Cognitive neuropsychology of alexithymia: Implications for personality typology

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Introduction. We examine the cognitive neuroscience of the five components of the alexithymia syndrome, and propose a classification of alexithymia types based on psychobiological traits.

Method. Literature review.

Results. The following neural structures have been shown to be prominent in emotional function: right and left hemisphere, corpus callosum, anterior commissure, anterior cingulate, prefrontal cortex, amygdala, and insular cortex. The specific relevance of these structures to alexithymia is discussed.

Conclusions. The following conclusions and/or propositions are presented: The right hemisphere produces a global, nonverbal overview of emotional information; the left hemisphere seems dedicated to analysing emotions and higher explicit emotional cognitions. Both orbitoprefrontal cortices are important in affective aspects of alexithymia, while right temporal cortex is involved in cognitive aspects. Two subparts of anterior cingulate fulfill functions in the affective and cognitive dimensions of alexithymia. The amygdalae are involved in both cognitive and affective aspects. All structures mentioned can modulate one another. The role of interhemispheric information transfer via the corpus callosum and the anterior commissure is also discussed. The evidence that cognitive processing of emotional information inhibits affective processing of such information is discussed in terms of its implications for a theory of alexithymia subtypes.

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We are obliged to Conner Dolan, Hubert Eleonora, and Martin Elton, University of Amsterdam, for their constructive criticism, and improvement of the manuscript.

© 2006 Psychology Press Ltd
http://www.psypress.com/cogneuropsychiatry DOI:10.1080/13546800500368607
In the late 1940s, MacLean (1949) noted that the emotional experience of many patients with psychosomatic complaints remained far below normal levels of conscious symbolic and verbal elaboration. This resulted in problems during psychoanalysis-based therapy (Groen, van der Horst, & Bastiaans, 1951; Ruesch, 1948; Sifneos, 1973b, 1975). Sifneos (1973a) introduced the term "alexithymia" (derived from the Greek α = lack, lex = work, thymos = mood or emotion) for this phenomenon. Since then, alexithymia has become an established concept (Ahrens & Deffner, 1986). In the accounts of Marty and M'Uzan, (1963), Nemiah and Sifneos (1970), Nemiah (1977, 1996), and Sifneos (1973a, 1991, 2000), five aspects of alexithymia have emerged. These are reduced capacities for emotionalising (the ease by which one experiences an emotional feeling, or becomes emotionally aroused—by external or internal stimuli), fantasising, identifying emotions, verbalising emotions, and thinking about or analysing emotions. This account of alexithymia is comparable to the accounts offered by others (Hendryx, Haviland, & Shaw, 1991; Krystal, 1988; Taylor, Ryan, & Bagby, 1985; Vorst & Bermond 2001). Although genetic factors appear to be involved in alexithymia (Heiberg & Heiberg, 1977; Valera & Berebaum, 2001), it has also been suggested that alexithymia could be the result of severe trauma (Krystal, 1988), sexual assault (Albach, Moormann, & Bermond, 1996; Berenbaum, 1996; Cloitre, Scarvalone, & Difede, 1997; Sher & Twain, 1999; Zeitlin, McNally, & Cassiday, 1993), or childhood in an emotionally dysfunctional family (Lumley, Mader, Gramzow, & Papineau, 1996; Mallinckrodt, King, & Coyle, 1998). It is often assumed—although the literature is far from clear—that emotional physiological regulation systems in alexithymics are characterised by a longer duration to respond, enhanced amplitude, or higher baseline values compared to controls (Bermond, 1997). In addition, it is assumed that alexithymia is associated with an increased probability of a variety of complaints and somatic illnesses. However, here again the literature is not clear. For instance, Lundh and Simonsson-Sarnecki (2001) failed to find the expected association between alexithymia and somatic complaints in a community sample of 137 individuals. The consensus is, however, that alexithymia and somatic complaints are correlated, albeit possibly weakly (de Gucht & Heiser, 2003; Güdel, Ceballos-Baumann, & von Rad, 2000; Taylor, Bagby & Parker, 1997). The exact interpretation of this correlation also remains unclear, as it may be due to an increased tendency to seek professional help, rather than an actual increase in somatic complaints (Lumley & Norman, 1996). This would be consistent with the notion that alexithymics suffer from hypochondria, rather than from psychosomatic disorders (Papciak, Feuerstein, Belar, & Pistone, 1987; Shipko, 1982; Wise, Mann, Hryvria, Mitchell, & Hill, 1990).

So far, two English review articles about the neuropsychology of alexithymia have appeared (Bermond 1997; Larsen et al., 2003). The aim of the present paper is to elaborate on these reviews by relating alexithymia to more neural structures, and by discussing more recent developments.
Alexithymia and left hemisphere preference or right hemisphere deficiencies

Processing emotional information has been shown to take place mainly in the right hemisphere (Adolphs, Damasio, Tranel, Cooper, & Damarsis, 2000; Banich, 1997; Bear 1983; Fricchione & Howanitz, 1985; Gainotti, 1972; Gainotti, Caltagirone, & Zoccolotti, 1993; Heilman, Scholes, & Wilson, 1975; Joseph 1982, 1992a, 1992b; Kolb & Whishaw, 1990; Ladas, Cimatti, del Pesce, & Tuozi, 1993; Malloy & Duffy, 1994; Lane, Kivley, du Bois, Shamasundaru, & Schwarz, 1995; Ross & Rush, 1981; Tucker 1981). The right hemisphere has been implicated in regulation of the subjective emotional experience, memorising emotions, in the communication of emotions to others, and in emotional physiological responses (Dimond & Farrington, 1977; Dimond, Farrington, & Johnson, 1976; Fricchione, & Howanitz, 1985; Gainotti, 1972, 2001; Ross & Rush, 1981; Wechsler, 1973). Blair, Morris, Frith, Perrett, & Dolan (1999) demonstrated right temporal lobe activation in reaction to sad facial expressions. These results suggest that alexithymia could involve: (a) a malfunctioning of the right hemisphere: or (b) a hyperactive left hemisphere (Bear, 1983; Berenbaum & Prince, 1994; Taylor, 1984a, 1984b; Voeller, 1986; Weintraub & Mesulam, 1983). Both positions have been supported (Aftanas, Varlamov, Reva, & Pavlov, 2003; Bermond, Bleys, & Stoffels, 2004; Kano et al., 2003; Parker, Taylor & Bagby, 1992; Spalletta et al., 2001).

