim. Behav. Sci. 205, 44–53.

Bus, J., Boumans, I., Webb, L., Bokkers, E., 2021. The potential of feeding patterns to assess generic welfare in growing-finishing pigs. Appl. Anim. Behav. Sci. 241, e105383.

Bus, J., Boumans, I., Webb, L., Bokkers, E., 2024. Exploring individual responses to welfare issues in growing-finishing pig feeding behaviour. Animal 18, e101192.

Bush, S., Clayton, D., 2018. Anti-parasite behaviour of birds. Philos. Trans. R. Soc. B-Biol. Sci. 373.

Cabanzo-Olarte, L., Bícego, K., Iannini, C., 2024. Behavioral responses during sickness in amphibians and reptiles: concepts, experimental design, and implications for field studies. J. Therm. Biol. 123.

Cai, K., van Mil, S., Murray, E., Mallet, J., Matar, C., Ismail, N., 2016. Age and sex differences in immune response following LPS treatment in mice. Brain Behav. Immun. 58, 327–337.

Caplen, G., Held, S., 2021. Changes in social and feeding behaviors, activity, and salivary serum amyloid A in cows with subclinical mastitis. J. Dairy Sci. 104, 10991–11008.

Capuron, L., Miller, A., 2011. Immune system to brain signaling: neuropsychopharmacological implications. Pharmacol. Ther. 130, 226–238.

Carrignon, S., Bentley, R.A., Silk, M., Fefferman, N.H., 2022. How social learning shapes the efficacy of preventative health behaviors in an outbreak. PLOS ONE 17, e0262505.

Chen, B., Cheng, T., Huang, Y., Hsieh, Y., Hsu, H., Lu, C., Huang, M., Nien, S., Kuo, Y., 2023. Developing an automatic warning system for anomalous chicken dispersion and movement using deep learning and machine learning. Poult. Sci. 102.

Colles, F., Cain, R., Nickson, T., Smith, A., Roberts, S., Maiden, M., Lunn, D., Dawkins, M., 2016. Monitoring chicken flock behaviour provides early warning of infection by human pathogen Campylobacter. Proc. R. Soc. B-Biol. Sci. 283.

Coutant, M., Villain, A., Briefer, E., 2024. A scoping review of the use of bioacoustics to assess various components of farm animal welfare. Appl. Anim. Behav. Sci. 275.

Cowton, J., Kyriazakis, I., Bacardit, J., 2019. Automated individual pig localisation, tracking and behaviour metric extraction using deep learning. IEEE Access 7, 108049–108060.

Custódio, C., Mello, B., Chaves, A., Lima, C., Cordeiro, R., Miyajima, F., Réus, G., Vasconcelos, S., Barichello, T., Quevedo, J., de Oliveira, A., de Lucena, D., Macedo, D., 2018. Neonatal immune challenge with lipopolysaccharide triggers long-lasting sex- and age-related behavioral and immune/neurotrophic alterations in mice: relevance to autism spectrum disorders. Mol. Neurobiol. 55, 3775–3788.

Cyktor, J., Turner, J., 2011. Interleukin-10 and Immunity against Prokaryotic and Eukaryotic Intracellular Pathogens. Infect. Immun. 79, 2964–2973.

Dantzer, R., Kelley, K., 2007. Twenty years of research on cytokine-induced sickness behavior. Brain Behav. Immun. 21, 153–160.

Dantzer, R., O'Connor, J., Freund, G., Johnson, R., Kelley, K., 2008. From inflammation to sickness and depression: when the immune system subjugates the brain. Nat. Rev. Neurosci. 9, 46–57.

Daw, F. (2024) PhD thesis "The impact of sweet itch on equine behaviour, with sweet itch status defined by owners and by IgE reactivity, and the confounding effects of rug-wearing. Royal Veterinary College.

Dawkins, M., 2008. The science of animal suffering. Ethology 114, 937–945.

des Roches, A., Lussert, A., Faure, M., Herry, V., Rainard, P., Durand, D., Wemelsfelder, F., Foucras, G., 2018. Dairy cows under experimentally-induced

Escherichia coli mastitis show negative emotional states assessed through Qualitative Behaviour Assessment. Appl. Anim. Behav. Sci. 206, 1–11.

Devevey, G., Niculita-Hirzel, H., Biollaz, F., Yvon, C., Chapuisat, M., Christe, P., 2008. Developmental, metabolic and immunological costs of flea infestation in the common vole. Funct. Ecol. 22, 1091–1098.

Dickson, E., Monk, J., Lee, C., Mcdonald, P., Narayan, E., Campbell, D., 2024. Loss of a grooming enrichment impacts physical, behavioural, and physiological measures of welfare in grazing beef cattle. Animal 18.

Doeschl-Wilson, A., Knap, P., Opriessnig, T., More, S., 2021. Review: livestock disease resilience: from individual to herd level. Animal 15.

Ekman, P., Cordaro, D., 2011. What is meant by calling emotions basic. Emot. Rev. 3, 364–370.

Escobar, J., Van Alstine, W., Baker, D., Johnson, R., 2007. Behaviour of pigs with viral and bacterial pneumonia. Appl. Anim. Behav. Sci. 105, 42–50.

Evans, J.C., Hodgson, D.J., Boogert, N.J., Silk, M.J., 2021. Group size and modularity interact to shape the spread of infection and information through animal societies. Behav. Ecol. Sociobiol. 75, e163.

Ezeoke, C.C., Morley, J.E., 2015. Pathophysiology of anorexia in the cancer cachexia syndrome. J. Cachex-.-. Sarcopenia dn Muscle 6, 287–302.

