

Chapter 5

On the Functions of Sadness and Grief

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An account of sadness and grief is offered that focuses on their evolutionary function (Lench, et al., 2015). Sadness and grief are distinct, yet complementary adaptive responses to stress. Sadness is characterized by low physiological arousal, whereas grief is characterized by higher physiological arousal and a propensity to weep. Three general responses to stress are proposed: (1) an immune response—principally an energy-conserving state that is coordinated with enhanced immune activity; (2) a cognitive response—principally a reflective disposition characterized by more realistic situational appraisals, ultimately encouraging adaptive actions; and (3) a social response—minimally an appeal to halt aggression, and more broadly, an appeal for altruistic assistance.

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In *The Expression of Emotions in Man and Animals*, Darwin (1872) observed how *sorrow* differs from *grief*: Darwin characterized grief as “frantic” and “energetic,” whereas sorrow was “languid” and “resigned.” In much modern emotion research, Darwin’s distinction between grief and sorrow is ignored, and “sadness” is regarded as a single basic affect. The difference between these two states is readily observed among children: when unhappy, a child may engage in sustained crying, or quiescent morose sadness.

Grief and *sadness* (the terms used here) are negative mood states precipitated by similar circumstances. However, this chapter will show that carefully delineating their physiological and psychological differences will lead to an evolutionary account that points to different, yet complementary, functions. It should be noted at the outset that the theory proposed here is not a theory of depression. Any biological system can assume pathological states. Freed, (2009) has characterized depression as a “sadness disorder”—a claim that Horwitz and Wakefield (2007) note has been expressed by Western thinkers

dating back to Hippocrates. Although the discussion will draw, from time-to-time, on research on depression, the focus here will be on normal rather than pathological behaviors. The chapter reviews evidence that (normal) sadness and grief are evolved behaviors that usually enhance inclusive fitness.

An appropriate place to begin is with detailed descriptions of sadness and grief. An integrated description may include at least seven different perspectives. A biological description focuses on genetic, neurologic, endocrine, and general physiological correlates. Etiological description focuses on precipitating stressors (biological, physical, cognitive, or social) that might induce the state. Affective description identifies subjective feelings that are presumed to amplify motivation. Cognitive description identifies patterns of thought that correlate with the psychological state. A developmental perspective focuses on the emergence and possible changes in how the psychological state is manifested over a lifespan. Behavioral description stresses the characteristic actions, postures, or expressions, associated with the state. Social-psychological description concentrates on how the state is communicated and interpreted by conspecifics within a social context. Finally, a cultural description identifies cultural manifestations of the state, including possible ritualization, as well as potential cultural origins of the state. As will be evident, the evolutionary account proposed here offers a plausible narrative at each of these descriptive levels.

Sadness

What causes sadness? Common etiology for sadness includes failures to achieve life goals, such as romantic, parental, or occupational goals. Sadness might be caused by the departure of a loved one, the breakup of a romantic relationship, parenting difficulties, loneliness, unemployment, financial problems, poor health, loss of social status, frustration in the pursuit of goals, or the inability to help others. Sadness can also arise from more basic vulnerabilities, such as from hunger, thirst, cold, injury, insecurity, or chronic fear. In general, sadness is associated with failure or powerlessness. In affluent societies, sadness is more likely to arise from social stressors—leading some researchers to conclude that sadness is exclusively social in origin (e.g., Allen & Badcock, 2003).

The main physiological symptom of sadness is *anergia* or low arousal. When sad, heart rate decreases and respiration is slower and shallower. Reduced levels of epinephrine, norepinephrine, acetylcholine, and serotonin are observed along with increased levels of the stress hormones cortisol and prolactin (although see Andrews & Thomson, 2009). In the peripheral nervous system, low acetylcholine is associated with poor muscle tone and slow muscle reactivity—making movement slow and lethargic (e.g., Siegel & Sapru, 2006). Low norepinephrine is associated with decreased attention and reduced engagement with the world (e.g., Viggiano, Ruocco, Arcieri, & Sadile, 2004). Low serotonin is linked to diminished self-esteem (e.g., Raleigh et al., 1991). Behaviorally, sadness commonly leads to reduced activity, slumped posture, slow movement, infrequent speech, weak voice, disrupted sleep, changes in appetite, diminished interest, and social withdrawal. Perhaps the most important behavioral change, however, is cognitive: sadness tends to lead to sustained reflection about one's life situation (Nesse, 1991; Andrews & Thompson, 2009).

Further behavioral characteristics are evident in the relaxation of all facial muscles. Relaxing the jaw causes the chin to drop downward. Relaxing the zygomatic muscles (involved in smiling) causes the cheeks to flatten—reducing the physical width of the face. The lowered chin and flattened cheeks contribute to the “long face” appearance—a description synonymous in several languages with being sad. Apart from the appearance of a longer face, the relaxation of the facial musculature is also associated with lowered head and drooping eyelids.

Sadness is also associated with characteristic changes in speech patterns. Sad or depressed speech is quieter, slower, with a lower overall pitch height. In addition, sad speech exhibits a more monotone prosody (narrow pitch movements), mumbled articulation, breathier voice, and a darker timbre or tone color (Kraepelin, 1899/1921; Murray & Arnott, 1993; Banse & Scherer, 1996; Scherer, Johnstone, & Klasmeyer, 2003; Erickson et al., 2006). However, the most important clinical observation regarding sad speech is the tendency for sad individuals to remain mute: sad people speak less (Kraepelin, 1899/1921).

The principal affective symptom of sadness is *anhedonia*. Many activities that are normally enjoyable lose their allure, including food, sex, play, and socializing. Apart from reduced pleasure in various activities, the phenomenal experience of sadness entails a distinctive gloomy qualia that is negatively valenced (Nesse, 1991).

Grief

The etiology of grief is similar to sadness. Like sadness, grief accompanies failures to achieve life goals, including romantic, social, or occupational goals. As with sadness, grief may be precipitated by extreme hunger, cold, poor health, insecurity, etc. In cultures around the globe, grief is most reliably induced by the death of a loved one (Rosenblatt, Walsh, & Jackson, 1976; p. 15). Whereas sadness can also be precipitated by failures to achieve life goals, grief more commonly accompanies the loss of already existing resources, including the reversal of current fortunes or the failure for highly anticipated outcomes to transpire. Interestingly, grief is more likely than sadness to occur in response to feelings of guilt or shame (Vingerhoets, 2013). Both grief and sadness are symptoms of adversity, failure, vulnerability, or powerlessness. Although grief and sadness are precipitated by similar circumstances, they differ principally in the magnitude of the loss or failure: grief is more likely to be associated with especially onerous failures or losses.

From a physiological perspective, grief (in contrast to sadness) is associated with an increase in epinephrine. The heart rate increases, blood pressure increases, breathing becomes deeper and more erratic. The most characteristic symptom of grief is weeping. In its full-blown expression, weeping entails a flushed face, nasal congestion, constricted pharynx, punctuated exhaling, vocalized wailing, and the shedding of tears. Pharyngeal constriction is typically described as either “a lump in one’s throat” or feeling “choked up”.

Tears probably represent the most stereotypic visual feature of weeping. Physiologists distinguish three types of tears: basal, reflex, and psychic (Frey, 1985). *Basal tears* serve to lubricate the eyeballs and are constantly being secreted. *Reflex tears* are

generated in response to irritation. *Psychic tears* are produced when we experience strong emotions (Lutz, 2001; 67-68). Apart from grief, psychic tears can also be produced from laughing (Frey, 1985).

With regard to the development of grief, the most notable change is the reduced amount of weeping that occurs with increased age. Babies cry a lot, children less so, and adults cry rarely. The amount of weeping appears to be related to the dependency on others. As an individual becomes more autonomous—able to fend for herself/himself—the frequency of weeping is reduced. Crying tends to increase among the elderly, but here too, it may be related to feelings of dependency or vulnerability (Rosenblatt et al., 1976; Vingerhoets, 2013).

In terms of observable behavior, grief is easy to recognize. Ekman has described the main facial features as including an open mouth, with the corners of the lips turned down, cheeks raised as if squinting, downward turned eyes, with drooping upper eyelids, and the inner corners of the eyebrows pulled up (Ekman, 2003; p. 95-96). Tears are a prototypical symptom of grief. When a person weeps for an extended period, the face tends to become red and puffy, with inflammation common surrounding the eyes, including redness of the eyes themselves—i.e., vasodilation of the blood vessels of the conjunctiva (Provine et al., 2011). Aside from the face, grief-related behaviors may include outstretched arms, and occasional acts of self-injury such as slapping one's face, pulling one's hair, or beating one's chest (e.g., Gertsman, 2011; Maguire, 1977).

Apart from the visible behaviors, grief is also associated with characteristic sounds. Vocalized wailing, combined with punctuated breathing produces the distinctive “ah-ah-ah” weeping sound. Nasal congestion leads to nasalized vocal timbre, while the constriction of the pharynx leads to characteristic resonances aptly described by linguists as “pharyngealized.” Post-nasal drip encourages rapid inhalation—producing a characteristic sniffing sound. Frick (1985, p. 420) notes that crying appears to involve a general tendency to contract the muscles of the face and neck. Pharyngealization, for example, involves strong contraction of the muscles of the pharynx. Extended weeping often leads to a feeling of soreness at the back of the throat due to the intense muscle contraction. Creaky voice involves muscle contractions that draw the arytenoid cartilages together. Falsetto phonation involves tensing the edges of the vocal folds. The strong contractions in the region of the vocal folds also account for the instability between falsetto and modal phonation that is responsible for cracking or “breaking” voice (Švec & Pešák, 1994). Constriction of the pharynx also appears to be the source of ingressive phonation. That is, the narrowing of the throat would naturally lead to sounds we associate with gasping (while inhaling).

It is possible to experience degrees of grief with only some of the symptoms appearing. For example, a person might simply feel a tightening in the throat (“choked up”) without any further symptoms. One might experience “incipient tears”—where tears simply well up along the lower eyelids without any tears actually dropping onto the cheeks; or where the eyes appear moist without any fluid evident. The corners of the mouth might turn downward without the mouth opening. In particular, the vocalizing can vary from no sound at all, to quiet whimpering, moaning, crying, or loud wailing.

Phenomenologically, grief is characterized by a strongly negative affective state. Grief vies with physical pain for the most negatively valenced affect. Although grief is normally regarded as an agonizing or miserable feeling, the experience of grief has led to some of the most touching and profound of human expressions. We find compelling expressions or portrayals in poetry, literature, visual art, and music.

Grief can be private and individual; but grief can also be public and communal, and therefore cultural. All over the world, cultures have shaped distinctive rituals for grieving, including funerary rites that provide both public and private contexts for grieving (e.g., Ebersole, 2000; Hockey, Katz, & Small, 2001; Marsella, Sartorius, Jablensky, & Fenton, 1985; Murphy, Wittkower, & Chance, 1964).

Many scholars have suggested that grief is an evolved behavior and a few scholars have proposed phylogenetic precursors for human crying (e.g., Frey, Ahern, Gunderson, & Tuason, 1986; Hasson, 2009; Montagu, 1960; Murube, 2009; Roes, 1989; Trimble, 2012). Panksepp and Bernatzky (2002), for example, have suggested that human grief might have biological origins in the phenomenon of separation distress (see also Zeifman, 2001; Vingerhoets, 2013). When removed from their mothers, offspring will often make distinctive calls (Panksepp, 1998).

Although something similar to grief may be experienced by many animals, weeping appears to be uniquely human. From time-to-time, there are reports of other animals shedding tears (such as elephants), however, no consistence evidence has been assembled (Bard, 2000; Vingerhoets, 2013). Watery eyes in response to psychic loss have not been observed in our closest relatives—chimpanzees, bonobos, or gorillas (Bard, 2000).

