DO SYMPTOMS OF ILLNESS SERVE SIGNALING FUNCTIONS? (HINT: YES)

Leonid Tiokhin

School of Human Evolution & Social Change and Center for Evolution & Medicine, Arizona State University

Tempe, Arizona 85287 USA

e-mail: ltiokhin@asu.edu

keywords

evolutionary medicine, disease, sickness behavior, symptom expression, social context, evolution of communication, signaling theory

abstract

Symptoms of illness provide information about an organism's underlying state. This notion has inspired a burgeoning body of research on organisms' adaptations for detecting and changing behavior toward ill individuals. However, little attention has been paid to a likely outcome of these dynamics. Once an organism's fitness is affected by others' responses to symptoms of illness, natural selection can favor individuals who alter symptom expression to influence the behavior of others. That is, many symptoms may originate as cues, but will evolve into signals. In this paper, I develop the hypothesis that symptoms of illness serve signaling functions, and provide a comprehensive review of relevant evidence from diverse disciplines. I also develop novel empirical predictions generated by this hypothesis and discuss its implications for public health. Signaling provides an ultimate explanation for otherwise opaque aspects of symptom expression, such as why symptoms fluctuate in social contexts, and can exist without underlying pathology, and why individuals deliberately generate symptoms of illness. This analysis suggests that signaling theory is a major organizing framework for understanding symptom etiology.

DOCTOR: I've looked at your X-rays . . . [a]nd I find that there's absolutely nothing wrong with you.
GEORGE: Hmm. Really? Nothing?
DOCTOR: Nothing that would indicate involuntary spasms.
GEORGE: Well, it's kind of a mystery, isn't it?
DOCTOR: No, not really.
GEORGE: How so?
DOCTOR: May I suggest the possibility that you're faking?
(*Seinfeld*. 1993. "The Non-Fat Yogurt." Season 5, Episode 7).

The Quarterly Review of Biology, June 2016, Vol. 91, No. 2

0033-5770/2016/9102-0003$15.00

INTRODUCTION

ALL living organisms experience disease and injury (i.e., illness), and exhibit phenotypic changes (i.e., symptoms) as a result. Symptoms are usually conceptualized as inadvertently providing information about an organism's underlying pathology. For instance, coughing, sneezing, and nasal congestion are reliably associated with respiratory infection (Monto et al. 2000). Limping is reliably associated physical injury. This reliable association between symptoms and somatic pathology makes much of medicine possible. This fact has also made it possible for organisms to evolve adaptations that adjust individual behavior as a function of others' symptoms. For example, many species avoid individuals exhibiting signs of infection (Curtis 2014). Such findings have led to a growing area of research that seeks to determine the structure of adaptations for detecting and responding to the symptoms of others (Schaller 2011; Curtis 2014).

Nonetheless, the assumption that symptoms only inadvertently provide information to others may not be valid. Once organisms use symptom-based information to adjust their own behavior, selection should favor individuals who manipulate this information content to alter others' behavior. This is the core thesis of this paper: natural selection can favor the evolution of symptoms into signals. Signaling hypotheses have previously been proposed to explain symptom form in many fields, including biology (Loehle 1995; Plesker and Mayer 2008; Shakhar and Shakhar 2015), psychology (Price et al. 2004; Vigil and Strenth 2014; Brown et al. 2015), anthropology (Nichter 1981; Fábrega 1999), and medicine (Wenegrat 2001; Halligan et al. 2003a; Kozlowska 2007). Yet, these proposals have largely been developed independently, without grounding in modern evolutionary theories of communication (but see Nock 2008), and without realization that their underlying logic has the potential to transform current conceptualizations of symptom etiology.

One recent paper provides an exception (Steinkopf 2015; this work was developed entirely independently of the current paper). Steinkopf proposes the idea that symptom expression has been shaped by natural selection for communicative purposes. He argues that discernible symptoms of human immune responses serve dual functions of fighting infection and signaling need for aid to conspecifics. Steinkopf argues that this explains the existence of the placebo effect: symptoms exist to honestly communicate need for aid and once these needs are addressed (via social support or beliefs about the health benefits of inert medicine), symptom expression subsides. He refers to this as the Signaling Theory of Symptoms or STS (Steinkopf 2015). Although STS is broadly similar to this paper's proposal, there are several notable differences. Like previous work, STS only focuses on a small subset of symptoms (i.e., those associated with human immune responses) and signaling functions (i.e., eliciting aid). In contrast, this review argues that all discernible symptoms in all species are candidates for signaling explanations, and that their signaling functions will be more diverse than mere aid elicitation. STS argues that symptoms are honest signals, and that the costs of immune function maintain this honesty. However, this claim is contradicted by both theoretical and empirical analyses of animal signaling: cost is neither necessary nor sufficient to maintain signal honesty, "honest signaling" is only one of many possible signaling equilibria, and signaling systems are rife with dishonesty (Searcy and Nowicki 2005; Számadó 2011; Zollman et al. 2013). In contrast, this review argues that symptoms will vary in their cost to signalers and signal value to receivers, the details of which will depend on species-specific selection pressures shaping signal form. The following section provides a brief overview of signaling theory, and forms the theoretical foundation for understanding the evolution of symptoms into signals.

SIGNALING THEORY AND THE EVOLUTION OF CUES INTO SIGNALS

Signaling theory provides a framework that explains information-transmission dynamics between organisms. Information is anything that results in the reduction

of uncertainty regarding some state of the world (Shannon and Weaver 1949). Organisms seek information because reducing uncertainty allows them to increase fitness by improving decision-making.

In addition to direct interactions with the environment, organisms obtain valuable information (in the form of cues and signals) from the phenotypes of others (Dall et al. 2005). Cues are aspects of organisms that provide information to others without having evolved for the purpose of information transmission. For example, animals produce CO_2 as a byproduct of respiration, and mosquitos use CO_2 emissions to reduce their uncertainty about the location of a biting target (Takken 1991). Signals are aspects of organisms (signalers) that have specifically evolved to influence the behavior of others (receivers) by transmitting information (Lachmann et al. 2001). For example, Thomson's gazelles respond to coursing predators by repeatedly leaping off the ground with all of their legs held stiff and straight (i.e., stotting), and do so as a function of predator type and distance (FitzGibbon and Fanshawe 1988). This leaping signals that the gazelle is difficult to catch, and therefore not worth pursuing.

Theoretical analyses of signal evolution have largely focused on two signal properties: honesty and cost. Honesty is a signal's information value. That is, how much is the receiver's uncertainty about a true state of the world reduced by receiving the signal? There are at least two kinds of signal costs: efficacy costs and strategic costs (Guilford and Stamp Dawkins 1991; Maynard Smith and Harper 2003). Efficacy costs are paid by signalers to ensure that a signal unambiguously reaches its target. For example, many species produce higher intensity signals in environments with high background noise (Leonard and Horn 2005; Parris et al. 2009). Strategic costs are paid on top of efficacy costs, and are sometimes necessary to ensure a signal's honesty. The honesty of "stotting" in Thomson's gazelles is ensured by such costs: only those gazelles in good condition can afford to waste time and energy by stotting when pursued by predators. The distinction between efficacy and strategic costs is important, as it means that signals can be costly solely because of the energy necessary to unambiguously transmit information.

As long as signalers benefit from producing signals and receivers benefit from responding, many different signaling systems can be evolutionarily stable. Modern signaling theory has yet to provide much guidance regarding the exact types of signals that should be common in nature (but see Kane and Zollman 2015). Shared interests can lead to the evolution of honest, cost-free signals (Maynard Smith 1991), as can repeated interactions and third-party sanctions, even when interests conflict (Silk et al. 2000; Boyd and Mathew 2015). If signals vary continuously as a function of signaler's state, and if the costs of lying outweigh the benefits, signals can have high information value at equilibrium, with costs ranging from negligible to high (Grafen 1990; Lachmann et al. 2001). If only a limited number of signals are possible, signals can be essentially cost-free and remain partially honest at equilibrium (Bergstrom and Lachmann 1998). Signals with low information value can also be evolutionarily stable (Számadó 2000; Mitri et al. 2009). If assessing signal honesty is costly enough, the optimal strategy for receivers may be to believe signals without checking their veracity (Stamp Dawkins and Guilford 1991). Signal detection theory predicts that asymmetric costs of false positives and false negatives will select for receivers who minimize the frequency of the costliest errors, which can also lead to high levels of signal dishonesty (Wiley 1994).

The relationship between cues and signals is not static. Because giving off cues that are used by others affects the fitness of organisms, cue production itself is under selection. In fact, many behavioral and physiological signals are thought to have originated as cues (Maynard Smith and Harper 2003). One study of experimental evolution with robots provides an apt example of this process (Mitri et al. 2009). Researchers created robots that emitted blue light, thus producing information. Robots

could also perceive the light emissions of other robots with light-sensitive cameras. These robots were placed in an arena containing a food source and a poison source, which they could identify at close range. The fitness of robots was a function of their time spent near food and poison sources: they gained or lost one point for every time unit spent near food or poison, respectively. Researchers then simulated experimental evolution by selecting the top performing 20% of robots, subjecting them to mutation and recombination, and assigning them to groups that made up the next generation. What happened? First, robots evolved to successfully locate food and avoid poison, while still emitting light randomly. This led to high light intensity near food. Light intensity thus became a cue to food availability, and other robots evolved an attraction to blue light. However, because robots were competing for food, this decreased the fitness of individuals emitting blue light near food sources. This led to selection for cue concealment: robots evolved to suppress light emission near food sources, but not near sources of poison. This work demonstrates how cues can evolve into signals via information suppression, resulting in signals with reduced information value (Mitri et al. 2009). The evolution of symptom expression may experience similar dynamics across evolutionary time.

