

Disgust as a transdiagnostic index of mental illness: A narrative review of clinical populations

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Disgust is a basic emotion of rejection, providing an ancestral defensive mechanism against illness. Based on research that documents altered experiences of disgust across several psychopathological conditions, we conducted a narrative review to address the hypothesis that altered disgust may serve as a transdiagnostic index of mental illness. Our synthesis of the literature from past decades suggests that, compared to healthy populations, patients with mental disorders exhibit abnormal processing of disgust in at least one of the analyzed dimensions. We also outline evidence of alterations in brain areas relevant to disgust processing, such as the insula and the interconnected limbic network. Overall, we provide preliminary support for the hypothesis that altered disgust processing may serve as a transdiagnostic index of mental illness. (Bulletin of the Menninger Clinic, 87(Suppl. A), 53–91)

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It's a dirty world outside! Try to remain impassive to an unpleasant odor in the air, the smell of spoiled food, or the sight of excrement. Surely your nose will wrinkle, your mouth will tighten, and you would probably try to protect yourself from possible contamination. Moreover, if you look around, you will recognize similar expressions and behaviors in others (Rozin, et al., 2008). Disgust is a basic emotion involving subjective feelings of revulsion and physiological, expressive, and behavioral reactions of avoiding or removing potentially contaminating entities (Rozin et al., 2008). In humans, disgust is often associated with thoughts and fear of potential contamination, and this probably best captures its evolutionary origin as one of the most ancestral defensive mechanisms against illness (Oaten et al., 2009). Indeed, disgust is thought to originate from the primitive sensation of distaste elicited by contaminated or bad-tasting foods—thus preventing the ingestion of harmful substances, and in turn protecting against diseases (Rozin & Fallon, 1987). Scholars have also proposed that during human evolution, the defensive role of disgust extended from the protection of the body against pathogens to the protection of the soul from certain moral offenses (Rozin & Fallon, 1987; Rozin et al., 2008; Vicario, Rafal, et al., 2022). In this sense, it has been suggested (e.g., Vicario, Rafal, Borgomaneri, et al., 2017; Vicario, Rafal, Martino, et al., 2017) that the experience of disgust is multidimensional, encompassing *core disgust* (i.e., a very basic aversive experience that is triggered by potentially toxic and contaminating stimuli), *moral disgust* (sensitivity to, and negative evaluations of, moral transgressions and socially inappropriate behaviors), and *social disgust* (the ability to recognize and evaluate social signs of disgust in others).

Because of its complex nature, disgust is relevant to several fields of research. In the clinical field, the term *self-disgust* has been introduced to describe feelings of disgust directed toward the self (Ille et al., 2014; Tsatali et al., 2019). The personality psychology literature also distinguishes between disgust propensity (how easily people are disgusted in response to a

disgust trigger; e.g., Olatunji, Berg, Cox, et al., 2017; van Overveld et al., 2006) and disgust sensitivity (how unpleasant the experience of disgust is to the individual; Ille et al., 2015; van Overveld et al., 2006; von Spreckelsen et al., 2018), which are two distinct dimensions typically assessed through self-report questionnaires (Haidt et al., 1994; Rozin et al., 1984; Schienle et al., 2010). Disgust is also considered relevant for monitoring internal (interoceptive) states. This is suggested by the evidence that cardiac activity predicts the experience of disgust (Gray et al., 2012), as well as evidence that disgust influences (Chan et al., 2014) and can be influenced by (Hoeftling et al., 2009; Vicario et al., 2018) appetite. Finally, neuroscience research has reported the existence of a complex disgust processing neural network (Scharmüller & Schienle, 2012; Wabnegger et al., 2018, 2022). While core disgust proneness (e.g., food-related disgust proneness) is associated with sectors of the insula considered to underpin the gustatory cortex, disgust proneness to decay and death negatively correlates with cognitive control in brain regions such as the dorsomedial cortex and the dorsolateral prefrontal cortex (DLPFC; Scharmüller & Schienle, 2012). Brain imaging studies of healthy populations have also shown involvement of the basal ganglia, the anterior cingulate cortex (ACC) (Klucken et al., 2012; Royet et al., 2016; Scharmüller & Schienle, 2012), and the orbito-frontal cortex (OFC) in disgust processing (Wabnegger et al., 2018). Moreover, it is important to mention the key role of the insula, which is thought to integrate information from multiple sensory modalities during disgust processing (Gan et al., 2022; Kelly et al., 2012; Miller, 2013; Nomi et al., 2018; Woolley et al., 2015).

In a recent review article by our group (Vicario, Rafal, Martino, et al., 2017; see also Vicario & Lucifora, 2021), we have addressed core, social, and moral disgust in clinical models. The results have outlined the existence of several neurological and psychiatric disorders affected by disgust processing deficits in at least one of the three above-mentioned domains. This pattern might be linked with the evidence of altered interoceptive—insula-based—functioning in these clinical populations (for a review, see also Vicario et al., 2020). Remarkably, in recent works (e.g., Olatunji, Armstrong, et al., 2017; Stasik-O'Brien et al., 2021), disgust has been proposed as a possible transdiagnostic

index for anxiety and related disorders. In light of the evidence of interoceptive/affective alterations encompassing the insula and the related neural network, in the present work we tested a transdiagnostic index hypothesis of disgust in a wider range of mental disorders. We addressed this hypothesis by providing a narrative synthesis of the past decade's literature on disgust (core, social, and self-disgust) processing in mental (neurological, psychiatric/clinical) disorders. The relevant literature was obtained by consulting the PUBMED and Google Scholar websites, including as keywords combinations of the name of the examined clinical disorders with the terms “disgust,” “disgust propensity,” and “disgust sensitivity.” We chose to conduct a narrative review because it represents a convenient approach for a preliminary and extensive exploration of the relevance of our hypothesis. We hypothesized the existence of altered disgust processing as a shared index, and the respective alteration of the disgust processing neural network among the examined clinical populations.