Conjugate lateral eye movements (CLEMs) are considered to be indicative of relative hemispheric activations. Movements to the left are considered to indicate higher right hemisphere activation, whereas movements to the right indicate a relative higher activation of the left hemisphere. Cole and Bakan (1985) and Parker et al. (1992) both registered CLEMs in alexithymics and non-alexithymics, and produced opposite results. However, Cole and Bakan measured alexithymia using the Schalling Sifneos Personality Scale (SSPS), which is considered to be lacking in reliability (Morrison & Phil, 1989; Norton, 1989; Taylor, et al., 1997). Parker et al. (1992) used both the Toronto Alexithymia Scale (TAS) and the SSPS. They found a relationship between right CLEMs (higher left hemisphere activation) and alexithymia as measured by the TAS. No such relationship was observed using the SSPS. Jessimer and Markham (1997) showed alexithymic and nonalexithymic subjects chimeric pictures of faces composed of conjoined emotive and nonemotive halves. Their results indicated that alexithymic subjects showed less leftward perceptual bias (indicating reduced right hemisphere responding) and poorer recognition of the facial expressions than nonalexithymic subjects. Lane et al. (1995), using the Levy Chimeric Faces Test (LCFT), demonstrated that greater right hemisphere
dominance in processing emotional information was related to higher “emotional awareness” as measured by the Levels of Emotional Awareness Scale (LEAS). The authors’ description of the scale suggests that the construct “emotional awareness” may be equivalent to “Identifying” and “Verbalising”, which are subscales in the TAS and Bermond Vorst Alexithymia Questionnaire (BVAQ). This implies that scores on LEAS are correlated negatively with scores on alexithymia. Thus, the results of Lane and co-workers are consistent with the results of Jessimer and Markham (1997). Spalletta et al. (2001) measured alexithymia in stroke patients with unilateral lesions. Lesions were located in various regions: neocortical areas, subcortical areas, brainstem, and cerebellum. Despite the variety in localisation, right hemisphere lesions were found to be more strongly associated with higher TAS scores, compared to left hemisphere lesions. Kano et al. (2003) measured brain activity, using positron electron tomography (PET), in alexithymic and nonalexithymic subjects, while they were shown angry, sad, happy, and neutral faces. Compared to nonalexithymics, areas of significantly lower cerebral blood flow (rCBF) in alexithymics were located in the neocortex of the right hemisphere, while areas of higher rCBF were generally located in the neocortex of the left hemisphere. Lumley and Sielky (2000), using a finger localisation task, demonstrated that individuals, who scored high on alexithymia as measured by the TAS, made more errors in localising fingers in the left hand (right hemisphere) compared to localisation of fingers of the right hand. Finally, Bermond et al. (2004) studied alexithymic and nonalexithymic subjects, using a visual-matching task paradigm with lateralised stimuli. They demonstrated that alexithymic subjects with severe reductions in “identifying” and “verbalising” emotions process emotional words—which are presented to the right hemisphere—in their left hemisphere. In contrast, nonalexithymic subjects process such information in the right hemisphere. This effect was independent of scores on emotionalising.

A magnetic resonance imaging (MRI) study conducted by Gündel et al. (2004) found a positive correlation between alexithymia scores and the size of the right anterior cingulate (see below) in healthy (nonalexithymic) men. In conclusion, these results, while not completely unequivocal, do support the notion that alexithymia could be associated with: (a) underfunctioning of the right hemisphere; or (b) a hyperactive left hemisphere.

Alexithymia and the corpus callosum

Various studies have indicated that verbal conscious and analytical information processing occurs predominantly in the left hemisphere, while unconscious, nonverbal and parallel holistic information processing generally takes place in the right hemisphere (Galin, 1974; Gazzaniga, 1989, 1995; Joseph, 1982, 1992a; Kalat, 1992; Kolb & Whishaw, 1990; Kupfermann, 1991; Miller, 1986; Tucker, Roth, & Bair, 1986). These findings, together with those stressing the
importance of the right hemisphere for emotions, suggest that alexithymia could be related to dysfunction of the corpus callosum (Galin, 1974; Krystal, 1988; Lădăvaz et al., 1993; Miller, 1986). This hypothesis has been supported in research (Buchanan, Waterhouse, & West, 1980; Dewaraja & Sasaki, 1990; Ernst, Key, & Koval, 1999; Hoppe & Bogan, 1977; Houtveen, Bermond, & Elton, 1997; Lumley & Sielky, 2000; Parker, Keightley, Smith, & Taylor, 1999; TenHouten, Hoppe, & Walter 1985, 1986; Zeitlin, Lane, O'Leary, & Schrift, 1989). The corpus callosum agenesis case study of Buchanan et al. (1980) provides a clear description of all alexithymia features in a patient who received maximum score on the Beth Israel Hospital Psychosomatic Questionnaire (BIHPQ; an 8-item therapist-evaluation alexithymia scale) from three independent raters. The study of Ernst et al. (1999) is also a corpus callosum agenesis case study. Their patient showed flat affect and scored in the alexithymic range on the TAS-20. However, this patient displayed many comorbid problems [spina bifida, hydrocephalus, central sleep apnea, and hypothalamic, pituitary, pineal gland dysfunction (requiring hormone replacement therapies), and chronic pain], so that the results regarding alexithymia should be interpreted with care. The studies of Hoppe, and Bogen, and TenHouten and co-workers all concern either a group of 8 patients with corpus callosum transections, or a group of 12 such patients and their matched controls. Patients were shown a 3 minute film about the death of a baby and of a boy. The combined results clearly indicate that, compared to their matched controls, commissurotomised patients used fewer affect-laden words, and fewer adjectives in their descriptions of the film. In addition, their descriptions of the film content were more concrete, and contained more nonemotional utilitarian details. These patients also demonstrated less creativity and fantasy. Finally, all scored as alexithymic on 6 of the 8 BIHPQ items.