Symptoms are Cues

There is an abundance of evidence that organisms attend to and adjust their behavior as a function of others' symptoms (see Schaller 2011 and Curtis 2014 for comprehensive reviews). Many organisms avoid individuals with symptoms of infection. Female mice discriminate between healthy males and those infected by a parasitic nematode, *Heligmosomoides polygyrus*, displaying aversions to the odors of parasitized males (Kavaliers and Colwell 1995). Male rats avoid females exhibiting sickness behaviors after interleukin-1 (IL-1) injection (Avitsur et al. 1997), and lactating rats spend less time with pups exhibiting sickness behaviors after lipopolysaccharide (LPS) injection (Walker et al. 2004). Gregarious lobsters avoid conspecifics infected with *Panulirus argus* virus 1 (Behringer et al. 2006). Bullfrog tadpoles avoid conspecific tadpoles infected with *Candida humicola*, a yeast pathogen (Kiesecker et al. 1999). House finches prefer to associate with healthy conspecifics over those exhibiting sickness behaviors after Complete Freund's Adjuvant (CFA) injection, an antigen solution that induces the acute phase sickness response (Zylberberg et al. 2013).

In humans, the emotion of disgust serves the core function of facilitating avoidance of infectious disease sources (Curtis et al. 2011; Tybur et al. 2013). Cross-cultural surveys reveal that people find disease-relevant stimuli more disgusting than disease-irrelevant stimuli (Curtis et al. 2004). Humans exhibit increased disgust sensitivity at times when they are most vulnerable to infection (Fessler et al. 2005; Fleischman and Fessler 2011), as well as stigmatize and force social isolation upon individuals suspected to be sick (Crandall and Moriarty 1995).

Avoiding diseased or injured individuals is not always adaptive. Predators can increase their probability of prey capture by targeting sick and injured individuals (Lafferty 1992; Mesa et al. 1994). Further, although individuals may want to avoid diseased mating partners, competing with sick individuals can increase one's probability of competitive success. In one study, researchers injected a subset of male and female finches with pink eye virus (*Mycoplasma gallisepticum*), then gave healthy same-sex finches a choice between feeding locations near sick or healthy conspecifics. Although females showed no feeding preference, males spent a much greater proportion of feeding time near diseased conspecific males than near healthy ones. They were also more likely to beat sick males in competitive interactions over food (Bouwman and Hawley 2010).

In species with kin-based nepotism, organisms benefit from assessing when recipients of altruism benefit most from a donor's altruistic act (Hamilton 1964). Additionally, if individuals interact repeatedly, the immediate costs of helping sick individuals can be outweighed by the benefits of

future interactions (Trivers 1971). Because the benefit of receiving altruism during times of illness is particularly high, small degrees of relatedness and few repeated interactions may be sufficient to facilitate the evolution of altruistic giving to sick individuals. A recent paper hypothesizes that the inclusive fitness benefits of preventing infection transmission to kin may be a major organizing principle shaping sickness behavior in vertebrates (Shakhar and Shakhar 2015). Sickness behaviors such as social disinterest, fatigue, hypersomnia, and anorexia restrict an organism's physical contact with nearby individuals and limit microbial contamination of surrounding resources. Reduced grooming and pheromonal changes also provide information about infection status to others. Selection could have favored the evolution of sickness behavior to prevent infection transmission, as long as the beneficiaries of these behaviors had higher average relatedness to infected individuals than the larger population, and direct fitness costs of sickness behaviors were outweighed by indirect fitness benefits (Shakhar and Shakhar 2015).

Evidence for aid provisioning during times of illness is sparse in nonhuman animals (Clutton-Brock 2009). Yet, it is a conspicuous feature of all human societies (Sugiyama 2004; Gurven et al. 2012). Given the frequency of severe health problems faced by people in small-scale societies, researchers have suggested that most people's survival will depend on provisioned foods at some point (Gurven et al. 2000). Among the Shiwiar of Ecuador and Peru, most people report experiencing a debilitating health crisis at some point in their lives (Sugiyama 2004). Among the Aché of Paraguay, almost 80% of people report having recently been "so hurt that they had to stay in bed" (Gurven et al. 2000:268) and report receiving substantial aid from kin and nonkin during these times (Gurven et al. 2000).

It is clear that, in a wide variety of species, individuals adjust their behavior as a function of others' symptoms. Further, these behavioral responses can strongly affect the fitness of symptomatic individuals (Gurven et al. 2012). Given these conditions, symptom production itself should be under selection. If symptoms can vary independently of underlying pathology, and if the fitness costs of altering symptom production are outweighed by socially mediated fitness benefits, symptoms will evolve into signals. These signals may take diverse forms. Ill individuals may suppress or exaggerate preexisting symptoms of illness. Healthy individuals may "pull a Costanza" and feign symptoms, without any underlying pathology. Individuals might also produce physiologically mediated symptoms, thereby creating actual pathology. Different signaling mechanisms will exist in different species and contexts, but their form will be shaped by a common logic: the fitness effects of social interaction cause an organism's optimal level of symptom production to be different than its optimal level when solely concerned with illness recovery (see Hennessy et al. 2014 and Lopes 2014 for similar arguments about life-history tradeoffs in symptom expression).

Symptoms are Signals

symptom feigning and exaggeration

Many precocial bird species use injury feigning as a nest-defense strategy to distract predators (Barash 1975; Montgomerie and Weatherhead 1988). Warblers feign symptoms of injury to distract predators approaching their nest by fluttering, hobbling around on the ground, drooping their wings, and spreading their tail (Grimes et al. 1936). Killdeer are famous for their broken-wing display, and feign injury by fanning their tail and bending their wings at an upward angle, simulating a broken wing (Brunton 1990). By feigning injury, birds incur the cost of increased predation risk, but gain inclusive fitness benefits by reducing the probability that predators find their offspring (Hamilton 1964; Sordahl 1990). These displays are effective because many predators preferentially target injured prey (Lafferty 1992; Mesa et al. 1994) and birds exploit this aspect of predator psychology. Anecdotal accounts of injury

feigning also exist in other taxa (Byrne and Stokes 2003; de Waal 2007), and YouTube videos of canine injury feigning abound.

Several plant species have evolved coloration patterns that resemble infestation by herbivores, predators, and pathogens (Schaefer and Ruxton 2009). For example, *Caladium steudneriifolium* (Araceae) has evolved leaves that mimic recent infestation by mining moth caterpillars (Soltau et al. 2009). Leaves of this species come in two morphs: plain green and variegated (i.e., whitish due to absence of chloroplasts). Mining moths are much less likely to choose variegated leaves for oviposition than plain leaves, and experimental manipulations reveal that the effect on moth deterrence is mediated by leaf color, not texture (Soltau et al. 2009). Variegation evolves for the same reasons as injury feigning. Plants produce symptoms at a cost (i.e., reduced photosynthetic ability), but this is outweighed by the benefit of moth deterrence.

Tonic immobility, or "death feigning," is one extreme form of feigning injury. This phylogenetically ancient response of "freezing" under conditions of extreme threat or physical entrapment has been conserved in a wide range of taxa (Gallup 1977). Ducks death feign when attacked by foxes, remaining immobile until their attacker has lost interest or is no longer nearby (Sargeant and Eberhardt 1975). Red flour beetles, *Tribolium castaneum* (Herbst), death feign when attacked by predators, and beetles from lineages artificially selected for longer death-feigning duration are much more likely to survive attacks (Miyatake et al. 2004). Although the reasons for the effectiveness of death feigning are unclear, leading explanations invoke prey exploitation of predator psychology via symptom feigning (Marx et al. 2008; Miyatake et al. 2009).

Humans feign and exaggerate symptoms in diverse contexts. Children feign illness to avoid going to school or gain additional social support (Libow 2000). Soccer and rugby players strategically "dive" and fake injury to get free kicks or otherwise illegal substitutions (David et al. 2011). Soldiers fabricate symptoms of disease during wartime to avoid participating in battle (Palmer 2003). Adults fake and exaggerate illness to avoid going to work, receive extra medical attention, and obtain increased disability compensation (Haccoun and Dupont 1987; Mittenberg et al. 2002; Halligan et al. 2003b). Increased benefits, such as financial incentives, further promote symptom exaggeration. People seeking legal compensation for chronic back pain report greater levels of disability than those not seeking compensation, despite being younger and having been in pain for less time (Suter 2002). Eliminating compensation for motor vehicle collision injuries reduces postcollision symptom severity, duration, and the total number of injury compensation claims filed (Cassidy et al. 2000; Ferrari and Schrader 2001). Social expectations about accident-related disability increase symptom severity and duration: chronic whiplash injury is nonexistent in countries that lack cultural beliefs that whiplash injuries result in chronic disability (Partheni et al. 2000; Ferrari and Schrader 2001). Existing estimates suggest that rates of illness feigning are staggeringly high. For instance, 20–40% of individuals seeking disability compensation are thought to fake or exaggerate their disability (Mittenberg et al. 2002; Larrabee 2003).