Disgust processing in neurodegenerative disorders

Huntington's disease

Huntington's disease (HD) is an inherited neurodegenerative disease that affects the central nervous system. Neurodegeneration starts with the atrophy of the striatum, involving the caudate nucleus and the putamen, and extends with the atrophy of widespread brain regions, causing loss of structural and functional connectivity in the basal ganglia loops responsible for motor, cognitive, and neuropsychiatric symptoms (for a review, see Snowden, 2017). Neuronal death is due to the mutated huntingtin protein located on an autosomal chromosome, consisting of cytosine-adenine-guanine (CAG) extensive repetition (Craufurd et al., 2015; Snowden, 2017). The characteristic feature of patients is hyperkinetic movement disorder, characterized by irregular involuntary contractions; in addition, the neurodegenerative process leads to other deficits in executive functions, memory, emotion processing, and social cognition (Snowden, 2017). HD patients also exhibit apathy, irritability, and depression, as assessed in a longitudinal study by Thompson et al.

(2012) using the Problem Behaviors Assessment for Huntington's Disease (PBA-HD). There is evidence of the central relevance of social disgust deficits in HD. For example, Dogan et al. (2014) observed less recruitment of the bilateral ventral putamen, dorso-ventral caudate, posterior insula, and inferior OFC in HD compared to controls in response to videos of faces expressing disgust. Furthermore, they found a positive association between insular activation and CAG repeats, indicating an increase in functional activity with higher genetic load in HD during disgust processing. Interestingly, a previous finding by Novak et al. (2012) reported negative correlations between disgust-related insular activity and CAG repeats in a pre-manifest disease sample. Together these findings suggest that increment of insular activity once the disease is manifested may represent the use of compensatory mechanisms needed to process disgust emotions. On the other hand, other works (Bora et al., 2016; Henley et al., 2012) support the conclusion that recognition of all negative emotions tends to be impaired in HD. For instance, Croft et al. (2014) reported lower accuracy of HD, compared to controls, in detecting faces expressing anger and disgust. Moreover, this difficulty included the no expression condition. As discussed by Eddy et al. (2016), emotion recognition problems with human faces, tone of voice, and body postures may lead to poor behavioral flexibility in social situations and antisocial or aggressive acts linked to misunderstanding of communication.

A recent study (Kordsachia et al., 2017) documented diminished levator labii activity in HD participants, compared to controls, in responses to disgusting scenes (but not to disgusted faces), suggesting HD patients' reduced sensitivity to core disgust. Finally, Labuschagne et al. (2018) found that in HD, the administration of intranasal oxytocin—a neuropeptide that improves emotional recognition (Heinrichs et al., 2009)—normalizes the activity of some brain regions (i.e., putamen) known to be related to disgust processing (Calder et al., 2000) during exposure to faces expressing disgust. Overall, the examined articles support the notion of altered core disgust in HD, underpinned by an altered disgust processing neural network. This is in line with the suggestion that disgust can be an early warning sign of HD symptoms. Regarding social disgust, the results are conflicting, and they could depend on the heterogeneity of the paradigms

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adopted for the investigation and on differences in the severity of the symptoms associated with the examined clinical samples.

Parkinson's disease

Parkinson's disease (PD) is one of the most common neurodegenerative disorders, along with Alzheimer's disease. It affects approximately 1% of adults over age 60, and it is distinguished by prodrome motor symptoms, including bradykinesia, rest tremor, and rigidity (McGregor & Nelson, 2019). It is well established that PD is characterized by neuronal death, mainly in the substantia nigra, which involves loss of dopamine and the consequent reduction of dopamine levels, causing dysfunctions of the basal ganglia and thalamocortical circuits (for a review, see McGregor & Nelson, 2019; Ponsi et al., 2021). Although the Movement Disorder Society PD Criteria retain motor symptoms as the core feature of the disease (Postuma et al., 2015), increasing evidence suggests that altered basal ganglia and thalamocortical circuitries also underpin impaired cognitive, emotional, and motivational processes in PD patients (for a review, see Ponsi et al., 2021). Hence, as discussed by Santangelo et al. (2017), dopamine depletion determines both motor and non-motor symptoms in the early stages of PD, and this imbalance in dopamine levels along with replacement therapy is associated with personality traits in PD patients, including "exploration of novelty" and "avoidance behavior," that are present even before the appearance of motor symptoms.

Despite many studies investigating recognition of disgust expression and disgust experience in PD patients, results are controversial. Comparing PD patients and healthy controls (HC) in the recognition of facial expressions depicting disgust among other emotions, Schienle, Ille, and Wabnegger (2015) observed similar activation (including amygdala, OFC, DLPFC, and entrolateral prefrontal cortex [VLPFC]) in both groups, and they found no differences in self-report scales for the assessment of disgust proneness measured with the Questionnaire for the Assessment of Disgust Proneness. Similar results were found in another study by the same group (Wabnegger et al., 2015). On the other hand, Sedda et al. (2017) observed a dissociation

