Psychiatry Research 175 (2010) 1–10

Contents lists available at ScienceDirect

Psychiatry Research

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

Review article

Is disgust associated with psychopathology? Emerging research in the anxiety disorders

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ARTICLE INFO

Article history:
Received 13 June 2008
Received in revised form 25 March 2009
Accepted 15 April 2009

Keywords:
Disgust
Neural mechanisms
Anxiety disorders
Contamination fear
Spider phobia
Blood-injection-injury phobia

ABSTRACT

Recent evidence indicates that the propensity towards experiencing disgust may contribute to the development and maintenance of some anxiety disorders. This article summarizes the empirical evidence with emphasis on illuminating potential mediators, moderators, and mechanisms of the disgust–anxiety disorder association that may inform the development of an integrative conceptual model. Early research using neuroimaging methods suggest that disgust processing is associated with activation of the insula. This research has the potential to facilitate progress in developing an empirically informed psychobiological theory on the causal role of disgust in the anxiety disorders.

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Contents

1. Introduction

Disgust is an emotion with response patterns that are universally recognized. Disgust appears to originate from the primitive sensation of distaste elicited by contaminated or bad-tasting foods. This view

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0165-1781/$ – see front matter © 2009 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.psychres.2009.04.007

suggests that disgust prevents ingestions of harmful substances, in turn protecting against diseases (Rozin and Fallon, 1987). Indeed, the characteristic facial expression of disgust involves muscles necessary to avoid consuming contaminants. However, disgust may have evolved to prevent contact with sources of contaminants in general. Although traditional models emphasize dysregulated fear processing in the development of anxiety disorders, a disease-avoidance account of disgust may also have implications for the development of anxiety disorders. Recent evidence points to neural specificity of disgust reactions in healthy individuals, particularly the ventroanterior and anterior insula (Calder et al., 2007; Mataix-Cols et al., 2008). Mataix-Cols et al. (2008) showed that exposure to disgust images also activated regions associated with emotion regulation, namely the dorsolateral and rostral prefrontal cortices. Findings such as these have begun to identify a neural basis for disgust that may account for its role in the development of anxiety disorders, leading to a noticeable shift in the emotional emphasis in anxiety disorders research.

Contemporary models conceptualize disgust as a multidimensional emotional state. Indeed, psychometric research has shown that the experience of disgust can be divided into three major categories: core, animal reminder, and contamination. Core disgust elicitors are characterized by a real or perceived threat of oral incorporation and a reactive sense of offensiveness. Rotting foods, bodily waste products, and small animals, particularly those associated with garbage and waste are subsumed within this category. Animal-reminder disgust elicitors consist of reminders of our own mortality and inherent animalistic nature. Attitudes and practices surrounding sex, injury to the body or violations of its outer envelope, and death are subsumed within this category. Contamination disgust is often elicited in interpersonal contexts such as contact with individuals who are unknown, ill, or tainted by disease, misfortune, or immorality. These components have been found to be dimensional in nature (Olatunji and Broman-Fulks, 2007) and relatively stable across cultures (Olatunji et al., 2009). Lastly, each dimension shows unique correlates with self-report, behavioral, psychophysiological assessments (Olatunji et al., 2008c). Individual differences in the tendency towards experiencing the emotion of disgust may also be viewed as a personality trait that may operate as a risk factor for anxiety conditions that are especially marked by aversion (Muris, 2006).

1.1. Disgust in specific phobia

It has generally been accepted that fear of spiders is biologically prepared as some have venom that is lethal to humans. However, Matchett and Davey (1991) suggest that a disease-avoidance model driven by disgust may better account for phobic avoidance of spiders. Consistent with this notion, several lines of research suggest that experiencing disgust is related to spider fear. First, self-report questionnaires of disgust (e.g., the Disgust Scale; Haidt et al., 1994) have consistently been found to correlate with self-report questionnaires of spider fear (Mulkens et al., 1996; de Jong and Merckelbach, 1998; Thorpe and Salkovskis, 1998). For example, Olatunji et al. (2007c) found that self-reported disgust predicted self-reported spider fears in a structural regression analysis even when controlling for trait anxiety ($n=352$; $\beta=0.63$). Second, individuals with elevated spider fears verbally report feelings of disgust when confronted with spiders (Tolin et al., 1997; Thorpe and Salkovskis, 1998; Sawchuk et al., 2002; Vernon and Berenbaum, 2002). For example, college students with elevated spider fear reported greater disgust during exposure to a realistic looking, but fake, spider compared with non-fearful college students (Olatunji and Deacon, 2008). Third, self-reported disgust is related to avoidance of spiders. For example, Woody et al. (2005) found that peak self-reported disgust during exposure to spiders was a better predictor of avoidance than was self-reported anxiety during the exposure. A more recent study also found that higher expectancies for disgust-relevant outcomes associated with exposure to a spider, compared to fear-relevant outcomes, better predicted avoidance of a spider on a behavioral task (Olatunji et al., 2008a). These data converge in suggesting that disgust is strongly related to spider phobia.

Another line of behavioral research suggesting a role of disgust in spider phobia is the 'contaminated cookie' paradigm. In this paradigm, participants observe a non-contaminated cookie come in contact with a spider. Participants are then asked to eat the cookie. Mulkens, de Jong and Merckelbach (1996) found that women with spider phobia were significantly less likely to eat a cookie after a spider had walked across it compared to women without spider phobia. Importantly, the two groups did not differ in their likelihood of drinking a contaminated cup of tea, suggesting that there were no differences between the groups in general sensitivity to dirtiness. This basic finding has since been replicated (de Jong and Muris, 2002; Woody et al., 2005) and suggests that spiders are perceived as a source of contamination, and thus related to disgust, among spider fearful individuals. Moreover, this effect mirrors the 'law of contagion' belief that characterizes disgust (Rozin and Fallon, 1987), which states that 'once in contact, always contaminated.'