Humans also exaggerate symptoms of pain, and much work suggests that human pain expressions serve communicative functions (Williams 2002; Craig 2009; Hadjistavropoulos et al. 2011; Vigil and Strenth 2014). Females report greater pain and have lower pain tolerance in the presence of females versus other males (Levine and De Simone 1991; Kállai et al. 2004; Aslaksen et al. 2007; Vigil and Coulombe 2011). The presence of a caring and solicitous spouse has a strong positive relationship with chronic pain severity and behavioral expressions of pain (Flor et al. 1987; Romano et al. 1992; Newton-John 2002). Women in labor report higher levels of pain during epidural catheter insertion when their partners are present (Orbach-Zinger et al. 2012). Further, people for whom pain is a particularly negative experience (i.e., catastrophizers) are especially likely to exaggerate symptoms of pain in the presence of others (Sullivan et al. 2004).

symptom suppression

Sickness behaviors refer to the stereotyped array of behaviors exhibited by many organisms during the acute-phase response to infectious agents (Hart 1988). These include increased body temperature, inactivity, lethargy, reduced intake of food and water, shivering, hunched body posture, and piloerection, and are thought to improve the host's ability to battle infective agents (Hart 1988). Such behaviors are cues to infection, which is why conspecifics often avoid individuals displaying them. Given the benefits of social interaction, selection should favor individuals who suppress sickness behaviors during infection, whenever the costs of doing so are outweighed by the social benefits (but see Shakhar and Shakhar 2015).

Several findings are consistent with this hypothesis. Zebra finches injected with LPS express sickness behaviors, such as reduced activity levels and increased resting time, when kept in isolation. However, those injected with LPS and returned to their breeding colony do not exhibit sickness behaviors, even though they maintain typical physiological responses to LPS injection, such as increased levels of interleukin-6 (IL-6; Lopes et al. 2012). In another study, male zebra finches were injected with saline or LPS and then either kept socially isolated or introduced to a novel female. Males injected with LPS exhibited sickness behaviors in isolation, but suppressed sickness behaviors when exposed to a novel female (Lopes et al. 2013). Isolated males injected with LPS reduced their number of hops and calls, and increased their resting time, whereas LPS-injected males introduced to novel females exhibited no changes. Such behavior suppression occurred despite similar levels of IL-1 expression in both groups.

Male song sparrows injected with LPS express a typical sickness behavior (reduced territorial defense behavior) during the nonbreeding season, but exhibit no changes during the active breeding season, when displaying sickness behaviors could compromise their ability to find mates (Owen-Ashley and Wingfield 2006). Male mice display a similar behavioral pattern. In a series of experiments, researchers administered saline, low-dose IL-1 injection, or high-dose IL-1 injections to both male and female rats and measured their sexual receptivity to novel partners. Although IL-1 injections suppressed female sexual activity and interest in novel males, they had negligible effects on male sexual activity and interest in novel females (Yirmiya et al. 1995), and females did not discriminate between IL-1-injected and saline-injected males on most measures of mate preference (Avitsur et al. 1997).

Many scholars claim that nonhuman animals have also evolved to suppress pain symptoms (Sherwin et al. 2003; Stasiak et al. 2003; Hellyer et al. 2007; Plesker and Mayer 2008; Collen 2014). Although this hypothesis lacks strong support, some findings are suggestive. Mice with experimentally induced nerve and tissue damage do not behave differently from sham controls (Urban et al. 2011). Conspecific stress odors cause mice to have reduced pain sensitivity (Fanselow 1985), and the presence of dominants causes suppressed pain behaviors in subordinate mice (Gioiosa et al. 2009). Such findings are consistent with pain masking. Nonetheless, pain masking cannot account for all of the effects of social context on pain (see Raber and Devor 2002; Langford et al. 2006).

There is strong evidence that humans suppress symptoms of pain in certain contexts. The presence of high professional status individuals causes subjects to endure painful stimuli for longer durations (Kállai et al. 2004). Children display reduced facial expressions of pain in the presence of peers and unfamiliar adults than in the presence of their parents (Zeman and Garber 1996; Vervoort et al. 2008). Males report less pain, have higher pain tolerances, and display reduced pain expressions in the presence of females (Levine and De Simone 1991; Kállai et al. 2004; Aslaksen et al. 2007; Vigil and Coulombe 2011), although how audience presence affects pain masking is not entirely understood (see Vigil and Strenth 2014).

Symptom suppression in social contexts is also consistent with nonsignaling hypoth-

eses, such as those based on life-history tradeoffs in energetic investment (Lopes 2014). Distinguishing between alternative accounts for symptom fluctuation is a formidable empirical challenge (but see the section, What Are Symptoms? Testing Alternative Accounts for Symptom Production).

SELF-INDUCED ILLNESS

Self-induced illnesses are potentially the costliest strategy for symptom signaling. Even without underlying pathology, organisms can benefit from generating actual physical symptoms, as long as their production costs are outweighed by the benefits of changes in the behaviors of others. Somatoform disorders and self-injury may represent two such strategies.

Somatoform disorders, also referred to as medically unexplained symptoms, functional somatic symptoms, or psychosomatic symptoms, are symptoms without an established basis in physical pathology (Mayou and Farmer 2002). They include headache, irritable bowel syndrome, fatigue, and various types of chronic pain, and are thought to be uniquely human (Henningsen et al. 2007). They exist worldwide, are highly comorbid, and covary with other aspects of psychological functioning, such as depression and anxiety (Kato et al. 2006).

The existence of symptoms without an established organic basis presented a puzzle for theories positing a mapping between organic pathology and symptom presentation. This spurred an interest in the effects of psychological functioning and social context on symptom etiology, and led to the development of new conceptual frameworks, such as "biopsychosocial" models (Engel 1981; Gatchel et al. 2007). The importance of interpersonal factors in the etiology of somatoform disorders also led several researchers to propose that they serve various signaling functions (Stuart and Noyes 1999; Price et al. 2004; Nock 2008).

Qualitative ethnographic research and empirical findings in western populations are consistent with the hypothesis that somatoform disorders signal need: individuals with the greatest need for aid can honestly signal their need by generating actual physical symptoms to elicit support (Godfray and Johnstone 2000). Anthropologists have long documented that people use physical symptoms to communicate distress (Linton and Devereux 1956; Nichter 1981; Kleinman 1982; Low 1985). Such symptoms vary depending on cultural context, and "idioms of distress" refers to this range of communicative strategies. For example, *nervios* (nerves) is a syndrome present in many Latin American societies. Its manifestation varies by region, but can include symptoms such as dizziness, headache, fainting, fatigue, hot/cold sensations, insomnia, trembling, and stomach ache (Low 1985). *Nervios* occurs when people have difficulty coping with stressful life events, such as trauma or family problems, and is a culturally acceptable way to elicit support (Low 1985). Far from an isolated phenomenon, distress-induced physical symptoms exist in many cultures (Yap 1967; Nichter 1981; Parsons and Wakeley 1991). Many "culture-bound syndromes" may be explicable in similar terms. Individuals display symptoms that are attributed to uncontrollable, supernatural causes, such as possession by spirits or the malicious acts of ghosts. Others then appease these agents by offering concessions to ill individuals or limiting their obligations. As a result, individuals benefit without being blamed for their actions (see Simons and Hughes 1985 and Wenegrat 2001 for comprehensive reviews).

In western populations, levels of physical and psychological stress are similarly related to somatoform disorders. People with greater levels of psychological distress report more psychosomatic symptoms, such as general pains, gastrointestinal issues, and headaches (Parsons and Wakeley 1991). People who report experiencing childhood trauma, abuse, serious injury, and poor health report greater numbers of psychosomatic symptoms later in life and score higher on measures of hypochondriasis (Noyes et al. 2002; Waldinger et al. 2006). Individuals who experience parental loss early in life have greater rates of psychosomatic symptoms later in life (Mallouh et al. 1995). People with lower levels of so-

cial support, who lack trust in social relationships, and who live in neighborhoods with low social capital, all report greater levels of psychosomatic symptoms (Stuart and Noyes 1999, 2006; Noyes et al. 2003; Åslund et al. 2010). Somatoform disorders are comorbid with anxiety and depression, both of which strongly relate to an individual's vulnerability to harm and need for social support (Kato et al. 2006; Bateson et al. 2011). Further, depression itself may function as a bargaining strategy to elicit social support (Hagen 2003).

Nonsuicidal self-injury (NSSI) is the deliberate destruction of bodily tissue in the absence of an intent to die (Nock 2008). The existence of NSSI is paradoxical: why should organisms ever deliberately harm themselves? Nonetheless, such behaviors exist in many mammalian species (Jones and Barraclough 1978). Human NSSI is strikingly common. Worldwide, people engage in ritual-motivated NSSI, including piercing and scarification (Sosis et al. 2007). Upwards of 50% of adolescents admit to engaging in some form of NSSI, such as self-mutilation, hair pulling, and inserting objects under the nails or skin (Lloyd-Richardson et al. 2007). Munchausen patients deliberately produce symptoms of illness, such as fever, by injecting themselves with noxious substances (Aduan et al. 1979; Huffman and Stern 2003). During times of war, soldiers deliberately expose their limbs to attract enemy fire and mutilate their hands, leaving them unable to operate weapons (Bourke 1996; Wessely 2003).