Mourning Cycle

In *Mourning and Melancholia*, Freud proposed that sadness is a variant of grief (Freud, 1917). Following after Freud, Bowlby (1961, 1973), proposed that the active (grief) and passive (sadness) responses represent stages or phases in a grief event. However, subsequent studies suggest that the two responses do not represent stages (Spencer-Booth & Hinde, 1971, Hinde & Spencer-Booth, 1971, as cited in Archer, 1999; p. 56). Instead, the two states frequently alternate back-and-forth. When in a state of mourning, for example, it is common for a person to experience periods of active weeping alternating with periods of passive sadness (Hofer, 1984), what we refer to as the *mourning cycle*.

Signals and Cues

In comparing sadness and grief, it is helpful to review the signal/cue distinction in ethology (Lorenz, 1937; Maynard Smith & Harper, 2003). A *signal* is a functional communicative act. An example of a signal is a rattlesnake's rattle. The rattle is used as a warning when the snake encounters another animal that could cause harm. The aim is to avoid unnecessary conflict. By contrast, a *cue* is an unintended conveyance of information. An example of a cue is the sound of a buzzing mosquito prior to an attack. In both cases, the sounds convey information—alerting the observer to the possibility of being attacked. However, the source of the information differs. In the case of the snake's rattle, the

communication is a functional behavior: the snake's interest is best served when the signal is perceived and recognized. In the case of the mosquito's buzzing, the communication (cue) is incidental—a byproduct of the need for the mosquito to move its wings.

In the case of signals, ethologists interpret these displays as evolutionary adaptations, and so ask “what is the adaptive value that a signal provides?” From an ethological perspective, if a smile is an innate behavior that serves an evolutionary purpose, then the smile must serve an adaptive purpose for the individual generating the smile. Accordingly, the purpose of a signal is to change the behavior of the observer to the benefit of the signaler (Bradbury & Vehrenkamp, 1998). Notice that if smiling led to reduced inclusive fitness, then smiling would be selected against and so disappear as a behavior.

If signals are intended to be overtly communicated, then signals should be obvious rather than subtle. The adaptive value of the signal depends on its successful communication. A signal is said to exhibit *redundancy* when the signal is repeated or sustained over time and over multiple channels (Wiley, 1983; Johnstone 1997). Communication is more likely to occur if the signal involves more than one modality (Partan & Marler, 1999). In the case of the rattlesnake's rattle, the signal entails both a distinctive auditory feature (the rattling sound) as well as a distinctive visual feature (the raised shaking tail). On theoretical grounds then, ethological signals are more likely to exhibit features in more than one sensory modality.

Since cues are artifacts, they may exhibit either unimodal or multimodal features. Since cues are not intended to be communicative, unlike signals, there exists no selection pressure for an existing unimodal cue to accrue multimodal features that would enhance conspicuousness. Tinbergen (1952, 1964) cogently argued that all signals evolve from cues (see also Maynard Smith & Harper, 2003). That is, if a cue offers an adaptive advantage for the animal exhibiting the cue, then selection pressures would lead to enhancing or amplifying the communicative properties of the cue. One of the best ways to enhance the conspicuousness of a newly evolved signal is to add features in another modality. As we will see, this process provides helpful clues that illuminate the adaptive functions of sadness and grief.

Apart from their conspicuous design, signals also differ from cues in how they influence observers. Signals are functional acts that “push” information into the environment. Cues are incidental phenomena where an observant individual “pulls” unintended information out of the environment. Cues are deciphered purely for the benefit of the observer. However, signals initiate a transaction that is commonly beneficial to both the signaling and observing animals (Maynard Smith & Harper, 2003).

Consider, by way of example, the submission or capitulation display in dogs. When one dog is attacked by another, the attacked animal may produce a submission display by rolling on its back and (commonly) making a whimpering sound. This behavior typically has a dramatic impact on the aggressor animal—immediately terminating the attack. The benefit for the signaling dog is that the aggression is stopped, reducing the likelihood of life-threatening injury. However, this favorable outcome is purchased at the cost of a loss of social status. The gain for the attacking dog is that it has established a relative social dominance over the submissive animal. Both animals benefit from the interaction.

Unlike cues, in the case of signals, both the signaling behavior and the response of the observer are stereotypic: they co-evolve. Under the appropriate circumstances, the signaling behavior is largely automatic—despite its costliness (Zahavi & Zahavi, 1997). At the same time, the response of the observer is similarly largely automatic. That is, the responses are biologically prepared. Once again, we will see that these widely accepted concepts in ethology provide helpful clues regarding the adaptive functions of sadness and grief.

Sadness as Cue

From an ethological perspective, an appropriate question is whether sadness and grief represent signals or cues. This review begins with sadness. Recall the seven acoustic features of sad speech: What—we might ask—do quieter, slower, lower pitch, small pitch movement, more mumbled, breathier, and dark timbre all share in common? The answer is that all seven features are associated with low physiological arousal. In the peripheral nervous system, low arousal is linked to reduced acetylcholine, which in turn reduces both tone and reactivity for skeletal muscles (Siegel & Sapru, 2006). Low arousal would therefore be associated with *relaxed* and *slow* muscles.

All of the peripheral muscles of the body are affected, including the muscles of the vocal folds, tongue, lips, chin, and pulmonary muscles. Reduced muscle tone will cause the vocal folds to be less tense, resulting in a lower overall pitch as well as breathier phonation (Hollien, 1960). A slower cricothyroid muscle will produce more sluggish pitch changes—and therefore generate a more monotone prosody (Sundberg, 1987). Relaxed pulmonary muscles result in lower subglottal air pressure, causing a quieter voice. Slower reactivity of tongue, lips and chin will result in a slower rate of speech and more slurred or mumbled articulation. When the zygomatic muscles of the face are relaxed, the lips tend to fall away from the teeth (in contrast to smiling); this results in a longer effective vocal tract length with a concomitant lower resonance—producing a darker timbre (Tartter, 1980). In short, *all* of the acoustical features of sad speech can be plausibly attributed to the effects of low physiological arousal—in particular, the effects of reduced acetylcholine.

Sadness is not the only state that will cause low arousal. Low arousal is most commonly experienced when people are relaxed, tired, or sleepy. In an unpublished experiment, we asked actors to distinguish sad voice from sleepy voice. Excluding the telltale sound of yawning, we found that listeners are unable to distinguish between feigned sadness and feigned sleepiness. Like sad speech, tired or sleepy speech exhibit quieter sound intensity, slower speaking rate, lower pitch, more monotone phonation, breathier voice, and mumbled articulation.

Consider now the visual aspects associated with sadness. Recall that sadness is associated with slumped posture and relaxed facial muscles. Eyelids may droop slightly and the eyes may gaze downward without focusing on particular objects. Relaxation of the zygomatic muscles tends to flatten the cheeks, and relaxation of the mouth tends to cause the chin to descend. As in the case of sad speech, all of these features are attributable to low physiological arousal.

By way of summary, the features associated with both sad speech and sad facial expressions appear to be indistinguishable from other low arousal states—notably sleepy, tired, or relaxed states. It may be that there exist reliable features that successfully distinguish sad from sleepy, tired or relaxed states. However, the apparent ease of confusion is not consistent with the properties of conspicuousness and redundancy that ethologists consider important for signaling (Johnstone, 1997; Partan & Marler, 1999; Wiley, 1983). Moreover, recall that the most characteristic auditory feature of sad voice is the tendency for sad individuals to remain mute. The tendency to reduce or avoid vocalization is not consistent with signaling. In summary, sadness better conforms to the concept of an ethological cue rather than an ethological signal.

Grief as Signal

Consider now the parallel ethological question regarding grief: Is grief a cue or a signal? Visual features include a furrowed brow, squinting eyes, down-turned corners of the mouth, flushed red face, and of course, tears (Ekman, 1982). Auditory features of grief include vocalized punctuated exhaling, long sustained tones (wails), ingressive vocalization, use of falsetto phonation, breaking voice, creaky voice, pharyngealization, and sniffing. When crying, sounds may range from quiet whimpering to loud sobbing or wailing. Tears can be shed without producing a sound, but the prototypical crying involves notable spontaneous sound production.

The strongest evidence regarding the compulsion to vocalize is the phenomenon of *ingressive phonation*—where the vocal folds are activated while inhaling. When crying, puffs of air are forced through the vocal folds while exhaling. When inhaling, it is common to hear a gasping sound akin to a neutral vowel or schwa sound. Phoneticians are well aware of how unusual this behavior is: 99.99 percent of all human vocalizations occur while exhaling. In a small number of languages, linguists have shown that on rare occasions, some phonemes involve ingressive phonation. However, across the majority of cultures, ingressive phonation is evident in only two behaviors: crying and laughing.

The phenomenon of ingressive phonation is highly suggestive. When weeping, the motor system appears steadfast in keeping the vocal folds engaged. Whether exhaling or inhaling, the air flows through the folds, ensuring that a sound is made. The physiological behavior is consistent with a resolute involuntary compulsion to vocalize. The contrast with sadness is striking: once again, when sad, people tend not to vocalize.

As noted earlier, the constriction of the pharynx changes the acoustic resonance of the vocal tract leading to a sort of “pinched” or pharyngealized sound. However, the pharyngeal constriction also leads to phonetic instability where the voice chaotically switches back-and-forth between modal and falsetto phonation. This results in a highly distinctive “cracking” or “breaking” voice, which—even more than whimpering or wailing—is the quintessential sound of weeping.

Perhaps the most compelling evidence that a particular behavior represents a true ethological signal is the presence of a dedicated anatomical feature or organ. In the case of the rattlesnake, the rattle represents a purpose-specific anatomical organ. This raises the question of whether weeping involves any purpose-specific anatomy? In general, the

various elements of grief do not appear to be behaviorally unique. Tear ducts are used to shed irritant tears as well as psychic tears; the motor actions involved in punctuated exhaling are shared with laughter; falsetto phonation is found in infant-directed speech; a red face is shared with blushing; nasal congestion is an unwelcome companion of most colds; and frowning is shared with anger. Nevertheless, there is at least one component that appears to be unique.

As noted earlier, ophthalmologists distinguish basal (lubricant) tears, reflex (irritant) tears, and psychic (emotional) tears. Reflex and psychic tears originate in the same lacrimal sacs. However, reflex and psychic tears appear to be evoked by different neural pathways. An ophthalmologist can anesthetize the entire eye so that basal and reflex tears are inhibited. However, given a suitably emotional stimulus, an anesthetized patient can still cry psychic tears because of the existence of a separate neural pathway (Kottler, 1996; p. 63; Earley, personal communication). The existence of a distinct limbic path for producing psychic tears implies an evolved neuro-anatomical feature, consistent with weeping as an evolved behavior—rather than being an artifact of some other process.

Recall that another characteristic feature of signals is that they tend to directly influence the behavior of others: signals are foremost intended to change the actions of the observer. Gelstein and her colleagues (2011) collected tears from women volunteers who were induced to weep by watching a sad scene from a movie. When asked to smell the collected tears, men were unable to distinguish actual tears from a control saline solution. Nevertheless, the real psychic tears produced a marked physiological effect: testosterone concentrations (as measured in the men's saliva) dropped significantly when the men were exposed to the real tears. In addition, other measures showed that smelling the tears significantly impeded sexual arousal. These behavioral changes suggest the presence of a pheromone in psychic tears. The effect is unconscious and automatic. In short, the work of Gelstein and colleagues (2011) is consistent with crying as an ethological signal that induces a biologically prepared stereotypic response.