between impaired recognition of disgusted faces and conserved processing for disgusting visual stimuli and sentences. Ricciardi et al. (2015) investigated a possible relationship between reduced facial expressiveness and altered emotion processing (i.e., facial recognition and alexithymia) in PD patients compared to controls. The authors found correlations between Ekman test scores (Ekman et al., 2002) and disgust expressiveness as well as a significant negative correlation between the expressiveness of disgust and scores on the Toronto Alexithymia Scale-20 (Bagby et al., 1994). A reduced volume of OFC gray matter in PD patients was found to be associated with reduced trait disgust proneness as measured with the Questionnaire for the Assessment of Disgust Proneness (Ille et al., 2015). The same research group (Ille, Wabnegger, et al., 2016) reported that PD patients exhibit less disgust toward poor hygiene and spoiled food on the Scale for the Assessment of Disgust Sensitivity (SADS). No differences were found between patients and controls in recognition and experiences of disgust emotion (Ille, Wabnegger, et al., 2016). This specific effect on disgust proneness might be explained according to the evidence that neurodegeneration in PD affects brain areas (OFC and piriform cortex) related to deficits in disgust processing mediated by olfaction (Ille et al., 2015; see also Lee et al., 2014). Finally, a recent study by Tsatali et al. (2019) found that PD patients reported higher baseline levels of self-disgust compared to controls, as well as higher levels of experimentally induced self-disgust, assessed through oral narration of experiences that made them feel ashamed, guilty, or self-disgusted; moreover, self-disgust was significantly and selectively predicted by the impulse control disorder of patients, measured by the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease Rating Scale (Weintraub et al., 2012).

Overall, the literature reviewed here suggests impaired disgust processing in PD, which may involve different domains and encompasses the disgust processing neural network. However, especially in the domain of social disgust, the results are inconsistent. As mentioned for HD, this may be due to the heterogeneity of the adopted paradigms and to differences in the severity of patients' clinical conditions.

Other neurological disorders

Abnormal disgust processing is also reported in several neurodegenerative disorders and neurological conditions that involve relevant neural regions for disgust processing, such as the insula, ACC, OFC, and ventromedial prefrontal cortex (Eckart et al., 2012; Ille, Wolf, et al., 2016; Kumfor et al., 2013; Verstaen et al., 2016; Vicario et al., 2021; Woolley et al., 2015). For example, Eckart et al. (2012) investigated the disgust responses in a sample of patients with the behavioral variant of frontotemporal dementia (bvFTD) during the observation of a video eliciting disgust. It was found that, compared to controls, bvFTD patients exhibit an overall reduced reactivity to disgust, as shown through different behavioral and psychophysiological measures. Moreover, Kumfor et al. (2013) observed a specific link between insular activation and the processing of disgust emotion compared to other emotions in a sample of bvFTD patients. However, the deficit also included the recognition of others negative emotions, depending on the neural structure involved. Similar results were found in subsequent investigations involving a heterogeneous sample of patients with neurodegenerative diseases (i.e., frontotemporal dementia, corticobasal syndrome, progressive supranuclear palsy, Alzheimer's disease) (e.g., Kumfor et al., 2013; Verstaen et al., 2016; Woolley et al., 2015). Finally, there is evidence of reduced disgust proneness toward spoilage/decay in olfactory (anosmia and hyposmia) dysfunction (Ille, Wolf, et al., 2016). However, the high heterogeneity of the sample examined does not allow a clear interpretation of the neurological origin of the reported results.

Overall, the literature examined in this section is in line with the results discussed in the previous paragraphs on movement disorders, suggesting that abnormal core disgust processing and altered activity of the disgust processing neural network could be considered relevant landmarks for other neurological disorders. However, evidence (Kumfor et al., 2013) of altered processing of other negative emotions suggests a more general emotion recognition deficit in neurological disorders. Much could depend on the neural network involved, which may depend on the stage of the neurological disease.

Psychiatric disorders

Specific phobias

Specific phobias (SP) are characterized by an intense and irrational aversive reaction triggered by avoidance of exposure to or anticipation of a particular fearsome stimulus or situation (American Psychiatric Association [APA], 2013). The *DSM-5* (APA, 2013) also categorizes specific phobias into five subtypes: animal, natural environment, blood-injection-injury, situational, and other.

Disgust is implicated in the development of specific phobias such as emetophobia (fear of vomiting), blood-injection-injury phobia (BII), and zoophobia such as fear of spiders or snakes, which can elicit disgust responses due to their physical characteristics, including being creepy, hairy, or slimy (Olatunji, Armstrong, & Elwood, 2017). The type of phobia more frequently associated with disgust sensitivity is BII, which is often accompanied by vasovagal syncope during exposure to specific phobic objects or situations. In the Brinkmann et al. (2017) experiment, a group of phobic BII women and a group of HC anticipated the presentation of neutral and specific images of the phobia. On the behavioral level, anxiety dominated the anticipatory period in the BII phobic women compared to controls, while both anxiety and disgust were elevated during image presentation. Results showed initial and sustained increases in activation-relevant neural regions for disgust processing such as the ACC, insula, and the amygdala for the BII phobic women compared to the control group.

A study conducted by Winder et al. (2021) examined the possibility of distinguishing between adverse reactions to fear-relevant stimuli (i.e., heights and small enclosed spaces) and disgust-relevant stimuli (i.e., spiders and blood/injury). The results demonstrated that anxiety sensitivity was related to adverse reactions to one of the fear-relevant stimuli, such as fear of heights, and was not related to adverse reactions to any of the disgust-relevant stimuli. In contrast, disgust sensitivity, as a personality trait, was relevant to adverse reactions to a broad range of stimuli, especially adverse reactions to blood/injury

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and spider stimuli. Therefore, the presence of a high sensitivity to disgust should also be considered in the treatment of specific phobias. Building tolerance of disgust may be an effective element in phobia treatments. Overall, the literature examined in this section confirms the existence of altered social and core disgust processing in this clinical population, together with altered activity of the disgust processing neural network.