Evidence for a role of disgust in spider phobia goes beyond self-report and behavioral measures. Spider fearful individuals consistently display disgust facial expressions during exposure to spiders (de Jong et al., 2002; Vernon and Berenbaum, 2002). de Jong et al. found that spider fearful individuals displayed greater activity of the levator labii muscle, which is activated during exposure to disgust stimuli (Vrana, 1993), towards spider-stimuli compared to non-fearful individuals. Vernon and Berenbaum did not measure muscle activity directly, but rather coded video tapes of participants' facial expressions during exposure to a spider. They found that spider fearful participants displayed more disgust and fear facial expressions relative to non fearful individuals. Accordingly, spiders elicit disgust in physiological domains as well as in self-report/appraisal domains.

Finally, studies of cognitive biases in spider phobia also suggest a critical role of disgust. Some studies have used the Implicit Association Tasks (IAT) to assess whether spider phobic individuals associate spiders with disgust-related stimuli or fear-related stimuli. Teachman et al. (2001) used an IAT with pictures of spiders as the target stimuli and disgust attributes (e.g., gross) and fear attributes (e.g., harm) as separate categories. Results revealed that spider fearful individuals associated spiders with both disgust and fear attributes to a greater degree than an anxious control group. Similarly, Huijding and de Jong (2007) employed an IAT with pictures of spiders as the target stimuli and disgust attributes (e.g., 'dirty') and fear attributes (e.g., 'attacking') as separate categories. Results revealed that individuals with spider phobia associated spiders with both fear and disgust attributes to a greater degree than did non-phobic individuals.

Other studies have used the expectancy bias paradigm to examine the role of fear and disgust in spider phobia. For example, van Overveld et al. (2006a,b) asked participants to rate whether they expected pictures of spiders, maggots, dogs, and rabbits to be followed by disgust outcomes (i.e., drinking a distasteful fluid), fear outcomes (i.e., an electric shock), or no outcome. The authors found that high spider fearful individuals expected both disgust and fear outcomes to follow spiders to a greater degree than low spider fearful individuals. Importantly, expectancies of disgust outcomes following spiders significantly predicted self-reported spider fear, but fear-expectancies did not. Using a similar methodology, de Jong and Peters (2007a) found that spider fearful individuals expected a disgust outcome to follow spider pictures, but did not expect a fear outcome to follow spider pictures. These studies suggest that spider fearful individuals associate spiders with disgust-related stimuli, which further support the important role of disgust in spider phobia.

Another large body of research also strongly implicates disgust in blood-injection injury (BII) phobia. First, self-report questionnaires of disgust positively correlate with questionnaires of BII phobia (de Jong and Merckelbach, 1998; Olatunji et al., 2006a, 2007c). For example,

Olatunji et al. (2006b) used structural equation modeling and found significant structural relations between disgust (particularly animal-reminder disgust) and BII fear independent of trait anxiety. Second, individuals with elevated BII phobia also respond with self-reported disgust when confronted with BII-relevant stimuli (Tolin et al., 1997; Sawchuk et al., 2002). Sawchuk et al. presented participants with surgery pictures and found that BII fearful individuals self-reported more fear and disgust relative to non-fearful individuals. Moreover, BII fearful individuals' self-reported disgust was greater than their self-reported fear. Third, behavioral avoidance of BII-related stimuli is positively related to self-reported disgust. Olatunji et al. (2008b) recently found that high BII fearful individuals displayed greater avoidance of a severed deer leg relative to a non-fearful group. Moreover, self-reported disgust, particularly for animal reminder disgust, predicted avoidance above and beyond the main effect of BII fear.

As with spider phobia, BII phobic individuals also display a disgust facial expression towards BII relevant stimuli. Lumley and Melamed (1992) videotaped BII phobic and non-phobic individuals' facial expressions while viewing a surgery film clip depicting an incision, a surgery film depicting tubes being placed in an abdomen, and neutral film clips. Results revealed that BII phobic individuals displayed more facial expressions of disgust (i.e., furrowed their eyebrows or raised their upper lip; raising of the upper lip indicates levator labii muscle activation) compared to non-phobic individuals, but only during the incision surgery film.

Further evidence for the role of disgust in BII phobia also comes from the unique heart rate response in BII phobia. The heart rate response to phobogenic stimuli in BII is characterized by a biphasic pattern (Page, 1994; Sarlo et al., 2002), in which there is a rapid acceleration of heart rate followed by a sharp decrease in heart rate. The latter half of the biphasic reaction seems responsible for the fainting response that uniquely characterizes BII phobia (Öst et al., 1984; Page, 1994; Sarlo et al., 2002). For example, Ost et al. presented BII phobics with surgery films and found an initial increase in heart rate, followed by a rapid decline in heart rate. Considering that disgust is also associated with heart rate deceleration (Levenson, 1992; Olatunji et al., 2008c; Stark et al., 2005), it seems likely that the emotional response in BII phobia and related fainting involves disgust to some extent. In support of this notion, Page (2003) found that for those that were distressed by blood, the diphasic response pattern (as assessed by systolic and diastolic blood pressure) was most evident among subjects high in disgust during exposure to threat-relevant stimuli.

Some research has failed to demonstrate the biphasic heart rate response in BII phobia (Gerlach et al., 2006; Ritz et al., 2005). However, the Ritz et al. study used strict criteria in defining the biphasic response (i.e., increase heart rate during viewing surgery videos relative to a generally unpleasant film and a subsequent decrease in heart rate relative to a neutral film). This strict criterion may have limited false positives but also maximized false negatives. The Gerlach et al. study failed to find a biphasic response in BII phobics during venopuncture; however, previous studies documenting the biphasic response used surgery films as the phobia-relevant stimulus. Accordingly, observation of the biphasic response may require more potent stimuli such as surgery exposure. Gerlach et al. also found that disgust sensitivity did not differ between BII phobics with a history of fainting and BII phobics without a history of fainting, which underscores the importance of differentiating between the different domain of disgust (e.g., animal reminder versus core; Olatunji et al., 2008b) when investigating the role of disgust in BII phobia.