Fruth, B., Ikombe, N., Matshimba, G., Metzger, S., Muganza, D., Mundry, R., Fowler, A., 2014. New Evidence for Self-Medication in Bonobos: *Manniophyton fulvum* Leaf- and Stemstrip-Swallowing From LuiKotale, Salonga National Park, DR Congo. Am. J. Primatol. 76, 146–158.

Fureix, C., Meagher, R., 2015. What can inactivity (in its various forms) reveal about affective states in non-human animals? A review. Appl. Anim. Behav. Sci. 171, 8–24.

Galef, B.G.Jr, 1996. Social enhancement of food preferences in Norway rats: a brief review. In: Heyes, C.M., Galef Jr, B.G. (Eds.), In: Social Learning in Animals: The Roots of Culture. Academic Press, London, pp. 49–64.

Gan, H., Ou, M., Zhao, F., Xu, C., Li, S., Chen, C., Xue, Y., 2021. Automated piglet tracking using a single convolutional neural network. Biosyst. Eng. 205, 48–63.

García, R., Aguilar, J., Toro, M., Pinto, A., Rodríguez, P., 2020. A systematic literature review on the use of machine learning in precision livestock farming. Comput. Electron. Agric. 179.

Garnett, B., Delahay, R., Roper, T., 2005. Ranging behaviour of European badgers (*Meles meles*) in relation to bovine tuberculosis (*Mycobacterium bovis*) infection. Appl. Anim. Behav. Sci. 94, 331–340.

Garrido, M., Adler, V., Pnini, M., Abramsky, Z., Krasnov, B., Gutman, R., Kronfeld-Schor, N., Hawlena, H., 2016. Time budget, oxygen consumption and body mass responses to parasites in juvenile and adult wild rodents. Parasites Vectors 9.

Gaskill, B., Pritchett-Corning, K., 2016. Nest building as an indicator of illness in laboratory mice. Appl. Anim. Behav. Sci. 180, 140–146.

Ghai, R., Fugère, V., Chapman, C., Goldberg, T., Davies, T., 2015. Sickness behaviour associated with non-lethal infections in wild primates. Proc. R. Soc. B-Biol. Sci. 282, 181–188.

Goessling, J., Guyer, C., Mendonça, M., 2017. More than fever: thermoregulatory responses to immunological stimulation and consequences of thermoregulatory strategy on innate immunity in gopher tortoises (*Gopherus polyphemus*). Physiol. Biochem. Zool. 90, 484–493.

Goldhawk, C., Chapinal, N., Veira, D., Weary, D., von Keyserlingk, M., 2009. Prepartum feeding behavior is an early indicator of subclinical ketosis. J. Dairy Sci. 92, 4971–4977.

González, M., Martínez-Carrasco, C., Sánchez-Zapata, J., Moleón, M., 2021. Smart carnivores think twice: red fox delays scavenging on conspecific carcasses to reduce parasite risk. Appl. Anim. Behav. Sci. 243.

Gordon, A.R., Lundström, J.N., Kimball, B.A., Karshikoff, B., Sorjonen, K., Axelsson, J., Lekander, M., Olsson, M.J., 2023. Human scent as a first-line defense against disease. Sci. Rep. 13, e16709.

Gormally, B., Bridgette, K., Emmi, A., Schuerman, D., Lopes, P., 2022. Female presence does not increase testosterone but still ameliorates sickness behaviours in male Japanese quail. R. Soc. Open Sci. 9.

Green, L., Lewis, K., Kimpton, A., Nicol, C., 2000. Cross-sectional study of the prevalence of feather pecking in laying hens in alternative systems and its associations with management and disease. Vet. Rec. 147, 233–238.

Hanotte, O., Ronin, Y., Agaba, M., Nilsson, P., Gelhaus, A., Horstmann, R., Sugimoto, Y., Kemp, S., Gibson, J., Korol, A., Soller, M., Teale, A., 2003. Mapping of quantitative trait loci controlling trypanotolerance in a cross of tolerant West African N'Dama and susceptible East African Boran cattle. Proc. Natl. Acad. Sci. USA 100, 7443–7448.

Hart, B., 2011. Behavioural defences in animals against pathogens and parasites: parallels with the pillars of medicine in humans. Philos. Trans. R. Soc. B-Biol. Sci. 366, 3406–3417.

HART, B., 1988. Biological basis of the behavior of sick animals. Neurosci. Biobehav. Rev. 12, 123–137.

Hayward, A., Nussey, D., Wilson, A., Berenos, C., Pilkington, J., Watt, K., Pemberton, J., Graham, A., 2014. Natural selection on individual variation in tolerance of gastrointestinal nematode infection. PLoS Biol. 12.

He, P., Chen, Z., Yu, H., Hayat, K., He, Y., Pan, J., Lin, H., 2022. Research progress in the early warning of chicken diseases by monitoring clinical symptoms. Appl. Sci. 12.

Healy, A., Hanlon, A., Weavers, E., Collins, J., Doherty, M., 2002. A behavioural study of scrapie-affected sheep. Appl. Anim. Behav. Sci. 79, 89–102.

Heil, M., 2016. Host manipulation by parasites: cases, patterns, and remaining doubts. Front. Ecol. Evol. 4.

Held, S., Spinka, M., 2011. Animal play and animal welfare. Anim. Behav. 81, 891–899.

Högberg, N., Hessle, A., Lidfors, L., Baltrusis, P., Claerebout, E., Höglund, J., 2021. Subclinical nematode parasitism affects activity and rumination patterns in first-season grazing cattle. Animal 15.

Hong, M., Ahn, H., Atif, O., Lee, J., Park, D., Chung, Y., 2020. Field-applicable pig anomaly detection system using vocalization for embedded board implementations. Appl. Sci. 10.