Obsessive-compulsive disorder

Obsessive-compulsive disorder (OCD) is a chronic and disabling disorder characterized by the presence of obsessions that are “recurrent and persistent thoughts, urges, or images that are experienced as intrusive and unwanted” (p. 235), and/or compulsions that are “repetitive behaviors or mental acts that an individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly” (p. 000) that cause distress and functional impairment (APA, 2013). OCD was recently removed from the anxiety disorders section of the *DSM-5* because it is associated with a range of non-anxiety affective experiences, including disgust (Stasik-O’Brien et al., 2021).

It has been suggested (e.g., Olatunji, Berg, Cox, et al., 2017; Stasik-O’Brien et al., 2021) that disgust propensity could be considered a transdiagnostic risk factor for OCD. Multiple studies suggest that disgust propensity mediates the relationship between OC symptoms and behavioral avoidance of contamination-related stimuli (Deacon & Olatunji, 2007; Olatunji et al., 2007). In addition, individuals with more severe OC symptoms and higher contamination fears report increased feelings of disgust as well as an increased heart rate response during exposure treatment sessions (Duncko & Veale, 2016). There are individual differences in disgust propensity that may influence the development of OCD (Knowles et al., 2018). For example, Muslim individuals with high scrupulosity are affected by greater disgust proneness and endorsed more symptoms of contamination fear after exposure to disgust-inducing images (Inozu et al., 2017). A study by Krause et al. (2022) highlights the relevance of disgust proneness across OCD symptom domains (i.e., the need to control thoughts in repugnant obsessions) and

suggests symptom-specific cognitive intervention targets. Moreover, Ching et al. (2018) showed that contamination-based disgust predicted OC concerns about sexual orientation. Finally, a study by Athey et al. (2015) of an adult OCD patient showed that changes in disgust propensity predicted changes in washing compulsion symptoms.

Bhikram et al. (2017) investigated clinical outcomes of OCD patients with high levels of disgust sensitivity and found that disgust may be more resistant to extinction than fear (Foa et al., 2005). In this sense, disgust plays a significant role in OCD symptoms, and some studies have shown that cognitive-behavioral treatment through cognitive reevaluation of emotions can lead to a significant decrease in learned discomfort and disgust among participants, compared to those who did not receive emotional suppression treatment (Olatunji et al., 2015; Olatunji, Berg, Cox, et al., 2017; see also Fink et al., 2018). A study by Inozu et al. (2023) suggests that treatment protocols should increase disgust tolerance by challenging dysfunctional appraisals related to the negative perceptions of contamination, which in turn may decrease the need for washing, particularly among individuals who have high disgust proneness. There is evidence that both primary and secondary reappraisal interventions may reduce disgust in individuals with moderate to high OCD-relevant contamination fears (Wong et al., 2021).

Regarding the neural correlates, a study conducted by Viol et al. (2019a) showed that while the evaluation of disgusting and neutral images was comparable between OCD patients and controls, patients' reactions were significantly different as they felt more agitated and could not cope with the situation due to the increased arousal. Furthermore, the results demonstrate the absence of hyperactivation of the insula in patients with OCD when they are facing disgusting images. In the disgust condition, no abnormal activity was found in any of the commonly overactive regions of OCD, neither in the ACC nor in the frontal cortex. However, the insula is active in these patients when they are experiencing stimuli that can cause OCD symptoms (i.e., patients become more agitated and feel less able to deal with the situation due to the heightened arousal) (Viol et al., 2019a). Therefore, a key neural region for disgust processing appears relevant to OCD. Overall, the literature examined in

this paragraph confirms the relevance of disgust in OCD, as the authors have observed a hypersensitivity to disgust-related triggers with a deficit in the processing of core disgust, along with altered activity of the insula, a key neural region of the disgust processing network. There is also evidence of altered social and moral disgust in OCD (Olatunji et al., 2011).

Posttraumatic stress disorder

Posttraumatic stress disorder (PTSD) is a psychiatric disorder that may develop following the experience of a traumatic event that evokes horror or fear (APA, 2013). Disgust, in addition to fear, sadness and anger, seems to contribute to PTSD symptoms (Coyle et al., 2014). Disgust sensitivity appears to be a risk factor for PTSD because it can affect the strength of classically conditioned associations during a traumatic event (Badour et al., 2013). Individuals with increased disgust sensitivity may be more likely to engage in acts of escape or avoidance in order to allay disgust in response to trauma cues (Badour & Feldner, 2018).

The experience of disgust in relation to trauma is more likely to occur when certain characteristics are present, such as body products (e.g., vomiting, blood), or odors related to the scene of the trauma, death, or experiences of sexual violence (Jones et al., 2020). Disgust is related to evaluative conditioning, in which the positive or negative value of a disgusting stimulus is transferred to a previously neutral stimulus that now becomes disgusting in itself (Jones et al., 2010). This type of conditioning explains why disgust is pervasive and difficult to extinguish and above all explains why some trauma victims feel disgusted and dirty even in the absence of potential contamination. For example, victims of sexual violence often resort to excessive cleaning and washing behaviors to feel cleansed of aggression (Mason & Richardson, 2012). Evidence suggests that disgust and mental contamination are particularly relevant to PTSD following various traumatic events such as sexual assault (Badour et al., 2013). Moral injuries can also be generated by disgust (Moon, 2019). Furthermore, moral injury is reliably associated with PTSD, and treatment should be focused on both PTSD-associated symptoms (fear, anxiety, and anger) and moral injury symptoms (guilt, shame) (Hall et al., 2022).