Although nonhuman-animal NSSI appears to be a captivity-related pathology (Novak 2003), the cause of human NSSI remains unclear. Despite its costs, people do obtain large benefits from NSSI. For example, illness allows soldiers to be removed from potentially lethal combat situations (Palmer 2003). Because self-injurers often obtain social benefits, several researchers have proposed signaling explanations for NSSI (Sosis et al. 2007; Hagen et al. 2008; Nock 2008). Many adolescent self-injurers do report using NSSI to manipulate the behavior of others (Brown et al. 2002; Rodham et al. 2004). As with somatoform disorders, NSSI is positively related to psychosocial stressors, such as early-life abuse (Yates 2004; Glassman et al. 2007), and is comorbid with depression (Nock and Prinstein 2005). NSSI is also expressed when other forms of communication are ineffective, suggesting that it may function as a higher intensity signal (Tulloch et al. 1997; Nock and Mendes 2008). Ethnographic analyses further suggest a role for signaling. People in diverse cultures self-harm and threaten suicide in contexts of fitness threats, such as conflicts, suboptimal relationships, and powerlessness, suggesting that NSSI is an honest signal of need (Syme et al. 2015). Further, societies where cooperation is especially important maintain the costliest forms of ritual NSSI, suggesting that NSSI may function as a signal of cooperative intent (Sosis et al. 2007).

What Are Symptoms? Testing Alternative Accounts for Symptom Production

Many symptoms plausibly serve signaling functions (see Table 1). Yet, determining whether a given symptom is a cue or a signal is difficult. Symptoms can evolve into signals if receivers have preexisting responses to them. Thus, one avenue for identifying candidate symptoms is to investigate the cognitive architecture of receivers. For instance, the fact that organisms change their behaviors toward conspecifics who exhibit sickness behaviors suggests that sickness-behavior expression should be under selection. Symptoms can only be signals if their expression is partially independent of the underlying state about which they convey information (Maynard Smith and Harper 2003). Open wounds are not signals, because the underlying state is the wound itself, but the act of creating a wound can be a signal, as can any act that transmits information about the wound's severity.

The relationship between signaler and receiver should influence signal form. Reduced conflicts of interest lead to the evolution of more accurate information transmission (Mitri et al. 2011). Symptoms should more accurately reflect an underlying disease state

TABLE 1
Symptoms and proposed signal functions

Taxa	Signaling strategy	Proposed function	Evidence
Birds (Aves)	Symptom feigning and exaggeration	Distract predators from finding bird's nest and offspring	Injury-feigning intensity increases with offspring value (Barash 1975; Brunton 1990)
Flowering plants (Araceae)	Symptom feigning and exaggeration	Herbivore deterrence	Mining moths are less likely to select variegated leaves for oviposition than healthy ones (Soltau et al. 2009)
Various taxa (fish, reptiles, amphibians, birds, mammals)	Symptom feigning and exaggeration	Deter predators from continued attack	Death feigning increases probability of surviving attack and causes predators to lose interest (Sargeant and Eberhardt 1975; Miyatake et al. 2004, 2009)
Humans (*Homo sapiens*)	Symptom feigning and exaggeration	Avoid costly obligations	Soldiers feign injury to avoid battle (Palmer 2003). Adults admit feigning illness to avoid work (Haccoun and Dupont 1987)
Humans (*Homo sapiens*)	Symptom feigning and exaggeration	Elicit aid/social support	Cultural beliefs about accident severity are related to the severity of postaccident disability (Partheni et al. 2000; Ferrari and Schrader 2001). Increased pain expression and sensitivity in social contexts (Romano et al. 2000; Vervoort et al. 2008; Vigil and Coulombe 2011; Orbach-Zinger et al. 2012)
Humans (*Homo sapiens*)	Symptom feigning and exaggeration	Obtain financial compensation	Injury compensation causes increased injury severity, duration, and increased pain reports (Cassidy et al. 2000; Suter 2002)
Zebra finch (*Taeniopygia guttata*)	Symptom suppression	Hide vulnerability to prevent conspecific aggression; prevent avoidance by conspecifics	Suppressed sickness behaviors in social contexts, without suppression of physiological immune response (Lopes et al. 2012)
Zebra finch (*Taeniopygia guttata*)	Symptom suppression	Prevent mate avoidance	Suppressed sickness behaviors in males when exposed to novel females, without suppression of physiological immune response (Lopes et al. 2013)
Song sparrow (*Melospiza melodia morphna*)	Symptom suppression	Hide vulnerability to prevent conspecific aggression; prevent avoidance by conspecifics	Suppressed sickness behaviors in males during the active breeding season (Owen-Ashley and Wingfield 2006)
Rats (*Rattus novegicus*)	Symptom suppression	Prevent mate avoidance	Suppressed sickness behaviors in males when exposed to novel females (Yirmiya et al. 1995)
Mice (*Mus musculus*)	Symptom suppression	Hide vulnerability to prevent conspecific aggression; deter predators	No change in pain behaviors after tissue and nerve damage (Urban et al. 2011). Reduced pain sensitivity after exposure to conspecific odors (Fanselow 1985). Reduced pain behaviors in presence of dominant individuals (Gioiosa et al. 2009)

continued

TABLE 1
Continued

Taxa	Signaling strategy	Proposed function	Evidence
Humans (*Homo sapiens*)	Symptom suppression	Hide vulnerability to prevent conspecific aggression; signal physical robustness	Decreased pain expression and sensitivity in social contexts (Levine and De Simone 1991; Kállai et al. 2004; Aslaksen et al. 2007; Vigil and Coulombe 2011)
Humans (*Homo sapiens*)	Self-induced illness	Elicit aid/social support	Social and physical stressors, as well as lack of communication, are positively related to the presence of somatoform disorders and rates of self-injury (Nichter 1981; Low 1985; Mallouh et al. 1995; Tulloch et al. 1997; Stuart and Noyes 1999; Noyes et al. 2002; Yates 2004; Glassman et al. 2007; Nock and Mendes 2008; Åslund et al. 2010)
Humans (*Homo sapiens*)	Self-induced illness	Avoid costly obligations	Soldiers induce injury to avoid battle (Bourke 1996; Wessely 2003). Adolescents report using self-injury to avoid obligations (Nock and Prinstein 2004)
Humans (*Homo sapiens*)	Self-induced illness	Signal cooperative intent	Costly male rites are more common in societies with frequent warfare (Sosis et al. 2007)

in the presence of individuals with shared interests, such as kin and long-term relationship partners. In contrast, symptoms should be least informative when interests conflict, and when it is difficult to discriminate between real and feigned symptoms (Wiley 1994). This may explain why symptoms of internal illness, such as pain and injury, are so frequently feigned.

Although symptoms that vary as a function of social context are likely candidates for signaling explanations, social contingency is neither necessary nor sufficient for identifying symptoms with signal functions. Not all signals vary as a function of social context (e.g., badges of status; Maynard Smith and Harper 2003). Further, nonsignaling hypotheses based in life-history tradeoffs in energetic investment also predict such fluctuations (Lopes 2014). For example, male zebra finches may suppress sickness behaviors when exposed to novel females, not as an attempt to deceive females, but because males shift energy allocation toward reproductive effort and away from sickness behaviors and immune responses (Lopes et al. 2013). These are not mutually exclusive hypotheses. Fitness is affected by life-history allocations and by information transmission to others, both of which shape optimal phenotype expression. It may be possible to differentiate between these hypotheses. If individuals adjust symptom expression to alter conspecific behavior, then the only symptoms that should fluctuate are those attended to by conspecifics. Alternatively, if individuals adjust symptom expression because of energy-allocation concerns, any energetically costly aspects of symptom expression may evolve to fluctuate in social contexts, regardless of conspecific responses. Differentiating between these hypotheses requires detailed studies of receiver psychology: only by determining how conspecifics attend and respond to specific symptoms can we understand the species-specific selection pressures shaping symptom form.

