Chapter 3
The Evolution of Disgust, Pathogens, and the Behavioural Immune System

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When considering factors that posed a threat to survival throughout evolutionary history, things like lions, tigers, bears, inclement weather, and environmental catastrophes readily come to mind. However, the most potent and ubiquitous selection pressure shaping human evolution arises from microorganisms (i.e., parasites, viruses, bacteria) that are typically too small to be seen by the naked eye. For instance, evidence from small scale forager-horticulturalist societies demonstrates that infectious diseases are responsible for over half of all deaths (Gurven et al. 2007). As such, humans and other animals have developed a repertoire of emotional, cognitive, behavioural, and physiological mechanisms that function as protection against the persistent threat posed by infection and illness.

In the current chapter, we first provide an overview of past literature supporting the reasoning that key aspects of our psychology—disgust and the behavioural immune system—represent adaptations to the selection pressures posed by infectious microorganisms. Although disgust has been shown to underlie moral reasoning, we limit our focus here to implications directly concerning infection and illness. We end by presenting innovative ideas bearing on disgust and the behavioural immune system, which incorporate perspectives from psychology, ecology, and psychoneuroimmunology.

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Disgust: An Infection-Avoidance Adaptation

Characterised by a feeling of revulsion, the emotion of disgust is a major player in our evolved strategies to minimise the risk of disease and illness. Evidence supporting this proposition emerges from different research methodologies exploring the various elicitors of disgust. Qualitative, cross-cultural work focusing on what people find disgusting shows that responses centre around potential vectors for disease and infectious microorganisms: bodily excretions (e.g., faeces), decay and rotten food, certain animals (e.g., cockroaches, lice, rats), and certain people (e.g., sick or unhygienic people; Curtis and Biran 2001). Other research including over 40,000 participants across a wide variety of cultures illustrates that people evaluate visual stimuli containing cues of disease to be more disgusting than similar stimuli without disease cues (Curtis et al. 2004). Cues of infection detected by other sensory systems similarly elicit disgust. For instance, people commonly find the smell of decay, faeces, and vomit to be disgusting (Croy et al. 2011; Glass et al. 2014), and tactile cues of moisture, which are indicative of pathogen presence, also elicit disgust (Oum et al. 2011; Saluja and Stevenson 2019). Together, these findings demonstrate remarkable consistency in the cues that elicit disgust, indicating that disgust is intimately tied to threats of disease and contamination.

Additional research finds that disgust is associated with distinct changes in perception and behaviour. For instance, when disgust is experimentally induced, people exhibit greater sensitivity to touch (Hunt et al. 2017) and smells (Chan et al. 2019). Such perceptual changes, which occur in the context of disgust, might function to better facilitate the detection of cues of contamination. Disgust is further related to increased behavioural avoidance of potential contaminants (Campbell et al. 2019; Dorfan and Woody 2011; Olatunji et al. 2014) and greater likelihood of hygienic behaviour after exposure to potential contaminants (e.g., handwashing; Porzig-Drummond et al. 2009). The results of this work are consistent with the reasoning that disgust represents an adaptation to the selection pressure posed by disease and infection, by motivating avoidance of potential contaminants (Curtis et al. 2011; Tybur et al. 2013).

Of importance to note here is that accurate knowledge regarding the transmission of illness and disease is not a prerequisite for feeling disgust towards or being motivated to avoid contaminants. Lay beliefs that disease and infection are spread by contact predate modern theories about disease transmission (Karamanou et al. 2012). For example, the set of rules for dealing with leprosy outlined in Leviticus emphasise quarantining and isolating individuals with the disease, presumably to prevent transmission (*King James Bible* 1769/ 2008, Leviticus 13). Moreover, disgust and hygienic practices that serve to reduce infection risk exist across history and cultures, long before scientists gained knowledge of bacteria and viruses (Curtis 2007). Our ancestors who felt disgust towards potential contaminants would have been better able to avoid disease and infection than those who did not, thus facilitating survival and reproduction (though, the trade-off between anti-pathogen defences and reproduction is not straightforward; Borg and de Jong, Chap. 9, this

volume). The disease-avoidance advantage of disgust would have led to such traits being more frequent in subsequent populations. Recent research finds approximately half of the variation in disgust sensitivity is due to underlying genetic factors (Sherlock et al. 2016), which suggests that disgust sensitivity is, to some degree, heritable. Obviously, it is impossible to invent a time machine to study the presence of disgust and the survival advantage it may have conferred in ancestral times. However, the presence of disgust across history, its functional correlates, and the cross-cultural ubiquity of disgust elicitors point to the existence of disgust as an evolved mechanism against disease.

