35 Disordered Social Cognition

Alexithymia and Interoception

REBECCA BREWER AND GEOFFREY BIRD

35.1 INTRODUCTION

Alexithymia is a condition characterized by difficulties identifying and describing one's own emotional states (Nemiah, Freyberger, & Sifneos, 1976). Individuals with alexithymia are often aware that they are experiencing an emotion, but struggle to determine whether it is fear, excitement, or anger, for example. Alexithymia is therefore associated with difficulties describing how one would feel in particular emotional scenarios (Lane et al., 1990), as well as with difficulties regulating one's emotions (Stasiewicz et al., 2012; Venta, Hart, & Sharp, 2013). This chapter details the behavioral and neurological characteristics of alexithymia, its etiology (including whether it may be evolutionarily adaptive), and its role in emotional impairment across clinical populations. The relationship between alexithymia and interoception (the ability to perceive and recognize the internal state of one's body) is also discussed, alongside evidence that alexithymia may represent a general deficit of interoception. Finally, the theory is proposed that poor interoception (and therefore alexithymia) is a candidate for the "P" factor of susceptibility to psychopathology.

35.2 BEHAVIORAL AND NEUROLOGICAL CHARACTERISTICS OF ALEXITHYMIA

Although many see alexithymia as primarily reflecting cognitive impairment in the representation of emotions (e.g., Luminet et al., 2006; Suslow & Junghanns, 2002), others have argued for the central role of affective difficulties, characterized by decreased ability to experience emotions (e.g., Bermond, 1997; Vorst & Bermond, 2001). A number of subtypes of alexithymia have been proposed, defined in terms of affective and cognitive abilities. The term "Type I" alexithymia has been used to describe individuals who are impaired in both the affective and cognitive domains, whereas "Type II" defines those who suffer impairment only in the cognitive domain (Bermond, 1997). More recently, Moorman and colleagues also identified "Type III" alexithymics, with affective but not cognitive impairment – "lexithymics," with high performance in the affective and cognitive domains, and "Modals," with average performance in both domains (Moormann et al., 2008). Impairment in the affective and cognitive domains has unsurprisingly been associated with different underlying neural atypicalities (Goerlich-Dobre et al., 2015). The majority of research focuses on the cognitive dimension and typically assesses alexithymia using the self-report Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994), which has become the gold-standard tool for the assessment of alexithymia. Research into affective difficulties is becoming more common, however, with many researchers aiming to separate the cognitive and affective dimensions using the Bermond–Vorst Alexithymia Questionnaire (BVAQ; Vorst & Bermond, 2001; although see Bagby et al., 2009).

Interestingly, the impairments associated with alexithymia extend beyond difficulties recognizing and interpreting emotions in oneself. A plethora of evidence suggests that alexithymia is also associated with deficits in recognizing others' emotional states, such as from facial (Grynberg et al., 2012; Jessimer & Markham, 1997; McDonald & Prkachin, 1990; Parker, Taylor, & Bagby, 1993; Parker, Prkachin, & Prkachin, 2005; Prkachin, Casey, & Prkachin, 2009; Swart, Kortekaas, & Aleman, 2009), vocal (Delle-Vigne et al., 2014; Goerlich et al., 2011; Heaton et al., 2012), and body posture cues (Borhani et al., 2016). If one does not possess clear representations of individual emotions in the self and the situations and behaviors usually associated with these emotions, it follows that one would also have difficulty identifying how another would feel in the same situations or from relevant behavioral cues. Notably, emotion recognition difficulties appear to be general rather than specific; despite many researchers suggesting that recognition of negative emotions may be especially impaired, numerous studies have also found diminished recognition of positive emotions in alexithymia (Jessimer & Markham, 1997; Lane et al., 2000; Parker, Taylor, & Bagby, 1993).