Hu, G., Do, D., Gray, J., Miar, Y., 2020. Selection for favorable health traits: a potential approach to cope with diseases in farm animals. Animals 10.

Huntingford, F., Rey, S., Quaggiotto, M., 2020. Behavioural fever, fish welfare and what farmers and fishers know. Appl. Anim. Behav. Sci. 231.

Hutchings, M., Knowler, K., McAnulty, R., McEwan, J., 2007. Genetically resistant sheep avoid parasites to a greater extent than do susceptible sheep. Proc. R. Soc. B-Biol. Sci. 274, 1839–1844.

Huzzey, J., Veira, D., Weary, D., von Keyserlingk, M., 2007. Prepartum behavior and dry matter intake identify dairy cows at risk for metritis. J. Dairy Sci. 90, 3220–3233.

Jenkins, H., Cox, D., Delahay, R., 2012. Direction of association between bite wounds and *Mycobacterium bovis* infection in badgers: implications for transmission. PLoS One 7.

Jog, M., Sackett, M., Kisty, S., Hansen, J., Stephenson, J., 2022. The behaviour of infected guppies depends on social context, parasite tolerance and host sex. Anim. Behav. 187, 97–104.

Johnston, A., Burne, T., Rose, S., 1998. Observation learning in day-old chicks using a one-trial passive avoidance learning paradigm. Anim. Behav. 56, 1347–1353.

Jones, T., Waitt, C., Dawkins, M., 2009. Water off a duck's back: showers and troughs match ponds for improving duck welfare. Appl. Anim. Behav. Sci. 116, 52–57.

Judge, J., Greig, A., Kyriazakis, I., Hutchings, M., 2005. Ingestion of faeces by grazing herbivores - risk of inter-species disease transmission. Agric. Ecosyst. Environ. 107, 267–274.

Kam, M., Khokhlova, I., Krasnov, B., Degen, A., 2011. Flea infestation does not cause a long-term increase in energy metabolism in *Gerbillus nanus*. J. Exp. Biol. 214, 3968–3971.

Kavaliers, M., Choleris, E., 2018. The role of social cognition in parasite and pathogen avoidance. Philos. Trans. R. Soc. B-Biol. Sci. 373.

Kavaliers, M., Colwell, D., Cloutier, C., Ossenkopp, K., Choleris, E., 2014. Pathogen threat and unfamiliar males rapidly bias the social responses of female mice. Anim. Behav. 97, 105–111.

Kavaliers, M., Wah, D.T.O., Bishnoi, I.R., Ossenkopp, K.P., Choleris, E., 2023. Disgusted snails, oxytocin, and the avoidance of infection threat. Horm. Behav. 155.

Kelly, C., Leroux, J., 2020. No evidence of sickness behavior in immune-challenged field crickets. Ecol. Evol. 10, 6049–6058.

Kempf, F., Cordoni, G., Chaussé, A., Drumo, R., Brown, H., Horton, D., Paboeuf, F., Denis, M., Velge, P., La Ragione, R., Kerouanton, A., 2023. Inflammatory responses induced by the monophasic variant of salmonella typhimurium in pigs play a role in the high shedder phenotype and fecal microbiota composition. Msystems 8.

Keymer, A., Crompton, D., Sahakian, B., 1983. Parasite-induced learned taste-aversion involving *nippostrongylus* in rats. Parasitology 86, 455–460.

Kim, C., Lee, K., Kang, M., Shin, D., Oh, J., Lee, C., Han, D., 2022. Artificial olfactory sensor technology that mimics the olfactory mechanism: a comprehensive review. Biomater. Res. 26.

Kis, A., Gergely, A., Galambos, A., Abdai, J., Gombos, F., Bodizs, R., Topal, J., 2017. Sleep macrostructure is modulated by positive and negative social experience in adult pet dogs. Proc. Royal. Soc. B. 284, 20171883.

Klasing, K., 1988. Influence of acute feed deprivation or excess feed-intake on immunocompetence of broiler chicks. Poult. Sci. 67, 626–634.

Krueger, J., Opp, M., 2016. Sleep and microbes. Gut Micro Behav. 207–225.

Kyriazakis, I., Tolkamp, B., Hutchings, M., 1998. Towards a functional explanation for the occurrence of anorexia during parasitic infections. Anim. Behav. 56, 265–274.

Langhans, W., 1996. Bacterial products and the control of ingestive behavior: clinical implications. Nutrition 12, 303–315.

Larson, S., Collins, S., Weingarten, H., 1996. Dissociation of temperature changes and anorexia after experimental colitis and LPS administration in rats. Am. J. Physiol. Regul. Integr. Comp. Physiol. 271, R967–R972.

Lasselin, J., Petrovic, P., Olsson, M., Göranson, S., Lekander, M., Jensen, K., Axelsson, J., 2018. Sickness behavior is not all about the immune response: possible roles of expectations and prediction errors in the worry of being sick. Brain Behav. Immun. 74, 213–221.

Laumer, I., Rahman, A., Rahmaeti, T., Azhari, U., Hermansyah, Atmoko, S., Schuppli, C., 2024. Active self-treatment of a facial wound with a biologically active plant by a male Sumatran orangutan. Sci. Rep. 14.

LeGrand, E., Alcock, J., 2012. Turning up the heat: immune brinksmanship in the acute-phase response. Q. Rev. Biol. 87, 3–18.

Lennon, R., Ronanki, S., Hegemann, A., 2023. Immune challenge reduces daily activity period in free-living birds for three weeks. Proc. R. Soc. B-Biol. Sci. 290.