More Broadly: The Behavioural Immune System

Disgust, however, is just the tip of the iceberg when it comes to evolved disease avoidance mechanisms. Within the last decade, research has established that, much like other animals, humans possess a suite of perceptual, cognitive, and behavioural components, which strategically function to defend against the threat of illness and infection. Together, these defences are referred to as the *behavioural immune system* (Schaller and Park 2011). Below, we discuss the evidence related to each of these components.

Beforehand, it bears noting that there is some disagreement in the literature regarding the relationship between disgust and the behavioural immune system. Some argue that these constructs are functionally identical (Lieberman and Patrick 2014). According to this perspective, given that disgust and the behavioural immune system rely on the same input (i.e., cues of contamination), involve the same computational processes (e.g., estimating potential cost of infection), and produce functionally similar outputs (e.g., avoidance), the distinction between the two is merely semantic. Others claim that, while disgust and the behavioural immune system can operate in concert, disgust is not a necessary prerequisite for the activities of the behavioural immune system (Schaller 2014). That is, people engage in prophylactic (e.g., handwashing) or culturally normative (e.g., hygiene) behaviour that pre-emptively serves to mitigate the threat of infection without consciously experiencing disgust. This disagreement is, as of yet, unsettled; future research is needed to delineate the extent to which disgust is or is not intrinsic to the activity of the behavioural immune system.

Perception and Cognition

The behavioural immune system is theorised to guide the detection of cues indicative of illness and infection. Research in this vein has explored whether individuals can detect the presence of illness in others through various sensory modalities. For instance, in one study, participants were shown faces of targets that had either

been injected with lipopolysaccharide (LPS), a bacterial stimulus that triggers an immune response, or a placebo, and were instructed to rate whether the target was sick or healthy (Axelsson et al. 2018). The results provided evidence that people identified the individuals injected with LPS as sick above chance level. Other research has used similar acute illness (vs. placebo) inductions to examine how people evaluate the health of targets based on smell (Olsson et al. 2014) or videos of their gait patterns (Sundelin et al. 2015). Compared to targets who were injected with the placebo, the scent and gait patterns of sick targets were rated to be less healthy by those injected with the immune trigger. The findings of this work demonstrate that our sensory systems are functionally tuned to detect cues of sickness and infection in conspecifics.

Other research has examined how the behavioural immune system influences key aspects of cognition, such as attention and memory. For instance, visual cues of contaminants (e.g., vomit) draw and hold people's attention (van Hooff et al. 2014; Vogt et al. 2011). Not only do people pay more attention towards potential sources of infection, they also exhibit increased memory for these stimuli. Research in this vein finds that images that elicit disgust are remembered better than neutral images or images that elicit fear (Chapman 2018). Moreover, people exhibit better memory for items that were touched by purportedly sick (vs. healthy) targets (Bonin et al. 2019; Fernandes et al. 2017).

Increased attention to, and memory for, sources of infection may help facilitate disease avoidance while individuals navigate their social world. For example, avoiding potential sources of infection first requires identifying them. Accordingly, being especially attuned to cues connoting disease risk—particularly when those cues are subtle—is key to appropriately activating emotional reactions, like disgust, that promote avoidance behaviours. Additionally, heightened memory for disgusting stimuli may help individuals avoid second encounters with situations conferring disease risk.

Behaviour and Sociality

As may be ascertained from the name, behaviour plays a major role in the behavioural immune system. Objects or other people containing cues of contamination are intuitively avoided. For instance, people exhibit greater behavioural avoidance—measured using an approach-avoid task—when viewing videos featuring contamination threats, as compared to videos eliciting threats of violence or danger (Newhagen 1998). This pattern of behaviour is found in human cultures removed from Western society and even in nonhuman animals. For instance, Apicella et al. (2018) demonstrate that Hadza and Tannese adults and children exhibit rejection of food that has come into contact with contaminants (Apicella et al. 2018). Nonhuman primates display similar aversion towards, and avoidance of, contaminated food (Sarabian et al. 2017, 2018).