Beyond emotion recognition difficulties, those with alexithymia demonstrate difficulties empathizing with others. Empathy has been defined in multiple ways, but

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is generally characterized by the sharing of another's emotional state, as well as attributing the emotion to the other and representing one's own emotion as being caused by that of the other (Bird & Viding, 2014; Singer & Lamm, 2009). Empathy therefore involves emotion contagion (affect sharing) and self–other distinction (representing the state of the self and the other separately). Self-reported empathy tends to decrease as alexithymia severity increases (Gleichgerrcht, Tomashitis, & Sinay, 2015; Grynberg et al., 2010; Guttman & Laporte, 2002; Neumann et al., 2014), although personal distress in response to others' negative states (likely reliant on the emotion contagion facet) increases (e.g., Guttman & Laporte, 2002; Moriguchi et al., 2007). Individuals with alexithymia also exhibit atypical responses in brain regions associated with empathy, such as the anterior insula (AI), when viewing others in pain (Moriguchi et al., 2007; Silani et al., 2008), although these findings are complicated by the fact that the pain stimuli used were likely to induce disgust in the observer, an emotion also associated with AI activity. Those with alexithymia also rate others' pain as less intense than do those with low alexithymia levels (Moriguchi et al., 2007). While the definition of empathy differs across studies, a consistent feature is the sharing of the other's affect. It is therefore unsurprising that alexithymia is associated with impaired empathy; if one cannot differentiate between one's own affective states, it is difficult both to associate those states with cues of affective states in others and to share the others' states (Bird & Viding, 2014). In line with difficulties empathizing and inferring emotions in oneself, alexithymia also seems to impact on one's ability to make typical moral judgments, a process shown to rely typically on emotion understanding. Individuals with alexithymia are more likely to make utilitarian moral decisions than those without alexithymia (Patil & Silani, 2014b; although see Gleichgerrcht et al., 2015) and are more likely to judge accidentally harming others to be acceptable (Patil & Silani, 2014a). Difficulties identifying emotions in oneself therefore appear to lead to further difficulties in the socio-affective domain.

At the neurological level, alexithymia has consistently been associated with the atypical structure and function of a number of brain regions. In particular, the anterior cingulate cortex (ACC) and AI, which play crucial roles in the processing of one's own emotions (e.g., Bush, Luu, & Posner, 2000; Etkin, Egner, & Kalisch, 2011; Kober et al., 2008; Lindquist et al., 2012; Phan et al., 2002), have been implicated. Much evidence suggests that alexithymia is associated with decreased gray matter volume of the ACC (Borsci et al., 2009; Grabe et al., 2014; Ihme et al., 2013; Koven et al., 2011; Paradiso et al., 2008; Sturm & Levenson, 2011; for a review, see van der Velde et al., 2013). Although one study found the opposite pattern of results (Gündel et al., 2004), the structural markers used in this study differed from others, and only individuals with low degrees of alexithymia were studied. ACC function during affective tasks has also been consistently associated with alexithymia severity (Berthoz et al., 2002; Deng, Ma, & Tang, 2013; Frewen et al., 2008b; Heinzel et al., 2010; Jongen et al., 2014; Kano & Fukudo, 2013; Moriguchi & Komaki, 2013; Moriguchi et al., 2007), although the direction of the effect has varied across studies (Pouga et al., 2010; van der Velde et al., 2013). Relatedly, investigation of neurotransmitters in the ACC indicated atypical concentrations of GABA in individuals with alexithymia (Ernst et al., 2014).

Where the insula is concerned, studies have produced mixed findings concerning structural atypicalities; although the majority of studies observed decreased insula volume in individuals with alexithymia (Borsci et al., 2009; Grabe et al., 2014; Ihme et al., 2013), increased insula volume has also been reported (Goerlich-Dobre et al., 2014a; Zhang et al., 2011). Alexithymia also appears to be associated with atypical insula function during affective processing (Deng et al., 2013; Frewen et al., 2006; Heinzel et al., 2010; Kano et al., 2003, 2007; Moriguchi et al., 2007; Reker et al., 2010; Silani et al., 2008), although the direction of the effect has varied between studies (Craig, 2009). This atypical function may be explained in part by atypical glutamate neurotransmitter concentrations in the insula (Ernst et al., 2014). Besides the ACC and AI, alexithymia has been found to be associated with amygdala structure (Grabe et al., 2014; Ihme et al., 2013) and function (Goerlich-Dobre et al., 2014b; Kugel et al., 2008; Miyake et al., 2012; Reker et al., 2010; Zotev et al., 2011), as well as atypicalities in the orbitofrontal cortex (OFC), fusiform gyrus, dorsomedial prefrontal cortex, precuneus, and limbic, paralimbic, and premotor areas (for a review, see van der Velde et al., 2013).