In studies that have demonstrated the biphasic heart rate response in BII phobia, the extent to which disgust accounts for this pattern is also not entirely clear. For example, a recent study found no evidence to indicate that BII phobic individuals demonstrate an increase in parasympathetic cardiac activity during disgust exposure and/or decrease in mean arterial pressure (van Overveld et al., 2009). Furthermore, a decrease in heart rate may only be observed as a response to disease-related disgust-induction (Rohrmann and Hopp, 2008) suggesting that the assessment of disgust as a unitary construct may yield underestimates of associations with fainting in BII phobia. In a related vein, Vogele et al. (2003) found a biphasic response during viewing surgery films among a BII phobia sample with a history of fainting, but did not find it in a BII phobia sample without a history of fainting. Moderating variables, such as history of fainting, domain of disgust, and type of stimulus used, may explain the relations between the biphasic heart rate response, disgust, and BII phobia. Prior research has also shown that trait anxiety and disgust sensitivity interact to generate symptoms of faintness in certain contexts (Exeter-Kent and Page, 2006). Thus, individual differences in other personality traits may moderate the association between disgust and fainting symptoms in BII phobia.

Research investigating cognitive biases in BII phobia also implicates disgust. Sawchuk et al. (1999) found that phobic participants were more likely to complete blood- and general disgust-related word stems than were nonphobic participants, suggesting an implicit memory bias toward those stimulus categories. More recently, de Jong and Peters (2007b) presented high and low BII fearful individuals with a series of fear-relevant (blood-related) and fear-irrelevant (rabbit and flower) slides that were randomly paired with either a harm-related outcome (shock), a disgust-related outcome (drinking a disgusting fluid), or nothing. Before the experiment, participants expected blood-related slides to be followed by disgust- and harm-relevant outcomes. These expectancy biases were readily corrected during the experiment. However, such biases did not differ between high and low BII fearful participants. These studies suggest that disgust may play a role in BII phobia. However, the extent to which robust associations between disgust and BII phobia may be observed appears to be partially dependent on the modality of assessment.

1.2. Disgust and contamination-based obsessive–compulsive disorder

Researchers have now begun to extend the examination of disgust in the anxiety disorders beyond specific phobias. The complexity and enduring nature of contamination fear observed in obsessive–compulsive disorder (OCD) has led to renewed interest in identifying factors that contribute to its development. While fear is central to contemporary models of washing compulsions (Rachman, 2004), recent findings suggest the tendency towards experiencing disgust emotions may also contribute to contamination-based OCD. As with spider and BII phobia, self-report measures implicate a relation between disgust propensity and symptoms of contamination-based OCD. First, self-report questionnaires of the tendency towards experiencing disgust correlate with self-report measures of contamination fear (Muris et al., 2000; Mancini et al., 2001; Olatunji et al., 2005b, 2007c; Cisler et al., 2007, 2008). Further, it has been shown that disgust propensity is a unique predictor of contamination fear, but not other symptom dimensions of OCD, and that this relationship with contamination fear is unmediated by trait anxiety (Moretz and McKay, 2008) or depression (Olatunji et al., submitted for publication, study 3). Second, individuals with heightened contamination fear report feelings of disgust when confronted with contamination-related stimuli (e.g., bedpans; Deacon and Olatunji, 2007). Similarly, self-reported disgust propensity is positively related to behavioral avoidance of contamination-related stimuli (Deacon and Olatunji, 2007; Olatunji et al., 2007b). Finally, individuals with heightened contamination fears display greater behavioral avoidance of disgusting objects relative to non-fearful and high trait anxious individuals (Tsao and McKay, 2004).

The available research clearly suggests that heightened disgust is related to contamination fears commonly observed in OCD. Contamination is defined as, “an intense and persisting feeling of having been polluted or infected or endangered as a result of contact, direct or

indirect, with a person/place/object that is perceived to be soiled, impure, infectious or harmful. The feeling of contamination is accompanied by negative emotions among which fear, disgust, dirtiness, moral impurity and shame are prominent" (Rachman, 2004, p. 1229). This definition suggests that disgust and contamination are related, but distinct concepts on a similar continuum (Olatunji et al., 2007b). Disgust, as a basic emotion, elicits a reliable physiological response, facial expression, and withdrawal/avoidance pattern. Contamination, on the other hand, is viewed as the evaluative/interpretive process that may occur upon the experience of disgust or anticipated exposure to elicitors of disgust.

In contrast to research on spider and BII phobia, there have been very limited physiological investigations of the role of disgust in contamination fear. However, research investigating cognitive biases in contamination fear appears to compliment findings that primarily employ self-report measures in demonstrating an important role for disgust in contamination fear. Connolly et al. (2009) used a covariation paradigm and found that individuals with elevated contamination fear overestimated the pairings of contamination-related pictures (e.g., vomit, dog feces) with pictures of fear facial expressions and disgust facial expressions to a greater degree than with neutral facial expressions. However, high fearful individuals did not overestimate disgust outcomes to a greater degree than low fearful individuals.

More empirical evidence that disgust is associated with contamination fear comes from a study demonstrating the 'law of contagion' belief in contamination fearful individuals. Tolin et al. (2004) asked individuals with contamination-related OCD, other anxiety disorders, and non-anxious controls to identify a contaminated object. The experimenter then rubbed a new pencil on the object and asked participants how contaminated the pencil was, then rubbed another new pencil on the previous pencil and again asked how contaminated this new pencil was. This process was repeated for 12 pencils. Results revealed that the anxious and non-anxious controls evidenced nearly a 100% decrease in appraisals of contamination across the 12 pencils, but the contamination-related OCD group displayed only a 40% reduction. Thus, contamination fearful individuals display the disgust related belief that 'once in contact, always contaminated.'