Other research has found that experimentally activating concerns of disease can influence hygiene behaviour. For instance, people wash their hands more when in the presence of a disgusting odour (Pellegrino et al. 2016). Disease concerns are additionally shown to reduce sociality. When disease concerns are made salient, people report lower extraversion, exhibit greater behavioural avoidance of human targets, and report less interest in affiliation (Mortensen et al. 2010; Sacco et al. 2014). Such shifts in sociality in the face of disease threats make adaptive sense, as contact with human targets pose a risk for disease transmission.

Smoke Detector Principle and Functional Flexibility

Although features of the behavioural immune system help detect cues of disease and serve to facilitate avoidance of sources of infection and illness, its responses are necessarily imperfect. Given the high cost of illness and infection across evolutionary time, our evolved disease-detection mechanisms are extremely sensitive and can be activated even in the absence of actual disease threats. For example, recent work presented participants with the sounds of coughs and sneezes from targets who reported currently experiencing an infectious illness (e.g., cold, flu) and from targets who were coughing and sneezing for reasons unrelated to illness (e.g., consumption of spices; Michalak et al. 2020). The study found that participants could not differentiate between the two sources, misidentifying non-contagious sounds of illness as posing an infection threat. Other research on this topic finds that people respond similarly towards contagious others as they do towards those who have non-contagious physical anomalies (Kouznetsova et al. 2012; Park et al. 2013; Ryan et al. 2012). For instance, people display similar behavioural avoidance towards objects touched by a target with influenza and a target with a facial birthmark (Ryan et al. 2012). Together this work demonstrates that the processes of the behavioural immune system can lead to psychologically categorising non-infectious others and objects as infectious and prompt behavioural avoidance, even when avoidance is unnecessary. As such, the responses of the behavioural immune system can themselves impose costs on one's ability to successfully navigate the challenges inherent in survival and reproduction (e.g., finding shelter, food, mates, friends, etc.).

Like other evolved threat management systems, the behavioural immune system is characterised by *functional flexibility*, such that the intensity of responses varies based on the costs and benefits of disease avoidance in a given context (Ackerman et al. 2018; Tybur and Lieberman 2016; Schaller and Park 2011). That is, the activities of the behavioural immune system are shown to be particularly amplified in contexts where benefits of disease avoidance more strongly outweigh the costs, such as when one is especially vulnerable to illness and infection (for a review see Schaller and Park 2011). For instance, the association between disease and physical anomalies is stronger for those who are chronically concerned about infection or when infection concerns are made salient (Miller and Maner 2012; Park et al. 2007).

Additionally, those who are chronically concerned about their health even evaluate relatively healthy individuals as being less healthy (Hedman et al. 2016).

On the other hand, behavioural immune system activity is downregulated in circumstances where avoidance restricts one's ability to meet other goals. For instance, taking care of children requires one to interact with potential contaminants (e.g., changing diapers, cleaning up vomit, etc.). Research finds that women who are parents of children report less disgust towards the smell of a dirty diaper from their baby, compared to a dirty diaper from an unrelated child (Case et al. 2006). Along similar lines, sexual intercourse involves close physical contact exposure to bodily fluids. When sexually aroused, both men and women report decreased disgust towards sex-related stimuli (Borg and de Jong, Chap. 9, this volume; Borg and De Jong 2012; Stevenson et al. 2011a). However, it is worth noting that one online study found that sexual arousal did not blunt women's disgust (Zsok et al. 2017). Other research finds that people who are food deprived exhibit less facial disgust in response to unpalatable food images (Hoefling et al. 2009). Together, this research demonstrates that responses of the behavioural immune system are contextually dependent, varying based on state vulnerability and salient goals.

Social Circumstances and Individual Differences in Disgust Sensitivity

Just as disgust varies transiently based on contextual factors that influence the relative costs of engaging in behavioural disease avoidance (e.g., when one is food deprived), recent research suggests that more stable social characteristics of individuals that affect these costs might also impact disgust sensitivity. That is, individuals whose social circumstances increase the costs associated with behavioural disease avoidance should demonstrate lower disgust sensitivity. In contrast, individuals whose social circumstances diminish the costs associated with behavioural avoidance should exhibit higher disgust sensitivity. For example, because disgust promotes avoidance of people or stimuli that elicit disgust, it could be costly because it reduces the number of options one has for social partners, romantic partners, or food options. Therefore, disgust should be higher for individuals who are better able to withstand the costs of losing opportunities in these domains compared those to who may have fewer options. In other words, individuals who can afford to be more selective should tend to exhibit greater disgust than those who cannot.