35.3 ETIOLOGY AND DEVELOPMENT OF ALEXITHYMIA

Alexithymia is likely to be a neurodevelopmental condition, meaning that it occurs in the absence of brain damage (Lane et al., 2015), but can also develop following neurological trauma (Henry et al., 2006; Hogeveen et al., 2016; Neumann et al., 2014; Williams & Wood, 2010). The etiology of developmental alexithymia is not, thus far, well understood, but both genetic and environmental factors have been identified as being involved in its development. Evidence from twin studies suggests that alexithymia is heritable, with genetic factors contributing to approximately 33 percent of the variation in alexithymia severity (Heiberg & Heiberg, 1978; Jørgensen et al., 2007; Picardi et al., 2011; Valera & Berenbaum, 2001). Recent research has also identified candidate genes for the condition (Ham et al., 2005; Mezzavilla et al., 2015; Walter et al., 2011). Of particular interest are findings implicating dopaminergic function in cortical areas known to be atypical in alexithymia, with some genetic variants implicating decreased dopaminergic function in ACC and OFC activation (Ham et al., 2005; Walter et al., 2011). Beyond genetic influences, some

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research has investigated the environmental causes of alexithymia. Alexithymia has been associated, for example, with decreased perceived parental care and increased perceived overprotectiveness (Fukunishi et al., 1997; Kooiman et al., 2004), as well as child abuse (Berenbaum, 1996), rural upbringing, and large family size (Joukamaa et al., 2003). These findings are confounded, however, by the subjective nature of self-reported parenting styles (which may be affected by alexithymia), as well as the genetic relationship between parents and participants; parenting style (as well as decisions over family size and location of upbringing) may be associated with alexithymia traits in parents, which are inherited by participants. Little reliable evidence therefore exists concerning the precise role of environmental factors in alexithymia, but heritability estimates do suggest that environmental factors contribute to its development.

From an evolutionary perspective, it is clear that the ability to form emotional concepts and identify one's own and others' emotions is adaptive. It is necessary to identify whether another individual is feeling angry, for example, in order to predict and avoid aggressive behavior. Identifying fear, on the other hand, in oneself and others allows one to evade a threatening situation. The fact that alexithymia has a genetic component suggests that impairment in this ability may be adaptive in some situations, or at least that alleles associated with susceptibility to alexithymia may offer an evolutionary advantage. One way in which alexithymia may be adaptive is following trauma, or when one is experiencing a highly aversive emotion or period of negative emotion, such as during depression. A decreased tendency to recognize and attend to negative emotions may lead to less interference of one's emotional state with one's daily functioning. Indeed, it has been suggested that traumatic life events such as childhood maltreatment can lead to alexithymia tendencies (Berenbaum, 1996) and that alexithymia is highly associated with depression (Honkalampi et al., 2001). This is also in line with the proposal that, when environmental inputs are heterogeneous (as is the case for humans), conditional adaptation (developmental plasticity) occurs such that one's phenotype and behavioral strategies develop to reflect one's context, optimizing fitness in that particular environment (Del Giudice, 2014; Del Giudice, Ellis, & Shirtcliff, 2011). These authors propose early stress in particular, such as from childhood maltreatment, violence, and unpredictability, as a determinant of later adaptive behavioral strategies.