Determining the signal function of symptoms is an additional challenge. If symptoms are designed to avoid costly obligations, they should be exaggerated when obligation costs are high. If symptoms function to elicit aid, they should be exaggerated in interactions with those who partially share

interests with the signaler and provide aid as a function of symptom severity. Kin, relationship partners, and social institutions may fit these criteria. If symptoms function to alert kin of infection status, they should be exaggerated when infections are most virulent and in the presence of close kin (Shakhar and Shakhar 2015). Without shared interests, selection will not favor receivers who provide aid to signalers, unless provisioners obtain other benefits (e.g., reputational gains) that outweigh the direct costs of provisioning. Individuals with the greatest need should express the most exaggerated forms of aid-eliciting symptoms, as they benefit most from receiving aid. Symptom exaggeration should be most common in species where individuals receive aid during times of illness. Given the ultra-cooperative nature of humans, it is no surprise that symptom feigning and exaggeration are so common. If humans self-induce symptoms to signal cooperative intent, symptoms should be most common among individuals without established reputations in their social group and before high-stakes cooperative ventures. If symptoms function as honest signals of individual robustness, such that only the highest quality individuals can afford to produce or maintain them, they should covary with other metrics of individual quality, such as body size or fluctuating asymmetry (Dongen 2006; Schwagmeyer and Mock 2008). Symptoms may also signal robustness by varying inversely to individual quality, such that the highest quality individuals display the least severe symptoms. Symptom suppression to hide vulnerability should occur in contexts where others benefit from exploiting such vulnerabilities, or selectively avoid symptomatic individuals (e.g., intraspecific competition, mate choice, and predator-prey interactions). Alternatively, individuals may benefit from exaggerating infectious disease symptoms to prevent the approach of unwanted individuals, or from generating symptoms to alert kin of infection status. Status differences between individuals may also affect symptom expression. Dominants may benefit from hiding vulnerability by suppressing symptoms, thereby preventing subordinates from making displacement attempts. If so, dominants should suppress symptoms in the presence of closely ranked individuals. Subordinates may benefit from feigning symptoms or "sandbagging" around dominants to signal that they do not pose a threat. On the other hand, ill subordinates may benefit from suppressing symptoms around dominants, if dominants preferentially exploit ill individuals.

Discussion

This paper has argued for the explanatory and predictive utility of conceptualizing symptoms within a signaling framework. Selection can favor symptom production to influence the behavior of others, even in the absence of organic disease. This means that "medically unexplained" symptoms may not be uniquely human. Such symptoms can evolve when individuals attend to the symptoms of others and when producing physiologically mediated symptoms has value added over mere feigning (e.g., feigners cannot perfectly mimic actual symptoms, or are less consistent and are "caught" more often). Selection can also favor symptom suppression, as long as the social benefits of suppression outweigh the costs of compromised illness recovery. Symptoms with high suboptimal symptom expression costs should be least likely to evolve into signals. Fever may be one such case (Kluger et al. 1975).

Humans use symptoms as signals in contexts that did not exist in ancestral environments. We can successfully do so because these contexts are not entirely novel: they resemble, along relevant parameters, those environments that were encountered by our species over its evolutionary history (Barrett 2006). For example, illness and disability were recurrent fitness threats in ancestral environments, and humans have long relied on conspecific aid during times of hardship (Gurven et al. 2000). Just as symptom exaggeration may have increased aid provisioning in ancestral environments, it can do so in "novel" modern environments: people can exaggerate signs of injury to receive sympathy from coworkers

or care from relationship partners. Natural selection can also adapt organisms to novel environments by favoring the evolution of developmental systems with open-ended reaction norms, where all inputs and outputs are not prespecified. Such developmental systems lead organisms to reliably develop certain phenotypes in the face of novelty. Humans reliably develop food concepts, even though items considered "food" are not entirely prespecified by natural selection, acceptable foods vary widely across different environments, and humans constantly encounter "novel" foods (Barrett 2015). Similarly, humans reliably develop to feign and exaggerate symptoms for financial or social gain, even though the symptoms they use and contexts in which they do so vary widely and did not exist ancestrally. The structure of these reaction norms remains an important question, and researchers can make substantial progress by evaluating symptom-signaling behaviors across the range of social and ecological environments in which humans live.

Conceptualizing symptoms as signals has important implications for understanding psychiatric conditions. If somatoform disorders serve signaling functions, their severity may not always be constant, but fluctuate as a function of social context. Although diagnostic classifications of mental disorders change over time, there is a longstanding distinction between different psychiatric syndromes associated with physical-symptom complaints (American Psychiatric Association 1994, 2013). Somatization disorder is characterized by the unconscious production of symptoms to gain attention and exploit the social benefits of the sick role. Factitious disorders include the conscious production of symptoms, with Munchausen syndrome as an extreme case. Malingering is the conscious production of symptoms for external gain, such as financial reward (Huffman and Stern 2003). A signaling analysis suggests that such disorders are not qualitatively different. Rather, they are maladaptive extremes of a continuum of adaptive human behavioral strategies that adjust symptom expression for personal gain. This implies that current psychiatric classifications are failing to carve nature at its joints.

The symptom-signaling hypothesis has implications for public health. If symptoms function to elicit aid, social support alone may relieve certain symptoms. Additionally, symptom feigning can be reduced by altering the benefits obtained by symptomatic individuals. Illness treatment and compensation, such as time off from work or financial benefits, can benefit both truly ill and healthy individuals. This incentivizes healthy people to feign illness, and ill people to ex-

TABLE 2
Fruitful directions for future research

Do organisms suppress symptoms near competitors or potential mates? Do life-history tradeoffs in energetic investment or signaling better explain such suppression?
Are hosts able to selectively downregulate the expression of symptoms that increase pathogen transmission in the presence of kin?
Can selection ever favor exaggeration, rather than suppression, of infectious disease symptoms?
How is symptom expression shaped by conflicts of interest between pathogen and host?
Do shared interests lead to more honest symptom expression?
Do individuals selectively adjust expression of those symptoms that most influence conspecific behavior?
How do interactions with higher-ranking or lower-ranking individuals affect symptom expression?
When do organisms self-induce injury as opposed to feigning symptoms?
Are internal pathologies more likely to be feigned than external ones?
What are the costs of symptom alteration and how do these vary as a function of the underlying pathology?
Why do symptoms vary in their information value regarding underlying pathology?
If symptoms can be feigned, then why do others continue responding to them?
Are organisms more likely to exaggerate symptoms in one-shot rather than repeated interactions?
To what extent are symptom-signaling strategies dependent on social learning?
How do the norms regarding treatment of ill individuals affect symptom expression?

aggerate it. Changing the structure of treatment and compensation to something that only benefits the truly ill will prevent such deception: if treatments are painful and time consuming, the costs of treatment may not be worth it for those feigning injury (Zahavi 1975).

Conceptualizing symptom expression within the framework of signaling theory raises novel questions and suggests important directions for future research (see Table 2). It also highlights the utility of evolutionary theory to medicine. In analyzing the ultimate causes of symptom expression, scholars in evolutionary medicine have focused on a handful of explanations: adaptive host defenses, pathogen manipulation of hosts, side effects of disease, constrains on optimality, or evolutionary disequilibrium (Ewald 1980; Nesse 2011). It is now time to update evolutionary medicine's analytical toolkit by adding signaling to the list of candidate explanations for symptoms of illness.

ACKNOWLEDGMENTS

I am grateful to Alex Danvers, Daniel Fessler, Willem Frankenhuis, Joseph Hackman, Randolph Nesse, Mariya Voytyuk, and two anonymous reviewers for providing constructive comments on earlier drafts. I am also grateful to Rob Boyd, Evelyn Cholakians, Willem Frankenhuis, Joseph Hackman, and Randolph Nesse for many useful discussions, and to Maciek (Zombieheart) Chudek for his intellectual guidance. Special thanks to Daniel Hruschka for all of the above. I also thank the School of Human Evolution and Social Change and the Center for Evolution and Medicine at Arizona State University for financial support.

REFERENCES

Aduan R. P., Fauci A. S., Dale D. C., Herzberg J. H., Wolff S. M. 1979. Factitious fever and self-induced infection: a report of 32 cases and review of the literature. *Annals of Internal Medicine* 90:230–242.

American Psychiatric Association. 1994. *Diagnostic and Statistical Manual of Mental Disorders: Diagnostic Criteria from DSM-IV.* Fourth Edition. Washington (DC): American Psychiatric Association.

American Psychiatric Association. 2013. *Diagnostic and Statistical Manual of Mental Disorders (DSM-5).* Fifth Edition. Arlington (Virginia): American Psychiatric Association.

Aslaksen P. M., Myrbakk I. N., Høifødt R. S., Flaten M. A. 2007. The effect of experimenter gender on autonomic and subjective responses to pain stimuli. *Pain* 129:260–268.

Åslund C., Starrin B., Nilsson K. W. 2010. Social capital in relation to depression, musculoskeletal pain, and psychosomatic symptoms: a cross-sectional study of a large population-based cohort of Swedish adolescents. *BMC Public Health* 10:715.

Avitsur R., Cohen E., Yirmiya R. 1997. Effects of interleukin-1 on sexual attractivity in a model of sickness behavior. *Physiology and Behavior* 63: 25–30.

Barash D. P. 1975. Evolutionary aspects of parental behavior: distraction behavior of the alpine accentor. *Wilson Bulletin* 87:367–373.

Barrett H. C. 2006. Modularity and design reincarnation. Pages 199–217 in *The Innate Mind, Volume 2: Culture and Cognition*, edited by P. Carruthers, S. Laurence, and S. Stich. Oxford (United Kingdom): Oxford University Press.

Barrett H. C. 2015. *The Shape of Thought: How Mental Adaptations Evolve.* Oxford (United Kingdom): Oxford University Press.

Bateson M., Brilot B., Nettle D. 2011. Anxiety: an evolutionary approach. *Canadian Journal of Psychiatry* 56:707–715.

Behringer D. C., Butler M. J., Shields J. D. 2006. Ecology: avoidance of disease by social lobsters. *Nature* 441:421.