One potential important determinant of an individual's ability to withstand the cost of elevated disgust responses is one's social status. This is because the wealth and prestige of having high social status endows an individual with a greater number of choices when it comes to romantic partners (Bereczkei et al. 1997; Stringhini et al. 2012; Von Rueden and Jaeggi 2016), housing (Dwyer 2007), and foods (Drewnowski and Specter 2004), compared to those of lower status. With these insights in mind, one recent set of studies examined relationships between social

status and disgust sensitivity (Bradshaw et al. n.d.). This work demonstrated that individuals reporting a higher social status also reported having greater disgust sensitivity. A set of experiments further revealed both that participants expected high status targets to exhibit greater disgust sensitivity than low status targets, and in reverse, that targets described as displaying disgust were rated as higher status than those not displaying disgust.

Together, this research suggests that social status and disgust are closely linked and sets the stage for future research to examine how additional social factors that influence one's ability to withstand the costs of disgust may impact its expression. Moreover, there are also a number of additional social and cultural factors related to socioeconomic status that might influence disgust sensitivity. Therefore, additional research is needed to determine the causal factors involved in this link. For example, individuals of high and low socioeconomic often differ in parenting behaviours (Hoff et al. 2002) and emotional styles (Manstead 2018), which might impact attitudes towards disgust-eliciting stimuli.

Relationships between the Biological Immune System and Behavioural Immune System

While a large body of work has demonstrated the far-reaching effects of the behavioural immune system on psychology and behaviour, research into how behavioural pathogen defence mechanisms intersect with the activities of the immune system is still in its nascent stage (Ackerman et al. 2018; Gassen et al. 2018; Gassen and Hill 2019; Murray et al. 2019). A leading hypothesis from the evolutionary psychology literature proposes that the biological and behavioural immune systems may operate in a compensatory manner (see compensatory prophylaxis hypothesis; Fessler et al. 2005; Fleischman and Fessler 2011), and some recent research in the field of psychoneuroimmunology has provided initial support for this possibility (e.g., Bradshaw et al. n.d.; Gassen et al. 2018). On the other hand, separate research suggests that—in some situations—disgust and biological immune activation may co-occur (e.g., Rubio-Godoy et al. 2007; Stevenson et al. 2011b). Here, we review previous theory and research on relationships between biological and behavioural pathogen defence mechanisms. We propose that progress in this domain has been limited by an over-reliance on indirect hypothesis testing (e.g., inferring immune status from hormone levels) and a failure to fully appreciate the complexity of immune function. We conclude by presenting future directions for disgust research that may help unravel complex relationships between immunity and behaviour that have evolved in the context of the host-pathogen arms race.

The Compensatory Prophylaxis Hypothesis

The compensatory prophylaxis hypothesis postulates that behavioural disease avoidance mechanisms have evolved to complement the activities of the biological immune system in protecting an individual from infection. However, because prophylactic disease avoidance behaviours bear energetic and social costs (as briefly outlined in the previous section), the extent to which individuals engage in these behaviours should not be constant, but should rather increase when the biological immune system is impaired and one is particularly vulnerable to the threat of infectious disease (Fessler et al. 2005; Fleischman and Fessler 2011). Support for this hypothesis includes research demonstrating that pregnant women reported elevated disgust sensitivity during the first trimester (compared to the second and third trimesters), a period when the maternal immune system is believed to be suppressed to prevent the maternal immune system from attacking the foetus (Fessler et al. 2005). Additional support for the compensatory prophylaxis hypothesis comes from research finding that, in naturally-cycling women, higher levels of salivary progesterone—a hormone that suppresses certain types of immune responses—were associated with greater disgust sensitivity, contamination-related rumination, and self-grooming behaviours (Fleischman and Fessler 2011; but see Jones et al. 2018).