A role for disgust in OCD does not appear to be entirely specific to the contamination subtype. Indeed, disgust also appears to be associated with OCD symptoms with religious focus. For example, Olatunji et al. (2005a) found that feelings of disgust towards stimuli with moral implications (i.e., death, unusual sexual practices) are significantly related with religious obsessions (i.e., fear that one has or will commit sin, intrusive blasphemous mental images, fear of punishment by God). Disgust has been directly linked to notions of purity which are incorporated into the moral codes of various religions (e.g., the Book of Leviticus in the Old Testament) where they function as protectors of the soul from moral pollution. In fact, there is evidence that disgust and moral violations are processed in overlapping brain regions (Moll et al., 2005). Consequently, a threat to one's moral purity may elicit disgust and obsessive processes among vulnerable individuals, including increased urges to wash (Zhong and Liljenquist, 2006).

1.3. Contextual variations of disgust in specific anxiety disorders

The questions remain as to what it is about disgust that may confer risk for the development of specific anxiety disorders. It may be the case that experiencing disgust directly influences the experience of fear and subsequent avoidance behavior or disgust may indicate the type of catastrophe that is anticipated ("the spider will get in my mouth"; "seeing blood means that I am just like an animal"). Disgust may also be experienced as a basic emotional response to [the anticipation of] real or imagined contaminants or it may be that the physiological component of disgust is what is crucial in some anxiety disorders (as is suggested for BII phobia). Addressing such concerns requires distinctions to be made between disgust as a basic emotion and disgust as a personality trait. Disgust in spider, BII, and contamination fear may be observed at the state emotional level during exposure to threat-relevant stimuli. However, the role of disgust in these focal fears is also reflected at the trait personality level, consisting of a predisposition towards experiencing disgust in general (in the absence of focal fears). In fact, research has shown that spider and BII phobics report higher repugnance across a range of stimuli unrelated to phobic concerns, such as rotting foods, bodily products, and revolting odours (Sawchuk et al., 2002).

This general proneness towards experiencing disgust in various contexts has been described as a specific genetically-based personality risk vulnerability factor that may contribute to the development of these phobic conditions (e.g., Muris, 2006), whereas excessive disgust emotional responding during exposure to threat-relevant stimuli may serve more of a maintenance function. Consistent with this notion, it has been shown that although spider phobics' stimulus-bound disgust reactions may decrease with treatment, their degree of disgust proneness, a personality trait, remains relatively unchanged even after successful exposure treatment, thereby implying that this trait may serve as a unique and independent role in the etiology and maintenance of spider phobia (de Jong et al., 1997). However, the personality trait of disgust proneness may also present with important variations that differentially predict specific anxiety disorder symptoms. For example, one may present with a heightened frequency/intensity of disgust experiences (*disgust propensity*; "When I experience disgust, it is an intense feeling") and/or one may present with overestimations of the potential negative consequences of experiencing disgust (*disgust sensitivity*; "When I notice that I feel nauseous, I worry about vomiting"). This latter view is akin to research showing that the perceived harmful consequence of experiencing anxiety (i.e., anxiety sensitivity) is a risk factor for panic disorder. There is preliminary data suggesting that such distinctions between disgust sensitivity and propensity may have some validity in differentiating anxiety disorder symptoms (Cisler et al., 2009b, 2009; van Overveld et al., 2006a,b; Olatunji et al., 2007a).

2. The neural bases of disgust in anxiety disorders

Functional neuroimaging techniques, including functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), have made it possible to examine neural regions associated with specific emotional processes. A large body of neuroimaging research now implicates the anterior insula as a critical neural substrate underlying the recognition, processing, and experience of disgust. Studies have found that disgust facial expressions activate the anterior insula (Phillips et al., 1997, 1998; Williams et al., 2005; Calder et al., 2007; Mataix-Cols et al., 2008) relative to neutral and fear facial expressions. For example, Phillips et al. (1997) revealed that both strong and mild expressions of disgust activated the anterior insular cortex but not the amygdala among non-clinical participants. Thus, the anterior insula is involved in recognizing disgust facial expressions. The insular cortex is functionally heterogeneous, thus it is important that research has demonstrated that the link between disgust and insula activity seems to be specific to the anterior region of the insular cortex. Indeed, a study using depth electrodes implanted into presurgery epilepsy patients recorded intracerebral potentials in response to pictures of facial expressions and found activation towards disgust stimuli specifically in the ventral anterior insula (Krolak-Salmon et al., 2003).

A correlation between anterior insula activation and facial expressions of disgust can only implicate the anterior insula in the *recognition* of disgust, but additional research also implicates the anterior insula in the *processing* and *experience* of disgust. Some studies demonstrate that the anterior insula is activated towards ecologically valid disgust stimuli (e.g., small animals, mutilation, spoiled food products, etc; Wright et al., 2004; Caseras et al., 2007; Calder et al., 2007). For example, Caseras et al. found anterior insula activation towards pictures of mutilated bodies, small animals, and body products compared with neutral pictures. Moreover, Mataix-Cols et al. (2008) found that individual differences in

disgust propensity predicted the magnitude of anterior insula responses towards disgusting pictures ($r = 0.41$). Finally, studies demonstrate that the anterior insula is activated during the subjective experience of disgust (Fitzgerald et al., 2004; Stark et al., 2007). For example, Fitzgerald et al. found that participants asked to recall and re-experience recent disgusting situations exhibited insula activation. These lines of research strongly suggest that the anterior insula is a critical neural substrate underlying disgust. The insula, which is part of the gustatory cortex, responds to unpleasant tastes. Thus, the association between disgust and the insula is consistent with the evolutionary function of disgust in avoidance of contaminated foods (Calder et al., 2001).