A second possibility is that a reduced ability to recognize negative emotions (e.g., signals of fear) in alexithymia allows one to take risks that others are not willing to, which may benefit the individual or the community as a whole. Indeed, alexithymia has been found to be associated with increased risk-taking (Barlow et al., 2015; Hahn, Simons, & Simons, 2016; Shishido, Gaher, & Simons, 2013), and while risky behavior increases the likelihood of harm to the individual, it can also lead to relatively large gains. It is possible that the survival advantages associated with alexithymia outweigh the disadvantages, as has been proposed for some psychological disorders, such as schizophrenia (for a review, see McClenon, 2011). It is also worth noting that alexithymia is associated with increased sexual risk-taking (including more sexual partners and intercourse without contraception), potentially leading to increased fertility in this population, causing the alexithymia phenotype to continue to be inherited.

Alternatively, the emergence of alexithymia may simply be due to polygenic mutation, as has been suggested to account for multiple psychological disorders (Keller & Miller, 2006). If this is the case, it is likely that the alexithymia phenotype simply emerges due to multiple combinations of mutations, rather than being specifically selected. Keller and Miller argue that, as over half of the genetic mutations carried by humans affect the brain, atypical neural functioning associated with psychological disorders may simply be attributable to a high number of mutations, which individually do not exert a large effect on behavior. Whether or not alexithymia is selected due to adaptive features therefore remains to be determined.

Alexithymia severity varies across the lifespan, with particularly high levels in young adolescence (Säkkinen et al., 2007). Alexithymia appears to be relatively stable in later adolescence (Karukivi et al., 2014), with severity tending to increase throughout adulthood (Mattila et al., 2006; for a review, see Murphy, Brewer, & Bird, 2017). Difficulties with the measurement of alexithymia across the lifespan, especially during childhood (Griffin, Lombardo, & Auyeung, 2016), however, can make it difficult to draw firm conclusions concerning the developmental trajectory of alexithymia. Future work should prioritize investigation into alexithymia across the lifespan and its relationship with associated difficulties; it is possible that alexithymia contributes to the development of varying forms of psychopathology at different stages of development (Murphy et al., 2017).

35.4 THE IMPACT OF ALEXITHYMIA ON SOCIAL AND EMOTIONAL ABILITIES ACROSS DISORDERS

Although alexithymia is an independent condition, it frequently co-occurs with multiple psychiatric disorders, such as autism spectrum disorder (ASD) (Hill, Berthoz, & Frith, 2004), feeding and eating disorders (EDs) (Cochrane et al., 1993), schizophrenia (Heshmati et al., 2010), depression (Honkalampi et al., 2001), post-traumatic stress disorder (Frewen et al., 2008a), and substance abuse (Mann et al., 1995), as well as neurological conditions such as Parkinson's disease (Assogna et al., 2012), frontotemporal dementia (Sturm & Levenson, 2011) and multiple sclerosis (Chahraoui et al., 2014; Prochnow et al., 2011) and physical illness such as diabetes (Abramson et al., 1991). Crucially, alexithymia is neither necessary nor sufficient for a diagnosis of these disorders and is not universal among individuals in these

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populations. Approximately 50 percent of individuals with ASD (Berthoz & Hill, 2005; Berthoz et al., 2013; Hill et al., 2004) and 60 percent of the ED population (Cochrane et al., 1993; for a review, see Nowakowski, McFarlane, & Cassin, 2013), for example, also have alexithymia, compared to approximately 10 percent of the typical population (Honkalampi et al., 2000; Kokkonen et al., 2001; Salminen, Saarija, & Rela, 1999). Furthermore, despite the association between alexithymia and depression (Honkalampi et al., 2000), evidence suggests that these are distinct constructs (Marchesi, Brusamonti, & Maggini, 2000; Parker, Bagby, & Taylor, 1991)