Leshchinsky, T., Klasing, K., 2001. Divergence of the inflammatory response in two types of chickens. Dev. Comp. Immunol. 25, 629–638.

Leung, N., Xu, C., Li, J., Ganguly, A., Meyerhof, G., Regimbald-Dumas, Y., Lane, E., Breault, D., He, X., Perrimon, N., Montell, C., 2024. Gut tumors in flies alter the taste valence of an anti-tumorigenic bitter compound. Curr. Biol. 34.

Lewis, K., Price, E., Croft, D., Green, L., Ozella, L., Cattuto, C., Langford, J., 2023. Potential role of biologgers to automate detection of lame ewes and lambs. Appl. Anim. Behav. Sci. 259.

Li, G., Hui, X., Lin, F., Zhao, Y., 2020. Developing and evaluating poultry preening behavior detectors via mask region-based convolutional neural network. Animals 10.

Littin, K., Acevedo, A., Browne, W., Edgar, J., Mendl, M., Owen, D., Sherwin, C., Würbel, H., Nicol, C., 2008. Towards humane end points: behavioural changes precede clinical signs of disease in a Huntington's disease model. Proc. R. Soc. B-Biol. Sci. 275, 1865–1874.

Liu, L., Li, B., Zhao, R., Yao, W., Shen, M., Yang, J., 2020. A novel method for broiler abnormal sound detection using WMFCC and HMM. J. Sens. 2020.

Lochmiller, R., Deerenberg, C., 2000. Trade-offs in evolutionary immunology: just what is the cost of immunity. Oikos 88, 87–98.

Loehle, C., 1995. Social barriers to pathogen transmission in wild animal populations. Ecology 76, 326–335.

Lopes, P., 2014. When is it socially acceptable to feel sick. Proc. R. Soc. B-Biol. Sci. 281.

Lopes, P., 2017. Why are behavioral and immune traits linked. Horm. Behav. 88, 52–59.

Lopes, P., Adelman, J., Wingfield, J., Bentley, G., 2012. Social context modulates sickness behavior. Behav. Ecol. Sociobiol. 66, 1421–1428.

Lopes, P., Carlitz, E., Kindel, M., König, B., 2020. Immune-endocrine links to gregariousness in wild house mice. Front. Behav. Neurosci. 14.

López-Colom, P., Castillejos, L., Rodríguez-Sorrento, A., Mainau, E., Puyalto, M., Mallo, J., Martín-Orúe, S., 2023. Behavioural changes in weaned piglets orally challenged with Escherichia coli F4 and supplemented with in-feed protected acid salts. Appl. Anim. Behav. Sci. 261.

Lough, G., Hess, A., Hess, M., Rashidi, H., Matika, O., Lunney, J., Rowland, R., Kyriazakis, I., Mulder, H., Dekkers, J., Doeschl-Wilson, A., 2018. Harnessing longitudinal information to identify genetic variation in tolerance of pigs to Porcine Reproductive and Respiratory Syndrome virus infection. Genet. Sel. Evol. 50.

Lowe, G., Sutherland, M., Waas, J., Cox, N., Schaefer, A., Stewart, M., 2021. Effect of milk allowance on the suitability of automated behavioural and physiological measures as early disease indicators in calves. Appl. Anim. Behav. Sci. 234.

MacIntosh, A., Alados, C., Huffman, M., 2011. Fractal analysis of behaviour in a wild primate: behavioural complexity in health and disease. J. R. Soc. Interface 8, 1497–1509.

Mandel, R., Harazy, H., Gygax, L., Nicol, C., Ben-David, A., Whay, H., Klement, E., 2018. Detection of lameness in dairy cows using a grooming device. J. Dairy Sci. 101, 1511–1517.

Mandel, R., Nicol, C., Whay, H., Klement, E., 2017. Detection and monitoring of metritis in dairy cows using an automated grooming device. J. Dairy Sci. 100, 5724–5728.

Mandel, R., Whay, H.R., Klement, E., Nicol, C.J., 2016. Environmental enrichment of dairy cows and calves in indoor housing. J. Dairy Sci. 99, 1695–1715.

Mandel, R., Whay, H., Nicol, C., Klement, E., 2013. The effect of food location, heat load, and intrusive medical procedures on brushing activity in dairy cows. J. Dairy Sci. 96, 6506–6513.

Marais, M., Maloney, S., Gray, D., 2013. Sickness behaviours in ducks include anorexia but not lethargy. Appl. Anim. Behav. Sci. 145, 102–108.

Marsella, R., White, S., Fadok, V.A., Wilson, D., Mueller, R., Outerbridge, C., Rosenkrantz, W., 2023. Equine allergic skin diseases: clinical consensus guidelines of the World Association for Veterinary Dermatology. Vet. Dermatol. 34, 175–208.

Marti, E., Novotny, E., Cvitas, I., Ziegler, A., Wilson, A., Torsteinsdottir, S., Fettelschoss-Gabriel, A., Jonsdottir, S., 2021. Immunopathogenesis and immunotherapy of *Culicoides* hypersensitivity in horses: an update. Vet. Dermatol. 32, 579–e156.

Martin, C., Mullens, B., 2012. Housing and dustbathing effects on northern fowl mites (*Ornithonyssus sylviarum*) and chicken body lice (*Menacanthus stramineus*) on hens. Med. Vet. Entomol. 26, 323–333.

Matthews, S., Miller, A., Clapp, J., Plötz, T., Kyriazakis, I., 2016. Early detection of health and welfare compromises through automated detection of behavioural changes in pigs. Vet. J. 217, 43–51.

McFarland, D. (1993) Animal Behaviour. Longman.

