LITERATURE REVIEW

NEUROCOGNITIVE CORRELATES OF ALEXITHYMIA: A REVIEW

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Abstract

Alexithymia is a construct used to describe a cluster of cognitive and affective characteristics present in individuals in both clinical and non-clinical populations who demonstrate difficulties in processing and regulating affect. Measures of alexithymia have been found to correlate with measures of psychological distress and it is considered to be a risk factor for a range of mental and psychosomatic illnesses. This review considers the evidence for a neurocognitive basis for alexithymia, drawing primarily on studies of cognitive bias, cerebral laterality and neural imaging. There is considerable theoretical and methodological divergence between studies in this area and whilst there has been some progress in recent years, the current state of knowledge does not support a definitive model. The review concludes by highlighting methodological issues raised by past studies and offers some considerations for future research in the area.

Introduction

The alexithymia construct is used to describe a cluster of cognitive and affective characteristics typical of many people with psychosomatic illnesses and mental health problems (Taylor & Taylor, 1997). The term was first introduced by Sifneos (1973) and is derived from Greek, and means having a lack of words for emotion. The construct evolved from earlier clinical observations (e.g. Reusch, 1948) that certain individuals responded poorly to forms of psychotherapy requiring a degree of insight into inner experiences (Taylor & Taylor, 1997). As a personality trait, alexithymia is characterised by a number of salient cognitive and affective features. Notable among these are a decreased ability to identify and communicate feelings, difficulty distinguishing between feelings and the bodily sensations of emotional arousal, a cognitive tendency toward external events or cues, and a paucity of imaginative thought as evidenced by a lack of experience of dreams or fantasy (Roedema & Simons, 1999; Taylor, 2000). In terms of clinical presentation, people scoring highly on measures of alexithymia typically report decreased or flattened emotional experiences and a reduced conception of emotions as part of their awareness. There is also a marked reduction in their performance on measures of emotional recognition, empathy, labelling and behaviour (Frawley & Smith, 2001).

Since its inception the alexithymia construct has attracted an increasing amount of attention from researchers, with the primary focus resting on examining its relationship with medical and psychiatric disorders (Parker, Keightley, Smith & Taylor, 1999). Measurement amongst clinical populations

indicates it to be a correlate of a range of physical, psychosomatic (see Taylor, 2000 for detailed descriptions) and psychiatric conditions (Parker, Taylor & Bagby, 1994). Furthermore, high measures of alexithymia have been associated with predictions both of the onset of illness and disease, and of poor outcomes in terms of psychotherapeutic intervention and prognosis (Taylor & Taylor, 1997; Roedema & Simons, 1999). Whilst some authors advocate a direct causal relationship between alexithymia and illness, notably Martin and Pihl's (1985) stress-alexithymia hypothesis, research findings have yet to demonstrate this with any degree of certainty. As most of the clinical studies have been cross-sectional in design, it is difficult to ascertain whether what is being measured as alexithymia is a stable personality trait or a symptom of the relevant illness.

Whether viewed as a 'primary' personality characteristic or a phenomenon secondary to pathological processes, increasing experimental research has been directed towards neurocognitive foundations of alexithymia. Such study serves the function of gaining a deeper understanding of the construct and informing clinical practice where alexithymia forms part of an individual's presentation. Drawing on the literature relating to experimental research in this field, this review critically evaluates the evidence for a neurocognitive basis for alexithymia. The paper considers evidence from both clinical and non-clinical populations before considering conclusions that can be drawn from the current state of knowledge in the field. An outline of the search strategy undertaken in construction of this review is included in Appendix 1.

Issues of definition and measurement

Before embarking on any examination of the alexithymia construct it is important to consider what is being measured and how measurement is achieved. As a psychological concept, alexithymia shares a degree of similarity with notions such as dissociation or Salovey and Mayer's (1989) emotional intelligence construct, and with diagnostic classifications such as Asperger's Syndrome (American Psychiatric Association, 1994). Whilst an alexithymic presentation may lead to questions about diagnosis (e.g. DeLuca & Daly, 2001), alexithymia is not a diagnosis in itself. As a hypothetical construct useful for characterizing clinical presentations alexithymia has attracted a number of attempts at measurement. A range of observer-rated measures, such as the Beth Israel Hospital Psychosomatic Questionnaire (Sifneos, 1973) and the Alexithymia Provoked Response Questionnaire (Krystal, Giller & Cicchetti, 1986), and self-rated measures including the MMPI Alexithymia Scale (Kleiger & Kinsman, 1980) and the Schalling-Sifneos Personality Scale (Apfel & SIfneos, 1979) have been published over the years. Many of these scales have received criticism related to poor reliability and provide little support for the validity of the alexithymia construct (Taylor, Bagby & Parker, 1997).