It is important to note that the insular cortex is heterogeneous in function (Augustine, 1996). Indeed, recent research has begun to elucidate the important role of the insular cortex in interoceptive awareness and the neural representation of internal bodily states (Critchley et al., 2004; Phan et al., 2004; Dalton et al., 2005; Wiens, 2005; Paulus and Stein, 2006). For example, Critchley et al. found that anterior insula activation while participants judged the timing of their heartbeats correlated with accuracy of heartbeat timing ($r = 0.62$). These data illuminate an important limitation of studies suggesting that the anterior insula is critically involved in disgust: activation of the anterior insula towards disgust stimuli may actually be due to the insula's role in interoceptive awareness. That is, disgust involves physiological changes, including a decrease in heart rate (Stark et al., 2005), that may be represented in the anterior insula. Thus, it is difficult to reliably discern whether activation of the insula towards disgust is due to interoceptive representations or the processing of disgust cues.

Tempered by this limitation, the following sections review studies examining neural responses towards disorder-relevant stimuli in spider phobia, BII phobia, and contamination-related OCD. If disgust is critically involved in the emotional response elicited from disorder-relevant stimuli, it follows that disorder-relevant stimuli will activate the anterior insula. Amygdala activation towards disorder-relevant stimuli suggests that fear is involved in the emotional response of these disorders. Neurobehavioral research (e.g., Walker and Davis, 1997) and theory (LeDoux, 2000; Rosen and Schulkin, 1998) typically suggest that the amygdala and functionally related structures (e.g., bed nucleus of the stria terminalis) are critical in understanding anxiety disorders. The current review can compliment and extend this body of work by examining whether disgust and the anterior insula are also involved in these disorders and perhaps even critical in understanding these disorders.

2.1. Spider phobia

Goossens et al. (2007) found greater amygdala and insula activation towards spider stimuli compared to a control group. These differences disappeared after exposure therapy. These results suggest that fear *and* disgust characterize spider phobia and also that fear and disgust may both decrease during therapy. Wendt et al. (2008) also found amygdala and insula activation towards spiders among spider fearful compared to nonfearful individuals. Schienle et al. (2007) found that only amygdala activation towards spider pictures was greater among spider fearful compared to nonfearful individuals, but amygdala and insula activation decreases following therapy, and that both correlated with decreases in self-reported somatic symptoms. Furthermore increased activation of the insula, as well as the dorsal anterior cingulate cortex (ACC), thalamus, and visual areas has been found among spider phobics compared to controls during anticipation of phobia-relevant versus anticipation of neutral stimulation (Straube et al., 2006).

Straube et al. (2006) found that spider phobics display amygdala and insula activation when attending to spider pictures, but only amygdala activation when the spider pictures are present but not part of the primary experimental task. Amygdala activation towards unattended spiders and insula activation towards only attended spiders demonstrates the relative ease with which spiders elicit fear compared to disgust. Thus, spiders may automatically elicit fear, whereas disgust may arise during more conscious, strategic processing. Similarly, Larson et al. (2006) found that immediate amygdala activation in response to spider stimuli differentiated between spider phobics and control participants; insula activation was not measured in this study.

2.2. Blood-injection-injury phobia

Hermann et al. (2007) found that BII phobic individuals display diminished medial prefrontal cortex activity in response to disorder relevant pictures compared to the control group. They did not find evidence of insula activation towards disorder-relevant pictures, but found marginally significantly greater activation of the amygdala towards disorder-relevant compared to neutral pictures. The medial prefrontal cortex has been implicated in the down-regulation of emotional processing (Phelps and LeDoux, 2005; Bishop, 2007), thus suggesting that BII phobia may be characterized by a generalized diminished ability to regulate emotions. As such, diminished medial prefrontal cortex activity could be consistent with either fear, disgust, or both underlying BII phobia.

2.3. Obsessive–compulsive disorder

Several recent neuroimaging studies suggest that disgust may be a dominant emotional response in contamination fear-related OCD. For example, one study found that patients with OCD showed greater activation than controls in the left ventrolateral prefrontal cortex, but reduced activation in the thalamus, to facial expressions of disgust and there were no between-group differences in response to fear (Lawrence et al., 2007). Phillips et al. (2002) also presented individuals with contamination-based OCD with disorder-relevant pictures and found heightened insula, but not amygdala, activation. Similarly, Shapira et al. (2003) found heightened insula and not amygdala activation towards generally disgusting pictures among a contamination fear sample.

Some studies implicate both fear and disgust in contamination fears. Schienle et al. (2005) used a heterogeneous OCD sample (i.e., not 'pure' contamination fear OCD) and found enhanced insula activation while viewing disorder relevant pictures, but amygdala activation towards these pictures correlated positively with a self-report measure of contamination fear as well as with the experience of disgust. Maitaix-Cols et al. (2004), however, used a heterogeneous OCD sample and found enhanced amygdala activation during a contamination fear provocation task, but that insula activation correlated with a self-report measure of contamination fear. Breiter et al. (1996) found both insula and amygdala activation in response to contaminated objects among contamination fearful individuals. Two studies found neither insula nor amygdala activation towards disorder-relevant objects (McGuire et al., 1994; Rauch et al., 1994), and one other study found only amygdala, and not insula, activation towards disorder-relevant pictures (van den Heuvel et al., 2004).