A role for the corpus callosum is also supported by the studies of Zeitlin et al. (1989), and Parker et al. (1999). They demonstrated that male alexithymic subjects make more mistakes in finger localisation tasks requiring callosal transfer of information. Lumley and Sielky (2000) have replicated these results, although they only found a significant relationship with “identifying emotions”. Furthermore, these authors failed to find this relationship in females. Huber et al. (2002) demonstrated in a PET study that alexithymic subjects showed less activation in the corpus callosum in response to emotional stimuli compared to nonalexithymics. However, Kano et al. (2003), who also measured brain activity using PET, reported the opposite result. Although there are various methodological differences in the experiments by Kano et al. and Huber et al., the main differences concern the technique of emotion induction. Huber et al. asked their patients to remember emotional experiences (happiness, sadness, and neutral), whereas Kano et al. presented to their patients pictures of faces with emotional expressions (anger, sadness, happiness, and neutral). In essence, individuals in the latter study were passively watching, while individuals in the former were
actively remembering. It is possible that passively watching requires little transfer of information from the right hemisphere to the left hemisphere (a strategy is expected in alexithymic subjects), whereas actively remembering would require more information exchange between the verbal left and emotional right hemisphere.

Some indirect support is provided by an EEG study of Aftanas et al. (2003). These authors observed in alexithymics, but not in nonalexithymics, deficient left hemisphere synchronisation in the upper theta band in response to emotional stimuli, along with excessive event-related synchronisation to emotional stimuli in the right hemisphere. Using Poffenberger’s (1912) paradigm for estimating the corpus callosum transmission times, Dewaraja and Sasaki (1990) observed longer right-to-left hemisphere transfer times in alexithymic persons than in nonalexithymic subjects. Although this is remarkable, it does not explain the alexithymic features. After all, alexithymia is defined by severe reductions in the experience of various components of emotions, and the results of Dewaraja and Sasaki do not indicate that less or incorrect information is sent to the left hemisphere. Moreover, the implicit assumption in the experimental design of Dewaraja and Sasaki, namely, that the information is processed in the hemisphere where it arrives, is dubious (Bermond et al., 2004). Contralateral processing, according to the relay model of Springer and Deutsch (1993) has to be expected if the opposite hemisphere is better suited for the cognitive task involved (Banich & Karol, 1992; Gazzaniga, Ivry, & Mangun, 1998). This could explain why Grabe et al. (2004) found more or less the opposite results; shorter transcallosal conduction times in male alexithymics compared to nonalexithymic males. However, the neural coherence results of Houtveen et al. (1997) implied less information transfer from the right frontal cortex to the left hemisphere in alexithymics. In conclusion, the idea that alexithymia could be related to a “functional commissurectomy” seems fairly well supported by experimental data, although the results are not unequivocal.

Alexithymia and the anterior commissure

Bermond (1997), referring to the work of Gazzaniga and LeDoux (1978), stressed the relation between the anterior commissure and alexithymia. Gazzaniga and LeDoux (1978) elaborated on earlier work of Roger W. Sperry, in which emotion-inducing pictures were presented to the right hemisphere of split-brain (commissurectomy) patients. Although these patients could not verbalise the content of these pictures, the pictures induced emotional feelings, and the patients could verbalise the emotional significance correctly. In the study of Gazzaniga and LeDoux (1978), words were presented either to the left or the right hemisphere of subjects with corpus callosum transections. These subjects could verbalise the words, if presented to the left hemisphere, but claimed that nothing was presented if the same words were presented to the right hemisphere.
However, when asked to evaluate the emotional valence of this "nothingness", they evaluated the "unseen" words exactly like the same words if presented to the left hemisphere. According to Gazzaniga and LeDoux this indicates that the emotional-cognitive information is sent from the right to the left hemisphere by the corpus callosum, whereas the emotional value is sent to the left hemisphere over the anterior commissure. These authors further substantiated their conclusion by referring to a study by Sperry, in which emotion-inducing pictures were presented to the right hemisphere of a patient in whom both the corpus callosum and the anterior commissure were transected. Although such pictures induced emotional behavioural responses in the patient, these responses were not accompanied by concomitant affect. The explanation of Gazzaniga and LeDoux fits with results showing that the anterior commissure connects the amygdala and the paleocortical parts in both hemispheres, as well as the anterior temporal lobes, a part of the temporal lobe to which the corpus callosum does not project (Kolb & Whishaw, 1990).

Thus, alexithymia, characterised by the presence of conscious emotional cognitions, together with severe reductions in affect (emotionalising), is expected as a result of dysfunctions in the anterior commissure. To date, no studies have related the anterior commissure directly to alexithymia. The fact that most research has been done with the TAS, which does not measure emotionalising, may explain this dearth of studies.

**Alexithymia and the anterior cingulate**

In 1937, Papez proposed that the anterior cingulate cortex (ACC) fulfils functions in the regulation of emotions. Specifically, he proposed that emotional experience emerges in this structure. More recently, Lane and co-workers (1998) related ACC activity to conscious awareness of emotions. Paus (2001) ascribes three main functions to the ACC: motor control, cognition, and arousal/drive regulation (related to conditioned emotional learning, vocalisations associated with the expression of internal states, assessment of motivational interactions, assignment of emotional valences to internal and external stimuli, and mother-infant interactions). Referring to the "emotional" functions, Paus underlined the dopaminergic, serotonergic, and noradrenergic innervations of the ACC. Further, Paus distinguishes two subparts of the ACC: a ventral tier covering Brodmann’s areas 24a and b and 25, and a dorsal tier covering 24c and 32. The ventral tier receives direct projections from the limbic system (amygdala and ventral striatum), whereas the dorsal tier receives direct projections from the hypothalamus (mediodorsal-and anterior nuclei, and midline nuclei), and indirect limbic projections via the ventral tier. Devinsky, Morrell, and Vogt (1995) and Bush, Luu, and Posner (2000) also distinguish two subparts in the anterior cingulate: an "affect" and a "cognition" component. Although they differ slightly in the assigned locations of the comparable subparts of the ACC,
their descriptions overlap: "Affect division" [Brodmann's areas; 25, 33, and 24; (Devinsky et al., 1995); 24 a–c, 32 & 25 (Bush et al., 2000)]; ‘cognition division’ [caudal areas 24 and 32, (Devinsky et al., 1995) 24 b–c and 32 (Bush et al., 2000)]. The cognitive subdivision maintains strong reciprocal interconnections with lateral prefrontal cortex, parietal cortex, and premotor and supplementary motor areas. The affective subdivision, by contrast, is connected to the amygdala, periaquaductal gray, nucleus accumbens, hypothalamus, anterior insula, hippocampus, and orbitofrontal cortex, and has outflow to autonomic, visceromotor and endocrine systems. Regarding alexithymia, it is of particular importance that these two subparts of the anterior cingulate show reciprocal suppression, and that cognitive and emotional information is processed separately (Bush et al., 2000).