Some recent research has provided additional support for compensatory prophylaxis by examining the relationship between inflammation—a crucial component of the immune system—and the activities of the behavioural immune system. For example, one study found that healthy individuals with lower levels of basal inflammation (both *in vivo* and *in vitro*) reported greater germ aversion than those with higher levels (Gassen et al. 2018). Other research shows that participants administered aspirin—a non-steroidal anti-inflammatory drug—reported feeling more negative towards pictures of disgusting stimuli than those administered a placebo (Bradshaw et al. n.d.). In both cases, these findings are consistent with the compensatory prophylaxis hypothesis, as they suggest that lower levels of inflammation are associated with increased sensitivity to disease-connoting contexts and stimuli.

Beyond the Compensatory Prophylaxis Hypothesis

Research finds that the biological and behavioural immune systems do not always operate in a compensatory manner, however. Instead, some studies have shown that experimentally activating the behavioural immune system via exposure to disgust-connoting stimuli can also elicit an immune response (Ersche et al. 2014; Schaller et al. 2010; Stevenson et al. 2011b, 2012). In other words, both the biological and behavioural immune systems appear to increase in concert when an individual is directly faced with stimuli that may confer infectious disease risk. For example, one

experiment found that exposure to disgusting visual stimuli (relative to neutral and negative, but not disgusting stimuli) led to increased oral inflammation (Stevenson et al. 2011b). Other work demonstrated that inducing disgust through a similar procedure led to both an increase in a marker of oral inflammation, as well as elevated body temperature (Stevenson et al. 2012).

The idea that the biological and behavioural immune systems may operate in parallel when an individual faces direct exposure to stimuli connoting infection risk is not mutually exclusive with insights from the compensatory prophylaxis hypothesis. Instead, it is possible, and likely, that interactions between these systems are highly context-dependent and moderated by individual-level factors, such as one's control over pathogen contact (see e.g., Bradshaw et al. n.d.), energetic status (see e.g., moderation by hormone leptin; Han et al. 2003; Iikuni et al. 2008), age (Fessler et al. 2005; Franceschi et al. 2007), and sex (Klein et al. 2015; Tybur et al. 2011a). However, a more comprehensive theoretical framework that accounts for each of these previous findings is needed to help guide predictions about when, how, and for whom relationships between the biological and behavioural immune systems follow a given pattern.

Current Limitations of Research Examining Links between Immune Function and Disgust

While research on disgust and the behavioural immune system has exploded in the last two decades, sparse effort has been devoted to integrate insights from immunology and related fields into this programme of study (with the exception of Schaller et al. 2010; and Stevenson et al. 2011b, 2012). Accordingly, there has been little progress towards elucidating the biological-behavioural immune system interface. This is particularly troublesome because the structure and function of evolved emotional, cognitive, and behavioural pathogen defence tools cannot be fully understood without knowing how they relate to our body's primary defence against infectious illness.

We argue that the first major limitation of research examining links between the biological and behavioural immune systems, to date, has been an over-reliance on indirect methods for measuring immune function. A strength of evolutionary psychology is its focus on considering ultimate explanations for behaviour. However, developing and testing evolutionary theories involves designing empirical research to test putative mechanisms. Without the appropriate methodology to do so, direct relationships and causality become difficult to interpret and both significant and null results can present more questions than answers. This has often been the case with extant research on relationships between the biological and behavioural immune systems. For example, studies testing the compensatory prophylaxis hypothesis have largely relied on menstrual cycle phase, pregnancy trimester, or levels of progesterone as a proxy for the activities of the immune system (e.g., Fessler et al. 2005;

Fleischman and Fessler 2011; Jones et al. 2018). However, each of these factors have complex, pleiotropic effects on immune function (i.e., rather than uniformly impairing immunity; Hall and Klein 2017; Hughes 2012; Lorenz et al. 2015; Mor and Cardenas 2010; Saito et al. 2010), and considered in isolation, likely provide a poor index of one's current infection risk.

Further limiting the utility of these measures is the fact that their effects on immunity are moderated by individual-level factors, such as whether or not one is sexually active (e.g., hormones and cycle phase; Prasad et al. 2014) and one's health/diet prior to and during pregnancy (e.g., Dellschaft et al. 2015; Sen et al. 2013). Therefore, it is vital that the activities of the immune system are directly measured when testing hypotheses about the biological-behavioural immune system interface. This also extends to measuring the behavioural immune system. Direct measurements of the behavioural immune system (e.g., approach-avoid tasks, behavioural tasks) should be prioritised to improve the external validity of such research.