McLennan, K., Rebelo, C., Corke, M., Holmes, M., Leach, M., Constantino-Casas, F., 2016. Development of a facial expression scale using footrot and mastitis as models of pain in sheep. Appl. Anim. Behav. Sci. 176, 19–26.

Mee, J., 2024. State-of-the-art sensors to monitor/manage dairy calf birth and calf health. Large Anim. Rev. 30, 61–66.

Meseguer, V., Alpizar, Y., Luis, E., Tajada, S., Denlinger, B., Fajardo, O., Manenschijn, J., Fernández-Peña, C., Talavera, A., Kichko, T., Navia, B., Sánchez, A., Señarís, R., Reeh, P., Perez-García, M., López-López, J., Voets, T., Belmonte, C., Talavera, K., Viana, F., 2014. TRPA1 channels mediate acute neurogenic inflammation and pain produced by bacterial endotoxins. Nat. Commun. 5.

Metz, J., 1985. The reaction of cows to a short-term deprivation of lying. Appl. Anim. Behav. Sci. 13, 301–307.

Miller, A., Maletic, V., Raison, C., 2009. Inflammation and its discontents: the role of cytokines in the pathophysiology of major depression. Biol. Psychiatry 65, 732–741.

Miller, L., Vicino, G., Sheftel, J., Lauderdale, L., 2020. Behavioral diversity as a potential indicator of positive animal welfare. Animals 10.

Millman, S., 2007. Sickness behaviour and its relevance to animal welfare assessment at the group level. Anim. Welf. 16, 123–125.

Moinard, C., Mendl, M., Nicol, C., Green, L., 2003. A case control study of on-farm risk factors for tail biting in pigs. Appl. Anim. Behav. Sci. 81, 333–355.

Morelle, K., Barasona, J., Bosch, J., Heine, G., Daim, A., Arnold, J., Bauch, T., Kosowska, A., Cadenas-Fernández, E., Aviles, M., Zuñiga, D., Wikelski, M., Vizcaino-Sanchez, J., Safi, K., 2023. Accelerometer-based detection of African swine fever infection in wild boar. Proc. R. Soc. B-Biol. Sci. 290.

Morris, A., Innocent, G., Cunningham, E., Athanasiadou, S., Hutchings, M., Smith, L., 2022. Early signals of parasitism expressed through behaviour but modulated by social context. Anim. Behav. 193, 157–179.

Munsterhjelm, C., Nordgreen, J., Aae, F., Heinonen, M., Valros, A., Janczak, A., 2019. Sick and grumpy: changes in social behaviour after a controlled immune stimulation in group-housed gilts. Physiol. Behav. 198, 76–83.

Murdock, C., Luckhart, S., Cator, L., 2017. Immunity, host physiology, and behaviour in infected vectors. Curr. Opin. Insect Sci. 20, 28–33.

Murillo, A., Abdoli, A., Blatchford, R., Keogh, E., Gerry, A., 2020. Parasitic mites alter chicken behaviour and negatively impact animal welfare. Sci. Rep. 10.

Murillo, A., Abdoli, A., Blatchford, R., Keogh, E., Gerry, A., 2024. Low levels of chicken body louse (*Menacanthus stramineus*) infestations affect chicken welfare in a cage-free housing system. Parasites Vectors 17.

Murillo, A., Chappell, M., Owen, J., Mullens, B., 2016. Northern fowl mite (*Ornithonyssus sylviarum*) effects on metabolism, body temperatures, skin condition, and egg production as a function of hen MHC haplotype. Poult. Sci. 95, 2536–2546.

Murillo, A., Mullens, B., 2016. Timing diatomaceous earth-filled dustbox use for management of northern fowl mites (*Acari: Macronyssidae*) in cage-free poultry systems. J. Econ. Entomol. 109, 2572–2579.

Murray, M., Murray, A., 1979. Anorexia of infection as a mechanism of host defense. Am. J. Clin. Nutr. 32, 593–596.

Nakov, D., Hristov, S., Stankovic, B., Pol, F., Dimitrov, I., Ilieski, V., Mormede, P., Nervé, J., Terenina, E., Lieubeau, B., Papanastasiou, D., Bartzanas, T., Norton, T., Piette, D., Tullo, E., van Dixhoorn, D., 2019. Methodologies for assessing disease tolerance in pigs. Front. Vet. Sci. 5.

Nasiri, A., Zhao, Y., Gan, H., 2024. Automated detection and counting of broiler behaviors using a video recognition system. Comput. Electron. Agric. 221.

Nicol, C.J., 1987. Behavioural-responses of laying hens following a period of spatial restriction. Anim. Behav. 35, 1709–1719.

Nicol, C.J., Caplen, G., Edgar, J., Browne, W., 2009. Associations between welfare indicators and environmental choice in laying hens. Anim. Behav. 78, 413–424.

Nilsson, A., Wilhelms, D., Mirrasekhian, E., Jaarola, M., Blomqvist, A., Engblom, D., 2017. Inflammation-induced anorexia and fever are elicited by distinct prostaglandin dependent mechanisms, whereas conditioned taste aversion is prostaglandin independent. Brain Behav. Immun. 61, 236–243.

Oliveira, D., Pereira, L., Bresolin, T., Ferreira, R., Dorea, J., 2021. A review of deep learning algorithms for computer vision systems in livestock. Livest. Sci. 253.

Olomski, F., Fettelschoss, V., Jonsdottir, S., Birkmann, K., Thoms, F., Marti, E., Bachmann, M., Kündig, T., Fettelschoss-Gabriel, A., 2020. Interleukin 31 in insect bite hypersensitivity-Alleviating clinical symptoms by active vaccination against itch. Allergy 75, 862–871.