While it has often been suggested that alexithymia is a risk factor for the development of psychiatric disorders (e.g., De Beradis et al., 2014; Parker et al., 2005; van der Velde et al., 2015), the nature of the relationship is not well understood at present (Grabe, Spitzer, & Freyberger, 2004; Honkalampi et al., 2010; Nowakowski et al., 2013). Interestingly, a number of the behavioral difficulties associated with alexithymia, such as emotion recognition, are also commonly observed in clinical populations, such as ASD, ED, and schizophrenia, although the literature has been relatively inconsistent (Edwards, Jackson, & Pattison, 2002; Harms, Martin, & Wallace, 2010; Oldershaw et al., 2011). Similarly, the core neurological regions implicated in alexithymia, such as the ACC and insula, have often been reported to be structurally and functionally atypical in clinical groups, though findings are again variable (Anagnostou & Taylor, 2011; Bauman & Kemper, 2005; Franklin et al., 2002; Kaye, 2008; Mundy, 2003; Nagai, Kishi, & Kato, 2007; Rogers et al., 2009).

Recent evidence suggests that alexithymia may be responsible for a number of the emotional impairments often thought to be associated with clinical disorders. In those with ASD and EDs, for example, co-occurring alexithymia (rather than ASD or EDs per se) appears to explain difficulties recognizing others' facial expressions (Brewer et al., 2015b; Cook et al., 2013) and atypical empathy (Bird et al., 2010; Brewer et al., 2019). These findings suggest that individuals with ASD or an ED are only impaired in the emotional domain if they also have co-occurring alexithymia and that inconsistent previous findings may be attributable to varying levels of alexithymia in clinical samples. Alexithymia also appears to influence behaviors that are not explicitly emotional but rely on emotional processes, such as judgments of personality traits from novel faces (Brewer et al., 2015a). As alexithymia co-occurs with multiple conditions, it is likely that a range of emotional difficulties, including emotion recognition and empathy, are explained by alexithymia, rather than disorder diagnosis or symptom severity, across a range of clinical populations (Brewer et al., 2015c). Further research is required, however, into the impact of alexithymia and disorder presence and severity on more complex emotional processes, such as moral reasoning. Evidence suggests, for example, that those with alexithymia in the typical population tend to make atypical moral judgments, while alexithymia is not associated with atypical moral judgments in individuals with ASD (Brewer et al., 2015d). Moral decision-making can involve both emotional and rational processes (Cushman, Young, & Greene, 2010; Ugazio, Lamm, & Singer, 2012), meaning that, as well as emotional abilities themselves, differences in the extent to which one relies on these two routes are likely to influence outcomes. The relationship between alexithymia and social abilities that rely on a combination of emotional and nonemotional processes therefore deserves further attention.

35.5 ALEXITHYMIA AS A GENERAL DEFICIT OF INTEROCEPTION

While the AI and ACC are clearly involved in emotion processing and alexithymia, these brain regions are also referred to as the "interoceptive cortex" due to their well-documented involvement in interoception (perception of the internal states of the body; Craig, 2002, 2003a, 2009; Critchley & Harrison, 2013; Garfinkel & Critchley, 2013). While interoception was initially defined as the perception of the condition of the viscera only (Sherrington, 1900), current definitions include the identification of numerous bodily states, such as heart rate, respiration, pain, temperature, itch, hunger, satiety, and fatigue (Craig, 2002, 2003a, 2009). The interoceptive cortex is centrally involved in the perception of these states and contributes to the human ability to subjectively represent one's own interoceptive condition, and therefore the "feeling self," via a spinothalamocortical pathway (Craig, 2002, 2003a). Recently, interoception has been divided into three distinct (though likely related) processes (Garfinkel & Critchley, 2013). Under this model, interoceptive sensitivity refers to one's ability to accurately perceive objective interoceptive states. Interoceptive sensibility, on the other hand, describes the propensity to introspect on internal states and subjective beliefs about one's own interoceptive states. Finally, interoceptive awareness refers to the accuracy of one's interoceptive sensitivity (one's metacognitive ability relating to interoception).