The main purpose of emotions is to act as motivational amplifiers (Tomkins, 1980). When we experience some affective state, we are more likely to behave in certain ways. When we encounter someone crying, feelings of compassion or sympathy encourage us to offer assistance (or terminate aggression). Consistent with an ethological signal, expressions of grief have a marked impact on the thoughts, feelings, and actions of those who witness them.

Finally, we need to ask whether grief expressions are subtle or obvious? A simple Internet search for photographs of people crying will confirm that weeping expressions are nearly always unmistakable. It is possible that confusion might arise for a person suffering from an allergy like hay fever. However, allergy sufferers typically do not show a furrowed brow, squinting eyes, or a down-turned mouth. Moreover, what photographs do not convey are the characteristic sounds of weeping: the punctuated vocalized exhaling, the sounds of whining or wailing, or the distinctive “cracking” voice. In contrast to sadness, grief appears to be obvious. Even when a grief-stricken person makes efforts to hide their face, or otherwise mask their grief, observers seem to have little difficulty detecting weeping. It is not simply the case that grief is conspicuous, it also seems to be the case that

people are especially sensitive and vigilant for the slightest indications that a person may be weeping.

By way of summary, human weeping appears to exhibit all of the hallmarks of an ethological signal. The features of weeping are multimodal, including distinctive visual, auditory, and olfactory elements. Together, the combination of elements makes weeping unmistakable. Weeping has a strong influence on the behavior of observers—an effect that appears limited to our own species. There appears to exist a separate limbic pathway for generating psychic tears—implying a unique evolved anatomical concomitant. In particular, the rare compulsion to vocalize while inhaling and the pheromonal effect of tears on the endocrine levels of observers provide especially strong evidence consistent with the notion that weeping is an ethological signal.

Weeping as Surrender/Solicit Signal

What is the biological function of weeping? The subject of human crying has attracted extensive theoretical speculation (e.g., Cornelius, 1997; Frey, 1985; Kottler, 1996; Lutz, 2001; Vingerhoets, 2013; Vingerhoets & Cornelius, 2001, Vingerhoets, Cornelius, Van Heck, & Becht, 2000). There exists a broad consensus that adult weeping is a form of help-seeking behavior (Becker, 1933; Bowlby, 1961; Engel, 1962; Farberow & Shneidman, 1961; Gorer, 1965; Henderson, 1974; Lewis, 1934; and many others). Moreover, this idea long predates modern research and can be found in ancient texts and commentaries in many cultures.

Kottler and Montgomery (2001; p.10) offer an important supplement to this theory. Specifically, they characterize adult weeping as a *surrender display* analogous to raising a “white flag.” The effect is illustrated in the following first-hand account relayed by Kottler (1996):

A male physician had been verbally abusing a female hospital administrator. The more she apologized, the more he berated her. It was clear he was not accepting her [apology].

All of a sudden, a tear welled up in her eye, just a single tear, and ran down her cheek. He stopped cold. This guy, big time surgeon and all, used to having his way and blustering onward, just stopped dead. This tiny spot of wetness communicated to him very clearly what he otherwise had not seen. He started backpedaling so fast, apologizing like crazy. That single tear had meaning for him a way that nothing else did. (Kottler, 1996, p. 68-69).

The important observation here is the transformation in the affective state (and consequent behavior) of the surgeon—from aggression to compassion (and perhaps even embarrassment). This single anecdotal example is consistent with more representative empirical research. For example, Lane (2006) showed that in interpersonal conflict situations, weeping tends to bring the conflict to a resolution, whereas the absence of weeping is likely to lead to an escalation of the conflict.

In short, weeping appears to exhibit at least two functions: a specific appeal to terminate aggression, and an appeal for assistance. With the surrender function, human weeping more closely resembles analogous displays that are ubiquitous among other social animals—such as the submission or capitulation display when a dog rolls over on its back in response to attack. As an ethological signal, weeping has a dramatic effect on observers, evoking feelings of compassion that commonly terminates aggression and encourages altruistic behaviors.

Depressive Realism

People commonly hold overly optimistic assessments of the likelihood of achieving certain goals (Alicke & Govorun, 2005; Brown & Marshall, 2000; Ross & Nisbett, 1991; Weinstein, 1987). When experiencing sadness, one might suppose that people tend to become pessimistic; however, research suggests that we are our most realistic when sad—a phenomenon called *depressive realism* (Alloy & Abrahamson, 1979; Moore & Fresco, 2012). Compared with happy or neutral affect, sadness promotes more detail-oriented thinking (Clore & Huntsinger, 2007), reduced stereotyping (Bless & Fiedler, 2006), less judgment bias (Clore & Huntsinger, 2007; Tan & Forgas, 2010), greater memory accuracy (Storbeck & Clore, 2005; Forgas, Goldenberg & Unkelbach, 2009), reduced gullibility (Forgas & East, 2008), more task perseverance (Goldenberg & Forgas, 2012), more social attentiveness and politeness (Forgas, 1995, 2002), more accurate assessments of the emotional states of others (Weary & Edwards, 1994; Yost & Weary, 1996), and improved reasoning related to social risks (Badcock & Allen, 2003). Andrews and Thomson (2009) suggest that sad feelings are adaptive for analyzing complex problems. (See Karnaze & Levine, Chapter 4, this volume, for a more detailed discussion of the cognitive benefits of sadness.)

Nesse (1991) has suggested that the optimism that characterizes normal mental life encourages individuals to strive to achieve goals that might be attainable with effort; conversely, depressive realism provides a mental “grounding” or “reality check” when those same goals prove elusive. That is, low mood is likely to discourage futile efforts that may squander crucial resources. The benefits of depressive realism have (over the past two decades) led a number of researchers to argue that ordinary sadness is commonly beneficial and that depression is overly diagnosed in Western cultures (e.g., Andrews & Thomson, 2009; Hagen, 2011; Horwitz & Wakefield, 2007; Keedwell, 2008; Nesse, 2000; Sharot, 2011; Wilson, 2008).

The apparent cognitive benefits associated with sadness appear to contradict classic adverse symptoms characteristic of major depressive disorders, namely *ruminatio*n. Rumination is a cognitive state in which the individual repeatedly recalls past situations or failures, dwelling on negative thoughts and self-assessments. Rumination is broadly regarded as destructive and unhelpful. In a ground-breaking study, Trapnell and Campbell (1999) carried out principal components analysis involving a large sample of responses for items on the Self-Consciousness Scale (Fenigstein, Scheier, & Buss, 1975; Scheier & Carver, 1985). They found a clear disassociation between two independent components they deemed *ruminatio*n and *reflectio*n. Rumination represents brooding thoughts

dominated by negative self-assessments. Reflection, by contrast, represents a pattern of thought related to self-awareness and self-knowledge (see also Joireman, Parrott & Hammersla, 2002).

The value of analytic processing is evident in several studies reviewed by Andrews and Thomson (2009). For example, encouraging depressed patients to reflect on their condition through expressive writing is more likely to alleviate depressive symptoms than amplify them (e.g., Gortner, Rude, & Pennebaker, 2006; Graf, Gaudiano, & Geller, 2008). Although reflective thoughts are typically more negatively valenced than thoughts experienced when not sad, reflective thinking exhibits greater verisimilitude. In short, the reflective thinking pattern described by Trapnell and Campbell converges with research on depressive realism. Ruminative thinking appears to be one of the main discriminators between (pathological) major depressive disorders and (normal) sadness. Unlike rumination, reflective thinking serves a useful role. On balance, normal sadness appears to be an adaptive behavior likely to enhance inclusive fitness (Andrews & Thomson, 2009; see also Karnaze & Levine, Chapter 4, this volume).

Purpose of Sadness and Grief

By way of summary, we have seen that sadness resembles an ethological cue, whereas grief resembles an ethological signal. Recall that cues are not intended to be communicative. Signals, by contrast, are overtly communicative and exist to change the behavior of observers to the benefit of the signaler. In short, sadness is a self-directed state, whereas grief is an other-directed state (see also Hagen, 2011).

When a person faces difficulties in life, there are several possible resources that can be recruited to help deal with the situation. One resource is our own mental capacities. We have seen that normal sadness is associated with reflection (as opposed to rumination). This reflective state is associated with cognitive abilities that exceed normal (optimistic) mentation in the accuracy of memory, cognition, and assessment. Through careful analysis, less biased perception, consideration of options, and realistic appraisal, an individual can strategize—forming a plan of action that may help resolve, overcome, or ameliorate a difficult situation. A second resource is our social network. The support of others can be solicited. Companions, partners, acquaintances, and even complete strangers may be induced to intervene and provide crucial support in dealing with a stressful circumstance.

The theory proposed here is that sadness and grief are distinct yet complementary states that arise in response to difficult conditions (stress). Both sadness and grief normally contribute to inclusive fitness. When we fail or encounter a problem, sadness is intended to optimize our own behaviors; grief is intended to favorably change the behavior of those around us.

This view has repercussions for interpreting the commonly observed mourning cycle. According to the theory proposed here, the alternation between periods of quiescent sadness and periods of active grieving represent phases of inward-directed and socially-directed behaviors. Notice that the proportion of time allotted to each behavior is likely to be shaped by the severity of the situation, the ability of the individual to cope with the situation alone, the capacity of others to be able to offer genuinely useful assistance, and

the willingness of the individual to incur the social cost associated with appeals for help. Minor failures are apt to lead to sadness without grief. Major failures are apt to require the help of others, and so result in grief. This chapter will consider the relative costs and benefits of various behaviors in more detail in a later section.

The suggestion that sadness is a self-oriented *cue* while grief is an other-oriented *signal* raises a number of questions. For example, if weeping is intended to change the behavior of observers, why would anyone cry alone? Similarly, if weeping is intended to be communicated, why would anyone attempt to suppress or hide their weeping? These, and other questions, will be addressed in due course.

Immune Response

Over the past decade, research on sadness and depression has drawn attention to the importance of the immune system. (Felger & Lotrich, 2013, Miller, Haroon, Raison & Felger, 2013; Raison, Capuron & Miller, 2006). Along with cognitive and social stressors, basic forms of stress also include injury and illness. Coping with these latter kinds of stressors has long been the province of the immune system.

When highly active, the immune system places considerable demands on metabolic resources. Indeed, an active immune system is comparable to the brain as a high-demand energy consumer. The effectiveness of the immune response is reduced if the immune system must compete for metabolic resources. Of various energy-commanding systems, voluntary motor movements can be singled-out as the most “optional.” By refraining from movement, more metabolic resources are available for the immune system (Engel, 1962; Miller et al., 2013). In order to achieve this state, the individual must lose his/her motivation to move. Two feelings contribute to this desirable state: anergia (in the form of fatigue) and anhedonia. Anergia discourages movement itself. Anhedonia reduces the incentive to engage in what otherwise might be beneficial behaviors.

The classic symptom of virtually every kind of pathology is inflammation—a response arising from pro-inflammatory cytokines. Recent research implicates pro-inflammatory cytokines in both feelings of anergia and anhedonia. Capuron and colleagues (2002) found that administering pro-inflammatory cytokines decreases putamen and caudate activity, consistent with decreased dopamine synthesis or dopamine release. As might be expected, suppression of dopamine has repercussions for both motor movement and motivation. Specifically, the reduced activity is correlated with feelings of fatigue and anhedonia. Normally rewarding activity is experienced as significantly less alluring.

An example of a pro-inflammatory cytokine is interferon IFN- α , a powerful endogenous antiviral used to treat various infectious diseases, notably hepatitis C. Injecting interferon into a healthy individual precipitates a series of changes including a rise in body temperature, feelings of fatigue, muscle pain, and possible headache. In short, the person will feel sick. Between 30 and 50 percent of patients receiving IFN- α in a clinical application will also exhibit symptoms of depression (Capuron et al., 2002; Musselman et al., 2001; Raison et al., 2006). In summary, feelings of sadness or depression appear to enhance the effectiveness of the immune response by discouraging competition for metabolic resources, principally through feelings of anergia and anhedonia (Nesse &

Williams, 1994). Activities that are normally enjoyable lose their appeal, and the person becomes less engaged in the world.