Paul, E., Browne, W., Mendl, M., Caplen, G., Trevarthen, A., Held, S., Nicol, C., 2022. Assessing animal welfare: a triangulation of preference, judgement bias and other candidate welfare indicators. Anim. Behav. 186, 151–177.

Perier, N., des Roches, A., Jensen, M., Proudfoot, K., 2019. Infectious disease does not impact the lying and grooming behaviour of post-parturient dairy cows. Animals 9.

Pickard, J., Maurice, C., Kinnebrew, M., Abt, M., Schenten, D., Golovkina, T., Bogatyrev, S., Ismagilov, R., Pamer, E., Turnbaugh, P., Chervonsky, A., 2014. Rapid fucosylation of intestinal epithelium sustains host-commensal symbiosis in sickness. Nature 514, 638.

PlataSalaman, C., 1996. Anorexia during acute and chronic disease. Nutrition 12, 69–78.

Pötzsch, C., Lewis, K., Nicol, C., Green, L., 2001. A cross-sectional study of the prevalence of vent pecking in laying hens in alternative systems and its associations with feather pecking, management and disease. Appl. Anim. Behav. Sci. 74, 259–272.

Proudfoot, K., Weary, D., von Keyserlingk, M., 2012. Linking the social environment to illness in farm animals. Appl. Anim. Behav. Sci. 138, 203–215.

Rao, S., Schieber, A., O'Connor, C., Leblanc, M., Michel, D., Ayres, J., 2017. Pathogen-mediated inhibition of anorexia promotes host survival and transmission. Cell 168, 503.

Reeves, M., Booth, N., Fox, N., Donbavand, J., Jack, M., Kenyon, F., Martin, J., Baxter, E., Dwyer, C., 2024. The impact of gastrointestinal parasitism on the behaviour and welfare of weaned housed lambs. Appl. Anim. Behav. Sci. 276.

Reiber, C., Shattuck, E., Fiore, S., Alperin, P., Davis, V., Moore, J., 2010. Change in human social behavior in response to a common vaccine. Ann. Epidemiol. 20, 729–733.

Reiner, G., Hübner, K., Hepp, S., 2009. Suffering in diseased pigs as expressed by behavioural, clinical and clinical-chemical traits, in a well defined parasite model. Appl. Anim. Behav. Sci. 118, 222–231.

Renault, J., Gheusi, G., Aubert, A., 2008. Changes in social exploration of a lipopolysaccharides-treated conspecific in mice: role of environmental cues. Brain Behav. Immun. 22, 1201–1207.

Rivas, F., Chervonsky, A., Medzhitov, R., 2014. ART and immunology. Trends Immunol. 35, 451-451.

Rohan, A., Rafaq, M.S., Hasan, M.J., Asghar, F., Bashir, A.K., Dottorini, T., 2024. Application of deep learning for livestock behaviour recognition: a systematic literature review. Comput. Electron. Agric. 224, e109115.

Russell, B., Adams, W., Hrelja, K., Zeeb, F., Taves, M., Kaur, S., Soma, K., Winstanley, C., 2022. Differential effects of lipopolysaccharide on cognition, corticosterone and cytokines in socially-housed vs isolated male rats. Behav. Brain Res. 433.

Rutherford, K., Haskell, M., Glasbey, C., Jones, R., Lawrence, A., 2004. Fractal analysis of animal behaviour as an indicator of animal welfare. Anim. Welf. 13, S99–S103.

Sadiq, M., Zaini, S., Mossadeq, W., Ramanoon, S., Syed-Hussain, S., 2024. Lying and rumination time as predictors of subclinical ketosis, metritis, and hypocalcaemia in dairy cows during the periparturient period: a systematic review and meta-analysis. Appl. Anim. Behav. Sci. 278.

Sakkas, P., Oikeh, I., Blake, D., Nolan, M., Bailey, R., Oxley, A., Rychlik, I., Lietz, G., Kyriazakis, I., 2018. Does selection for growth rate in broilers affect their resistance and tolerance to *Eimeria maxima*? Vet. Parasitol. 258, 88–98.

Sarabian, C., Wilkinson, A., Sigaud, M., Kano, F., Tobajas, J., Darmaillacq, A., Kalema-Zikusoka, G., Plotnik, J., MacIntosh, A., 2023. Disgust in animals and the application of disease avoidance to wildlife management and conservation. J. Anim. Ecol. 92, 1489–1508.

Sauer, E., Trejo, N., Hoverman, J., Rohr, J., 2019. Behavioural fever reduces ranaviral infection in toads. Funct. Ecol. 33, 2172–2179.

Scantlebury, M., Waterman, J., Hillegass, M., Speakman, J., Bennett, N., 2007. Energetic costs of parasitism in the Cape ground squirrel *Xerus inauris*. Proc. R. Soc. B-Biol. Sci. 274, 2169–2177.

Schaller, M., Miller, G., Gervais, W., Yager, S., Chen, E., 2010. Mere visual perception of other people's disease symptoms facilitates a more aggressive immune response. Psychol. Sci. 21, 649–652.

Schaller, M., Murray, D., 2008. Pathogens, personality, and culture: disease prevalence predicts worldwide variability in sociosexuality, extraversion, and openness to experience. J. Personal. Soc. Psychol. 95, 212–221.

Schaller, M., Park, J., 2011. The behavioral immune system (and why it matters). Curr. Dir. Psychol. Sci. 20, 99–103.

Schoenle, L., Schoepf, I., Weinstein, N., Moore, I., Bonier, F., 2018. Higher plasma corticosterone is associated with reduced costs of infection in red-winged blackbirds. Gen. Comp. Endocrinol. 256, 89–98.