Studies of patients with insula lesions have generally observed interoceptive difficulties (for a review, see Ibañez, Gleichgerrcht, & Manes, 2010), and stimulation of the insula in humans has been found to elicit unpleasant sensations (Krolak-Salmon et al., 2003), gastrointestinal motility, abdominal sensations, and nausea (Penfield & Faulk, 1955), while insula inhibition has been associated with reduced awareness of one's heart beat and respiratory effort (Pollatos et al., 2016; but see Coll et al., 2017). While much evidence therefore suggests that the insula is integral to consciously representing feeling states, it should be noted that typical experience of interoceptive states, such as itch, tickle, pain, and temperature, as well as emotions, has been observed following bilateral insula lesion (Damasio, Damasio, & Tranel, 2013). Damasio and colleagues therefore emphasize the role of the brain stem and the

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thalamic, hypothalamic, and somatosensory regions (alongside interoceptive cortices) in representing these states. Similarly, ACC and AI lesions do not appear to impair the detection of heartbeats (Khalsa et al., 2009a), although it is possible that exteroceptive signals (e.g., from the chest wall) may contribute to this ability (Khalsa et al., 2009a). Overall, it is undeniable that interoceptive cortical areas, such as the AI and ACC, are involved in interoceptive awareness, but subcortical and somatosensory regions also contribute to the representation of internal states.

As alexithymia is associated with AI and ACC atypicalities, and as these brain regions are central to interoception, it is possible that alexithymia is associated with deficits of non-affective as well as affective interoception. Indeed, alexithymia is often described as involving difficulties differentiating one's emotions from other bodily sensations (Nemiah et al., 1976; Parker et al., 2003). It is therefore possible that alexithymia is better characterized as a general failure of interoception, rather than of affective interoception only (Brewer et al., 2015c; Brewer, Cook, & Bird, 2016). Some evidence does indeed suggest a negative relationship between alexithymia and identification of interoceptive states. Alexithymia appears to be associated, for example, with less accurate perception of one's heart rate (Herbert, Herbert, & Pollatos, 2011; Shah et al., 2016), as well as delayed responses to acute myocardial infarction (Carta et al., 2013). Erratic consumption of alcohol (Lyvers et al., 2012) and other substances (de Haan et al., 2014; Taylor, Parker, & Bagby, 1990) is also common in alexithymia, likely due to decreased awareness of the effects of these substances on the state of the body. Individuals with alexithymia also struggle to estimate their objective levels of arousal (Gaigg, Maurice, & Bird, 2018). Recent evidence suggests that increased alexithymia is associated with self-reported difficulties distinguishing between a range of nonemotional internal states, as well as differentiating these states from emotions (Brewer et al., 2016; Longarzo et al., 2015). While further investigation is needed into whether interoception is a unitary construct (Garfinkel et al., 2016a), it appears that alexithymia may be associated with numerous interoceptive difficulties beyond the emotional domain and may therefore be conceptualized as a general deficit of interoception, rather than one specific to emotion (Brewer et al., 2015c, 2016). Indeed, novel tests assessing the use of interoceptive information when judging one's speed of respiration and interoceptive accuracy in the domains of muscular effort and taste also reveal an association with levels of alexithymia (Murphy, Catmur, & Bird, 2018).