Current findings seem to provide strong evidence for a role of the insula (and ventrolateral prefrontal cortex) in contamination-related OCD, and further suggest that abnormally elevated activity in these neural regions may be largely associated with heightened sensitivity to disgust. However, activation of the amygdala, commonly associated with fear, is not an uncommon observation. Such discrepant findings may likely emerge as a functional of key methodological differences across studies. For example, important differences may emerge in neural activation in response to disgusted faces versus disgusting objects. Cross-study comparisons are further complicated by the use of a homogenous sample of OCD patients in some studies and the use of a heterogeneous sample of OCD patients in others. Current discrepancies in studies on neuroimaging of emotion in OCD may also be resolved by

consideration of chronometry of activation. That is, responses associated with timing but not magnitude of activation may ultimately reveal a synergistic relation between the amygdala and the insula in OCD. There is mounting evidence highlighting differences between fear and disgust at an early, more automatic, stage of processing, and also at a later post-categorical, more conscious stage. Clarification of amygdala and insula involvement in OCD will ultimately require attention to the temporal course of recruitment of these structures known to be part of a neural network recruited to process fear and disgust information.

3. Specificity of disgust's role in spider, BII, and contamination-related OCD

The data reviewed above indicates a clear relation between disgust and spider phobia, BII phobia, and contamination-related OCD. It is likely, however, that the manner by which disgust is causal depends on the specific disorder. Cisler et al. (2009a) recently reviewed the role of disgust and fear in cognitive, neural, facial expression, and heart rate responses in spider phobia, BII phobia, and contamination-related OCD. The available evidence suggests a prominent role of disgust in the cognitive and facial expression responses of spider phobics, but a prominent role of fear in the heart rate and neural responses of spider phobics. For example, spider phobic individuals tend to not eat cookies if a spider has walked across it (Mulkens et al., 1996), which demonstrates a cognitive feature of disgust called the 'law of contagion' (i.e., once in contact, always contaminated; Rozin and Fallon, 1987). However, spider phobics demonstrate a heart rate *increase* when exposed to spiders, which indicates a fear response. In contrast, the review suggested a prominent role of disgust in all response domains currently investigated in BII phobia and contamination-related OCD, and the role of fear in these disorders appeared to emerge mostly in the cognitive (i.e., self-reported emotion during exposure to disorder-relevant stimuli) domains. For example, contamination-related OCD tends to be associated with anterior insula activation (Phillips et al., 2002) as well as the law of contagion cognitive style (Tolin et al., 2004), both of which indicate a disgust response. Accordingly, the magnitude of the role of disgust may depend on the specific disorder and the response domain measured.

The pattern of findings may also emerge due to the nature of the disorder-relevant stimuli (Cisler et al., 2009a). That is, spiders are *moving* objects in the environment. Thus, rapid physiological mobilization (i.e., heart rate increase, automatic amygdala activation) may be needed to rapidly increase physical distance from the spider, thus necessitating a fear response in physiological domains. Disgust may emerge in the more controlled response domains of facial expression and cognitive processes in order to motivate passive avoidance of objects that have come in contact with spiders in order to protect against contagion transmission (cf. Matchett and Davey, 1991). The disorder-relevant stimuli in BII phobia and contamination-related OCD, however, are relative *static*. The potential for harm from these stimuli (e.g., blood, feces) may be more related to the possibility for contagion; thus, rapidly increasing the physical distance from the stimulus is not necessary. Instead, a disgust response may be called for in order to prevent physical contact with these stimuli and stimuli that have contacted these stimuli. Thus, disgust may operate as the primary emotion in BII phobia and contamination-based OCD with fear being mobilized as a secondary emotion, whereas the inverse is true of spider phobia. (Sawchuk et al., 2002). Distinct physiological profiles may also be observed as a function of the primacy of fear and disgust in specific anxiety disorders. This line of reasoning may also explain why the magnitude of fear and disgust responses in these disorders depends on the specific disorder as well as the response domain measured.

4. Disgust and anxiety disorders: towards an integrative model

Descriptive, experimental, and neuroimaging evidence demonstrates that disgust is a critical factor in spider phobia, BII phobia, and contamination-related OCD. However, there is a need for a comprehensive model to guide future research. Development of such a model may be informed by integrating 1) studies demonstrating that disgust mediates the relation between higher-order constructs (e.g., negative affect) and anxious responding, 2) studies demonstrating factors that moderate/potentiate the relation between disgust and anxious symptoms, 3) and studies investigating the precise mechanism by which disgust may cause increases in symptoms of anxiety disorders. Another critical feature of developing an integrated model is predicting whether there is a general or a specific route by which disgust is involved in the development of specific anxiety disorders. Given that a number of studies have been conducted demonstrating a link between disgust and specific brain regions (Calder et al., 2007) as well as emotional regulation (Mataix-Cols et al., 2008), and the research pointing to a connection between disgust and specific psychopathology, an important next step would involve integrating these approaches. There are several important directions that the conjoint examination of neural response to disgust in specific anxiety disorders may take that builds on the available evidence.

4.1. Mediation studies

Recent evidence demonstrates that disgust is a critical mediating factor in spider phobia, BII phobia, and contamination-related OCD. Olatunji et al. (2007c) employed structural equation modeling and tested the relation between latent factors of negative affectivity, disgust propensity, spider phobia, BII phobia, and contamination-related OCD. The results demonstrated that disgust fully mediated the relation between negative affectivity and spider phobia and BII phobia, and disgust partially mediated the relation between negative affectivity and contamination-related OCD. Deacon and Olatunji (2007) engaged participants in behavioral avoidance tasks (BAT) involving a used comb, a cookie on the floor, and a bedpan filled with toilet water. Regression analyses demonstrated that disgust propensity fully mediated the relation between self-reported contamination-related cognitions (e.g., the likelihood that touching a rail would result in contamination) and avoidance in the BAT tasks. Disgust propensity also mediated the relation between contamination-related cognitions and self-reported anxiety during the BATs.