Further evidence of a close relationship between depression and the immune system is suggested by the effects of sleep deprivation. Sleep deprivation has long been known to interfere with immune function (Rogers, Szuba, Staab, Evans, & Dinges, 2001). Ultimately, sleep deprivation leads to death through a catastrophic failure of immune responses (Everson, 1993). Interestingly, sleep deprivation has been used as an effective therapy for short-term treatment of major depressive disorders (Dallaspazia & Benedetti, 2011; Pflug & Tölle, 1971). That is, the relief of depressive symptoms is correlated with reduced immune function.

This simple story may make sense for injury and illness, but much sadness or depression in humans arises from other stressors, notably social stress. Although one might expect that inflammatory responses make no sense in responding to cognitive forms of stress, the research suggests otherwise. The physiological mechanisms involved in cognitive or social stress appear to be elaborations of immune responses. Cytokines have been shown to target the dorsal anterior cingulate cortex (dACC)—a region associated with social pain. For example, Eisenberg and Lieberman (2004) showed that the dACC is activated when experiencing social rejection. Moreover, Slavich and colleagues (2010) found that the intensity of the inflammatory response is predictive of the magnitude of negative feelings in response to subsequent social rejection. In addition, antidepressants that are effective for cognitively induced depression have been shown to inhibit the production and release of pro-inflammatory cytokines, and stimulate the production of anti-inflammatory cytokines (Kenis & Maes, 2002). Altogether, the research implies that ancient immune responses provide the foundation for responses to cognitive and social stressors as well as injury and microbial stressors.

Although ordinary sadness may commonly enhance fitness, a proportion of the population is disposed to suffer from major depressive disorders that are clearly pathological. The etiology of major depressive disorders remains a complex problem addressed by ongoing research. One part of the puzzle appears to involve a genetic concomitant. Meta-analyses of genome-wide association studies suggest that many of the genes implicated in depression are known to enhance immune effectiveness (Raison & Miller, 2013). Miller and colleagues (2013) argue that depression-related genes have been preserved in the human gene pool because of their adaptive value in protecting against pathogens.

Overall, recent research suggests a linkage between immune responsiveness and the negative feeling states associated with sadness and depression. In particular, the negative feeling states are closely linked with pro-inflammatory cytokines. There remains the question of why sadness would be associated with social withdrawal, rather than social approach or appeal. Like voluntary movement, social engagement is likely to incur an energy cost; if the social cost of an appeal for help (via crying) is deemed too high, then avoidance of social interaction would be warranted as a means of conserving energy.

Ritualization

With this background, consider once again the symptoms of weeping: watery eyes, nasal congestion, constriction of the pharynx (lump-in-throat), and erratic breathing. When people cry for an extended period, they typically also experience red swollen eyes, and some individuals may experience urticaria (hives) in response to sustained weeping (Saul & Bernstein, 1941). As noted earlier, these are classic symptoms of a systemic allergic response. In short, weeping itself resembles yet another immune response, linked predominantly to yet another class of pro-inflammatory cytokines—histamines. Consequently, the characteristic features of weeping—from tears to cracking voice, amount to downstream manifestations of immune activity in response to stressors.

Ethologists have long proposed that signals evolve from cues through a process dubbed *ritualization* (Tinbergen, 1952, 1964). Through repetition and amplification, ritualization renders a cue more conspicuous, and so more communicative (Maynard Smith & Harper, 2003). For example, the signaling of aggression by lowering the pitch of the voice originates from the association between large size and low frequency of oscillation. This association begins as a cue, proves useful, and so is subsequently amplified to make it more conspicuous (Huron & Shanahan, 2013).

In light of the recent discoveries identifying the immunological concomitants of sadness, we propose an evolutionary-historical scenario of the process of ritualization by which crying or weeping arose as a signal among *Homo sapiens*. Long before the appearance of social animals, the principal response to attack (either injury or illness) was immunological. Dealing with tissue damage or fighting pathogens was the main function of pro-inflammatory cytokines. As noted, the effectiveness of the immune response is enhanced if there are no other behaviors vying for metabolic resources. Notably, reduced motor movement is beneficial. Accordingly, the benefits from releasing cytokines are augmented when they influence motivation—mainly by evoking lethargic feelings that lead to reduced motor movement, and by discouraging other normal goal-related behaviors by rendering hedonic rewards less appealing. In short, we might expect that at some point in evolutionary history, anergia and anhedonia were added to the effects of the release of pro-inflammatory cytokines. This would have arisen long before the appearance of humans.

At some point in hominini evolution, the release of pro-inflammatory cytokines was broadened to include the release of histamines, with predictable symptoms—including watery eyes, nasal congestion, and pharyngeal constriction. Notice that these allergic symptoms are among the most easily observed signs of immunological stress. Although overt tears may not be common, watery eyes are symptomatic of infection or stress in many mammals, and can be readily observed, for example, in sick dogs and cats. Accordingly, observant conspecifics learn to interpret these symptoms as suggesting that the observed individual is experiencing a stressed (or saddened) state. That is, allergic symptoms initially served as an ethological *cue* for dejected or depressed states—more generally indicating a reduced fitness in the observed individual.

Like many (artifactual) cues, this cue would have been easily misinterpreted. Notably, a dejected or saddened state would not have been distinguishable from a genuine

allergic response. As discussed below, there are advantages for both observer and observed to transform the cue into a signal. Hence the symptoms involved in the cue (tears, congestion, constricted pharynx) undergo selection pressure that enhances their conspicuousness (i.e., ritualization). The small quantity of tears arising from the allergic response is amplified, so psychic tears become more profuse than is the case for an allergy. Most important in the transition from cue to signal is the introduction of an acoustical component that increases conspicuousness by adding another sensory modality. With the exception of occasional sniffing and nose-blowing, the allergy itself exhibits few sonic features. Added to the allergic response, then, is the compulsion to vocalize. This compulsion is evident, first, in sobbing, whining, or wailing. A second distinctive sound is ingressive phonation. As mentioned earlier, the rare behavior of vocalizing while inhaling testifies to a powerful motivation to keep the vocal folds engaged. Finally, two other characteristic sounds are directly attributable to the constricted pharynx. In the first instance, constricting the pharynx leads to the distinctive pharyngealized vocal resonance. In addition, the constricted pharynx produces phonetic instability, leading to abrupt transitions between modal and falsetto phonation producing the quintessential cracking or breaking voice. In effect, the constricted pharynx (originating from the allergic response) affords an exaptation for the ensuing acoustical components of a bona fide weeping signal (Gould & Vrba, 1982).

Vocalization is most communicative when it is loud. Loud vocalization, however, requires relatively high physiological arousal. As noted, sadness and depression are associated with low arousal. Engel (1962) argued that sadness-related anergia serves a resource-conserving function that is an appropriate response to stress. Miller and colleagues (2013) have argued more explicitly that anergia frees metabolic resources for wound healing and fighting infection. Consequently, in the case of crying, there arises a conundrum regarding optimum arousal levels: the goal of tissue repair conflicts with the goal of communication. It is possible that these competing goals account for the wide energy range found in grief-related vocalizations—expressions that can range from quiet/subdued sobbing to loud/energetic wailing.

Co-evolved Responsiveness

Apart from the changes in the signaling animal, ethological signals also entail the co-evolution of a stereotypic response in conspecific observers. The selection pressures for the signaler are clear: weeping benefits the grief-stricken individual either by terminating an attack or by receiving support from an observer. However, what selection pressure exists for the observer to co-evolve a helping behavior? In what way does the signal benefit the observer? Why would an observer experience a feeling such as compassion?

When encountering an individual in need, opportunities arise for the observer to benefit through both kin-related altruism as well as through reciprocal altruism (Trivers, 1971). Kin-related altruism contributes directly to inclusive fitness. However, altruistic acts with non-kin can also contribute to inclusive fitness. Enhanced social status and implied reciprocal obligations for future resource-sharing can often outweigh the proximal cost of helping someone in need.

In short, the act of weeping has the potential to benefit both signaling and observing individuals. It is this potential for mutually beneficial interaction that provides converging selection pressure that propels the ritualization—from cue to signal. What begins as an artifact of histamine release is reshaped into the ethological signal we call weeping.

By way of summary, crying or weeping behavior can be plausibly traced to an evolutionary history beginning with pro-inflammatory responses arising from injury, illness, or defeat. Expanded to include histamines, the readily perceived symptoms offer an ethological cue, informing observers of the sad, stressed, or depressed state of an individual. Altruistic behaviors benefit the signaler, and can also benefit the observer. Consequently, allergy-related symptoms come under selection pressure that amplifies them, and extends them to include multimodal characteristics. An ethological cue is transformed into an ethological signal in accordance with the classic process of ritualization (Tinbergen, 1952, 1964).

The Differential Costs and Benefits of Weeping

The principal cost of weeping is the loss of social status. The principal benefit of weeping is the increased likelihood of terminating aggression and/or the increased likelihood of receiving altruistic assistance. These costs and benefits are not the same for all individuals. High social position is biologically important insofar as it impacts reproductive success (Ellis, 1995; Hopcroft, 2006). Moreover, high social rank disproportionately benefits males. A high-ranked male can father many more offspring than a high-ranked female. In summary, those who pay the greatest costs for weeping are reproducing individuals of highest social rank, with males incurring a greater cost than equivalently ranked females. Those individuals who have the least to lose by weeping are non-reproducing individuals and those at the bottom of the social hierarchy.

These theory-derived predictions accord remarkably well with empirical observations concerning the frequency of crying. The greatest amount of crying is evident in infancy. Crying decreases around 2-3 years of age, when toddlers become more socially engaged, and so are more likely to suffer from the associated social penalty incurred by crying. Sex-linked differences in crying frequency are not evident in early life. Landreth (1941) carried out an extensive developmental study of crying among children between 2 and 5 years of age. She was the first researcher to observe that, in this age group, boys cry (slightly) more often than girls.

At puberty, a dramatic reduction in the frequency of crying is evident for both males and females. At the same time, marked sex-related differences appear at puberty, with males much less likely to cry than females (Delp & Sackeim, 1987). The difference in crying frequency between males and females is evident in nearly every culture observed. Similarly, in many cultures, people of high social station (both male and female) exhibit less crying (Rosenblatt, Walsh & Jackson, 1976).

With the onset of old age, sex-related differences are attenuated. Elderly men are slightly more likely to weep than younger men (Vingerhoets, 2013). These differences are consistent with changes in reproductive fitness among older people. Elderly men

commonly retain some reproductive capacity, but with typically reduced social status. A further reduction of social status due to weeping is less likely to impact reproductive success.

In conjunction with the costs incurred by weeping, one also needs to consider the benefits. Those who have the most to gain are those who are being attacked, those who have little access to resources, and those who lack the skills to acquire resources. Landreth (1941) found a significant negative correlation between crying frequency and IQ. To the extent that IQ correlates with a capacity for acquiring resources, this relationship is consistent with an increased value of crying for those individuals less able to provide for themselves.

Executive Control

In ethology, both the etiology of a signal (also known as a releaser) and the observer's responses are commonly stereotypic. The apparent automaticity of these behaviors led Lorenz (1937) to dub them "innate releasing mechanisms." However, subsequent research has deemphasized the notion that the behaviors are fixed and automatic. For mammals in particular, some degree of executive control may override or modify behaviors that otherwise appear to be instinctive (Immelmann & Beer, 1989).