Measuring immune function, however, is easier said than done. The immune system is unbelievably complex, and classes of immune responses can be categorised in a variety of ways, whether it be innate versus adaptive immunity (Hoebe et al. 2004), cellular versus humoral immunity (Xu et al. 2004), Th_1 versus Th_2 versus Th_{17} responses (Romagnani 1992), or pro-inflammatory versus anti-inflammatory (Dinarello 1997), among many more. The second limitation of research examining relationships between the biological and behavioural immune systems is that literature has largely ignored this intricacy, leaving gaps in our understanding of how these systems truly interact. For example, the immune system is often described as being broadly "elevated" or "diminished" (e.g., Bradshaw et al. n.d.; Fessler et al. 2005; Fleischman and Fessler 2011; Gassen et al. 2018; Jones et al. 2018), but these states likely only reflect a specific facet of immune function, such as inflammatory activity, not the immune system as a whole. Moreover, even lower levels of inflammation do not necessarily indicate diminished immune function. In fact, lower systemic levels of inflammation within the normal range may actually indicate the opposite (see e.g., Cohen et al. 1999, 2012).

While on the surface it may appear daunting, appreciating the complexity of immune function is necessary for developing informed hypotheses and designing appropriate tests of relationships between the biological and behavioural immune system. Viewed more optimistically, the complexity of immunity provides a wealth of opportunities to generate novel and nuanced hypotheses about how specific aspects of immune function relate to behavioural pathogen avoidance mechanisms. For example, we might find that disgust is related to the function of innate immune cells, such as natural killer cells, but not adaptive immunity, like the production of antibodies by B cells. Further, we might find that relationships between behavioural immune system activity and different facets of immunity differ by age, sex, or ecology. Each of these shapes both disgust responses, as well as immune responses. Recent advancements in immunology that increased the affordability and accessibility of technologies like flow cytometry and transcriptomics now make it possible for more scientists—even social scientists—to test these and other hypotheses.

Future Directions for Disgust Research

In the previous sections, we presented evidence suggesting that disgust and the activities of the behavioural immune system represent a suite of evolved cognitive, psychological, and behavioural mechanisms that function to reduce exposure to potential sources of infection. We also briefly summarised theory and research on how these mechanisms might interact with the biological immune system. Below, we build on this past research and integrate insights from evolutionary biology, ecology, and psychoneuroimmunology to present new evolution-informed hypotheses about how environmental-, host-, and pathogen-level factors may interact to influence both the activities of the behavioural immune system and the course of the arms race between host sensory systems and harmful microorganisms.

Different Behavioural Responses to Different Classes of Pathogens

The behavioural immune system is believed to be relatively domain general, promoting avoidance of a broad range of stimuli connoting infection risk (Schaller 2011). However, it is possible that, like the activities of the biological immune system, behavioural pathogen avoidance strategies may be fine-tuned to the type of disease threat encountered and its primary vector (Abbas and Janeway 2000; Everett and McFadden 1999; Girardin et al. 2002; Romagnani 1991; Sher et al. 2003; Wang et al. 2016). For instance, the threat posed by intestinal parasites differs from that posed by viruses and bacteria. As such, we may expect that visual exposure to threats of intestinal parasites may elicit a different reaction (e.g., disgust plus gastrointestinal distress) than would viewing pictures of vectors of viruses or bacteria (e.g., general disgust).

The threat posed by ectoparasites (e.g., lice, ticks, fleas) illustrates the utility of specialised behavioural responses to specific classes of pathogen threats. Ectoparasites are common vectors for infectious diseases, and, given that they attack the host via attaching to external surfaces (i.e., the skin), the traditional avoidance defence of the behavioural immune system offers little protection against this class of threat. Kupfer and Fessler (2018) suggest that humans and other animals possess specifically tailored behavioural defences (e.g., scratching and grooming) that function to counter the threat posed by ectoparasites. Consistent with this reasoning, initial research finds parasites and pathogens that are primarily transmitted via the skin elicit emotional responses and behavioural reactions—such as a skin-crawling sensation—that are distinct from general disgust (Blake et al. 2017). A separate study found that exposure to live maggots—also ectoparasites—led to increased tactile sensitivity relative to exposure to control stimuli (i.e., rice) presented in the

same container (Hunt et al. 2017). Future research is needed to examine whether exposure to stimuli connoting risk for infection by different classes of pathogens evokes different emotional, cognitive, and behavioural responses, as well as the role that learning plays in such processes.