Olatunji and Deacon (2008) similarly engaged participants in a BAT towards a realistic-appearing, but fake, tarantula. Consistent with the disease avoidance model (Matchett and Davey, 1991), regression analyses demonstrated that disgust propensity fully mediated the relation between self-reported contamination fear and self-reported fear during the BAT. Overall, these data suggest that disgust is a necessary mechanism by which widely-accepted higher order factors (i.e., negative affect, contamination cognitions, and contamination fear) contribute to spider phobia, BII phobia, and contamination-related OCD. Importantly, the mediating function of disgust generalizes across both self-report and behavioral indices. Although a mediational model implies causality, there may not be sufficient data to confirm a causal path between cognitions-disgust or contamination fear-disgust. Perhaps one tentative conclusion is that the influence of general vulnerabilities on the development of some anxiety disorders may be transmitted via disgust.

4.2. Moderation studies

In the context of disgust research, moderators facilitate the prediction of 1) when disgust will be related to these specific anxiety disorders, and 2) the degree disgust will be related to these anxiety disorders. Cougle et al. (2007) engaged college students who had elevated scores on a contamination-related OCD measure in a prolonged exposure task and measured the decline in disgust, anxiety, and urge to wash across the exposure trial. The authors found that for participants whose contamination fear was primarily due to a fear of contracting

illness, the decline in disgust during the exposure task did not predict concurrent declines in the urge to wash. Conversely, for the participants whose contamination concerns were primarily related to a fear of being overwhelmed with disgust (73% of the sub-sample) or anxiety (20% of the sub-sample), the decline in disgust did predict declines in the urge to wash. Thus, the primary threat underlying the contamination fear moderated the degree to which disgust predicted positive outcome during exposure.

In a related vein, Cisler et al. (2007) have demonstrated that anxiety sensitivity (AS; i.e., a fear of symptoms of anxiety) potentiates the ability of disgust to predict contamination fears. This effect is specific to contamination fear and does not generalize to BII phobia (Cisler et al., 2008). Similarly, Cisler et al. (2009b) found that self-reported difficulties in general emotion regulation potentiate the ability of disgust to predict both spider and contamination fear, but not BII fear. In contrast, self-reported difficulties regulating disgust potentiated the ability of disgust to predict BII fear and not spider or contamination fear. These data suggest that individual difference variables, such as anxiety sensitivity and emotion regulation, moderate the relation between disgust and anxiety disorder symptoms.

4.3. Mechanism studies

Recent research has also begun to investigate the precise mechanisms by which disgust may causally contribute to these specific anxiety disorders. One possible mechanism whereby disgust may contribute to anxious responding is via information processing biases. Davey et al. (2006) found that priming a disgusted mood led to greater threat-related interpretation biases relative to priming a neutral or positive mood. Provided that interpretation biases have been shown to causally increase anxiety (Mathews and Mackintosh, 2000), Davey and colleagues argued that disgust may cause anxiety through the induction of negative interpretation biases. For example, disgust stimuli may elicit disgust and subsequently cause an individual to interpret neutral stimuli in a negative manner. Interpreting neutral stimuli in a negative manner may then cause an increase in anxiety.

Research has generally demonstrated attentional biases towards disgust stimuli (Charash and McKay, 2002; but see Thorpe and Salkovskis, 1998 for contradictory results in the Stroop task). For example, Cisler et al. (2009) recently demonstrated that the attentional bias characteristics for disgust stimuli differ from the attentional bias characteristics for fear stimuli. Specifically, disgust stimuli appear to elicit an exaggerated difficulty disengaging attention away from disgust stimuli relative to fear stimuli or neutral stimuli. They also found that this exaggerated difficulty disengaging attention from disgust stimuli may be specific to individuals who have a high propensity to respond with disgust. This exaggerated difficulty disengaging away from disgust stimuli may serve as an etiological mechanism by which excessive anxiety may develop. For example, a highly disgust prone individual's attention may be captured by disgust cues in a kitchen and disrupt ongoing goal directed behavior. Difficulty removing attention away from those disgust cues, due to concerns of the spread illnesses, may lead the individual to excessively clean the kitchen until disgust cues no longer capture attention. The individual may then excessively clean in the future as a means to prevent disgust cues from capturing attention and disrupting ongoing purposeful behavior.

It is important to note that some studies also show only a main effect of valence (i.e. an attentional bias for disgust) that does not significantly interaction with disgust sensitivity. This suggests that the preferential processing of disgusting stimuli may not always be uniquely observed among vulnerable (high in disgust-related personality traits) individuals. This may imply that an information processing bias mechanism for disgust may not fully explain why some individuals develop an anxiety disorders and others not. However, the absence of an interaction between an attentional bias for disgust and trait disgust sensitivity/propensity may also be partially explained by divergent methods in studies that have found such an interaction. Consistency across experimental methods and operational definitions of disgust as an individual difference personality trait will be vital in future studies examining the feasibility of information processing parameters and potential causal mechanisms.

Another possible mechanism by which disgust may lead to anxiety is via dread of contagion, which is consistent with the disease-avoidance model of small animal phobias (Matchett and Davey, 1991). Muris et al. (2008) presented Dutch children with pictures of Australian marsupials unfamiliar to the Dutch culture. The children were either told that the marsupials were 'dirty' (e.g., pelt full of disease, rubs feces on his felt, urinates in his bed) or 'clean' (e.g., has a clean and soft pelt, sleeps in a bed of flower petals). The 'dirty' information led children to endorse more fear towards the animals relative to the 'clean' information. These data suggest that one mechanism by which disgust may lead to fears of small animals is via dread of contagion. Importantly, prior research has demonstrated that merely experiencing disgust does not cause an increase in experienced anxiety (Marzillier and Davey, 2005). Thus, the mechanism by which children's fears of the animals increased is not due to merely feeling disgusted; rather, the disgust *information* appears to be necessary to motivate the avoidance of possible contagion.

4.4. Specific versus non-specific pathways

Another critical feature of developing a theory for disgust in these anxiety disorders is predicting whether there is a general (i.e., non-specific) route by which disgust may be involved in each of these three anxiety disorders, or whether there is a specific route for each anxiety disorder. For example, Olatunji et al. (2007c) demonstrate that disgust may be a necessary pathway by which a predisposition towards negative affect develops into either spider, BII, or contamination fears, but what determines whether an individual develops spider fears instead of BII or contamination fears?