Compared with other animals, humans have a greater capacity for self-control. Large regions of the frontal cortex are known to serve inhibitory functions—suppressing, modifying, or masking otherwise compelling behaviors. Weak connections from the frontal lobes are implicated in impulsive behavior (Miller & Cummings, 2007).

Despite the fact that weeping appears to be largely involuntary, since weeping incurs a social cost, if the individual assesses the social cost as too burdensome, then the person may attempt to suppress, modify, or mask their weeping (Kraemer & Hastrup, 1988). Such executive control of weeping behavior is evident in several ways. The weeping signal can be physical masked by turning away, hiding one's face, or seeking privacy. If physical masking is impossible, various strategies exist for psychological masking. For example, a person moved to the edge of tears may consciously think of something else—typically mundane—such as the need to fuel the car, or contemplate what to prepare for dinner. That is, prefrontal "executive" control is used to mask or circumvent the propensity to weep.

Conversely, an individual might conclude that, under the circumstances, weeping would be advantageous. In this case, a reverse psychological strategy might be employed, such as thinking sad or tragic thoughts—as in recalling the death of a loved one.

In light of the costs and benefits associated with weeping, one might expect a complex calculus to exist concerning whether or not to weep in a given circumstance. Note, however, that the very fact that people hide their faces, pretend to have something in their eye, or think thoughts that either inhibit or facilitate weeping attests to the comparatively automatic etiology of the behavior—despite its malleability through executive control. That is, the modifications afforded by executive control notwithstanding, weeping exhibits the stereotypic tendencies seen in signaling among nonhuman animals.

The same arguments apply to how individuals respond when they observe weeping. Recall that the principal benefit to the observer is the long-term inclusive fitness conferred by engaging in kin-related or reciprocal altruistic assistance. Notice that potential benefits from reciprocal altruism depend on future interaction with the weeping individual. If the interaction is unique or “one-off,” then the fitness advantage of altruistic assistance is negligible. Indeed, we find considerable anecdotal evidence of the failure for weeping to suspend an attack when the attacker and crier are strangers, or when the potential for future interaction is limited. Both experimental research and game-theoretic models show that the likelihood for cooperation depends on the probability of repeated future interaction (Ahn, Janssen, & Ostrom, 2004; Andreoni & Miller, 1993; Axelrod & Hamilton, 1981). At the same time, executive control is apt to be motivated by such factors as the perceived honesty of the signal, reputation and history of past interactions, and the marginal cost to the observer of offering assistance.

In summary, there is a notable degree of automaticity both with regard to the circumstances leading to weeping, and with regard to observer responses to weeping. However, humans exhibit a considerable capacity for executive control in which such biologically prepared behaviors can be inhibited or masked.

Crying Alone

The foregoing discussions provide answers to two questions posed earlier: If weeping is an ethological signal, and if signals are intended to be communicative, why would anyone weep alone? Moreover, if signals are intended to be communicative, why do people often attempt to mask their weeping—such as seeking privacy, hiding their face, or wiping away tears?

As was just noted, the very fact that people mask or hide their weeping suggests that the act of weeping is largely automatic and involuntary. Circumstances can dispose people to weep, whether they want to or not. This automaticity contributes to the honesty of the signal—reassuring the observer that the weeping is unlikely to arise from intentional manipulation. However, the largely involuntary character of weeping increases the likelihood that a person might weep, even when there is no audience to witness the signal.

Similarly, the second question has already been answered. Since weeping incurs a cost, cognitive appraisals (including cultural norms) might be expected to contribute to a complex calculus of whether the cost is prohibitively high. In these cases, efforts to mask or disguise weeping may be expected.

Infant Crying

As noted, the principal cost for weeping is the loss of social status. The loss of social status is more costly for high status individuals of reproductive age. The least cost is incurred by non-reproducing individuals at the bottom of the social ladder, a group whose members include infants and children. Since these individuals incur the least cost associated with weeping, it follows that they have the most to gain from the altruistic responses evoked by weeping.

To the extent that adults are biologically prepared to respond to weeping with acts of compassion, and since children suffer little cost when they weep, children have much to gain from weeping behaviors. In short, there is a period in human development, when the emergence of weeping allows children to tap into adult compassion at relatively little cost. This window of opportunity closes slightly when toddlers begin earnest social interaction, and closes more fully with sexual maturity when social costs have greater consequences on reproductive success.

At birth, crying is most frequent in the *absence* of a caregiver, suggesting that the main purpose of infant crying is to maintain infant-caregiver contact (e.g., Panksepp & Bernatzky, 2002). However, by two years of age, crying is maximum in the *presence* of a caregiver, suggesting that the main purpose of toddler crying is to promote caregiver investment. Evidently, weeping provides an extraordinary tool through which an enterprising child can loosen the adult grip on resources through a biologically prepared disposition to acts of compassion.

The use of crying by infants to solicit resources is consistent with a further feature of infant crying—namely, the tendency for crying to be contagious. The crying of a single infant is apt to induce crying among nearby infants (Geangu, Benga, Stahl, & Striano, 2010; Hatfield, Cacioppo, & Rapson, 1994). This crying “contagion” might simply be a response to the unpleasantness of the sound of others crying. However, another possibility is that crying might represent a form of sibling competition. If caregiver attention is given disproportionately to the infant that cries, then the silent infant is apt to receive less.

The costs incurred by infants and children from weeping can be so low, that a real danger is the possibility of caregiver neglect, abuse, and even infanticide (Frodi & Lamb, 1980; Frodi, 1985). Such scenarios are consistent with classic parent-offspring conflict described by Trivers (1974).

As noted, reproducing adults pay a considerable cost for crying through the loss of social status. This chapter has suggested that the reason why infants and children incur so little cost for weeping is that the weeping behavior is crucial for adult social interaction; consequently, weeping simply must appear at some point in human development, and that moment of appearance necessarily occurs when social costs are low. Colic would be a non-adaptive artifact of this evolutionary scenario.

Cultural Coda

If weeping is an ethological signal, then it must be species-wide, and therefore truly cross-cultural. The notion that weeping has a biologically prepared meaning might be expected to raise concerns among cultural anthropologists. Surprisingly, some anthropologists agree that weeping is an innate behavior. American anthropologist Jules Henry has commented on the “striking resemblance” of grief-related behaviors across cultures (Henry, 1941/1964, pp. 66; quoted in Rosenblatt et al., 1976, p.18). Similarly, Greg Urban has remarked on the common humanity of grief expressions, referring to them as “natural” and “transparently understandable, not in need of detailed ethnographic description.” (Urban, 1991, p. 151).

Nevertheless, weeping expressions exhibit a number of differences within and between cultures, and these differences raise challenges for any theory claiming that

weeping has a biologically prepared social meaning. For example, in many cultures people experience tears of joy—such as those produced by beauty pageant winners. As noted earlier, in several cultures, weeping is sometimes accompanied by various forms of self-injury, including tissue damage that appears to contradict the claim the grief behaviors enhance inclusive fitness. There also exist ritualized forms of weeping, such as the “fake crying” used by the Tupinamba Indians to greet strangers—the so-called *Welcome of Tears*. These and other cultural behaviors raise challenges for any theory that purports any innate foundation in weeping behaviors.

In the space available here, we might examine a single cultural expression—the crying of beauty pageant winners. If weeping is regarded as a response to loss or tragedy, then the tearful pageant winner appears to make no sense. Having triumphed over the competition, the winner is certainly not experiencing feelings of loss or disappointment. However, ethology tells us that the purpose of a signal is not to communicate the feeling state of the individual: the purpose of a signal is to change the behavior of observers to the benefit of the signaler. Once again, if weeping is a signal, we need to attend, less to the feelings of the weeping person, and more to the effect the signal has on observers.

If weeping is a signal of surrender, then the weeping beauty pageant winner is communicating submission. Unlike the winner of a sports competition who thrusts her arms into the air in joyful celebration (and possible gloating), the weeping pageant winner is exhibiting a remarkable display of humility. When a culture expects a winner to be magnanimous, gracious, and grateful, there is arguably no more powerful expression than to voluntarily mark-oneself-down in the social hierarchy.

By way of summary, the argument here is that the tears shed by a beauty pageant winner are not tears of joy. The feeling of joy is surely real, but the tears are not part of the joy. Tears may flow in response to intense stress, especially when executive control is relaxed or abandoned. However, the main effect would be how observers interpret the tears—as communicating humility and gratitude (as opposed to entitlement), which, for many observers, would make the pageant winner more appealing rather than less appealing.

This single example is offered merely as an illustration of how the analysis of cultural expressions related to weeping might be profitably approached from an ethological perspective.

Phenomenology

Evolutionary arguments such as those offered here have a long history of poor reception by the general public. It is not simply that Machiavellian-like motives to maximize inclusive fitness are offensive when viewed from common moral standards. The accounts themselves simply do not accord with subjective experience when we observe weeping or when we ourselves weep.

Humans exist in a physical world of competing molecular patterns whose dynamics shape the deep motivations underlying behavior. However, our subjective experience has us living in a world of people and objects, social networks, and ineffable feelings. Love

may simply be Nature's way of encouraging pair-bonding and procreation, but the feeling of love is no less profound an experience despite its prosaic bio-chemical origin.

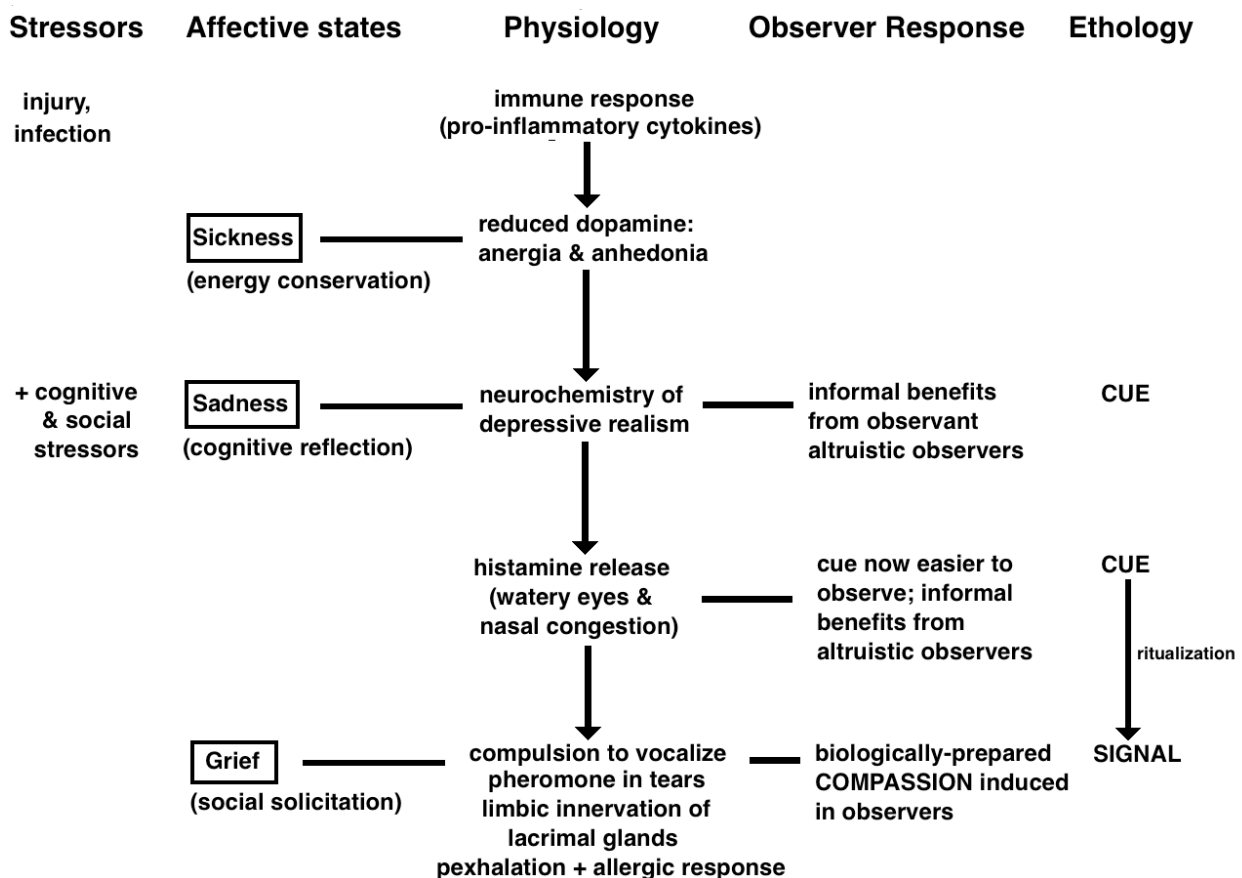
When we weep, our subjective experience is not one of manipulating others to help. Instead our experience is one of expressing true tragedy—accompanied by feelings of profound capitulation to an unhappy human condition. Similarly, when we observe others weeping, our phenomenological experience is not one of gleefully helping others—confident that they are incurring a great debt to us that may be repaid later. Instead our experience is one of expressing true compassion and empathy, often accompanied by a powerful feeling of social connection.