Schofield, G., Papafitsoros, K., Haughey, R., Katselidis, K., 2017. Aerial and underwater surveys reveal temporal variation in cleaning-station use by sea turtles at a temperate breeding area. Mar. Ecol. Prog. Ser. 575, 153–164.

Schreier, K., Grindstaff, J., 2020. Repeatable behavioural and immune defence strategies against infection are not traded off. Anim. Behav. 162, 11–22.

Schütz, K., Latimer, B., Mcdonald, N., Hunter, L., Huddart, F., Watson, T., Saunders, L., Kells, N., Cox, N., Monaghan, R., 2024. Effects of 2 wintering practices on behavioral and physiological indicators of welfare of nonlactating, pregnant dairy cattle in a pasture-based system. J. Dairy Sci. 107, 7079–7091.

Seal, S., Dharmarajan, G., Khan, I., 2021. Evolution of pathogen tolerance and emerging infections: a missing experimental paradigm. ELIFE 10.

Sepúlveda-Varas, P., Proudfoot, K., Weary, D., von Keyserlingk, M., 2016. Changes in behaviour of dairy cows with clinical mastitis. Appl. Anim. Behav. Sci. 175, 8–13.

Sepúlveda-Varas, P., Weary, D., von Keyserlingk, M., 2014. Lying behavior and postpartum health status in grazing dairy cows. J. Dairy Sci. 97, 6334–6343.

Sherwin, C., Heyes, C., Nicol, C., 2002. Social learning influences the preferences of domestic hens for novel food. Anim. Behav. 63, 933–942.

Siivonen, J., Taponen, S., Hovinen, M., Pastell, M., Lensink, B., Pyörälä, S., Hänninen, L., 2011. Impact of acute clinical mastitis on cow behaviour. Appl. Anim. Behav. Sci. 132, 101–106.

Silverman, M., Macdougall, M., Hu, F., Pace, T., Raison, C., Miller, A., 2007. Endogenous glucocorticoids protect against TNF-alpha-induced increases in anxiety-like behavior in virally infected mice. Mol. Psychiatry 12, 408–417.

Sinha, P., Guerrant, R., 2023. The costly vicious cycle of infections and malnutrition. J. Infect. Dis. 229, 1611–1613.

Soares, M., Teixeira, L., Moita, L., 2017. Disease tolerance and immunity in host protection against infection. Nat. Rev. Immunol. 17, 83–96.

Somagond, A., Patel, B., Pattanaik, A., Verma, M., Krishnaswamy, N., Periyasamy, T., Gaur, G., Biswal, P., Yadav, S., Dutt, T., Bhanuprakash, V., 2023. Effect of physical form of the therapeutic diet on the behaviour of crossbred calves experimentally infected with foot-and-mouth disease virus. Prev. Vet. Med. 212.

Spencer, S., Heida, J., Pittman, Q., 2005. Early life immune challenge - effects on behavioural indices of adult rat fear and anxiety. Behav. Brain Res. 164, 231–238.

Stachowicz, J., Umstätter, C., 2021. Do we automatically detect health- or general welfare-related issues? A framework. Proc. R. Soc. B-Biol. Sci. 288.

Stivanin, S., Vizzotto, E., Matiello, P., Machado, F., Campos, M., Tomich, T., Pereira, L., Fischer, V., 2021. Behavior, feed intake and health status in Holstein, Gyr and Girolando-F1 cows during the transition period Behavior and health of dairy cows in the transition period. Appl. Anim. Behav. Sci. 242.

Stockmaier, S., Ulrich, Y., Albery, G., Cremer, S., Lopes, P., 2023. Behavioural defences against parasites across host social structures. Funct. Ecol. 37, 809–820.

Sullivan, K., Fairn, E., Adamo, S., 2016. Sickness behaviour in the cricket *Gryllus texensis*: comparison with animals across phyla. Behav. Process. 128, 134–143.

Sundman, E., Millman, S., Schwartzkopf-Genswein, K., Dewell, R., Perrett, T., Booker, C., Erickson, S., Silva, G., Thomson, D., Johnson, A., Dewell, G., 2024. Assessment of chronically ill or injured feedlot cattle populations at eight United States and nine Canadian feedlots and implications for cattle welfare. J. Anim. Sci. 102, 11-11.

Svensson, C., Jensen, M., 2007. Identification of diseased calves by use of data from automatic milk feeders. J. Dairy Sci. 90, 994–997.

Swartz, T., Schramm, H., Petersson-Wolfe, C., 2020. Short communication: association between neonatal calf diarrhea and lying behaviors. Vet. Anim. Sci. 9.

Swift, B., Barron, E., Christley, R., Corbetta, D., Grau-Roma, L., Jewell, C., O'Cathail, C., Mitchell, A., Phoenix, J., Prosser, A., Rees, C., Sorley, M., Verin, R., Bennett, M., 2021. Tuberculosis in badgers where the bovine tuberculosis epidemic is expanding in cattle in England. Sci. Rep. 11.

Szyszka, O., Tolkamp, BJ., Edwards, SA., Kyriazakis, I., 2012. he effects of acute versus chronic health challenges on the behavior of beef cattle. J. Anim. Sci. 90, 4308–4318.

Taylor, J., Sakkas, P., Kyriazakis, I., 2022. Starving for nutrients: anorexia during infection with parasites in broilers is affected by diet composition. Poult. Sci. 101.

Thomas, A., Orsel, K., Cortés, J., Pajor, E., 2021. Impact of digital dermatitis on feedlot cattle behaviour. Appl. Anim. Behav. Sci. 244.