Davey et al. (1993) previously demonstrated that parental levels of disgust propensity predicted offspring levels of small animal fears. One possible theory integrating Davey et al.'s (1993) findings with Muris et al.'s (2008) findings is that parents with high levels of disgust may describe small animals (e.g., spiders, rodents, etc) with disgusting terms (e.g., dirty, disease-ridden, gross, etc), thus inducing disgust-related instructional conditioning, dread of contagion from these animals, and consequently motivating excessive fear of these animals. However, it cannot be determined yet whether this is a specific pathway by which disgust leads to spider fears because research has not yet tested whether disgust instructional conditioning also leads to BII or contamination fears.

Similarly, it is not yet clear whether the other possible mechanisms by which disgust may exert an influence on anxiety (i.e., interpretation and attentional biases) are general or specific mechanisms. For example, Cisler et al. (2009) discussed attentional biases as a mechanism for disgust in contamination-related OCD, but it may similarly be the case that attentional biases are a mechanism for disgust in spider phobia or BII phobia. Given the current state of evidence, it is only possible to propose that these mechanisms are general processes by which disgust may lead to any of these anxiety disorders.

The current state of evidence does, however, allow for one specific prediction. Cougle et al. (2007) and Cisler et al. (2008, 2009b) studies suggest that AS and general emotion dysregulation may be specific potentiating factors for the role of disgust in contamination and spider fears and not BII fears. Accordingly, one factor that may determine whether heightened disgust leads an individual to develop contamination fears instead of BII fears is AS or general emotion dysregulation.

4.5. Summary analysis

The available research suggests that disgust mediates the relation between negative affect and spider phobia and BII phobia, but partially

mediates the relation for contamination fears (Olatunji et al., 2007c). Disgust may contribute to these anxiety concerns via interpretation biases (Davey et al., 2006), attentional biases (Cisler et al., 2009), or dread of contagion (Matchett and Davey, 1991; Muris et al., 2008). Whether disgust contributes to the development of contamination fears versus other fears may also be moderated by AS (Cisler et al., 2008). Parental disgust propensity may be a pathway towards the development of spider phobia (Davey et al., 1993), which is mediated by dread of contagion (Muris et al., 2008). However, the extent to which parental disgust propensity may be implicated in BII phobia and OCD has yet to be investigated. Preliminary research suggests that emotion (dys)regulation may be a higher-order factor that may potentiate the effect of disgust on the development of anxiety-disorder symptoms (Cisler et al., 2009b). Previous theoretically diverse research has identified crucial mediators, moderators, and potential mechanisms that may partially explain the association between the experience of disgust and anxiety disorder symptoms. An integration and synthesis of such research will prove to be instrumental in the development of a unified theoretical model in which more definitive causal inferences can be made regarding the role of disgust in the development of specific anxiety disorders.

5. Future directions

Emerging research has implicated disgust in spider phobia, BII phobia, and contamination-based OCD. While disgust may be a cause of these anxiety conditions it is likely that excessive disgust responding may be a major consequence of other anxiety disorders. Such distinctions will require the examination of anxiety disorders where the experience of disgust in not intricately linked to specific objects. For example, Dalgleish and Power (2004) argue that some forms of posttraumatic stress disorder (PTSD) involve disgust reactions to reminders of a traumatic event and physiological concomitants of disgust such as nausea and vomiting, as well as vigilance for signs of contamination. Indeed, elevated disgust has generally been linked with PTSD among Vietnam veterans (Foy et al., 1984) and Shin et al. (1999) have found that women with a history of childhood sexual abuse and associated PTSD reported significantly more disgust during the recollection and imagery of a traumatic event than those without PTSD. Furthermore, when exposed to memories of sexual assault, women report elevated feelings related to disgust, including “feelings of dirtiness” and “urge to wash” (Fairbrother and Rachman, 2004; Fairbrother et al., 2004), and disgust is a significant predictor of feelings of dirtiness associated with imagining a sexual assault among women (Herba and Rachman, 2007). Although these initial findings suggest that disgust may play an important role in PTSD, the mechanism and neurobiological correlates of disgust in PTSD remain unclear. However, extending the examination of disgust to anxiety disorders beyond those marked by specific fear and avoidance of aversive objects/situations will prove to be instrumental in better understanding the origins and boundaries of the disgust in the anxiety disorders.

In addition to illuminating the potential role of disgust in other anxiety disorders, empirical evidence for an integrative model to guide future research is clearly needed. The success of such a research agenda will depend, in part, on identifying operations that are unique to disgust. The available research suggests that the insula is involved in the decoding of disgust, whereas fear engages the amygdala. This dissociation appears to be related to the modulation of attention that emerges at a later stage for disgust, whereas fear processing may be relatively independent from attention (Anderson et al., 2003; Bishop et al., 2007). Experimental psychopathology research has been instrumental in illuminating important differences between fear and disgust as well as the relevance of such differences for better understanding specific anxiety conditions. Experimental psychopathology is a subfield of psychopathology research that aims to elucidate processes that contribute, either in whole or in part, to the etiology, exacerbation, or maintenance of abnormal behavior (Forsyth and Zvolensky, 2002). Experimental psychopathologists have devised numerous laboratory methods designed to elucidate clinical conditions. Adoption of this approach by neuroscientists could yield a truly translational approach to examining the role of disgust (and emotions more broadly) in the anxiety disorders. A central feature of experimental psychopathology is the identification and manipulation of variables and processes so as to induce features of psychopathology. Experimental manipulation of disgust-relevant processes in neuroscience will ultimately inform a unified theoretical model with an empirically informed biological basis. Integration of experimental psychopathology and neuroscience research certainly holds promise in advancing research on disgust in the anxiety disorders.

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