ACC activity has been related to mood changes, and ACC lesions have consistently been associated with change in affect (Luu & Posner, 2003). Emotional lability has been related to reduced cerebral blood flow in the ACC (Lopez et al., 2001). Changes in subjective emotional experiences and deficits in the recognition of emotional expressions have been described in patients with lesions in the anterior cingulate (Hornak et al., 2003). In a PET study, Lane et al. (1998) observed significantly increased ACC activation in response to watching emotion inducing films. Blair et al. (1999), again in a PET study, demonstrated that activation in the ACC was positively correlated to the intensity of images of faces expressing anger. Phillips et al. (2004), in a fMRI study, obtained similar results with fearful expressions. Finally, Sanfey et al. (2003) demonstrated increased cingulate activations in subjects who were angered by unfair offers made by others.

There is thus ample reason to think that the anterior cingulate plays a role in the regulation of emotions, both emotional cognitions and emotionalising. Thus, dysfunction of the ACC could be related to various features of alexithymia. Lane et al. (1998) reported positive associations between LEAS scores and cerebral blood flow in the ACC during film-induced negative emotions. As argued above, the LEAS might measure the opposite of important alexithymia features. Thus, their results suggest that alexithymia is related to less ACC activation. In line with this, Huber et al. (2002) and Kano et al. (2003), using PET, both demonstrated that alexithymics showed less activation in the anterior cingulate cortex than nonalexithymics, while looking at angry faces or during autobiographical recall of emotional situations. Berthoz et al. (2002) obtained results that are more complicated. In a functional magnetic resonance imaging (fMRI) study, they observed higher activation in alexithymics, compared to nonalexithymics, in the anterior cingulate in reactions to positive emotional pictures, and lower activation in the left anterior cingulate in alexithymics in reaction to negative emotional pictures. As Kano et al. (2003) used angry face stimuli, this suggests that reduced cingulate activation in alexithymics is limited to negative emotions. However, it is also possible that the anterior cingulate
reacts specifically to angry faces, since Blair et al. (1999) registered an increase in cingulate activity in reaction to angry faces, but not in reaction to sad faces (using PET in a group of normal subjects). However, another explanation is possible. Phan, Wager, Taylor, and Liberzon (2002) published a meta-analysis on 55 PET or fMRI studies regarding emotions. The results indicated that positive emotions (happiness) result in activation in the basal ganglia, not in activation of the ACC. It is thus possible that the alexithymic subjects, in the study of Berthoz and co-workers, had difficulties in appraising the positive pictures as positive stimuli. Gündel et al. (2004) reported conflicting results. In a MRI study, they found a positive correlation between the volume of the right anterior cingulate and TAS scores. The authors explained their results by supposing that the anterior cingulate might be involved in a suppression mechanism that pushes unwanted associations out of awareness. This notion is consistent with the finding that the cognitive and emotional parts of the ACC show reciprocal inhibition (Bush et al., 2000). Given this otherwise reasonable hypothesis, one would expect an increase in ACC activation in alexithymic subjects, when they process emotional information. The studies by Huber et al. (2002), Kano et al. (2003) and Lane et al. (1998) indicated just the opposite.

In conclusion, although the literature is unequivocal regarding a role of the anterior cingulate in the regulation of emotions, the literature is far less clear concerning the relationship between the anterior cingulate and alexithymia.

Alexithymia and the prefrontal cortex

In 1947, Freeman and Watts concluded on basis of the literature that frontal lobotomy was particularly successful in patients with strong emotional complaints (Kucharski, 1984). This was unsurprising, as the intervention was originally meant to reduce emotions (Kolb & Whishaw, 1990). In 1946, Freeman introduced the transorbital method, in which mainly the medial orbitofrontal parts of the prefrontal cortex (O-PFC) were isolated from the rest of the brain. Restriction to this part of the brain turned out to be clinically more effective and also resulted in fewer adverse effects (Freeman, 1971; Livingston, 1969). Trigg (1970), who reviewed the literature, cited a great number of similar case studies. Generally before surgery, patients were preoccupied with their emotional feeling, which they experienced as overwhelming. Following the operation, the patients experienced flatness of affect and absence of spontaneous emotional reflection (Damasio, 1994; Damasio, Tranel, & Damasio, 1990; Trigg, 1970). However, the emotional cognitions remained unchanged (Trigg, 1970). Such prefrontal lesions, however, also induce a disinhibition of emotional behaviour, resulting in short periods of violent emotional behaviour (Fuster, 1989; Jarvie, 1954; Malloy & Duffy, 1994; Valenstein, 1990). Violent emotional behaviour in absence of accompanying affect has also been described in alexithymic patients (Krystal, 1988; Nemiah, Fryberger, & Sifneos, 1976). Furthermore, other work
has suggested that patients with prefrontal lesions also show a reduction in facial expression and an impoverished imagination, features which have also been described in alexithymic patients (Kolb & Whishaw, 1990; Krystal, 1988; Levine & Alberts, 1948; McDonald & Prkachin, 1990; Sifneos, 1975; Taylor, Ryan, & Bagby, 1985). Moreover, deficits in the recognition of emotional expressions have been described in patients with orbitofrontal lesions (Hornak et al., 2003), and reduction in empathy has been observed in patients with ventromedial prefrontal lesions (M-PFC; covering both the O-PFC and ACC, Bechara, 2004) (Shamay-Tsoory, Tomer, Berger, & Aharon-Peretz, 2003). Furthermore, the orbitofrontal cortex has been implicated in the decoding of negative as well as positive reinforcers (Rolls, 2000), and specific O-PFC activations in reaction to emotional stimuli have also been demonstrated (Taylor, Liberonz, Decker, & Koepppe, 2002). The O-PFC has been implicated in the regulation of facial expression and emotional learning (Angrilli, Palomba, Cantagallo, Maletti, & Stegagno, 1999; O’Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001). Finally, the functional presence of PFC is not only required for emotionalising, but also for various forms of fantasising (creativity, originality, imaginative powers, mental imagery, and divergent thinking) (Damasio, 1994; Damasio & Anderson, 1993; Kolb & Whishaw, 1996).

The O-PFC projects to the hypothalamus, where orbitofrontal neurons connect with neurons projecting to the brainstem and spinal autonomic centres. It is by these connections that the O-PFC has control over primitive emotional autonomic responses (Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Simpson et al., 2001). Further the O-PFC and the amygdala, another important neural structure in the regulation of emotions (Davidson, Jackson, & Kalin, 2000), have strong reciprocal connections (Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Compton, 2003; Ghashghaei & Barbas, 2002), so can modulate one another.