Brake et al. (2017) investigated the connection between PTSD, self-disgust, and suicide, showing that disgust can be a potential suicide risk factor. The results showed that negative alterations in cognition and mood, such as the inability to feel positive emotions and reliving the traumatic event, can lead to self-disgust, which increases the risk of suicide (Brake et al., 2017). Vidotto et al. (2014) investigated the relationship between anhedonia (the reduced ability to experience pleasure) and neural correlates of disgust in two clinical populations (those with PTSD, and those experiencing the first episode of a psychosis). The study results showed that individuals with PTSD have greater activation in the right superior frontal gyrus (rSFG), right inferior frontal gyrus (rIFG), right anterior insula (rAI), medial frontal gyrus (MFG), right amygdala (rA), and left inferior frontal gyrus (lIFG) in response to disgusting images, compared to the group experiencing the first episode of a psychosis and the control group. These data provide evidence of the involvement of the medial prefrontal cortex (MPC) in the pathophysiology of PTSD. Finally, a recent MRI study has shown that patients with PTSD are affected by greater gray matter volume in the putamen and the insula (Vicario, Martino, et al., 2022), two regions frequently associated with disgust processing (Calder et al., 2000; Papagno et al., 2016). Overall, the literature examined in this paragraph suggests the relevance of disgust in PTSD regarding the self and the core domains, as well as altered activity of the disgust processing neural network.

Borderline personality disorder

Borderline personality disorder (BPD) is defined by a pattern of instability in one's identity, relationships, and emotions, and is characterized by impulsiveness, self-harm, fear of abandonment, anger, emptiness, and pronounced fear of abandonment/rejection in one's social relationships (APA, 2013).

As discussed by Schmahl et al. (2014), BPD is characterized by the experience of intense negative feelings and difficulty in regulating emotion broadly, especially aversive states. A meta-analysis of fMRI studies by Ruocco et al. (2013) showed that BPD patients have greater activation within the insula and posterior cingulate cortex (PCC) during negative emotion processing;

moreover, this increased arousal may interfere with patients' ability to identify facial expressions of disgust in others. Thereby, in everyday life, for example, when confronted with poor hygiene, these patients also have a reduced recognition of disgust emotions in others (Schienle, Leutgeb, & Wabnegger, 2015). Because recognizing emotional cues in others is critical to social functioning, a deficit in disgust recognition is likely to be a potential factor involved in the difficulty in building relationships that these individuals often experience (Veague & Hooley, 2014). Furthermore, patients with BPD seem to experience marked self-disgust, the tendency to negatively judge their own actions and personal characteristics (Rüsch et al., 2010). Armstrong et al. (2017) compared the incremental validity of anxiety sensitivity and disgust sensitivity in predicting concomitant symptoms of BPD; however, the results did not demonstrate a relationship between disgust sensitivity and BPD symptoms. These results contrast with those of a previous study (Schienle et al., 2013), which found that disgust sensitivity, in addition to predicting symptoms in the BPD group, was higher in this group of patients, who received inpatient treatment, compared to HC. One possible explanation is that self-disgust rather than disgust sensitivity is the relevant aspect of disgust in BPD. Related to a neural level, in addition to the insula, the amygdala is a fundamental structure for the perception of information relevant to disgust (Gan et al., 2022). A voxel-based morphometry (VBM) study (Schienle, Leutgeb, & Wabnegger, 2015) showed that women diagnosed with BPD have a higher volume of the latero-basal amygdala (LBA), compared to HC. Furthermore, LBA volume was positively correlated with syndrome severity, whereby the increase in LBA volume of BPD patients likely reflected a greater propensity to experience negative stimuli more intensely. Overall, the results suggest the relevance of self-disgust for BPD, whereas there is less consensus in the literature on the relevance of core and social disgust. The literature also reported an altered involvement of the disgust processing neural network.

Depression

Depression is the leading cause of disability and one of the most common worldwide mental health problems (World Health

Organization, 2017). One of the risk factors for depression is loneliness, as feeling socially isolated induces negative affective states, such as greater attention to threatening stimuli (Cacioppo et al., 2015). There is evidence that individuals diagnosed with major depressive disorder (MDD) report feeling disgust toward themselves more frequently than feelings of guilt or shame (Zahn et al., 2015). A study conducted by Ypsilanti et al. (2019) evaluated the additive and mediating role of self-disgust in the relationship between loneliness and depression; the study demonstrated that self-disgust is greater among individuals with higher loneliness scores and that self-disgust may explain the association between loneliness and depression. Interestingly, Powell et al. (2013) showed that self-disgust can exist even in the absence of a depressive experience, as reported by one patient despite the lessening of the patient's depressive symptoms. The authors suggested that feelings of disgust may exist as a vulnerability factor only in some cases of depression and depressive relapse.

A similar relationship between depression and disgust has also been reported for social disgust. A recent study (Lv et al., 2022) compared the performance of a group of adolescents with MDD and a HC group who completed a facial emotion processing task (FEP) using the Chinese Facial Affective Picture System. The MDD group had significantly lower accuracy and perception intensity of happiness expression and significantly higher accuracy of disgust expression and higher perception intensity of sad and fearful faces in the FEP task. These results support the understanding that in depression there is hypersensitivity to all negative emotional facial expressions. Furthermore, according to Douglas and Porter (2010), patients with depression show a specific deficit in the recognition of facial expressions of disgust, compared with HC. The literature reports several studies linking the insula with disgust in depression (e.g., see Vicario, Rafal, Martino, et al., 2017, for a review). More recently, Korgaonkar et al. (2019) reported a specific pattern of amygdala hypoconnectivity to the insula in response to disgust threat emotions (i.e., subliminal processing of facial expressions) in bipolar depression compared to unipolar depression.