Bergstrom C. T., Lachmann M. 1998. Signaling among relatives. III. Talk is cheap. *Proceedings of the National Academy of Sciences of the United States of America* 95:5100–5105.

Bourke J. 1996. *Dismembering the Male: Men's Bodies, Britain and the Great War.* Chicago (Illinois): University of Chicago Press.

Bouwman K. M., Hawley D. M. 2010. Sickness behaviour acting as an evolutionary trap? Male house finches preferentially feed near diseased conspecifics. *Biology Letters* 6:462–465.

Boyd R., Mathew S. 2015. Third-party monitoring and sanctions aid the evolution of language. *Evolution and Human Behavior* 36:475–479.

Brown M. Z., Comtois K. A., Linehan M. M. 2002. Reasons for suicide attempts and nonsuicidal self-injury in women with borderline personality disorder. *Journal of Abnormal Psychology* 111:198–202.

Brown S. G., Shirachi S., Zandbergen D. 2015. Health selection theory: an explanation for the paradox between perceived male well-being and mortality. *Quarterly Review of Biology* 90:3–21.

Brunton D. H. 1990. The effects of nesting stage, sex, and type of predator on parental defense by

killdeer (*Charadrius vociferous*): testing models of avian parental defense. *Behavioral Ecology and Sociobiology* 26:181–190.

Byrne R. W., Stokes E. 2003. Can monkeys malinger? Page 54 in *Malingering and Illness Deception*, edited by P. W. Halligan, C. Bass, and D. A. Oakley. Oxford (United Kingdom): Oxford University Press.

Cassidy J. D., Carroll L. J., Côté P., Lemstra M., Berglund A., Nygren A. 2000. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *New England Journal of Medicine* 342:1179–1186.

Clutton-Brock T. 2009. Cooperation between non-kin in animal societies. *Nature* 462:51–57.

Collen M. 2014. Pain and treatment from a human primate perspective. *Journal of Pain and Palliative Care Pharmacotherapy* 28:152–157.

Craig K. D. 2009. The social communication model of pain. *Canadian Psychology/Psychologie Canadienne* 50:22–32.

Crandall C. S., Moriarty D. 1995. Physical illness stigma and social rejection. *British Journal of Social Psychology* 34:67–83.

Curtis V., Aunger R., Rabie T. 2004. Evidence that disgust evolved to protect from risk of disease. *Proceedings of the Royal Society B: Biological Sciences* 271: S131–S133.

Curtis V., de Barra M., Aunger R. 2011. Disgust as an adaptive system for disease avoidance behaviour. *Philosophical Transactions of the Royal Society B: Biological Sciences* 366:389–401.

Curtis V. A. 2014. Infection-avoidance behaviour in humans and other animals. *Trends in Immunology* 35:457–464.

Dall S. R. X., Giraldeau L.-A., Olsson O., McNamara J. M., Stephens D. W. 2005. Information and its use by animals in evolutionary ecology. *Trends in Ecology and Evolution* 20:187–193.

David G. K., Condon C. H., Bywater C. L., Ortiz-Barrientos D., Wilson R. S. 2011. Receivers limit the prevalence of deception in humans: evidence from diving behaviour in soccer players. *PLOS ONE* 6:e26017.

de Waal F. B. M. 2007. *Chimpanzee Politics: Power and Sex Among Apes*. Baltimore (Maryland): Johns Hopkins University Press.

Dongen S. V. 2006. Fluctuating asymmetry and developmental instability in evolutionary biology: past, present and future. *Journal of Evolutionary Biology* 19:1727–1743.

Engel G. L. 1981. The clinical application of the biopsychosocial model. *Journal of Medicine and Philosophy* 6:101–124.

Ewald P. W. 1980. Evolutionary biology and the treatment of signs and symptoms of infectious disease. *Journal of Theoretical Biology* 86:169–176.

Fábrega H. Jr. 1999. *Evolution of Sickness and Healing*. Berkeley (California): University of California Press.

Fanselow M. S. 1985. Odors released by stressed rats produce opioid analgesia in unstressed rats. *Behavioral Neuroscience* 99:589–600.

Ferrari R., Schrader H. 2001. The late whiplash syndrome: a biopsychosocial approach. *Journal of Neurology, Neurosurgery and Psychiatry* 70:722–726.

Fessler D. M. T., Eng S. J., Navarrete C. D. 2005. Elevated disgust sensitivity in the first trimester of pregnancy: evidence supporting the compensatory prophylaxis hypothesis. *Evolution and Human Behavior* 26:344–351.

FitzGibbon C. D., Fanshawe J. H. 1988. Stotting in Thomson's gazelles: an honest signal of condition. *Behavioral Ecology and Sociobiology* 23:69–74.

Fleischman D. S., Fessler D. M. T. 2011. Progesterone's effects on the psychology of disease avoidance: support for the compensatory behavioral prophylaxis hypothesis. *Hormones and Behavior* 59: 271–275.

Flor H., Kerns R. D., Turk D. C. 1987. The role of spouse reinforcement, perceived pain, and activity levels of chronic pain patients. *Journal of Psychosomatic Research* 31:251–259.

Gallup G. G. 1977. Tonic immobility: the role of fear and predation. *Psychological Record* 27:41–61.

Gatchel R. J., Peng Y. B., Peters M. L., Fuchs P. N., Turk D. C. 2007. The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychological Bulletin* 133:581–624.

Gioiosa L., Chiarotti F., Alleva E., Laviola G. 2009. A trouble shared is a trouble halved: social context and status affect pain in mouse dyads. *PLOS ONE* 4:e4143.

Glassman L. H., Weierich M. R., Hooley J. M., Deliberto T. L., Nock M. K. 2007. Child maltreatment, non-suicidal self-injury, and the mediating role of self-criticism. *Behaviour Research and Therapy* 45: 2483–2490.

Godfray H. C. J., Johnstone R. A. 2000. Begging and bleating: the evolution of parent-offspring signalling. *Philosophical Transactions of the Royal Society B: Biological Sciences* 355:1581–1591.

Grafen A. 1990. Biological signals as handicaps. *Journal of Theoretical Biology* 144:517–546.

Grimes S. A., Cruickshank A. D., Rutter R. J., Cottam C., Laskey A. R., Roads M. K. 1936. "Injury feigning" by birds. *The Auk* 53:478–482.

Guilford T., Stamp Dawkins M. 1991. Receiver psychology and the evolution of animal signals. *Animal Behaviour* 42:1–14.

Gurven M., Allen-Arave W., Hill K., Hurtado M. 2000. "It's a wonderful life": signaling generosity among the Ache of Paraguay. *Evolution and Human Behavior* 21:263–282.

Gurven M., Stieglitz J., Hooper P. L., Gomes C., Kaplan H. 2012. From the womb to the tomb: the role of transfers in shaping the evolved human life history. *Experimental Gerontology* 47:807–813.

Haccoun R. R., Dupont S. 1987. Absence research: a critique of previous approaches and an example for a new direction. *Canadian Journal of Administrative Sciences* 4:143–156.

Hadjistavropoulos T., Craig K. D., Duck S., Cano A., Goubert L., Jackson P. L., Mogil J. S., Rainville P., Sullivan M. J. L., de C. Williams A. C., Vervoot T., Fitzgerald T. D. 2011. A biopsychosocial formulation of pain communication. *Psychological Bulletin* 137:910–939.

Hagen E. H. 2003. The bargaining model of depression. Pages 95–123 in *Genetic and Cultural Evolution of Cooperation*, edited by P. Hammerstein. Cambridge (Massachusetts): MIT Press.

Hagen E. H., Watson P. J., Hammerstein P. 2008. Gestures of despair and hope: a view on deliberate self-harm from economics and evolutionary biology. *Biological Theory* 3:123–138.

Halligan P. W., Bass C., Oakley D. A. 2003a. *Malingering and Illness Deception*. Oxford (United Kingdom): Oxford University Press.

Halligan P. W., Bass C., Oakley D. A. 2003b. Wilful deception as illness behaviour. Pages 3–28 in *Malingering and Illness Deception*, edited by P. W. Halligan, C. Bass, and D. A. Oakley. Oxford (United Kingdom): Oxford University Press.

Hamilton W. D. 1964. The genetical evolution of social behaviour. I. *Journal of Theoretical Biology* 7:1–16.

Hart B. L. 1988. Biological basis of the behavior of sick animals. *Neuroscience and Biobehavioral Reviews* 12:123–137.

Hellyer P., Rodan I., Brunt J., Downing R., Hagedorn J. E., Robertson S. A., AAHA/AAFP Pain Management Guidelines Task Force Members. 2007. AAHA/AAFP pain management guidelines for dogs and cats. *Journal of Feline Medicine and Surgery* 9: 466–480.

Hennessy M. B., Deak T., Schiml P. A. 2014. Sociality and sickness: have cytokines evolved to serve social functions beyond times of pathogen exposure? *Brain, Behavior, and Immunity* 37:15–20.

Henningsen P., Zipfel S., Herzog W. 2007. Management of functional somatic syndromes. *The Lancet* 369:946–955.

Huffman J. C., Stern T. A. 2003. The diagnosis and treatment of Munchausen's syndrome. *General Hospital Psychiatry* 25:358–363.