Damasio and co-workers have also underlined the importance of the O-PFC/M-PFC cortex in emotion regulation (e.g., Bechara, 2004; Bechara, Damasio, & Damasio 2000; Damasio, 1994, 1999; Tranel, Bechara, & Denburg, 2002). These studies stress both the reduction in emotionalising due to such lesions, and deficiencies in emotional decision making. It seems that emotional decision making is more related to emotional cognitions than emotionalising itself. This seems to conflict with older literature, which stresses that destruction of the O-PFC does not affect emotional cognitions. However, as argued above, such lesions impair spontaneous emotional reflection. This is consistent with the idea that the emotional experience forces people to reflect upon their emotions (Laird & Bressler, 1992). In other words, a dysfunctioning of the O-PFC could result in a severe reduction in emotionalising together with the presence of emotional cognitions. Such patients lack the internal drive to reflect upon their emotional cognitions. However, they will do so if stimulated by others (Damasio et al., 1990). This lack of spontaneous reflection, together with the fact that most of the
patients studied by Damasio and co-workers had M-PFC lesions, could explain disrupted emotion decision making. Thus, PFC dysfunction could be related to the features “emotionalising”, “fantasising”, and “analysing” of alexithymia.

A few recent studies have directly addressed the relationship between the PFC and alexithymia. Aftanas et al. (2003) demonstrated that in response to emotional stimuli, alexithymics manifest a decrease in frontal left hemisphere event related potentials (ERPs) in the upper theta band in the early test period of 0–200 ms. According to the authors, this indicates a dysregulation of the appraisal of emotional stimuli. The PET study of Kano et al. (2003), demonstrated lower activations in the right O-PFC in alexithymics, compared to nonalexithymics, in reaction to negative emotional stimuli. Huber et al. (2002), also using PET, reported in alexithymics lower activations in the right frontal cortex and higher activations in the left frontal cortex during recall of autobiographic emotional experiences, results that may seem contradictory. However, there are indications that activation in one hemisphere inhibits activation in the other hemisphere, no matter the cause of the activations (Kolb & Whishaw, 1990, 1996; Sperry, Zaidel, & Zaidel, 1979). Thus, left hemisphere to right hemisphere inhibitions have occurred in the processing of emotional information, resulting in lower subjective emotional experiences and lower emotional physiological responses (Dimond & Farrington, 1977; Dimond et al., 1976). The fMRI results of Berthoz et al. (2002) are complex. They report in alexithymics less activation in the left mediofrontal cortex in reaction to highly negative emotional stimuli, but more bilateral activation in the mediofrontal cortex in reaction to positive stimuli.

In sum, although there is general agreement about the function of the PFC, especially the O-PFC/ M-PFC, in emotional experience, literature that directly relates alexithymia to the prefrontal cortex is limited and equivocal.

Alexithymia and the amygdala

Both the amygdala and the PFC receive massive projections from sensory cortices, and from higher order temporal lobe association areas specialised in processing features of stimuli and their memory. Both structures appear to have a global overview of the environment, which is probably necessary in processing and remembering emotional significance (Ghashghaei & Barbas, 2002). The amygdala also receives direct projections from the accessory olfactory bulb and thalamus. The amygdala projects to neocortical structures, and directly or indirectly to various subcortical structures involved in emotional physiological responses and emotional behaviours (hypothalamus, hippocampus, bed nucleus of the stria terminalis, preoptic area and central grey) (Baum, 2002; LeDoux, 1996, 2000; McCarthy & Becker, 2002; Young & Insel, 2002). This subcortical network may be viewed as a phylogenetically older emotion regulation system.
Various studies indicate that the amygdala is involved in detection of emotional significance of stimuli (Adams, Gordon, Baird, Ambady, & Kleck, 2003; Breiter et al., 1996; Compton, 2003; Davidson & Irwin, 1999; Gainotti et al., 1993; Ono, Nishijo, & Nishino, 2000; Phillips et al., 2003), and in recognition of emotion facial expressions (Alphonse et al., 1999; Alphonse & Tranel, 2003). Gainotti (2001) argues that external stimuli are appraised in the amygdala. In line with this, Morris, Öhman, and Dolan (1998) demonstrated that subliminally projected images of angry faces, which were not consciously perceived, resulted in a selective activation of the right amygdala. Likewise, de Gelder et al. (1999) demonstrated in a “blind-sight study” that emotional expressions of faces presented in the “blind” part of the visual field were identified correctly well above chance. These authors explained their results by stating that the “unseen” stimuli activate the amygdala via the colliculopulvinar pathway. Furthermore, Morris et al. (1996, 1998b) presented PET results indicating that amygdala activation is emotion specific, and that activations of other neural structures by the amygdala are also emotion-specific. The studies of Bar-On, Tranel, Denburg, and Bechara (2003) and Bechara, Damasio, and Damasio (2003), indicate that patients with amygdala lesions show very low levels of “emotional intelligence”, and are poor in emotional decision making. In contradistinction to this, Anderson and Phelps (2002) presented results indicating that patients with unilateral and bilateral amygdala lesions report emotional feelings of normal intensities. Furthermore, Keightley et al. (2003) reported results based on fMRI-data, which indicated that the amygdala is more active during emotional processing. Taylor et al. (2002) reported similar results based on PET data. Whalen et al. (1998) reported significant fMRI modulations in the amygdala in response to emotional stimuli that were not consciously apperceived. The modulations involved increases in reaction to negative, and decreases in reaction to positive, emotional stimuli. Recently Williams, McGlone, Abbott, and Mattingley (2005) reported results indicating that the amygdala response is modulated by attention. Amygdala activation was found to increase given positive stimuli (happy faces) when subjects were asked to attend to these stimuli. The increase in amygdala activation to negative stimuli (fearful faces) was enhanced when subject did not attend to such stimuli. Finally, Phan et al. (2002) stated that glucose metabolism in the M-PFC is strongly inversely associated with metabolic rate of the amygdala. In addition, they reported that amygdala activation is attenuated during cognitive appraisal and tasks demanding higher cognitive processing, whereas the activity of the M-PFC is increased during cognitive appraisal. More complicated, but still comparable fMRI results are reported by Ochsner, Bunge, Gross, and Gabrieli (2002). Their results indicated that reappraisal of negative emotion-inducing pictures as emotionally neutral pictures results in increased activation of the lateral as well as medial PFC and decreased activation in the amygdala and orbito-PFC. Opposite results in M-PFC and O-PFC suggest that successful reappraisal-induced increase in M-PFC activity is attributable to an
increase in anterior cingulate activation, whereas such reappraisal simultaneously results in decreased activation of the orbito-PFC. As argued above, this subpart of the medial-PFC is directly linked to emotionalising or emotional feelings.