In our phenomenological world, sadness is about loss and regret; weeping is about vulnerability and compassion. In our shared world of phenomenological experience, sadness and grief are feelings of ultimate depth that touch and inspire us. Indeed, these feelings find expression in some of the most exquisite and affecting moments in poetry, drama, literature, and music (e.g., Kaufmann, 1992; Larson, 2010; Lewis, 1961; Young, 2010). When compared to the profundity of our subjective feelings, the presumed evolutionary origins will necessarily appear mundane and trivial.

Reprise

With this background, it is possible to offer a more systematic statement of the evolutionary theory proposed here for sadness and grief. Figure 5.1 provides a schematic summary of the proposed evolutionary history of stress-related responses. The following numbered summary is intended to clarify the logic of the theory, with the potential to better expose weakness. The summary may also provide a guide for identifying components of the theory amenable to empirical testing.

- 1. Three responses to stress.** Animals experience many kinds of stress. These include physical injury, pathogens, hunger, cognitive challenges, and social threats—such as social exclusion. When faced with difficulty, an individual can draw on at least three broad types of resources: *physiological resources* that fight infection, repair injury, and conserve energy, *cognitive resources* that encourage realistic reflection and strategizing, and *social resources* where conspecifics are induced to terminate aggression and/or offer assistance.



2. **Immune responsiveness.** Physical injury and illness provoke ancient immune responses. These responses are metabolically expensive, accounting for nearly 20% of metabolic energy consumption when fully activated.
3. **Anergia.** When injured or ill, energetic motor behaviors reduce fitness by competing with the immune system for metabolic resources. Fitness is enhanced if voluntary energetic activities are suppressed. Energy conservation can be achieved through the proximal feelings of fatigue.
4. **Anhedonia.** Many behaviors are motivated by hedonic rewards. A second effective strategy for reducing energy-depleting motor movement is to render normally enjoyable activities less enjoyable or appealing. This is achieved through the proximal feelings of anhedonia or apathy.
5. **Dopamine and lethargy.** The principal neurochemical concomitant of decreased motivation and motor activity (collectively “lethargy”) is reduced dopamine.
6. **Pro-inflammatory cytokines.** In order to facilitate tissue repair and defense against pathogens, the immune system releases pro-inflammatory cytokines. These cytokines are implicated in anergia, anhedonia, dopamine suppression, and

depressive feelings. In addition, pro-inflammatory cytokines are implicated in social pain, such as arising from social exclusion.

7. **Reflection.** When experiencing stress, appropriate mental activities may include cognitive reflection, where alternative adaptive strategies are formed and assessed.
8. **Depressive realism.** In difficult conditions, normally “optimistic” thinking has less utility than “realistic” thinking. Accurate memory recall, unbiased judgment, and realistic assessment are favored.
9. **Sadness versus grief.** Sadness and grief are different yet complementary states. Sadness is a quiescent state whose purpose is to promote cognitive reflection and strategizing. Grief is a socially directed signal intended to terminate aggression and/or solicit the assistance of others.
10. **Mourning cycle.** For minor stresses, the cost of weeping is likely to outweigh the benefits, and so minor stresses are apt to lead to sadness without grief. For major stresses, the benefits of weeping are more likely to outweigh the costs, and so sadness is apt to be supplemented by bouts of grief, where inward-directed sadness alternates with outward-directed grief, producing a “mourning cycle.”
11. **From inflammation to lethargy to sadness to grief.** Since injury and illness are ubiquitous threats, immune responses (“inflammation”) must have appeared very early in animal evolution. The beneficial consequences of anergia and anhedonia (“lethargy”) are likely to have arisen next. The compulsion for cognitive reflection (“sadness”) would have appeared subsequently. Finally, a grief signal (“weeping”) appears to be a ritualization of histamine release, unique to the hominini line.
12. **Histamines.** One class of pro-inflammatory cytokines includes histamines. Histamine release leads to allergic symptoms, including watery eyes, nasal congestion, and pharyngeal constriction. Note that histamines also disrupt sleep, reduce libido, and interfere with attention and vigilance—classic symptoms in depression (Cará et al., 1995; Falus, Grosman, & Darvas, 2004).
13. **Manifest allergies.** Of the various immunological responses, allergic symptoms are among the most easily perceived by observers. Nasal congestion and moist eyes are symptoms of immunological stress.
14. **Allergy as cue.** In the ancient past, watery eyes and nasal congestion acted as ethological cues—indexing a robust immune response to microbial stress. However, as these immune responses broadened to deal with other stressors (notably social stress), these symptoms also became observable cues indicating cognitive or social stress. Observant conspecifics learned to associate these symptoms with an individual experiencing stress. However, this cue was easily confused with a true allergic response. That is, an observer could not confidently distinguish those symptoms arising from microbial stress from those symptoms arising from cognitive or social stress.
15. **Ritualization.** Signals evolve from cues through the process of ritualization. Allergy-like cues would occasionally provoke altruistic assistance from observers. In light of the benefits for the stressed individual, the allergic symptoms arising from histamine release underwent selection pressure, transforming a *stress-induced-allergy-cue* into a *stress-induced-weeping-signal*.

16. **Conspicuousness.** Since signals are intended to be communicative, signals evolve toward conspicuousness and redundancy.
17. **Psychic tears.** The most conspicuous visual feature of weeping is the proliferation of tears. Although the neuroanatomy remains obscure, independent limbic activation of lacrimal sacs implies an anatomical connection, which in turn suggests a genetic (therefore innate) basis for psychic tears.
18. **Compulsion to vocalize.** An important aspect of conspicuousness is making a signal multimodal. Apart from sniffing and nose-blowing, the allergy exhibits no distinctive sonic element. The *compulsion to vocalize* (which is not part of a histamine-engendered allergic response) enhances the communicative effect of tears, contributing to the transformation from an ethological cue into an ethological signal.
19. **Ingressive phonation.** Especially compelling evidence in support of the compulsion to vocalize is found in ingressive phonation, where the vocal folds remain activated even while inhaling. This leads to distinctive gasping sounds.
20. **Breaking voice; pharyngealization.** As part of the allergic response, the constriction of the pharynx originally functioned to impede the entry of allergens. With the development of weeping, pharyngeal constriction affords two characteristic acoustical features: breaking or cracking voice (due to instability between modal and falsetto phonation) and a pharyngealized acoustic resonance or formant. In the evolution from a *stress-induced-allergy-cue* to a *stress-induced-weeping-signal*, pharyngeal constriction qualifies as an exaptation.
21. **Pant-laughter.** The punctuated vocalized exhaling responsible for the distinctive ‘ah-ah-ah-ah’ sound of weeping appears to be phylogenetically related to primate pant-laughter.
22. **Mutualism.** Signals evolve only if they serve both the signaling animal and the observing animal. The advantage of weeping for the signaling individual is the termination of attack and the effective soliciting of social assistance. The advantage for the observing individual is an enhanced social status, improved reputation, possible kin-selection, and/or future opportunities to benefit from reciprocal altruism.
23. **Compassion.** For the observing individual, the proximal motivation to terminate aggression and to engage in resource sharing is a feeling of compassion, commonly followed by feelings of pride or virtue.
24. **Pheromonal tears.** Psychic tears appear to contain a pheromone that encourages compassionate behaviors. This discovery holds three important implications. First, the research indicates that weeping activates at least three sensory modalities—visual, auditory, olfactory—consistent with the multimodal conspicuousness of ethological signals. Second, since exposure to tears changes the behavior of the observer to the benefit of the signaler, this effect is consistent with the claim that weeping is an ethological signal. Third, the apparent presence of a pheromone provides further indirect evidence of genetic factors underlying weeping behaviors, with the further implication that weeping is an evolved behavior.

25. **Biologically prepared.** Taken altogether, the anatomical, physiological, phenomenological, behavioral, and social evidence is consistent with weeping exhibiting the stereotypic tendencies commonly observed in signaling among non-human animals. That is, there are biologically prepared tendencies to weep under certain circumstances, and biologically prepared tendencies for observers to respond in certain ways toward weeping individuals. There is a notable degree of automaticity to these behaviors.
26. **Executive control.** Compared with other animals, humans exhibit greater executive control. Among humans, otherwise compelling behaviors (such as weeping) are susceptible to suppression, modification, or masking.
27. **Frequency of crying.** The frequency of crying is proportional to the magnitude of the stressor and inversely proportional to a person's social status and reproductive fitness. Crying is least common during reproductive years, less common among reproductive males, and more common among those with lower social status.
28. **Responsiveness to weeping.** The tendency to respond to weeping by terminating attack and/or offering assistance is mediated by several factors, including the degree of relatedness between observer and crier, the likelihood of future sustained interaction, the perceived honesty of the signal, the reputation and history of past interactions, the presence of an audience, and the marginal cost to the observer of offering assistance.
29. **Infant weeping.** Weeping incurs much lower costs for infants and children than for adults. As a result, there are few limits to the amount of infant weeping.
30. **An autoimmune disorder.** Depression appears to be another type of autoimmune disorder.

References

- Ahn, T. K., Janssen, M. A., & Ostrom, E. (2004). Signals, symbols, and human cooperation. In R. W. Sussman & A. R. Chapman (Eds.), *The origins and nature of sociality* (pp. 122–140). Hawthorne, New York, NY: Aldine de Gruyter.
- Alicke, M. D., & Govorun, O. (2005). The better-than-average effect. In M. D. Alicke, & J. I. Krueger (Eds.), *Studies in self and identity. The self in social judgment* (pp. 85–108). New York, NY: Psychology Press.
- Allen, N. B., & Badcock, P. B. T. (2003). The social risk hypothesis of depressed mood: Evolutionary, psychosocial, and neurobiological perspectives. *Psychological Bulletin*, 129, 887–913.
- Alloy, L. B., & Abramson, L. Y. (1979). Judgment of contingency in depressed and nondepressed students: Sadder but wiser? *Journal of Experimental Psychology: General*, 108, 441–485.
- Andreoni, J., & Miller, J. H. (1993). Rational cooperation in the finitely repeated prisoner's dilemma: Experimental evidence. *Economic Journal*, 103, 57–85.
- Andrews, P. W., & Thomson, J. A., Jr. (2009). The bright side of being blue: Depression as an adaptation for analyzing complex problems. *Psychological Review*, 116, 620–654.
- Archer, J. (1999). *The nature of grief: The evolution and psychology of reaction to loss*. London, England: Routledge.
- Axelrod, R., & Hamilton, W. D. (1981). The evolution of cooperation. *Science*, 211, 1390–1396.