The most widely accepted and utilised instrument for measuring alexithymia is The Twenty-Item Toronto Alexithymia Scale (TAS-20). The TAS-20 (Bagby, Taylor & Parker, 1994a; Bagby, Taylor & Parker, 1994b) is a revised version of the earlier 26-item version and consists of 20 self-descriptive statements. The TAS-20 contains three sub-scales that can be scored

separately: (F1) difficulty identifying and distinguishing between feelings and bodily sensations, (F2) difficulty describing feelings to others, and (F3) externally orientated thinking (Taylor, Bagby & Luminet, 2000). The TAS-20 has demonstrated good internal consistency (Cronbach's alpha=0.81) and test-retest reliability (r=0.77) and a three-factor structure theoretically congruent with the alexithymia construct (Taylor et al., 1997). Although the TAS-20 has been criticised for an uneven distribution of items between the factors, a vulnerability to response tendencies due to the imbalance between positively and negatively phrased items and not covering the ability to experience feelings or fantasy within its factorial structure (Bermond, Vorst, Vingerhoets & Gerritsen, 1999), it remains the measure most frequently adopted throughout the literature. Recent findings support the use of the TAS-20 for empirical research although suggest that separate analyses using the subscales may be of limited value (Kooiman, Spinhoven & Trijsburg, 2002). Bermond et al.'s (1999) own measure, the five-factor Amsterdam Alexithymia Scale, has received little support since its publication. Consistent with the established trend in alexithymia research, this review will only include studies that have used the TAS-20, aside from a few exceptions which will be noted in the text

Experimental studies of alexithymia

Historically, evidence for the alexithymia construct came from clinical observations of patients with a range of psychiatric and psychosomatic disorders. These observations formed the basis for a relatively large body of research

correlating measures of alexithymia with various measures of pathology (see Taylor & Taylor, 1997 for an overview). Since the early 1990s, more studies have sought to link alexithymia to proposed deficits in emotional processing through the employment of experimental designs. This has allowed researchers to test predictions about ways in which alexithymic and non-alexithymic individuals might differ (Parker, Taylor & Bagby, 1993a). Montreuil, Jouvent, Carton and Bungener (1991) reported that a comparison of psychosomatic patients and healthy controls on the Parallel Visual Information Processing Test confirmed that alexithymia was characterised by impaired recognition and recall, with alexithymics favouring concrete and non-symbolic tasks. In a study of ability to recognise affect in images of posed facial expressions, Parker, Taylor and Bagby (1993b) reported that alexithymic college students were less accurate in identifying emotions. Mann, Wise, Trinidad & Kohanski (1994) later found that a group of high alexithymic hospital staff performed significantly less well than medium or low groups on a similar task, using the earlier version of the TAS. Both of these studies contradicted the earlier findings of McDonald and Prkachin (1990) who found no relationship between alexithymia and emotion recognition in a non-clinical sample, although the use of a less reliable alexithymia measure and a small sample (n=10) in this study renders these results questionable. Lane, Sechrest, Reidel, Weldon, Kaszniak and Schwartz (1996) examined the ability to match verbal and non-verbal emotional stimuli and responses. On verbal and non-verbal tasks, alexithymic participants matched stimuli less accurately than controls, suggesting impaired emotional processing. In a study using images of facial expressions, Pandey and Mandal (1997) found no differences between alexithymic and non-alexithymic right-handed males on

matching or labelling tasks although they observed that alexithymic participants were less able to verbally describe the stimuli. Using the earlier version of the TAS, Troisi, Delle Chiaie, Russo, Russo, Mosco and Pasini (1996) found a relationship between alexithymia and non-verbal behaviour. They concluded that in comparison to controls, alexithymic participants' poor capacity for processing emotions led to reduced non-verbal expressivity and patterns of behaviour indicative of anxiety in a structured interview situation. In a recent study of emotion situation priming, Suslow & Junghanns (2002) found that high alexithymics from a non-clinical population took longer to name an emotional target word if it was preceded by a prime sentence that was descriptive of an emotionally charged situation. This was viewed as evidence of poor integration of affective–cognitive schemata, reflecting clinical observations that alexithymia is characterised by impairment in linking emotion-eliciting scenarios and emotion concepts.