Taken together, these results are comprehensible. The amygdala mediates both emotional physiological and inborn and acquired behavioural responses (Iversen, Kupfermann, & Kandel, 2000), whereas the prefrontal cortex (O-PFC/M-PFC) is related to emotional feeling. Emotional feeling, furthermore, is in some way related to emotional reflection (Buck, 1993; Damasio, 1999; Laird & Bressler, 1992). If the M-FPC did not inhibit the amygdala, then this phylogenetically older structure would initiate preprogrammed emotional behaviour. This would render emotional feeling and reflection purposeless. Conversely, if the emotional stimuli are of such intensity that an immediate behavioural response is required, emotional feeling and reflection are diversions, which should be blocked. Although reciprocal facilitations of the amygdala and medial-PFC cannot be ruled out, the main function of the connections between these structures seems to be inhibitory. Thus, very strong activations of the amygdala can result in auto-execution of preprogrammed emotional behaviour. Because the PFC is bypassed, this behaviour is accompanied neither by emotional feelings, nor by emotional reflection.

The combination of these results, furthermore, suggests that with respect to emotional experience, the amygdala is primarily involved in “implicit identifying emotions”, and, through connections to cortical areas, also involved in “explicit identifying emotions”. However, Iversen et al. (2000) stress the idea that the “emotion-identifying” modules for visual stimuli in the inferior temporal cortex can make the appropriate responses autonomously, and forward this information to the amygdala. This suggests that explicit (neocortical) emotional cognitions do not depend on implicit (amygdala) emotional cognitions. Furthermore, the amygdala is, through projections to the PFC, involved in “emotionalising”. Thus, amygdala dysfunction could also have an effect on emotionalising, while hardly affecting explicit emotional cognitions (Alphonse & Tranel, 2003).

Surprisingly, there are almost no studies relating the amygdala directly with alexithymia. Berthoz, Ouhayoun, Perez-Diaz, Consoli, and Jouvent (2000) reported that they failed to find differences between alexithymics and nonalexithymics in amygdala activation. Likewise, Kano et al. (2003) reported the absence of such differences in subcortical structures. We predict that subjects with extremely low capacities to emotionalise will show, in response to innate or conditioned emotional stimuli, strong activations in the amygdala, while subjects with extremely high capacities to emotionalise and reflect upon their emotions, will show low amygdala activations in reaction to such stimuli.
Insular cortex and alexithymia

Gainotti (2001) related the insula to the regulation of autonomic components of emotions. Although the exact function of the insula in emotional experience remains unclear, various studies have implicated the insular cortex in the regulation of negative emotional experiences, such as pain, distress, hunger, thirst, fear, anger, sadness, and disgust (Hennenlotter et al., 2004; Ishai, Pessoa, Bikle, & Ungemleider, 2004; Phan et al., 2002; Phillips et al., 2003; Reiman et al., 1997; Sanfey, Rilling, Aronson, Nystrom, & Cohen, 2003; Taylor, Phan, Decker, & Koepepe, 2003), emotional decision making (Ernst et al., 2003), and the production of facial emotional expressions (Carr, locoboni, Duban, Maziotta, & Lenzi, 2003). To our knowledge, only one study has directly addressed the role of the insula in alexithymia. Kano et al. (2003) demonstrated reduced insula activations in alexithymics compared to nonalexithymics in response to angry faces.

Other neural areas and alexithymia

The two PET studies cited above (Huber et al., 2002; Kano et al., 2003) also related alexithymia to other neural structures. Kano et al. reported that alexithymics, compared to nonalexithymics, show less activation in the right inferior parietal and occipital cortices, and more activation in the left inferior parietal cortex and cerebellum (all in response to angry and sad facial expressions). Huber et al. asked subjects to recall autobiographic emotional situations. They report higher activations in alexithymics, compared to nonalexithymics, in cuneus and precuneus, thalamus (pulvinar), right inferior temporal and left superior temporal regions, and the cerebellum.

The lower activations in the right inferior parietal cortex and right occipital cortex, and higher activations in the left superior temporal regions, and left inferior parietal cortex are consistent with the idea of a left hemisphere preference in alexithymics. Further, the lower activation in the occipital cortex may be explained by assuming that alexithymics show less top-down facilitation, from higher brain centres to more primary sensory nuclei. This could be of interest for the field of alexithymia, since there are indications that feedback of sensory information from higher brain centres to more primary sensory areas is a prerequisite for conscious perception (Lamme & Roelfsema, 2000). The increased activation of the thalamus in alexithymics may indicate an increased activation of a phylogenetically older emotion regulation system, since the thalamus projects directly upon the amygdala (LeDoux, 1996). However, if this were the case, one might also expect increased activations in the amygdala, and such results have not been published. The increased activation in the right inferior cortex (Huber et al., 2002) is hard to explain in the framework of alexithymia. One could argue that right inferior temporal cortex modules
involved in aspects of emotional experience (Iversen et al., 2000) are, in alexithymic subjects, not well suited for this function and, therefore, demand more neural activity and, thus, more oxygen and glucose utilisation, in order to fulfill this function properly. This is conceivable because Huber et al. forced their subjects to actively remember autobiographical emotional experiences. The cuneus, precuneus, and cerebellum have been implicated in various emotional functions in other research (concerning the (pre-)cuneus, see Fernandez et al., 2001; Gardner, Martin, & Jessel, 2000; Hennenlotter et al., 2004; Juengling et al., 2003; Kihlstrom, Gideon, Ely, & Holtman, 2003; concerning the cerebellum, see Critchley, Elliott, Mathias, & Dolan, 2000a; Fernandez et al., 2001; Leroy et al., 2002; Paravizi, Anderson, Martin, Damasio, & Damasio, 2001; Parsons et al., 2000). Increased activation in the cuneus is of interest, as ascending pain fibres synapse here (Gardner et al., 2000). These results therefore suggest that alexithymics might experience more pain than nonalexithymics, which might relate to the increased proclivity to seek help (Lumely & Norman, 1996) and hypochondria seen in alexithymics (Papciak et al., 1987; Shipko, 1982; Wise et al. 1990). Decreased cerebellum activations have been implicated in autism and Asperger syndrome (Critchley et al., 2000b; Frith, 2004), suggesting that the cerebellum may be involved in mentalising. However, its exact function in emotional experience remains unclear.