Jones I. H., Barraclough B. M. 1978. Auto-multilation in animals and its relevance to self-injury in man. *Acta Psychiatrica Scandinavica* 58:40–47.

Kállai I., Barke A., Voss U. 2004. The effects of experimenter characteristics on pain reports in women and men. *Pain* 112:142–147.

Kane P., Zollman K. J. S. 2015. An evolutionary comparison of the handicap principle and hybrid equilibrium theories of signaling. *PLOS ONE* 10:e0137271.

Kato K., Sullivan P. F., Evengård B., Pedersen N. L. 2006. Chronic widespread pain and its comorbidities: a population-based study. *Archives of Internal Medicine* 166:1649–1654.

Kavaliers M., Colwell D. D. 1995. Discrimination by female mice between the odours of parasitized and non-parasitized males. *Proceedings of the Royal Society B: Biological Sciences* 261:31–35.

Kiesecker J. M., Skelly D. K., Beard K. H., Preisser E. 1999. Behavioral reduction of infection risk. *Proceedings of the National Academy of Sciences of the United States of America* 96:9165–9168.

Kleinman A. 1982. Neurasthenia and depression: a study of somatization and culture in China. *Culture, Medicine, and Psychiatry* 6:117–190.

Kluger M. J., Ringler D. H., Anver M. R. 1975. Fever and survival. *Science* 188:166–168.

Kozlowska K. 2007. The developmental origins of conversion disorders. *Clinical Child Psychology and Psychiatry* 12:487–510.

Lachmann M., Számadó S., Bergstrom C. T. 2001. Cost and conflict in animal signals and human language. *Proceedings of the National Academy of Sciences of the United States of America* 98:13189–13194.

Lafferty K. D. 1992. Foraging on prey that are modified by parasites. *American Naturalist* 140:854–867.

Langford D. J., Crager S. E., Shehzad Z., Smith S. B., Sotocinal S. G., Levenstadt J. S., Chanda M. L., Levitin D. J., Mogil J. S. 2006. Social modulation of pain as evidence for empathy in mice. *Science* 312:1967–1970.

Larrabee G. J. 2003. Detection of malingering using atypical performance patterns on standard neuropsychological tests. *Clinical Neuropsychologist* 17: 410–425.

Leonard M. L., Horn A. G. 2005. Ambient noise and the design of begging signals. *Proceedings of the Royal Society B: Biological Sciences* 272:651–656.

Levine F. M., De Simone L. L. 1991. The effects of experimenter gender on pain report in male and female subjects. *Pain* 44:69–72.

Libow J. A. 2000. Child and adolescent illness falsification. *Pediatrics* 105:336–342.

Linton R., Devereux G. E. 1956. *Culture and Mental Disorders*. Springfield (Illinois): Thomas.

Lloyd-Richardson E. E., Perrine N., Dierker L., Kelley M. L. 2007. Characteristics and functions of non-suicidal self-injury in a community sample of adolescents. *Psychological Medicine* 37:1183–1192.

Loehle C. 1995. Social barriers to pathogen transmission in wild animal aopulations. *Ecology* 76: 326–335.

Lopes P. C. 2014. When is it socially acceptable to feel sick? *Proceedings of the Royal Society B: Biological Sciences* 281:20140218.

Lopes P. C., Adelman J., Wingfield J. C., Bentley G. E. 2012. Social context modulates sickness behavior. *Behavioral Ecology and Sociobiology* 66: 1421–1428.

Lopes P. C., Chan H., Demathieu S., González-Gómez P. L., Wingfield J. C., Bentley G. E. 2013. The impact of exposure to a novel female on symptoms of infection and on the reproductive axis. *Neuroimmunomodulation* 20:348–360.

Low S. M. 1985. Culturally interpreted symptoms or culture-bound syndromes: a cross-cultural review of nerves. *Social Science and Medicine* 21:187–196.

Mallouh S. K., Abbey S. E., Gillies L. A. 1995. The role of loss in treatment outcomes of persistent somatization. *General Hospital Psychiatry* 17:187–191.

Marx B. P., Forsyth J. P., Gallup G. G., Fusé T., Lexington J. M. 2008. Tonic immobility as an evolved predator defense: implications for sexual assault survivors. *Clinical Psychology: Science and Practice* 15:74–90.

Maynard Smith J. 1991. Honest signalling: the Philip Sidney game. *Animal Behaviour* 42:1034–1035.

Maynard Smith J., Harper D. 2003. *Animal Signals*. Oxford (United Kingdom): Oxford University Press.

Mayou R., Farmer A. 2002. Functional somatic symptoms and syndromes. *BMJ: British Medical Journal* 325:265–268.

Mesa M. G., Poe T. P., Gadomski D. M., Petersen J. H. 1994. Are all prey created equal? A review and synthesis of differential predation on prey in substandard condition. *Journal of Fish Biology* 45(Supplement):81–96.

Mitri S., Floreano D., Keller L. 2009. The evolution of information suppression in communicating robots with conflicting interests. *Proceedings of the National Academy of Sciences of the United States of America* 106:15786–15790.

Mitri S., Floreano D., Keller L. 2011. Relatedness influences signal reliability in evolving robots. *Proceedings of the Royal Society B: Biological Sciences* 278:378–383.

Mittenberg W., Patton C., Canyock E. M., Condit D. C. 2002. Base rates of malingering and symptom exeggeration. *Journal of Clinical and Experimental Neuropsychology* 24:1094–1102.

Miyatake T., Katayama K., Takeda Y., Nakashima A., Sugita A., Mizumoto M. 2004. Is death-feigning adaptive? Heritable variation in fitness difference of death-feigning behaviour. *Proceedings of the Royal Society B: Biological Sciences* 271:2293–2296.

Miyatake T., Nakayama S., Nishi Y., Nakajima S. 2009. Tonically immobilized selfish prey can survive by sacrificing others. *Proceedings of the Royal Society B: Biological Sciences* 276:2763–2767.

Montgomerie R. D., Weatherhead P. J. 1988. Risks and rewards of nest defence by parent birds. *Quarterly Review of Biology* 63:167–187.

Monto A. S., Gravenstein S., Elliott M., Colopy M., Schweinle J. 2000. Clinical signs and symptoms predicting influenza infection. *Archives of Internal Medicine* 160:3243–3247.

Nesse R. M. 2011. Ten questions for evolutionary studies of disease vulnerability. *Evolutionary Applications* 4:264–277.

Newton-John T. R. O. 2002. Solicitousness and chronic pain: a critical review. *Pain Reviews* 9:7–27.

Nichter M. 1981. Idioms of distress: alternatives in the expression of psychosocial distress: a case study from south India. *Culture, Medicine, and Psychiatry* 5:379–408.

Nock M. K. 2008. Actions speak louder than words: an elaborated theoretical model of the social functions of self-injury and other harmful behaviors. *Applied and Preventive Psychology* 12:159–168.

Nock M. K., Mendes W. B. 2008. Physiological arousal, distress tolerance, and social problem-solving deficits among adolescent self-injurers. *Journal of Consulting and Clinical Psychology* 76:28–38.

Nock M. K., Prinstein M. J. 2004. A functional approach to the assessment of self-mutilative behavior. *Journal of Consulting and Clinical Psychology* 72: 885–890.

Nock M. K., Prinstein M. J. 2005. Contextual features and behavioral functions of self-mutilation among adolescents. *Journal of Abnormal Psychology* 114:140–146.

Novak M. A. 2003. Self-injurious behavior in rhesus monkeys: new insights into its etiology, physiology, and treatment. *American Journal of Primatology* 59:3–19.

Noyes R. Jr., Stuart S., Langbehn D. R., Happel R. L., Longley S. L., Yagla S. J. 2002. Childhood antecedents of hypochondriasis. *Psychosomatics* 43:282–289.

Noyes R. Jr., Stuart S. P., Langbehn D. R., Happel R. L., Longley S. L., Muller B. A., Yagla S. J. 2003. Test of an interpersonal model of hypochondriasis. *Psychosomatic Medicine* 65:292–300.

Orbach-Zinger S., Ginosar Y., Sverdlik J., Treitel C., MacKersey K., Bardin R., Peleg D., Eidelman L. A. 2012. Partner's presence during initiation of epidural labor analgesia does not decrease maternal stress: a prospective randomized controlled trial. *Anesthesia and Analgesia* 114:654–660.

Owen-Ashley N. T., Wingfield J. C. 2006. Seasonal modulation of sickness behavior in free-living northwestern song sparrows (*Melospiza melodia morphna*). *Journal of Experimental Biology* 209:3062–3070.

Palmer I. P. 2003. Malingering, shirking, and self-inflicted injuries in the military. Pages 42–53 in *Malingering and Illness Deception*, edited by P. W. Halligan, C. Bass, and D. A. Oakley. Oxford (United Kingdom): Oxford University Press.

Parris K. M., Velik-Lord M., North J. M. A. 2009. Frogs call at a higher pitch in traffic noise. *Ecology and Society* 14:25.

Parsons C. D. F., Wakeley P. 1991. Idioms of distress: somatic responses to distress in everyday life. *Culture, Medicine, and Psychiatry* 15:111–132.