Whilst there is a degree of variation in findings due in part to differences in sample groups and methodology, these studies provide experimental evidence for clinically observed components of the alexithymia construct. They represent an important step in the identification of a neurocognitive basis for alexithymia in that they provide empirical support for a relationship between the construct and deficits in information processing. However, they do not satisfactorily address underlying mechanisms for such deficits although this has been attempted in other contributions to the field.

Cognitive bias in affective processing

One contribution to understanding the apparent deficits in alexithymia has developed in the form of studies of cognitive bias. These have drawn on a number of general cognitive concepts of information processing including limited capacity models and selective attention (Williams, Watts, MacLeod & Matthews, 1998), and specific theories such as the adaptive advantage of automatic vigilance to undesirable stimuli (Fiske, 1980). Whilst most of the studies reviewed above are concerned with conscious and verbalisable aspects of affective processing, studies of cognitive bias draw on the well-supported notion that emotional stimuli may be processed without conscious awareness (Wells & Mathews, 1994; Pandey, 1995).

Emotional Stroop studies

Based on the theoretical assumption that alexithymia reflects a deficit in the capacity to cognitively process emotions, Parker et al. (1993a) tested the prediction that alexithymic individuals would be less able to attend to a task when presented with an emotionally arousing distracter stimulus. They used a modified Stroop colour-naming task with a non-clinical population and found that the alexithymic group took significantly longer than the non-alexithymic group to colour-name arousal words. As the groups did not differ in their ability to colour-name neutral words or baseline stimuli, the authors concluded that the processing delays in the arousal word condition were caused by the arousal words demanding the allocation of processing resources, making attention to the naming task more difficult (Williams et al., 1988). These results suggested an

attentional bias towards affectively salient words in the alexithymic group and a reduced capacity to modulate their response (Parker et al., 1993a). Parker et al.'s (1993a) findings were later replicated by Pandey (1995), with a non-clinical sample of bi-lingual students using the Hindi version of the TAS-20. A limitation of these two studies was the failure to assess the psychological state of the participants, as anxiety and depression, both common correlates of the TAS-20 (Honkalempi, Hintikka, Tanskanen, Lehtonen & Viinamaeki, 2001; Lundh & Simonsson-Sarnecki, 2002), have been found to contribute to impaired performance on Stroop tasks (Williams et al., 1988). In addition, the method of manually presenting words on cards and recording response times using a stopwatch raises questions about the accuracy of the response time data in both studies. In a computerised Stroop task with a non-clinical population, Sanchez and Serrano (1997) reported the opposite finding, in that alexithymics took *less* time than non-alexithymics to colour-name arousal words. Interestingly, akin to Parker et al. (1993a) they interpreted their results as providing further support for the alexithymia construct, albeit for a different reason, namely that alexithymics seemed less able to recognise emotional stimuli.

Lundh and Simonsson-Sarnecki (2002) attempted to clarify this apparent dichotomy of explanations by comparing Stroop interference effects in response to both illness words and negative emotion words. They hypothesised that in line with Taylor et al.'s (1997) assertion that alexithymia involves an impaired capacity to construct mental representations of emotions and a corresponding tendency to focus on somatic sensations, alexithymics would be slow to colour-name illness words but not negative emotion words. Using a combined Stroop

and implicit memory task with a Swedish translation of the TAS-20, Lundh and Simonsson-Sarnecki (2002) found that compared to their low alexithymia group (TAS-20≤30) the high alexithymia group (TAS-20≥50) were significantly slower to colour-name illness words than negative emotion words. This was consistent with their hypothesis that alexithymia is associated with an attentional bias toward illness-related (but not emotion-related) information. The failure to find a correlation among the whole sample may indicate that the observed effect is only applicable to those scoring highly on the TAS-20. Comparison between this and other Stroop studies is hampered by the use of a community sample containing a mixture of non-clinical participants and those with physical illnesses. After controlling for the effects of somatic anxiety, the observed effect was only marginally significant.

The Stroop studies seem to point to a degree of attentional bias to certain types of information among individuals with high levels of alexithymia, although there is disagreement about the nature of this information and what such results might indicate. Considered within the broader literature pertaining to Stroop methodology, the most widely accepted explanation would be that high alexithymia is characterised by an attentional bias toward affectively relevant stimuli. Processing these stimuli captures attentional resources, making attention to the colour-naming task more difficult (Williams, Mathews & McLeod, 1996). Suslow (1998) interpreted the results of Parker et al.'s (1993a) study as indicating that alexithymic individuals demonstrate a heightened vigilance for emotional stimuli in a similar manner to that for threatening stimuli as observed in

people with generalised anxiety disorder or post-traumatic stress disorder (McNally, Kaspi, Rieman & Zeitlin, 1990; Mogg, Matthews & Weinman, 1989).