**DISCUSSION**

The mass of neuropsychological work presented above leads to the following suggestions. Both orbito-prefrontal/medial-prefrontal cortices, although the right more than the left, are important in emotionalising and fantasising. Modules in the right temporal cortex (Frith, 2004) are involved in emotional cognitions, and, mainly by their connections to the O-PFC, marginally involved in emotionalising. With respect to emotional cognitions, these neural structures, as well as the right O-PFC/M-PFC contact modules, related to consciousness and language, in the left hemisphere by the corpus callosum. With respect to “emotionalising”, these structures contact modules related to consciousness and language in the left hemisphere via the anterior commissure. The two subparts of anterior cingulate have a role in emotionalising and emotional cognitions, respectively. The amygdalae are directly involved in implicit emotional cognitions. By their projections to neocortical structures, they are involved in explicit emotional cognitions. By their connections with the prefrontal cortex, they are involved in emotionalising. All structures mentioned are interconnected, so can modulate one another (Frith, 2004; Ghassghaei & Barbas, 2002; Iversen et al., 2000; Phan et al., 2002; Phillips et al., 2004). The affective subpart of the anterior cingulate is strongly connected to the O-PFC. It is therefore possible that this structure derives its role in emotionalising from its projections to the O-PFC.
In the mature brain, both the amygdala and PFC receive information on various levels of processing. Both structures control structures involved in emotional physiological responses and in emotional behavior (Barbas et al., 2003; Iversen et al., 2000), but they differ in their function in the regulation of emotions. The amygdalae can induce auto-executing of preprogrammed emotional behaviour, accompanied neither by emotional feelings, nor by emotional reflection.

With respect to emotional cognitions, the right hemisphere is strikingly incapable of making even the simplest deductions, although this hemisphere does produce a global, nonverbal overview of the emotional information. As Gazzaniga (1995) demonstrated, this hemisphere fails to make the simplest of inferences (e.g., the combination of firewood and matches may give rise to a campfire). According to Gazzaniga (1995) the human inferential system is limited to the left hemisphere: “The left hemisphere, on the other hand, is constantly, almost reflexively, labeling experiences, making inferences as to cause, and carrying out a host of other cognitive activities”. Thus the left hemisphere is required in analyzing emotions, and for explicit emotional cognitions.

As argued above, there are clear indications that activation of structures involved in emotional cognition inhibit activations in structures involved in affective aspects of emotions. Reciprocal inhibitions have been demonstrated between the two hemispheres, between the two subregions of the anterior cingulate, and between amygdala and PFC. Furthermore, Keightley et al. (2003) reported increased activity in various parts of the left hemisphere (dorsal portion of the frontal lobe and parietal lobe) in subjects who were asked to indicate whether presented facial expressions were positive or negative. No such increased activity was found, when the subjects were asked to passively look at these expressions. Taylor et al. (2003) demonstrated in a PET study that the activation of brain structures involved in processing emotional information was reduced in subjects who were asked to rate of the intensities of aversive stimuli. Again, this reduction was absent when subjects were asked to passively look at the stimuli. Taylor et al. concluded: “This finding is consistent with reports showing that detached analytical appraisal of emotional stimuli can reduce both subjective and psychophysiological responses”. Pessoa, Kastner, and Ungerleider (2003), in a review of the attention literature, arrived at two interesting conclusions. First, they presented results indicating that there is a ventral system, strongly lateralised to the right hemisphere, which detects behaviourally relevant stimuli and works as an alerting mechanism for the dorsal frontal attention system. This right hemisphere system is also important in emotions, because this system is involved in drawing attention to emotionally salient stimuli. Second, in the light of recent fMRI investigations, they concluded that the stimulus-evoked fMRI response is essentially absent in individuals who engage in a competing task with high attentional load.
Kolb and Whishaw (1996) described two very different personality styles in cognitive problem solving, one with a left hemisphere preference, and the other with a right hemisphere preference. The left-hemisphere-preference-type is viewed as analytical, logical, neat and tidy, interested in details, and directed to cognitive control. The right-hemisphere-preference-type is viewed as a synthesiser, interested in generalisations, with a facility to perceive a gestalt quickly, and to integrate diverse concepts into a meaningful whole.

In line with Kolb and Whishaw, and given the neuropsychological data presented above, the following four different personality types are conceivable within the broader framework of the alexithymia concept. (1) Persons characterised by a strong proclivity to activate neural modules involved in the cognitive processing of nonemotional information will inhibit the neural modules involved in "emotionalising" and the modules involved in cognitive aspects of the emotional experience. Such persons will ignore their emotions and direct their attention to other things. (2) Persons characterised by a strong proclivity to activate neural modules involved in the cognitive processing of emotional information, will inhibit the neural modules involved in "emotionalising". Such persons will mainly experience emotionally detached emotion-related cognitions. (3) Persons, with a strong proclivity to activate neural modules involved in emotionalising, will inhibit the neural modules involved in cognitive aspects of the emotional experience. Such persons will go with the flow of the emotion, and not analyse their emotions. (4) Persons, who activate modules involved in emotionalising first, and subsequently activate the cognitive-emotion-modules. Such persons will go with the flow of the emotion, and thereafter analyse their emotions.

It is conceivable that these very different ways of handling emotions develop into more or less stable personality traits, due to childhood experiences. However, genetic predispositions to one of the four types may not be ruled out (Heiberg & Heiberg, 1977; Valera & Berebaum, 2001).