35.6 ALEXITHYMIA (POOR INTEROCEPTION) AS THE "P" FACTOR OF PSYCHOLOGICAL DISORDERS

As previous research suggests that alexithymia explains emotional impairments across clinical populations, alexithymia may also account for interoceptive impairments across the multiple disorder populations with which it co-occurs. Where ASD is concerned, for example, sensory atypicalities, such as hypersensitivity to touch, are often exhibited (Leekam et al., 2007; Tomchek & Dunn, 2007). As itch and slow, affective touch can be considered to be interoceptive states, alexithymia (and impaired interoception) may account for atypical sensory profiles in those with ASD. Gastrointestinal difficulties are also common in those with ASD (Torrente et al., 2002; Wakefield et al., 2000, 2005; White, 2003; Williams et al., 2011) and may be attributable to alexithymia due to the well-documented role of interoception in gastric sensitivity (Stephan et al., 2003; Vandenbergh et al., 2005) and atypical visceral sensitivity in alexithymia (Kano et al., 2007). Evidence also suggests atypical neural responses (implicating the ACC and insula in particular) during reward processing in those with ASD (Bellebaum, Brodmann, & Thoma, 2014; Cascio et al., 2012; Dichter et al., 2012; Kohls et al., 2013; Larson et al., 2011; Schmitz et al., 2008). Thus, difficulties in processing reward may also rely on interoceptive abilities (and therefore be affected by alexithymia), rather than ASD itself. Finally, although evidence concerning imitation in those with ASD has varied depending on task demands (Cook & Bird, 2012; Leighton et al., 2010; Press, Richardson, & Bird, 2010), it is possible that alexithymia can account for any differences between those with and without ASD due to the role of interoception in the ability to represent the self (Craig, 2009; Critchley et al., 2004; Quattrocki & Friston, 2014; Seth, Suzuki, & Critchley, 2011), and therefore likely the ability to distinguish between the self and other. Indeed, both alexithymia and interoceptive sensitivity to heartbeats have been found to predict control of the tendency to automatically imitate others' actions (Ainley, Brass, & Tsakiris, 2014; Sowden et al., 2016). It should be noted that the impact of interoceptive difficulties on the self–other distinction is also particularly relevant for schizophrenia, which is characterized by difficulties distinguishing the self from others (Ebisch et al., 2013; Jardri et al., 2011; Sass & Parnas, 2003).

Interoception and the interoceptive cortex, such as the AI and ACC, are also thought to play a role in processes such as the perception of time (Bushara, Grafman, & Hallett, 2001; Livesey, Wall, & Smith, 2007; Magnani et al., 2014), reward processing (Furl & Averbeck, 2011; Tanaka et al., 2004; Wittmann et al., 2010), cognitive control (Brass & Haggard, 2007; Cai et al., 2014; Cole & Schneider, 2007; Dosenbach et al., 2007; Eichele et al., 2008; Ghahremani, Rastogi, & Lam, 2015), metacognition (Fleming & Dolan, 2012), attention (Chen et al., 2015; Mason et al., 2007; Weissman et al., 2006), decision-making (Botvinick, 2007; Coricelli et al., 2005; Critchley, Mathias, & Dolan, 2001; Dunn et al., 2010), and cravings (relating to addiction; Gray & Critchley, 2007; Naqvi & Bechara, 2009, 2010; Naqvi et al., 2007). These abilities relate to Damasio's somatic marker hypothesis, which posits that interoceptive signals of arousal (including

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emotional responses) can influence cognition and behavior, particularly decision-making, through learned associations between behaviors and outcomes (Damasio, Tranel, & Damasio, 1991). It is likely that, if alexithymia is characterized by poor interoception, those with alexithymia may exhibit impairments in all of these domains.

Beyond ASD, a similar interoceptive explanation may also hold for impairments observed in other disorders. Difficulties with reward processing and decision-making, for example, are characteristic of those with depression (Davidson et al., 2002; Forbes & Dahl, 2012), substance abuse and other addiction disorders (Bechara & Damasio, 2002; Bechara, Dolan, & Hindes, 2002; Koob & Le Moal, 2001; Noble, 2000; Reuter et al., 2005; Schoenbaum, Roesch, & Stalnaker, 2006), EDs (Broft et al., 2011; Cowdrey et al., 2011; Keating et al., 2012; Wagner et al., 2007), and schizophrenia (Abi-Dargham et al., 2000; Heerey, Bell-Warren, & Gold, 2008; Simon et al., 2010), and may all be attributable to alexithymia (and therefore interoceptive impairment) rather than to disorder presence or severity per se. Indeed, alexithymia has been associated with poor decisions on gambling tasks in those with gambling disorder (Aïte et al., 2014), in line with the widely accepted role of interoception in addiction (Naqvi & Bechara, 2010). Similarly, atypical decision-making has been associated with alexithymia in the typical population (Shah, Catmur, & Bird, 2016). Alexithymia may also explain the interoceptive deficits characteristic of EDs, such as difficulties detecting signals of hunger and satiety (Fassino et al., 2004; Lilenfeld et al., 2006), as well as perceiving one's own heartbeat (Pollatos et al., 2008). Alexithymia may therefore predict these impairments across the broad range of disorders it co-occurs with and possibly contribute to the development of these disorders in some individuals. Determining whether the role of alexithymia in interoceptive impairment is comparable across populations should be a priority for future work.