Overall, the literature supports the conceptualization of self-disgust as a factor with a significant etiological role in depression. Moreover, the literature provides insight into abnormal

social disgust appraisal and altered activity of the disgust processing neural network.

Anorexia and bulimia nervosa

Anorexia nervosa (AN) is a psychiatric disorder characterized by a restriction of food intake that leads to a reduction in body weight; furthermore, binge episodes may be followed by food elimination behaviors in addition to a distorted perception of body shape and weight (APA, 2013). A related eating disorder is bulimia nervosa (BN), which refers to a mental illness characterized by repetitive overeating and purging behaviors to prevent weight gain (APA, 2013).

Numerous studies have investigated the relationship between disgust and eating disorders. Aharoni and Hertz (2012) found that in the AN group, there was greater sensitivity to disgust in the majority of the disgust domains (six out of eight) as measured by the Disgust Sensitivity Questionnaire. Moreover, a recent study clarifies the direct link between altered disgust processing and food. While disgust perception is maintained in AN, if there is a particular focus on food-related aspects, patients with AN perceive disgust much more markedly than HC (Marzola et al., 2020; Vicario, 2013). Because disgust is instrumental in modulating food intake, it is interesting to analyze how the brain of ED patients processes disgusting and pleasant stimuli. In a study by Monteleone et al. (2017), patients with AN and BN and HC underwent fMRI while tasting a solution of sucrose (sweet taste, pleasant) and a solution of quinine hydrochloride (bitter taste, aversive). The results demonstrated that in patients with AN, both stimuli activated relevant regions for disgust processing, such as the insula, the striatum, and the OFC. The most interesting finding was that patients with AN and patients with BN both showed a greater brain response in the areas of the taste-reward pathways when they experienced a pleasant taste compared to an aversive one. A possible explanation could be that these subjects attribute a greater salience to pleasant gustatory stimuli than to foods with a high caloric content, and the latter are therefore considered a potential danger by patients with eating disorders (Monteleone et al., 2017). Regarding unpleasant/disgusting stimuli, AN patients showed a reduced response to

the bitter stimulus in the right amygdala and in the left ACC compared to HC; BN patients exhibited reduced activity in the right amygdala and the left insula compared to HC. The authors suggested that there may be an impairment of physiological aversion to unpleasant bitter tastes in patients with eating disorders.

Another fMRI study (Jiang et al., 2019) found overall increased disgust sensitivity in patients with AN and BN while they were smelling food odors compared to controls. This study also associated impaired disgust sensitivity with a different dysfunction of the mesolimbic circuit: Both groups of patients showed reduced activation of the anterior ventral pallidum and the insula compared to controls.

Finally, we note the theoretical model proposed by Glashouwer and de Jong (2021) that calls into question body-related self-disgust to explain the onset and maintenance of AN. According to this model, disgust is elicited by feelings of revulsion related to the confrontation with the body, so that limiting food intake may serve to avoid self-disgust. Greater self-disgust and disgust sensitivity in anorexic and bulimic patients, compared to controls, has also been documented recently by Kot et al. (2021). In addition, self-disgust has been shown to predict the severity of ED characteristics and to mediate the links of depressive symptoms and trait anxiety with ED characteristics in both AN and BN groups. To prevent feelings of revulsion arising from confrontation with their own body, AN patients can engage in typical disgust-related behaviors, such as avoiding touching or hiding their body with loose clothing (Nikodijevic et al., 2018). Furthermore, the belief that ingesting food can cause a change in the body, with consequent weight gain and self-disgust, leads to food restriction, which in turn results in cognitive impairment, less cognitive flexibility (Hatch et al., 2010), and higher levels of stress, depression, and anxiety (Vicario & Felmingham, 2018). Bell et al. (2017) have shown a partial dissociation between AN and BN about the experience of self-disgust. They reported a positive correlation between self-disgust and anxiety symptoms, avoidance, and seeking behaviors in the BN group, whereas there was a negative correlation between self-disgust and seeking behaviors in the AN group.

Overall, the literature suggests higher disgust sensitivity and self-disgust in individuals with AN and BN compared to HC

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(for a recent meta-analysis, see Bektas et al., 2022). Differences in disgust recognition have also been found in individuals with AN and BN compared to controls. These abnormalities in disgust processing are related to abnormalities in the activity of the disgust processing neural network.

Hyperphagia

Hyperphagia is characterized by a strong desire to eat and consume foods especially high in caloric content (Heymsfield et al., 2014). In the past decade, only a few studies have investigated disgust emotion processing in hyperphagia and related conditions such as overweight and obesity. A study by Houben and Havermans (2012) examined the relationship between disgust and obesity. The authors found that women with a higher body mass index exhibited less core and contamination disgust. Similar results were found by Vicario and Rafal (2017), not only for disgust sensitivity but also for moral disgust. A more recent study by Palmeira et al. (2019) examined the associations among self-disgust, self-compassion, and psychopathological eating symptoms in overweight and obese individuals. The authors found a link between self-disgust and self-compassion in psychopathological eating, highlighting a key role of self-disgust in the development of eating pathology. The authors also noted the importance of developing a more compassionate attitude toward the self to promote healthy eating behaviors.