Most research in the field of alexithymia has been done with the Toronto Alexithymia Scale (TAS), or later versions of TAS, the TAS-R, and TAS-20 (Bagby, Parker, & Taylor, 1994; Taylor et al., 1992, 1985). None of these scales measure "emotionalising". In addition, the TAS-R and TAS-20 do not measure "fantasising". The Bermond-Vorst Alexithymia Questionnaire (BVAQ) was developed to measure all five alexithymia features. The BVAQ has also good psychometric properties (Berthoz et al., 2000; Müller, Bühner, & Elligring, 2004; Vorst & Bermond, 2001; Zech, Luminet, Rime, & Wagner, 1999). Principal component analysis of the BVAQ subscale scores produced two uncorrelated factors: an affective dimension (involving the features emotionalising and fantasising), and a cognitive dimension (involving identifying, and verbalising emotions). The feature analysing loaded on both factors (Vorst & Bermond, 2001). These two orthogonal dimensions have now been replicated in confirmatory factor analyses in seven countries and six different languages.
Given the fact that the two alexithymia dimensions are orthogonal to one another, four extreme groups are conceivable. Three of these show clear, but distinct, deficits in their experience of emotions, and are designated alexithymia types 1 through 3. Type 1 is characterised by low affective and low (emotion related) cognitive capacities, Type 2 by high affective and low cognitive capacities, and type 3 by low affective and high cognitive capacities. The fourth group, characterised by high affective and high cognitive capacities, has been dubbed the lexicithymic group (Vorst & Bermond, 2001; Bermond et al., in press). The characteristics of these four groups fit with those described above. It has further been demonstrated that each of these four extreme groups are clearly linked to specific other personality traits (Niessen, 2001).

The fact that the two alexithymia dimensions are orthogonal may explain the unexpected lack of correlation, observed by Lane et al. (1998), between scores on the LEAS ("identifying" and "verbalising" emotions) and the intensity of self-reported affect ("emotionalising").

Lumley et al. (1996), Mallinckrodt et al. (1998), and Fitzgerald (2003) proposed that a childhood in an emotionally dysfunctional family could result in alexithymia. This calls for an ontogenetic explanation of alexithymia. The amygdala and anterior commissure are functional very early in life, the neocortex matures later, while the prefrontal cortex and corpus callosum are fully matured only towards the end of adolescence, as is the case for the higher socio-emotional-cognitive capacities (Kolb & Whishaw, 1990, 1996). Thus, in the infant brain emotions are regulated only by the amygdalae and other subcortical structures that receive direct or indirect amygdala projections. Subsequent emotional development depends mainly upon the maturation rate of the functions of the PFC and the corpus callosum. However, our knowledge of the maturation rates of these structures with regard to emotional functions is very limited. Given the principle of fixation, it is possible that, due to childhood experiences, the two components of the system regulating the emotional experience become fixated at various stages of maturity, resulting in the four extreme groups mentioned above, and in various intermediate personality types. This idea is supported by studies concerning brain plasticity during early childhood. Davidson, Jackson, and Kalin (2000) state: "Some of the most impressive evidence for brain plasticity is emotional learning (LeDoux, 1996). Plasticity in the neural circuitry underlying emotion is also likely to play an important role in understanding the impact of early environmental factors in influencing later individual differences and risk for psychopathology". These authors refer to studies in Long Evans rats, which can show two styles of maternal care, which are denoted high licking/grooming and low licking/grooming. These maternal styles are not genetically preprogrammed, but transmitted to the next generation on the basis of the received amount of licking during childhood (Francis, Dorio, Liu, & Meaney, 1999). Pups that have
received low levels of licking are more fearful and more stress-responsive in adulthood. As adults they also show neurobiological changes, which explain the increased stress responsivenes. This includes higher levels of ACTH and glucocorticoids in response to acute stress, decreased glucocorticoid feedback sensitivity and increased levels of hypothalamic ACTH-RH mRNA expression (Liu & Dirio, 1997), fewer benzodiazepine receptors in various subnuclei of the amygdala and the locus coeruleus, increased ACTH-RH receptor densities in the locus coeruleus, (suggesting enhanced hormonal stress responses), and less GABA inhibition of emotions (especially fear; Caldji et al., 1988). Comparable effects, including unstable physiological stress mechanisms (high variability in heart and respiratory rates, high blood pressure, and high cortisol levels in response to stress), have been reported in adult male rhesus monkeys of low-ranking mothers. This also suggests that these males experience more stress during their lives (Nelson, 1995). Finally, the combination of high levels of ACTH-RH and adrenal androgens with low levels of cortisol during development, as in humans with congenital adrenal hyperplasia, has been shown to result in significantly smaller amygdalae (Merke et al., 2003). It is of interest here that enhanced glucocorticoid levels have been shown to result in enhanced right hemisphere frontal activity, and greater negative affect (Schmidt, Fox, Goldberg, Smith, & Schulkin, 1999). This suggests that subjects with highly responsive adrenal cortices (as described above) will, in their reaction to emotional stimuli, show more emotionalising, more right hemisphere activation, and thus less left hemisphere activation.

These results illustrate that differences in early childhood experiences may have far-reaching consequences, leading to lifelong changes in the functioning of the central nervous system, and the formation of stable, neurologically based, personality traits. This idea is shared by other authors, including Joseph (1999), who states: "Deprived or abnormal rearing conditions induce severe disturbance in all aspects of social and emotional functioning, and effect the growth and survival of dendrites, axons, synapses, interneurons, neurons, and glia". The study of the influences of early (neurophysiological) stress responses upon the ontogeny of the affective and cognitive alexithymia dimensions will advance our understanding of alexithymia greatly.

In summary, neuropsychological data suggest that modules involved in emotionalising and modules involved in emotional cognitions tend to inhibit one another. Furthermore, modules involved in processing nonemotional information tend to inhibit both these emotion modules. On the basis of this, four extreme ways of managing emotions are conceivable. (1) Activating only neural modules involved in the cognitive processing of nonemotional information, which would result in negation of the emotions. (2) Activating only neural modules involved in the cognitive processing of emotional information, which would result in emotionally detached emotion-related cognitions. (3) Activating only neural modules involved in emotionalising, which would
result in intense emotional feelings that are not accompanied by emotional cognitions. (4) Full-blown activation of neural modules involved in emotiona-lising, followed by a full-blown activation of neural modules involved in the cognitive processing of emotional information. Persons characterised by this fourth strategy, will first go with the flow of the emotion, and only thereafter analyse their emotions. Recently, it was demonstrated that second order factor analyses of BVAQ scores resulted in two independent factors: an affective alexithymia dimension and a cognitive alexithymia dimension. This is thus compatible with the notion of four extreme groups. The characteristics of the four extreme groups based upon the two alexithymia dimensions (low affective + low cognitive; low affective + high cognitive; high affective + low cognitive; high affective + high cognitive) are consistent with the groups described above. Finally, it has also been demonstrated that these four extreme groups are each clearly linked to specific other personality traits. We therefore believe that the two alexithymia dimensions may form the foundation of other personality traits.

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