In line with evidence for interoceptive difficulties across a number of disorders, we have recently proposed that alexithymia (and therefore poor interoception) encompasses the "P" factor of psychopathology (Brewer et al., 2016). The "P" factor has been described as a unitary factor that can account for general susceptibility to psychopathology, with symptom severity being associated with the degree of neural atypicality (Caspi et al., 2015; Lahey et al., 2012). The P factor model currently includes 11 disorder symptoms, comprising depression, anxiety, addiction, conduct disorder, obsessive–compulsive disorder, mania, and schizophrenia, the majority of which have been associated with interoceptive impairment (for a review, see Khalsa & Lapidus, 2016). Atypical interoception has been associated, for example, with depression (Harshaw, 2015), addiction (Naqvi & Bechara, 2010; Verdejo-Garcia, Clark, & Dunn, 2012), schizophrenia (Ardizzi et al., 2016), OCD (Lazarov et al., 2010; Stern, 2014), and anxiety (associated with atypically high rather than low interoceptive abilities; Domschke et al., 2010; Ehlers & Breuer, 1992; Paulus & Stein, 2006), as well as with disorders that are not currently included, such as ASD (Garfinkel et al., 2016b) and EDs (Pollatos et al., 2008). Interoceptive impairment appears, therefore, to be a likely candidate for increased disorder susceptibility. In fact, as interoceptive awareness is required for physical as well as mental health, an extension of this hypothesis may suggest that alexithymia accounts for susceptibility to health issues in general. Indeed, elevated levels of alexithymia have been observed in diabetes, which is characterized by atypicalities in the implicit interoception required for homeostatic maintenance of blood glucose levels. Clearly, this hypothesis requires substantial testing, but existing evidence points to a role for interoception (and alexithymia) in at least a broad range of symptoms that are observed in multiple conditions (for a full description of this theory, see Murphy et al., 2017). As it is possible to improve interoceptive abilities through training (Schandry & Weitkunat, 1990), investigation of this theory may provide opportunities for therapeutic interventions that may be applied across psychological and medical disorders.

35.7 CONCLUSION

Alexithymia is a condition that has traditionally been defined in terms of difficulties understanding one's own emotions, and much research has focused on its relationship with affective abilities, such as emotion regulation, recognition of others' emotions, empathy, and moral decision-making. The neurological regions implicated in alexithymia (e.g., the ACC and AI) are also centrally involved, however, in more general interoceptive processes. This chapter reviewed evidence that alexithymia is responsible for the emotional difficulties experienced by many individuals with psychological disorders, such as ASD and EDs, and theorized that alexithymia may also account for the interoceptive difficulties often assumed to be core features of clinical disorders. We suggest that alexithymia may be characterized by a general deficit of interoception rather than an impairment of affective interoception alone, and that interoceptive deficits may be a reasonable candidate for the "P" factor, a unitary factor representing susceptibility to psychopathology. We therefore suggest that alexithymia, and by inference interoception, may explain both the symptom commonalities between psychiatric conditions and the symptom heterogeneity within conditions. If it is the case that alexithymia, or difficulties with interoception, predisposes one to developing psychological disorders, the question remains as to why this condition continues to be inherited. Due to its relatively high prevalence, it is possible that there are adaptive aspects of alexithymia. Determining whether this is the case should be a priority for future research.

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