- Badcock, P. B. T., & Allen, N. B. (2003). Adaptive social reasoning in depressed mood and depressive vulnerability. *Cognition and Emotion, 17*, 647–670.
- Banse, R., & Scherer, K. R. (1996). Acoustic profiles in vocal emotion expression. *Journal of Personality and Social Psychology, 70*, 614–636.
- Bard, K. A. (2000). Crying in infant primates: Insights into the development of crying in chimpanzees. In R. G. Barr, B. Hopkins, & J. A. Green (Eds.), *Crying as a sign, a symptom, and a signal: Clinical, emotional and developmental aspects of infant and toddler crying*. Clinics in Developmental Medicine No. 152 (pp. 157–175). New York, NY: Cambridge University Press.
- Becker, H. (1933). The sorrow of bereavement. *Journal of Abnormal and Social Psychology, 27*, 391–410.
- Bless, H., & Fiedler, K. (2006). Mood and the regulation of information processing and behavior. In J. P. Forgas (Ed.), *Affect in social thinking and behavior* (pp. 65–84). New York, NY: Psychology Press.
- Bowlby, J. (1961). Process of mourning. *International Journal of Psycho-Analysis, 42*, 317–340.
- Bowlby, J. (1973). *Attachment and loss; Vol. 2: Separation*. London, England: Hogarth.
- Bradbury, J. W., & Vehrenkamp, S. L. (1998). *Principles of animal communication*. Sunderland, MA: Sinauer.
- Brown, J. D., & Marshall, M. A. (2000). Great expectations: Optimism and pessimism in achievement settings. In D. C. Change (Ed.), *Optimism and pessimism: Implications for theory, research, and practice*. (pp. 239–256). Washington, DC: American Psychological Association.
- Capuron, L., Gummnick, J. F., Musselman, D. L., Lawson, D. H., Reemsnyder, A., Nemeroff, C. B., & Miller, A. H. (2002). Neurobehavioral effects of interferon-alpha in cancer patients: Phenomenology and paroxetine responsiveness of symptom dimensions. *Neuropsychopharmacology, 26*, 643–652.
- Cará, A. M., Lopes-Martins, R. A., Antunes, E., Nahoum, C. R., & De Nucci, G. (1995). The role of histamine in human penile erection. *British Journal of Urology, 75*, 220–224.
- Clore, G. L., & Huntsinger, J. R. (2007). How emotions inform judgment and regulate thought. *Trends in Cognitive Science, 11*, 393–399.
- Cornelius, R. R. (1997). Toward a new understanding of weeping and catharsis? In A. J. J. M. Vingerhoets, F. J. Van Bussel, & A. J. W. Boelhouwer (Eds.), *The (non)expression of emotions in health and disease*. (pp. 303–321). Tilburg, Netherlands: Tilburg University Press.
- Dallaspezia, S., & Benedetti, F. (2011). Chronobiological therapy for mood disorders. *Expert Review of Neurotherapeutics, 11*, 961–970.
- Darwin, C. R. (1872). *The expression of emotions in man and animals*. London, England: John Murray.
- Delp, M. J., & Sackeim, H. A. (1987). Effects of mood on lacrimal flow: Sex differences and asymmetry. *Psychophysiology, 24*, 550–556.
- Ebersole, G. L. (2000). The function of ritual weeping revisited: Affective expression and moral discourse. *History of Religions, 39*, 211–246.
- Eisenberger, N. I., & Lieberman, M. D. (2004). Why rejection hurts: A common neural alarm system for physical and social pain. *Trends in Cognitive Science, 8*, 294–300.
- Ekman, P. (1982). *Emotion in the human face*. New York, NY: Cambridge University Press.
- Ekman, P. (2003). *Emotions revealed: Understanding faces and feelings*. New York, NY: Times Books.
- Ellis, L. (1995). Dominance and reproductive success among nonhuman animals: A cross-

- species comparison. *Ethology and Sociobiology*, *16*, 257–333.
- Engel, G. L. (1962). Anxiety and depression-withdrawal. The primary affects of unpleasure. *International Journal of Psychoanalysis*, *43*, 89–97.
- Erickson, D., Yoshida, K., Menezes, C., Fujino, A., Mochida, T., & Shibuya, Y. (2006). Exploratory study of some acoustic and articulatory characteristics of sad speech. *Phonetica*, *61*, 1–25.
- Everson, C. A. (1993). Sustained sleep deprivation impairs host defense. *American Journal of Physiology*, *265*, R1148–R1154.
- Falus, A., Grosman, N., & Darvas, Z. (2004). *Histamine: Biology and medical aspects*. Budapest, Hungary: SpringMed.
- Farberow, N. L., & Shneidman, E. S. (1961). *The cry for help*. New York, NY: Blakiston Division, McGraw-Hill.
- Felger, J. C., & Lotrich, F. E. (2013). Inflammatory cytokines in depression: Neurobiological mechanisms and therapeutic implications. *Neuroscience*, *246*, 199–229.
- Fenigstein, A., Scheier, M. F., & Buss, A. H. (1975). Public and private self-consciousness: Assessment and theory. *Journal of Consulting and Clinical Psychology*, *43*, 522–527.
- Forgas, J. P. (1995). Mood and judgment: The Affect Infusion Model (AIM). *Psychological Bulletin*, *116*, 39–66.
- Forgas, J. P. (2002). Feeling and doing: Affective influences on interpersonal behavior. *Psychological Inquiry*, *13*, 1–28.
- Forgas, J. P., & East, R. (2008). How real is that smile? Mood effects on accepting or rejecting the veracity of emotional facial expressions. *Journal of Nonverbal Behavior*, *32*, 157–170.
- Forgas, J. P., Goldenberg, L., & Unkelbach, C. (2009). Can bad weather improve your memory? A field study of mood effects on memory in a real-life setting. *Journal of Experimental Social Psychology*, *54*, 254–257.
- Freed, P. (2009). Is sadness an evolutionarily conserved brain mechanism to dampen reward seeking? Depression may be a “sadness disorder.” *Neuropsychoanalysis*, *11*, 61–66.
- Freud, S. (1917). Trauer und Melancholie. *Internationale Zeitschrift für Psychoanalyse*, *4*, 288–301.
- Frey, W. H. (1985) *Crying: The mystery of tears*. Minneapolis, MN: Winston Press.
- Frey, W. H., Ahern, C., Gunderson, B. D., & Tuason, V. B. (1986). Biochemical behavioral and genetic aspects of psychogenic lacrimation: The unknown function of emotional tears. In E. J. Holly (Ed.), *The preocular tear film*. Lubbock, TX: Dry Eye Institute.
- Frick, R. W. (1985). Communicating emotion: The role of prosodic features. *Psychological Bulletin*, *97*, 412–429.
- Frodi, A. (1985). When empathy fails: Aversive infant crying and child abuse. In B. Lester & C. F. Boukydis (Eds.), *Infant crying*. (pp. 263–277). New York, NY: Plenum Press.
- Frodi, A., & Lamb, M. E. (1980). Child abusers’ responses to infant smiles and cries. *Child Development*, *51*, 238–241.
- Geangu, E., Benga, O., Stahl, D., & Striano, T. (2010). Contagious crying beyond the first days of life. *Infant Behavior and Development*, *33*, 279–288.
- Gelstein, S., Yeshurun, Y., Rozenkrantz, L., Shushan, S., Frumin, I., Roth, Y., & Sobel, N. (2011). Human tears contain a chemosignal. *Science*, *331*, 226–230.
- Gertsman, E. (2011). *Crying in the Middle Ages: Tears of history*. London, England: Routledge.
- Goldenberg, L., & Forgas, J. P. (2012). *Can happiness make us lazy? Hedonic discounting can make us reduce perseverance and the motivation to perform*. Sydney, Australia: University of New South Wales.
- Gorer, G. (1965). *Death, grief, and mourning*. New York, NY: Doubleday.

- Gortner, E. M., Rude, S. S., & Pennebaker, J. W. (2006). Benefits of expressive writing in lowering rumination and depressive symptoms. *Behavior Therapy, 37*, 292–303.
- Gould, S. J., & Vrba, E. S. (1982). Exaptation – a missing term in the science of form. *Paleobiology, 8*, 4–15.
- Graf, M. C., Gaudiano, B. A., & Geller, P. A. (2008). Written emotional disclosure: A controlled study of the benefits of expressive writing homework in outpatient psychotherapy. *Psychotherapy Research, 18*, 389–399.
- Hagen, E. H. (2011). Evolutionary theories of depression: A critical review. *Canadian Journal of Psychiatry, 56*, 716–726.
- Hasson, O. (2009). Emotional tears as biological signals. *Evolutionary Psychology, 7*, 363–370.
- Hatfield, E., Cacioppo, J. T., & Rapson, R. L. (1994). *Emotional contagion*. New York, NY: Cambridge University Press.
- Henderson, S. (1974). Care-eliciting behavior in man. *Journal of Nervous and Mental Disease, 159*, 172–181.
- Henry, J. (1964). *Jungle people: A Kaingáng tribe of the highlands of Brazil*. New York, NY: Vintage Books. (Original work published 1941)
- Hinde, R. A., & Spencer-Booth, Y. (1971). Effects of brief separation from mother on rhesus monkeys. *Science, 173*, 111–118.
- Hockey, J. L., Katz, J., & Small, N. (2001). *Grief, mourning, and death ritual*. Buckingham, England: Open University Press.
- Hofer, M. A. (1984). Relationships as regulators: A psychobiologic perspective on bereavement. *Psychosomatic Medicine, 46*, 183–197.
- Hollien, H. (1960). Some laryngeal correlates of vocal pitch. *Journal of Speech and Hearing Research, 3*, 52–58.
- Hopcroft, R. L. (2006). Sex, status, and reproductive success in the contemporary United States. *Evolution and Human Behavior, 27*, 104–120.
- Horwitz, A., & Wakefield, J. (2007). *The loss of sadness: How psychiatry transformed normal sadness into depressive disorder*. Oxford, England: Oxford University Press.
- Huron, D., & Shanahan, D. (2013). Eyebrow movements and vocal pitch height: Evidence consistent with an ethological signal. *Journal of the Acoustical Society of America, 133*, 2947–2952.
- Immelmann, K., & Beer, C. (1989). *A dictionary of ethology*. Cambridge, MA: Harvard University Press.
- Johnstone, R. A. (1997). The evolution of animal signals. In J. R. Krebs & N. B. Davies (Eds.), *Behavioural ecology* (pp. 155–178). Oxford, England: Oxford University Press.
- Joireman, J., Parrott, L., & Hammersla, J. (2002). Empathy and the self-absorption paradox: Support for the distinction between self-rumination and self-reflection. *Self and Identity, 1*, 53–65.
- Kaufmann, W. (1992) *Tragedy and philosophy*. Princeton, NJ: Princeton University Press.
- Keedwell, P. (2008). *How sadness survived: The evolutionary basis of depression*. Oxford, England: Radcliffe.
- Kenis, G., & Maes, M. (2002). Effects of antidepressants on the production of cytokines. *International Journal of Neuropsychopharmacology, 5*, 401–412.
- Kottler, J. A. (1996). *The language of tears*. San Francisco, CA: Jossey-Bass.
- Kottler, J. A., & Montgomery, M. J. (2001). Theories of crying. In A. J. J. M. Vingerhoets, & R. Cornelius (Eds.), *Adult crying: A biopsychosocial approach* (pp. 1–7). East Sussex, England: Brunner-Routledge.
- Kraemer, D. L., & Hastrup, J. L. (1988). Crying in adults: Self-control and autonomic correlates.