Emotion priming studies

This theory has been tested further in a series of studies that have used the emotion priming paradigm developed from the work of Neely (1977). He suggested that the presentation of a lexical prime activated pre-existing associations that would influence the response to the presentation of a subsequent lexical target. In a preliminary study, Suslow, Arolt & Junghans (1998) used a word-word priming task with a non-clinical population, finding a negative correlation between the TAS-20 subscale 'difficulty describing feelings' and a facilitation effect of negative prime words. The authors concluded that negatively-valenced prime words facilitated response times to congruent target words but, contrary to expectation, this effect reduced with alexithymia. In addition to the low TAS-20 scores of the participants, which render any conclusions about alexithymia suspect, Suslow (2002) has since questioned these findings both on methodological and theoretical grounds. Suslow's (1998) subsequent emotion priming study, again with a non-clinical population used a pronunciation task and an evaluation task to further explore the automatic vigilance theory. In accordance with this hypothesis, two TAS-20 subscales were positively correlated with faster responses to affective stimuli. 'Difficulty describing feelings' showed a correlation with affective facilitation based on negative stimuli, and 'externally oriented thinking' and the TAS-20 sum score correlated with affective facilitation based on positive stimuli only. The interpretation of these results was that alexithymia might serve a protective

function in that an individual is able to pre-attentively process the affective valence of the communication of others and avoid communicating their own emotional state for fear of devaluation of the self. A tendency to prioritise positive affective information was interpreted as being advantageous in terms protecting an individual from conflict-laden negative material. As in Suslow et al. (1998) a rationale for selection of the stimulus words was not described and the sample had low TAS-20 scores, falling well within the non-alexithymic range as identified by Taylor et al.'s (1997) cut-off, thereby limiting clinical relevance of the findings.

Automatic vigilance effects have been associated with the adaptive advantage of an organism's ability to quickly and efficiently process threatening cues (Fiske, 1980; Pratto & John, 1991). Whilst this theory may explain affective facilitation in response to negative stimuli it does not explain Suslow's (1998) observation of an association between the TAS-20 and positive affective facilitation effects. Hermans, De Houwer and Eelen's (1996) two stage model of semantic activation was initially offered by Suslow (1998) as an explanation of these findings although these two theories predict differential priming effects. Suslow, Ohrmann and Arolt (2001) sought to examine automatic affective priming effects further in a non-alexithymic population. Their results were consistent with those from the Stroop studies, concluding that only negative primes interfered with processing of subsequent target words, undermining the semantic activation model as an account of priming effects. With the implication from this study that a bias toward evaluating negatively valenced affective stimuli exists independently from alexithymia, Suslow, Junghanns, Donges and Arolt's (2001) revised hypothesis was that automatic vigilance for negative information should

be expected in low alexithymics whilst in high alexithymics the effect should extend to both positive and negative stimuli. This was tested using a non-clinical sample, divided into high and low alexithymia groups and compared on a verbal and pictorial (emotional faces) evaluation task. The results of the verbal task confirmed the hypothesis and indicated that high alexithymics were able to evaluate affective stimuli at an automatic processing level but compared to low alexithymics they showed less processing engagement toward negative stimuli. This is consistent with Sanchez and Serrano's (1997) conclusions and the results are more compelling than previous priming studies as formation of the alexithymia groups was more rigorous and the effects of depression and anxiety were controlled for.

All the above studies of cognitive bias have used reaction times to stimuli as the dependent variable. In a recent study of a non-clinical population, Bennett (2003) extended the analysis of the emotion priming paradigm to include the effects of emotion priming on response errors as well as response times in a word evaluation task. The results of the automatic processing conditions indicated that in addition to showing slower responses to affective and neutral primes, high alexithymics made more classification errors than low alexithymics. The error rate of both groups was reduced by positive prime-target congruence and this effect also applied to negative prime-target congruence in the high alexithymic group. Whilst from the perspective of response times, the findings from this study are inconsistent with those of Suslow, Junghanns, Donges and Arolt (2001) as little evidence for an automatic priming effect was found, the

response error data offers further evidence for their notion of impaired engagement to affective stimuli at a strategic processing level.

Inconsistencies between findings in studies of cognitive bias are likely to be a function of methodology as each of the priming studies listed above have differed in terms of sample group characteristics, stimuli and the precise nature of the task completed by participants. Consequently, direct comparisons are difficult to make and more replication studies with a greater degree of methodological standardisation are required before firm conclusions can be drawn. The methodological improvements in recent studies of affective priming are encouraging although theoretical divergence in interpreting the results of different studies remains a barrier to progress.