The fact that different classes of pathogen threats elicit distinct behavioural responses may provide some clarification for recent work regarding the lack of a relationship between regional variability in infectious disease prevalence and disgust sensitivity (Tybur et al. 2016). If disgust sensitivity is an evolved solution to the threat of infection, one may expect individuals who reside in areas where the threat of infectious disease is relatively high to exhibit greater disgust sensitivity than those in areas where the threat of infectious disease is lower. That no such relationship has been found, at first blush, appears to suggest that disgust sensitivity does not serve its alleged adaptive function. However, this research used an index of infectious disease prevalence that did not differentiate between diseases caused by different classes of pathogens. This index is necessarily simplistic, as specific classes of pathogens vary as a function of certain environmental features (e.g., temperature and precipitation; Guernier et al. 2004). As such, it is possible that relationships between disgust sensitivity and pathogen richness or prevalence may emerge when considering pathogen classes and their most common vectors separately. For example, individuals living in regions especially rich in viral-induced illnesses (but not macroparasites) may be more attuned to infection cues in conspecifics—a primary vector for viral transmission—than those living in regions where macroparasites pose more of a burden. Further, given that unique immune responses to different types of pathogens also have distinct metabolic and oxidative costs (e.g., see defence vs. tolerance: Allen and Sutherland 2014; Medzhitov et al. 2012; Wang et al. 2019), the selection pressure that pathogen density poses on behavioural disease avoidance mechanisms likely varies based on types of micro- or macro-organisms most commonly encountered in a given region.

An additional consideration, here, also arises when taking into account the relationship between pathogen prevalence and immune system activity. That is, the Tsimane, who live in a pathogen-dense ecology, have elevated levels of many immune parameters compared to industrialised populations, such as immunoglobulins and certain cells types including eosinophils (involved in parasite defences), B cells (involved in adaptive immunity), and natural killer cells (involved in innate immunity; Blackwell et al. 2016). Given recent evidence of a negative relationship between inflammation and behavioural immune system activity (e.g., Bradshaw et al. n.d.; Gassen et al. 2018), disgust sensitivity may actually be lower in regions rich in pathogens that elicit systemic inflammatory responses (e.g., intracellular pathogens). More research is needed to understand how various types of pathogen threats might differentially influence both the biological and behavioural immune system. Research in this vein, moreover, will hopefully provide opportunities for cross-disciplinary and cross-cultural collaboration.

Co-evolution between Pathogens and Host Sensory Systems

Humans and other animals are in a never-ending evolutionary arms race with pathogens. This is evident in the myriad strategies disease-causing microorganisms have for subverting and down-regulating host immune responses (Lucas et al. 2001; Olivier et al. 2005). Animals also use various sensory modalities to detect and avoid cues connoting infection risk, chief among them being olfaction (Poirotte et al. 2017; Tybur et al. 2011b). Little attention has been paid to the possibility that our ability to smell and avoid contamination may exert a selection pressure on microorganisms to make themselves and their vectors less aversive, or "smelly" and disgust-inducing (for more on olfaction and disgust see Liuzza, Chap. 7, this volume).

With food in particular, we often know almost instantaneously (via visual or olfactory inspection) whether our food is undercooked or spoiled and harbouring bacteria that may do us harm. However, research finds that these "smelly" bacteria that render your food inedible are not typically the types that cause illness in humans (Gram et al. 2002). In contrast, some of the most dangerous bacteria living in your food can only be detected in the lab. For example, *Listeria monocytogenes*, which infects approximately 1600 Americans each year, cannot be smelled, tasted, or seen (Farber and Peterkin 1991). The same is true for many of the other top causes of foodborne illness, including *Salmonella* (nontyphoidal), *Campylobacter* (multiple species), and *Escherichia coli* (O157) (Centre for Disease Control 2018). Although wildly speculative, is it possible that a taxonomy of bacterial "smelliness" would reveal a history of host olfaction-pathogen co-evolution that has endowed disease-causing bacteria with the ability to downregulate production of compounds hosts find aversive so that they can live right under our noses?