- Journal of Social and Clinical Psychology*, 6, 53–68.
- Kraepelin, E. (1921). *Psychiatrie. Ein Lehrbuch für Studierende und Ärzte [Manic-depressive insanity and paranoia]*. In G. M. Robertson (Ed.), R. M. Barclay (Trans.), Text-book of psychiatry vols. iii. and iv. (8th ed.). Edinburgh, Scotland: Livingstone. (Original work published 1899)
- Landreth, C. (1941). Factors associated with crying in young children in the nursery school and the home. *Child Development*, 12, 81–97.
- Lane, C. J. (2006). *Evolution of gender differences in adult crying*. (Doctoral dissertation). University of Texas at Arlington, Arlington, TX.
- Larson, T. (2010). *The saddest music ever written: The story of Samuel Barber's Adagio for Strings*. New York, NY: Pegasus Books.
- Lench, H. C., Bench, S. W., Darbor, K. E., & Moore, M. (2015). A functionalist manifesto: Goal-related emotions from an evolutionary perspective. *Emotion Review*, 7, 90–98.
- Lewis, A. J. (1934). Melancholia: A clinical survey of depressive states. *British Journal of Psychiatry*, 80, 277–378.
- Lewis, C. S. (1961) *A grief observed*. New York, NY: Seabury Press.
- Lorenz, K. (1937). Über die Bildung des Instinkt-begriffes. *Naturwissenschaften*, 25, 289–300, 307–318, 324–331.
- Lutz, T. (2001). *Crying: A natural and cultural history of tears*. New York, NY: Norton.
- Maguire, H. (1977). The depiction of sorrow in Middle Byzantine art. *Dumbarton Oaks Papers*, 31, 123–74.
- Marsella, A. J., Sartorius, N., Jablensky, A., & Fenton, F. R. (1985). Cross-cultural studies of depressive disorders: An overview. In A. Kleinman & B. Good (Eds.), *Culture and depression: Studies in the anthropology and cross-cultural psychiatry of affect and disorder*. (pp. 299–324). Berkeley, CA: University of California Press.
- Maynard Smith, J. M., & Harper, D. (2003). *Animal signals*. Oxford, England: Oxford University Press.
- Miller, A. H., Haroon, E., Raison, C. L., & Felger, J. C. (2013). Cytokine targets in the brain: Impact on neurotransmitters and neurocircuits. *Depression and Anxiety*, 30, 297–306.
- Miller, B. L., & Cummings, J. L. (2007). *The human frontal lobes: Functions and disorders*. New York, NY: Guilford Press.
- Moore, M. T., & Fresco, D. (2012). Depressive realism: A meta-analytic review. *Clinical Psychology Review*, 32, 496–509.
- Montagu, A. (1960). Natural selection and the origin and evolution of weeping in man. *Journal of the American Medical Association*, 174, 392–397.
- Murray, I. R., & Arnott, J. L. (1993). Toward the simulation of emotion in synthetic speech: A review of the literature on human vocal emotion. *Journal of the Acoustical Society of America*, 93, 1097–1108.
- Murphy, H. B. M., Wittkower, E., & Chance, N. (1964). Cross-cultural inquiry into the symptomatology of depression. *Transcultural Psychiatric Research Review*, 1, 5–21.
- Murube, J. (2009). Hypotheses on the development of psychoemotional tearing. *The Ocular Surface*, 7, 171–175.
- Musselman, D. L., Lawson, D. H., Gumnick, J. F., Manatunga, A. K., Penna, S., Goodkin, R. S., ... Miller, A. H. (2001). Paroxetine for the prevention of depression induced by high-dose interferon alfa. *New England Journal of Medicine*, 344, 961–966.
- Nesse, R. M. (1991). What good is feeling bad? The evolutionary benefits of psychic pain. *The Sciences*, 31, 30–37.
- Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry*, 57, 14–20.

- Nesse, R. M., & Williams, G. C. (1994). *Why we get sick: The new science of Darwinian medicine*. New York, NY: Times Books.
- Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. Oxford, England: Oxford University Press.
- Panksepp, J., & Bernatzky, G. (2002). Emotional sounds and the brain: The neuro-affective foundations of musical appreciation. *Behavioural Processes*, *60*, 133–155.
- Partan, S., & Marler, P. (1999). Communication goes multimodal. *Science*, *283*, 1272–1273.
- Pflug, B., & Tölle, R. (1971). Disturbance of the 24-hour rhythm in endogenous depression and the treatment of endogenous depression by sleep deprivation. *International Pharmacopsychiatry*, *6*, 187–196.
- Provine, R. R., Cabrera, M. O., Brocato, N. W., & Krosnowski, K. A. (2011). When the whites of the eyes are red: A uniquely human cue. *Ethology*, *117*, 1–5.
- Raison, C. L., Capuron, L., & Miller, A. (2006). Cytokines sing the blues: Inflammation and the pathogenesis of depression. *Trends in Immunology*, *27*, 24–31.
- Raison C. L., & Miller A. H. (2013). The evolutionary significance of depression in Pathogen Host Defense (PATHOS-D). *Molecular Psychiatry*, *18*, 15–37.
- Raleigh, M. J., McGuire, M. T., Brammer, G. L., Pollack, D. B., & Yuwiler, A. (1991). Serotonergic mechanisms promote dominance acquisition in adult male vervet monkeys. *Brain Research*, *559*, 181–190.
- Roes, F. L. (1989). On the origin of crying and tears. *Human Ethology Newsletter*, *5*, 5–6.
- Rogers, N. L., Szuba, M. P., Staab, J. P., Evans, D. L., & Dinges, D. F. (2001). Neuroimmunologic aspects of sleep and sleep loss. *Seminars in Clinical Neuropsychiatry*, *6*, 295–307.
- Rosenblatt, P. C., Walsh, R. P., & Jackson, D. A. (1976). *Grief and mourning in cross-cultural perspective*. New Haven, CT: Human Relations Area Files.
- Ross, L., & Nisbett, R. E. (1991). *The person and the situation: Perspectives of social psychology*. New York, NY: McGraw-Hill.
- Saul, L. J., & Bernstein, C. (1941). The emotional settings of some attacks of urticaria. *Psychosomatic Medicine*, *3*, 349–369.
- Scheier, M. F., & Carver, C. S. (1985). The self-consciousness scale: A revised version for use with general populations. *Journal of Applied Social Psychology*, *15*, 687–699.
- Scherer, K. R., Johnstone, T., & Klasmeyer, G. (2003). Vocal expression of emotion. In R. J. Davidson, K. R. Scherer, & H. Goldsmith (Eds.), *Handbook of the affective sciences* (pp. 433–456). Oxford, England: Oxford University Press.
- Sharot, T. (2011). *The optimism bias: A tour of the irrationally positive brain*. New York: Pantheon Books.
- Siegel, A., & Sapru, H. N. (2006). *Essential neuroscience*. London, England: Lippincott Williams & Wilkins.
- Slavich, G. M., Way, B. M., Eisenberger, N. I., & Taylor, S. E. (2010). Neural sensitivity to social rejection is associated with inflammatory responses to social stress. *Proceedings of the National Academy of Sciences*, *107*, 14817–14822.
- Spencer-Booth, Y., & Hinde, R. A. (1971). Effects of brief separations from mothers during infancy on behavior of rhesus monkeys 6-24 months later. *Journal of Child Psychology and Psychiatry*, *12*, 157–172.
- Storbeck, J., & Clore, G. L. (2005). With sadness comes accuracy; with happiness, false memory: Mood and the false memory effect. *Psychological Science*, *16*, 785–791.
- Sundberg, J. (1987). *The science of the singing voice*. DeKalb, IL: Northern Illinois University Press.

- Švec, J. G., & Pešák, J. (1994). Vocal breaks from the modal to the falsetto register. *Folia Phoniatrica et Logopaedica*, *46*, 97–103.
- Tan, H. B., & Forgas, J. P. (2010). When happiness makes us selfish, but sadness makes us fair: Affective influences on interpersonal strategies in the dictator game. *Journal of Experimental Social Psychology*, *46*, 571–576.
- Tartter, V. C. (1980). Happy talk: Perceptual and acoustic effects of smiling on speech. *Perception & Psychophysics*, *27*, 24–27.
- Tinbergen, N. (1952). “Derived” activities; their causation, biological significance, origin, and emancipation during evolution. *Quarterly Review of Biology*, *27*, 1–32.
- Tinbergen, N. (1964). The evolution of signaling devices. In W. Etkin (Ed.), *Social behavior and organization among vertebrates* (pp. 206–230). Chicago, IL: University of Chicago Press.
- Tomkins, S. S. (1980). Affect as amplification: some modifications in theory. In R. Plutchik & H. Kellerman (Eds.), *Emotion: Theory, research and experience* (pp. 141–164). New York, NY: Academic Press.
- Trapnell, P. D., & Campbell, J. D. (1999). Private self-consciousness and the five-factor model of personality: Distinguishing rumination from reflection. *Journal of Personality and Social Psychology*, *76*, 284–304.
- Trimble, M. (2012). *Why humans like to cry: Tragedy, evolution, and the brain*. Oxford, England: Oxford University Press.
- Trivers, R. L. (1971). The evolution of reciprocal altruism. *Quarterly Review of Biology*, *46*, 35–57.
- Trivers, R. L. (1974). Parent-offspring conflict. *American Zoologist*, *14*, 249–264.
- Urban, G. (1991). *A discourse-centered approach to culture: Native South American myths and rituals*. Austin, TX: University of Texas Press.
- Viggiano, D., Ruocco, L. A., Arcieri, S., & Sadile, A. G. (2004). Involvement of norepinephrine in the control of activity and attentive processes in animal models of attention deficit hyperactivity disorder. *Neural Plasticity*, *11*, 133–149.
- Vingerhoets, A. J. J. M. (2013). *Why only humans weep: Unravelling the mysteries of tears*. Oxford, England: Oxford University Press.
- Vingerhoets, A. J. J. M., & Cornelius, R. R. (2001). *Adult crying: A biopsychosocial approach*. East Sussex, England: Brunner-Routledge.
- Vingerhoets, A. J. J. M., Cornelius, R. R., Van Heck, G. L., & Becht, M. C. (2000). Adult crying: A model and review of the literature. *Review of General Psychology*, *4*, 354–377.
- Weary, G., & Edwards, J. A. (1994). Social cognition and clinical psychology: Anxiety, depression, and the processing of social information. In R. S. Wyer Jr., & T. K. Srull (Eds.), *Handbook of social cognition* (pp. 289–338). Hillsdale, NJ: Erlbaum.
- Weinstein, N. D. (1987). Unrealistic optimism about susceptibility to health problems: Conclusions from a community-wide sample. *Journal of Behavioral Medicine*, *10*, 481–500.
- Wiley, R. H. (1983). The evolution of communication: information and manipulation. In T. R. Halliday, & P. J. B. Slater (Eds.), *Communication* (pp. 82–113). Oxford, England: Blackwell.
- Wilson, E. (2008). *Against happiness: In praise of melancholy*. New York, NY: Farrar, Straus and Giroux.
- Yost, J. H., & Weary, G. (1996). Depression and the correspondent inference bias: Evidence for more effortful cognitive processing. *Personality and Social Psychology Bulletin*, *22*, 192–200.
- Young, K. (Ed.). (2010). *The art of losing: Poems of grief and healing*. New York, NY: Bloomsbury.

Zahavi, A., & Zahavi, A. (1997). *The handicap principle: A missing piece of Darwin's puzzle*.
Oxford, England: Oxford University Press.

Zeifman, D. M. (2001). An ethological analysis of human infant crying: Answering Tinbergen's four questions. *Developmental Psychobiology*, 39, 265–285.