On the neural level, an fMRI study (Watkins et al., 2016) investigating the neural correlates of disgust proneness showed lower activation in the right insular region of the obese group compared to the thinner one, along with decreased disgust sensitivity scores associated with reduced insular activation. Interestingly, Cassidy et al. (2012) examined the brain response to visual representations of disgusting food in a group of patients with Prader Willi syndrome (PWS), a genetic, multisystem disorder that involves altered control of appetite with the tendency to overeat. Blanco-Hinojo et al. (2019) observed less extensive engagement of brain regions typically related to visually evoked disgust (i.e., the anterior insula, the frontal operculum, the

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lateral frontal cortex, and visual areas), as well as very low activation in limbic structures (i.e., hypothalamus, amygdala/hippocampus, and periaqueductal gray) in PWS patients compared to HC. According to the authors, the absence of responses in deep limbic structures may explain the tendency to overeat because signals of disgust do not consistently reach the core brain structures that promote appetite control. Overall, the literature suggests abnormal (decreased) core disgust sensitivity and altered activity of the disgust processing network in obesity and related clinical conditions.

Discussion

In this review, we examined the relevant literature to address the role of disgust as a transdiagnostic index for mental disorders. Overall, our work confirms the existence of altered disgust processing in the examined clinical disorders. The main results are summarized in Table 1. In the following paragraphs, we will discuss the rationale to support the hypothesis of disgust as a transdiagnostic index—that is, a cognitive marker—for mental illness.

Disgust as a transdiagnostic index for mental illness: Insight from neuroimaging evidence

The insular cortex and the basal ganglia are the key structures in the functional neurocircuitry of disgust in healthy individuals as well as in those with pathological conditions (Chapman & Anderson, 2012; Holtmann et al., 2020; Vicario, Rafal, Martino, et al., 2017). The literature on mental disorders that we have examined indicates that the reported deficits in core, social, and self-disgust might be related to dysfunctions in insular cortex and cortico-basal ganglia circuitries (involving sensory, interoceptive, and reward systems). This is confirmed by research with HD patients, which documents an increment of insular activity along with disease severity (Dogan et al., 2014; Novak et al., 2012) or increased activity in the middle frontal gyrus and the putamen, when the pattern of activation to disgust stimuli was normalized by intranasal oxytocin administration (Labuschagne

Table 1. An overview of disgust processing in the examined clinical and personality disorders

	HD	PD	bvFTD	svPPA	SP	PTSD	OCD	BPD	Depression	AN/BN	Hyperphagia
Core disgust	↓	↓	↓	↓	↑	↑	↑	—	—	↑	↓
Social disgust	↑↓	↑↓	↓	↓	↑	↑	—	↓	—	↓	↓
Self-disgust	—	↓	—	—	—	↑	↑	↑	↑	↑	↑
Disgust propensity	—	↓	—	—	—	—	↑	—	—	—	—
Disgust sensitivity	—	↑	—	—	↑	↑	—	↑↓	—	↑	↓

Note: ↑ = an increased rating in clinical population compared to controls; ↓ = a decreased rating in clinical population compared to controls; ↑↓ = controversial results; — = no evidence found; HD = Huntington's disease; PD = Parkinson's disease; bvFTD = behavioral variant of frontotemporal dementia; svPPA = semantic variant of primary progressive aphasia; SP = specific phobias; PTSD = posttraumatic stress disorder; OCD = obsessive-compulsive disorder; BPD = borderline personality disorder; AN = anorexia nervosa; BN = bulimia nervosa.

et al., 2018). PD patients expressed stronger recruitment of somatosensory regions compared to controls (Wabnegger et al., 2015), although no differences between groups were found in disgust proneness and trait anxiety scores; in addition, similar activation of the amygdala, OFC, DLPFC, and VLPFC was observed in both groups (Schienle, Ille, & Wabnegger, 2015). Nevertheless, the insula plays a role in PD, as suggested by a meta-analysis (Criaud et al., 2016). Decreased gray matter volume in the anterior insula associated with reduced core and social disgust was also observed in patients with bvFTD and svPPA (Verstaen et al., 2016; Woolley et al., 2015). Hyperactivity of the insular cortex may explain the higher disgust sensitivity and propensity in individuals with OCD (Berlin et al., 2017; Mataix-Cols et al., 2004; Viol et al., 2019a, 2019b), specific phobias (Brinkmann et al., 2017), PTSD (Mazza et al., 2013), depression (Surguladze et al., 2010), BPD (for a review on fMRI studies see Ruocco et al., 2013), AN, and BN (Knyahnytska et al., 2019; Setsu et al., 2017), while reduced activity in the insula is associated with decreased disgusted sensitivity, as seen in obese individuals (Watkins et al., 2016). Hyperactivation of cortico-basal ganglia circuits, including frontal regions, OFC, and ACC, was also found during the processing of abnormal disgust emotion in the analyzed disorder, and such increases were linked to higher trait disgust proneness and self-disgust in individuals with OCD (Viol et al., 2019b), specific phobias (Brinkmann et al., 2017), PTSD (Vidotto et al., 2014), AN, and BN (Frank et al., 2013, 2016; Titova et al., 2013), while atrophy in these brain regions, in particular OFC, were found in PD patients (Ille et al., 2015; Lee et al., 2014) and in individuals with anosmia and hyposmia (Ille, Wolf, et al., 2016). Concerning the ACC, Woolley et al. (2015) found that it is the downstream of sensory representations that are generated in the insula so that behavioral responses to disgusting stimuli are likely predicated upon the individuals' viscerosceptive experience of the stimuli. According to these authors, dysfunctions in this region may decrease the individual's ability to stimulate the visceral responses associated with disgust stimuli. These findings provide a neural rationale for the hypothesis that disgust can serve as a transdiagnostic index of mental illness.