Partheni M., Constantoyannis C., Ferrari R., Nikiforidis G., Voulgaris S., Papadakis N. 2000. A prospective cohort study of the outcome of acute whiplash injury in Greece. *Clinical and Experimental Rheumatology* 18:67–70.

Plesker R., Mayer V. 2008. Nonhuman primates mask signs of pain. *Laboratory Primate Newsletter* 47:1–3.

Price J. S., Gardner R. Jr., Erickson M. 2004. Can depression, anxiety and somatization be understood as appeasement displays? *Journal of Affective Disorders* 79:1–11.

Raber P., Devor M. 2002. Social variables affect phenotype in the neuroma model of neuropathic pain. *Pain* 97:139–150.

Rodham K., Hawton K., Evans E. 2004. Reasons for deliberate self-harm: comparison of self-poisoners and self-cutters in a community sample of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry* 43:80–87.

Romano J. M., Turner J. A., Friedman L. S., Bulcroft R. A., Jensen M. P., Hops H., Wright S. F. 1992. Sequential analysis of chronic pain behaviors and spouse responses. *Journal of Consulting and Clinical Psychology* 60:777–782.

Romano J. M., Jensen M. P., Turner J. A., Good A. B., Hops H. 2000. Chronic pain patient-partner interactions: further support for a behavioral model of chronic pain. *Behavior Therapy* 31:415–440.

Sargeant A. B., Eberhardt L. E. 1975. Death feigning by ducks in response to predation by red foxes (*Vulpes fulva*). *American Midland Naturalist* 94:108–119.

Schaefer H. M., Ruxton G. D. 2009. Deception in plants: mimicry or perceptual exploitation? *Trends in Ecology and Evolution* 24:676–685.

Schaller M. 2011. The behavioural immune system and the psychology of human sociality. *Philosophical Transactions of the Royal Society B: Biological Sciences* 366:3418–3426.

Schwagmeyer P. L., Mock D. W. 2008. Parental provisioning and offspring fitness: size matters. *Animal Behaviour* 75:291–298.

Searcy W. A., Nowicki S. 2005. *The Evolution of Animal Communication: Reliability and Deception in Signaling Systems.* Princeton (New Jersey): Princeton University Press.

Shakhar K., Shakhar G. 2015. Why do we feel sick when infected—can altruism play a role? *PLOS Biology* 13:e1002276.

Shannon C. E., Weaver W. 1949. *The Mathematical Theory of Communication.* Urbana (Illinois): University of Illinois Press.

Sherwin C. M., Christiansen S. B., Duncan I. J., Erhard H. W., Lay D. C. Jr., Mench J. A., O'Connor C. E., Petherick J. C. 2003. Guidelines for the ethical use of animals in applied ethology studies. *Applied Animal Behaviour Science* 81:291–305.

Silk J. B., Kaldor E., Boyd R. 2000. Cheap talk when interests conflict. *Animal Behaviour* 59:423–432.

Simons R. C., Hughes C. C. 1985. *The Culture-Bound Syndromes: Folk Illnesses of Psychiatric and Anthropological Interest.* Dordrecht (The Netherlands): D. Reidel.

Soltau U., Dötterl S., Liede-Schumann S. 2009. Leaf variegation in *Caladium steudneriifolium* (Araceae): a case of mimicry? *Evolutionary Ecology* 23:503–512.

Sordahl T. A. 1990. The risks of avian mobbing and distraction behavior: an anecdotal review. *Wilson Bulletin* 102:349–352.

Sosis R., Kress H. C., Boster J. S. 2007. Scars for war: evaluating alternative signaling explanations for cross-cultural variance in ritual costs. *Evolution and Human Behavior* 28:234–247.

Stamp Dawkins M., Guilford T. 1991. The corruption of honest signalling. *Animal Behaviour* 41:865–873.

Stasiak K. L., Maul D., French E., Hellyer P. W., Vandewoude S. 2003. Species-specific assessment of pain in laboratory animals. *Journal of the American Association for Laboratory Animal Science* 42:13–20.

Steinkopf L. 2015. The signaling theory of symptoms: an evolutionary explanation of the placebo effect. *Evolutionary Psychology* 13:1474704915600559.

Stuart S., Noyes R. Jr. 1999. Attachment and interpersonal communication in somatization. *Psychosomatics* 40:34–43.

Stuart S., Noyes R. Jr. 2006. Interpersonal psychotherapy for somatizing patients. *Psychotherapy and Psychosomatics* 75:209–219.

Sugiyama L. S. 2004. Illness, injury, and disability among Shiwiar forager-horticulturalists: implications of health-risk buffering for the evolution of human life history. *American Journal of Physical Anthropology* 123:371–389.

Sullivan M. J. L., Adams H., Sullivan M. E. 2004. Communicative dimensions of pain catastrophizing: social cueing effects on pain behaviour and coping. *Pain* 107:220–226.

Suter P. B. 2002. Employment and litigation: improved by work, assisted by verdict. *Pain* 100:249–257.

Syme K. L., Garfield Z. H., Hagen E. H. 2015. Testing the bargaining vs. inclusive fitness models of suicidal behavior against the ethnographic record. *Evolution and Human Behavior.* doi:10.1016/j.evolhumbehav.2015.10.005.

Számadó S. 2000. Cheating as a mixed strategy in a simple model of aggressive communication. *Animal Behaviour* 59:221–230.

Számadó S. 2011. The cost of honesty and the fallacy of the handicap principle. *Animal Behaviour* 81:3–10.

Takken W. 1991. The role of olfaction in host-seeking of mosquitoes: a review. *International Journal of Tropical Insect Science* 12:287–295.

Trivers R. L. 1971. The evolution of reciprocal altruism. *Quarterly Review of Biology* 46:35–57.

Tulloch A. L., Blizzard L., Pinkus Z. 1997. Adolescent-parent communication in self-harm. *Journal of Adolescent Health* 21:267–275.

Tybur J. M., Lieberman D., Kurzban R., DeScioli P. 2013. Disgust: evolved function and structure. *Psychological Review* 120:65–84.

Urban R., Scherrer G., Goulding E. H., Tecott L. H., Basbaum A. I. 2011. Behavioral indices of ongoing pain are largely unchanged in male mice with tissue or nerve injury-induced mechanical hypersensitivity. *Pain* 152:990–1000.

Vervoort T., Goubert L., Eccleston C., Verhoeven K., De Clercq A., Buysse A., Crombez G. 2008. The effects of parental presence upon the facial expression of pain: the moderating role of child pain catastrophizing. *Pain* 138:277–285.

Vigil J. M., Coulombe P. 2011. Biological sex and social setting affects pain intensity and observational coding of other people's pain behaviors. *Pain* 152: 2125–2130.

Vigil J. M., Strenth C. 2014. No pain, no social gains: a social-signaling perspective of human pain behaviors. *World Journal of Anesthesiology* 3:18–30.

Waldinger R. J., Schulz M. S., Barsky A. J., Ahern D. K. 2006. Mapping the road from childhood trauma to adult somatization: the role of attachment. *Psychosomatic Medicine* 68:129–135.

Walker F. R., Brogan A., Smith R., Hodgson D. M. 2004. A profile of the immediate endocrine, metabolic and behavioural responses following a dual exposure to endotoxin in early life. *Physiology and Behavior* 83:495–504.

Wenegrat B. 2001. *Theater of Disorder: Patients, Doctors, and the Construction of Illness*. Oxford (United Kingdom): Oxford University Press.

Wessely S. 2003. Malingering: historical perspectives. Pages 31–41 in *Malingering and Illness Deception*, edited by P. W. Halligan, C. Bass, and D. A. Oakley. Oxford (United Kingdom): Oxford University Press.

Wiley R. H. 1994. Errors, exaggeration, and deception in animal communication. Pages 157–189 in *Behavioral Mechanisms in Evolutionary Ecology*, edited by L. A. Real. Chicago (Illinois): University of Chicago Press.

Williams A. C. de C. 2002. Facial expression of pain, empathy, evolution, and social learning. *Behavioral and Brain Sciences* 25:475–480.

Yap P. M. 1967. Classification of the culture-bound reactive syndromes. *Australian and New Zealand Journal of Psychiatry* 1:172–179.

Yates T. M. 2004. The developmental psychopathology of self-injurious behavior: compensatory regulation in posttraumatic adaptation. *Clinical Psychology Review* 24:35–74.

Yirmiya R., Avitsur R., Donchin O., Cohen E. 1995. Interleukin-1 inhibits sexual behavior in female but not in male rats. *Brain, Behavior, and Immunity* 9:220–233.

Zahavi A. 1975. Mate selection—a selection for a handicap. *Journal of Theoretical Biology* 53:205–214.

Zeman J., Garber J. 1996. Display rules for anger, sadness, and pain: it depends on who is watching. *Child Development* 67:957–973.

Zollman K. J. S., Bergstrom C. T., Huttegger S. M. 2013. Between cheap and costly signals: the evolution of partially honest communication. *Proceedings of the Royal Society B: Biological Sciences* 280:20121878.

Zylberberg M., Klasing K. C., Hahn T. P. 2013. House finches (*Carpodacus mexicanus*) balance investment in behavioural and immunological defences against pathogens. *Biology Letters* 9:20120856.

Associate Editor: Paul W. Ewald
Handling Editor: James D. Thomson