Toaff-Rosenstein, R., Gershwin, L., Zanella, A., Tucker, C., 2016. The sickness response in steers with induced bovine respiratory disease before and after treatment with a non-steroidal anti-inflammatory drug. Appl. Anim. Behav. Sci. 181, 49–62.

Toaff-Rosenstein, R., Tucker, C., 2018. The sickness response at and before clinical diagnosis of spontaneous bovine respiratory disease. Appl. Anim. Behav. Sci. 201, 85–92.

Toscano, M., Sait, L., Jorgensen, F., Nicol, C., Powers, C., Smith, A., Bailey, M., Humphrey, T., 2010. Sub-clinical infection with Salmonella in chickens differentially affects behaviour and welfare in three inbred strains. Br. Poult. Sci. 51, 703–713.

Townsend, A., Johansson, E., Danielson, A., Boyd, A., Egey, E., Winner, K., 2022. Infection impairs problem-solving performance in American crows. Anim. Behav. 184, 11–17.

Tu, T., Phillips, A., Koski, K., Scott, M., 2008. Acute phase nematode infection reduces resting metabolic rate in both protein-sufficient and protein-deficient mice. Can. J. Zool. 86, 1432–1436.

Turnbull, A.V., Rivier, C.l., 1999. Regulation of the hypothalamic-pituitary-adrenal axis by cytokines: Actions and mechanisms of action. Physiol. Rev. 79, 1–71.

van der Zande, L., Guzhva, O., Rodenburg, T., 2021. Individual detection and tracking of group housed pigs in their home pen using computer vision. Front. Anim. Sci. 2.

van Leeuwen, F., Petersen, M., 2018. The behavioral immune system is designed to avoid infected individuals, not outgroups. Evol. Hum. Behav. 39, 226–234.

Veit, C., Foister, S., Valros, A., Munsterhjelm, C., Sandercock, D., Janczak, A., Ranheim, B., Nordgreen, J., 2021. The use of social network analysis to describe the effect of immune activation on group dynamics in pigs. Animal 15.

Vezzoli, G., Mullens, B., Mench, J., 2015a. Dustbathing behavior: do ectoparasites matter? Appl. Anim. Behav. Sci. 169, 93–99.

Vezzoli, G., Mullens, B., Mench, J., 2015b. Relationships between beak condition, preening behavior and ectoparasite infestation levels in laying hens. Poult. Sci. 94, 1997–2007.

Wagner, N., Antoine, V., Mialon, M., Lardy, R., Silberberg, M., Koko, J., Veissier, I., 2020. Machine learning to detect behavioural anomalies in dairy cows under subacute ruminal acidosis. Comput. Electron. Agric. 170.

Wang, S., Jiang, H., Qiao, Y., Jiang, S., Lin, H., Sun, Q., 2022. The research progress of vision-based artificial intelligence in smart pig farming. SENSORS 22.

Warwick, C., 1991. Observations on disease-associated preferred body temperatures in reptiles. Appl. Anim. Behav. Sci. 28, 375–380.

Watkins, L., Wiertelak, E., Goehler, L., Smith, K., Martin, D., Maier, S., 1994. Characterization of cytokine-induced hyperalgesia. Brain Res. 654, 15–26.

Weary, D., Huzzey, J., von Keyserlingk, M., 2009. Using behavior to predict and identify ill health in animals. J. Anim. Sci. 87, 770–777.

Webster, J., Kaushik, M., Bristow, G., McConkey, G., 2013. *Toxoplasma gondii* infection, from predation to schizophrenia: can animal behaviour help us understand human behaviour? J. Exp. Biol. 216, 99–112.

Weeks, C., Nicol, C., Titchener, R., 1995. Effects of the sucking louse (*Linognathus-Vituli*) on the grooming behavior of housed calves. Vet. Rec. 137, 33–35.

Williams, E., Davis, C., Williams, M., Jones, D., Cutress, D., Williams, H., Brophy, P., Rose, M., Stuart, R., Jones, R., 2022. Associations between gastrointestinal nematode infection burden and lying behaviour as measured by accelerometers in periparturient ewes. Animals 12.

Wilson, A., 2014. Immune responses to ectoparasites of horses, with a focus on insect bite hypersensitivity. Parasite Immunol. 36, 558–570.

Wing, E., Young, J., 1980. Acute starvation protects mice against *Listeria-monocytogenes*. Infect. Immun. 28, 771–776.

Wingfield, J., 2003. Control of behavioural strategies for capricious environments. Anim. Behav. 66, 807–815.

Wolf, T., Lazarus, D., Opperman, P., Heath, L., Ganswindt, A., Fosgate, G., 2020. Impact of foot-and-mouth-disease on goat behaviour after experimental infection with serotype SAT1 virus. Prev. Vet. Med. 176.

Woodroffe, R., Donnelly, C., Ham, C., Jackson, S., Moyes, K., Chapman, K., Stratton, N., Cartwright, S., 2017. Ranging behaviour of badgers *Meles meles* vaccinated with Bacillus Calmette Guerin. J. Appl. Ecol. 54, 718–725.

Zanella, R., Settles, M., McKay, S., Schnabel, R., Taylor, J., Whitlock, R., Schukken, Y., Van Kessel, J., Smith, J., Neibergs, H., 2011. Identification of loci associated with tolerance to Johne's disease in Holstein cattle. Anim. Genet. 42, 28–38.

Zou, A., Nadeau, K., Wang, P., Lee, J., Guttman, D., Sharif, S., Korver, D., Brumell, J., Parkinson, J., 2020. Accumulation of genetic variants associated with immunity in the selective breeding of broilers. BMC Genet. 21.