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Insight from behavioral studies*

Our examination suggests that it is possible to distinguish disorders in which disgust sensitivity and propensity are deficient with respect to the dimensions of core, social, and self-disgust. In the case of core disgust, we found a general reduction in sensitivity or propensity to disgust in individuals with neurodegenerative diseases compared to controls (Ille et al., 2015; Ricciardi et al., 2015; Wabnegger et al., 2015), while core disgust increased in those with psychiatric disorders, especially those related to anxiety disorders (Olatunji, Armstrong, & Elwood, 2017; Stasik-O'Brien et al., 2021; Winder et al., 2021). In EDs core disgust sensitivity was higher in AN and BN while in obesity it was reduced (for a meta-analysis, see Bektas et al., 2022); these differences in disgust sensitivity in EDs may contribute to explain the predisposition to overeat or avoid eating (Houben & Havermans, 2012).

Social disgust was abnormal in most of the examined populations, including those with HD, bvFTD, svPPA, Sp, and BPS, and in all those with eating disorders. Disgust sensitivity may be relevant in the classification of specific phobias (Winder et al., 2021); for example, BII is associated with disgust sensitivity and is often accompanied by vasovagal syncope during exposure to specific objects or situations related to the phobia. The study by Ille, Wabnegger, et al. (2016) showed that PD patients have a greater disgust sensitivity on the SADS and more problems in managing feelings of anger and disgust than controls. Increased disgust sensitivity in PD was associated with less healthy functioning in daily life and less disgust about poor hygiene and spoiled food.

Regarding self-disgust, there is general agreement that this is predominant in ED (Ammann et al., 2018; Egolf et al., 2018; Hay & Katsikitis, 2014; Ille et al., 2014), especially in AN and BN (Bektas et al., 2022). Self-disgust can also predict the severity of ED characteristics and mediate the links of depressive symptoms and trait anxiety with ED characteristics in both groups (Kot et al., 2021). Self-disgust can be associated with avoidance-based strategies toward certain parts of self that do not align with internalized social norms (Rinker, 2019).

Moreover, self-disgust has been shown to be involved in depression (Olatunji et al., 2012; Overton et al., 2008) and can predict depressive symptoms. The association between self-disgust and depression is stronger for individuals with higher scores on expressive suppression. Maladaptive emotion regulation strategies reinforce the association between self-disgust and depressive symptoms (Ypsilanti et al., 2019). In PTSD, the inability to feel positive emotions along with persisting memories of the traumatic event can lead to self-disgust, increasing risk of suicide (Brake et al., 2017). Patients with BPD seem to experience marked self-disgust and show an inability to identify facial expressions of disgust in others (Schienle et al., 2014); this can explain the difficulty in building stable relationships. The disgust propensity trait could be considered a risk factor for OCD (Stasik-O'Brien et al., 2021). Furthermore, Athey et al. (2015) show that changes in disgust propensity predicted changes in wash-compulsion symptoms. Olatunji et al. (2015) further assessed self-disgust and its subsequent impact on the development of obsessive-compulsive symptoms, showing a correlation between self-disgust and symptoms of OCD, general anxiety, and bulimia. In conclusion, similar to social disgust, self-disgust is compromised in the majority of analyzed disorders. While disgust propensity is a specific risk factor for OCD, disgust sensitivity plays a key role in the development of specific phobia, eating disorders, and some neurogenerative disorders.

Limitation and conclusions

This narrative synthesis provides preliminary evidence for the relevance of disgust as a transdiagnostic index for mental illness. However, several limitations related to the literature we reviewed should be mentioned. First, we did not evaluate the specificity of the different disgust domains across the clinical disorders we examined. Therefore, our suggestion arises from a general picture of neurocognitive impairment for disgust processing that could involve one or more domains, depending on the examined clinical population and the available literature. In addition, there was low consistency and high heterogeneity of tasks, procedures, study designs, and sample sizes among the clinical populations we examined. Moreover, the clinical

populations we investigated had heterogeneous patterns of pharmacological treatment, which might have contributed to inconsistent results from the same clinical condition (e.g., in the literature on PD patients). A further limitation involving some of the examined studies is the absence of a separate analysis for social disgust, because the data associated with recognition of disgust expressions were merged with those associated with other negative expressions. This may have obscured evidence of a more severe deficit in social disgust than that reported for other negative emotions. Finally, we did not consider other clinical categories that can be linked with disgust processing deficits, such as schizophrenia (e.g., Lindner et al., 2014) and irritable bowel syndrome (IBS; Formica et al., 2022). The latter study, which refers to a clinical condition closely related to abnormal visceral processing (Fournier et al., 2020), prompts suggestive insights into the key relevance of interoceptive deficits as a physiological landmark for explaining the disgust processing deficits of the clinical populations we examined. Disgust was found to predict symptom severity only in IBS, while no relationship was found to another psychosomatic condition (i.e., chronic stress urticaria). This suggests that disgust may play the role of a transdiagnostic index only for the clinical conditions involving alteration of the neural network commonly activated in disgust and interoception processing.

In conclusion, our review provides evidence for the hypothesis that disgust might serve as a transdiagnostic index for mental illness. Our results also suggest that structural and/or functional alteration of the insula and the respective neural network might be responsible for the abnormal disgust processing reported in the examined clinical populations. Further systematic approaches (i.e., systematic review and/or meta-analyses) are needed to explore to what extent the hypothesis of disgust as a transdiagnostic index for mental illness can be supported with respect to the several facets of this emotion.

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