Similar reasoning applies when considering the evolution of infectious microorganisms that are spread primarily through social contact. Such pathogens, according to Nesse and Williams (1994), may downregulate virulence in order to facilitate their spread. While illness and infection are typically accompanied by a feeling of lassitude, motivating the host to conserve energy (Schrock et al. 2020), certain viruses may have evolved longer asymptomatic periods before the onset of symptoms. Take, for example, the recent COVID-19 pandemic. Individuals infected with COVID-19 are most infectious prior to the onset of any symptoms (He et al. 2020). Moreover, because viral infections induce inflammation (Kawai and Akira 2006), and inflammation has been linked to greater impulsivity (Gassen et al. 2019a), these viruses may also have evolved to manipulate their hosts' social behaviour to increase transmissibility. This reasoning, however, is entirely speculative. Future research would benefit from examining how various diseases might exploit the behaviour of hosts.

Disease Seasonality and Host Defences

One important parameter that has been essentially neglected in the behavioural immune system literature is the seasonality of disease. Rather than being constant,

infectious diseases vary cyclically across seasons. For instance, vector-borne diseases and parasitic infections occur at higher levels during the warm and rainy seasons (Altizer et al. 2006; Amin 2002), and in temperate climates, viral respiratory infections predictably increase during the winter months (Moriyama et al. 2020). These seasonal peaks in infection are thought to be driven by various environmental factors that influence pathogen transmissibility and host susceptibility (Altizer et al. 2006; Dowell 2001; Fisman 2007). For example, contexts of relatively low temperature and humidity not only increase the transmissibility of the influenza virus but can also increase the host's susceptibility to infection (Moriyama et al. 2020).

Furthermore, research finds that the activities of the immune system also exhibit seasonal variation (Demas and Nelson 1998a, 1998b; Dopico et al. 2015; Gassen et al. 2019b). In the wild, the stressful conditions that accompany winter—such as shorter days, thermoregulatory demands, and reduced food availability—together compromise immune function and leave animals more susceptible to infectious illnesses (Nelson 2004; Demas and Nelson 1998a, 1998b). Winter, however, is reliably predicted annually by the shortening of days. To combat the effects of seasonal stress on infection risk, research finds that animals, even in the lab when exposed artificially to shortened days, tend to upregulate investment in immune function as winter approaches (Nelson 2004; Demas and Nelson 1998a, 1998b). More recent research finds that humans, too, exhibit more robust immune responses as the days shorten (Dopico et al. 2015; Gassen et al. 2019a).

Given that seasonality both influences pathogen transmissibility and the activities of the immune system, disgust and the behavioural immune system may also exhibit seasonal variation. For example, people may display greater disgust and behavioural avoidance in response to cues of viral infection (e.g., coughs, sneezing) during the winter months than the summer months. Behavioural immune system activity might further be influenced by changes in ambient environmental temperature. Foodborne bacteria, for instance, is more likely to grow in relatively high ambient temperature (Han et al. 2016). As such, people may exhibit more disgust towards questionable food sources when in environments with a high (vs. low) ambient temperature. To the best of our knowledge, there has been no research examining the relationship between activities of the behavioural immune system and seasonality or temperature. While these possibilities are, at the moment, mere speculations, they represent a potentially fruitful direction for researchers seeking to more fully understand how environmental factors might influence our disease avoidance psychology.

Conclusions

Diseases have been present throughout human history. Although medical advances, such as vaccines, have increased our ability to withstand the threat of illness and infection, viruses and bacteria evolve rapidly, and, despite our best efforts, are likely to pose a threat to our survival for the foreseeable future. The strength of this selection pressure has shaped both our body and our psychology to counter the

persistent threat of infection. Disgust and the behavioural immune system represent aspects of our psychology that are perhaps best understood through an evolutionary lens. An evolutionary perspective bearing on how disease threats have shaped behaviour can not only provide an explanation for why we find certain things more disgusting than others, but can also help provide insights into individual-and context-based differences in disgust sensitivity. The research on disgust is still in its infancy, and there is much left to learn. Future research into the nature of disgust will continue to benefit from applying evolutionary thinking, as well as integrating theory and methods from fields like immunology, psychoneuroimmunology, and behavioural ecology, to further uncover the complex bio-behavioural tools that protect the body from the omnipresent threat of infectious illness.

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