The role of cerebral laterality in affective processing

A further group of studies has sought to locate the deficits observed in alexithymia within specific structures of the brain. There is a growing body of evidence that an alexithymic cognitive style may reflect poor integration of the information processing of the two cerebral hemispheres (Parker et al., 1999). This research stemmed from the observation that individuals with right hemisphere damage have difficulties in processing affective information, such as images of posed facial expressions (Bowers, Bauer, Coslett & Heilman, 1985) and the consequent theory that the right hemisphere is superior to the left in processing a range of emotional stimuli (Berenbaum & Prince, 1994). A variety

of theorists have attempted to explain the differential involvement of the cerebral hemispheres in emotion recognition and regulation and experimental studies of alexithymia have formed part of this enquiry. It should be noted that on the basis of evidence pointing to a lesser degree of cerebral lateralisation in left-handed individuals, many studies have chosen to exclude this group when identifying participants (Dewaraja & Sasaki, 1990; Parker et al., 1999). Parker, Taylor and Bagby (1992) reported an association between high alexithymia and a bias toward higher levels of left hemisphere arousal during ongoing cognitive processing in a study of conjugate lateral eye movements. These findings were supported by Berenbaum and Prince (1994) using a chimeric face task with a non-clinical population, although only when individuals with extreme difficulties in identifying and communicating their emotions were compared to others. On analysis of the whole sample, alexithymia was not found to correlate with hemispheric bias. Jessimer and Markham (1997) studied the relationship between alexithymia and leftward perceptual bias on chimeric tasks involving pictures of faces among a non-clinical population. They hypothesised that individuals with high alexithymia would show less leftward bias (indicative of reduced right hemisphere arousal) for affective information, as had been suggested in previous studies. They confirmed the hypothesis although the trends they reported were not specific to emotional stimuli and were more suggestive of a general association between alexithymia and reduced right hemisphere activity. Neither Berenbaum and Prince (1994) or Jessimer and Markham (1997) presented TAS-20 scores for their high and low alexithymia groups and some questions remain over the levels of significance that were accepted in the latter study.

Taylor et al. (1997) reported a shift in cerebral laterality theory and research away from a concern with cerebral asymmetry toward a view that emotional processing relies on the integration of functions provided by both hemispheres. This has led to the strengthening of an interhemispheric transfer deficit model of alexithymia, first mooted following the observation that patients with epilepsy who had undergone cerebral commissurotomies (Hoppe & Bogen, 1977; TenHouten, Warren, Hoppe, Bogen, & Walter, 1985) and a patient with agenesis of the corpus callosum (Buchanan, Waterhouse & West, 1980) exhibited alexithymic characteristics. Further support for the model came from a series of studies that used experimental tasks to examine the efficiency of interhemispheric communication. Zeitlin, Lane, O'Leary & Schrift (1989) studied combat veterans with post-traumatic stress disorder (PTSD), concluding that alexithymic veterans' performance on a tactile finger localisation task indicated a bidirectional interhemispheric transfer deficit. The attribution of this result to alexithymia is questionable given Spivak, Segal, Mester and Weizman's (1998) finding that PTSD itself is associated with a hemispheric imbalance. Zeitlin et al.'s (1989) findings were followed by Dejawara and Sasaki's (1990) study of a non-clinical population, which concluded that the results of linguistic and non-linguistic lateralised visual matching tasks demonstrated an association between alexithymia and reduced transfer of non-linguistic information. None of the afore-mentioned interhemispheric transfer studies used the TAS-20 as the measure of alexithymia, thereby raising questions about the validity of their conclusions and whether the same phenomena were being measured. However, Parker et al. (1999) sought to replicate Zeitlin et al.'s (1989) findings with a non-clinical

population using the TAS-20. Their findings were consistent with the previous study and suggested that the deficits found among the alexithymic participants were bidirectional and not located in one particular hemisphere, although the possibility of right hemisphere information processing dysfunction was not entirely excluded. The tactile finger localisation paradigm measures the transfer of sensorimotor information only, so care should be taken in generalising the results to affective stimuli (Parker et al., 1999).

Whilst right hemisphere and interhemispheric transfer deficit models are often studied and presented separately, Taylor et al. (1997) suggested that as there is much that remains to be understood concerning the function and integration of both cerebral hemispheres, it is possible that the same neurological factors may underpin the evidence that has been provided for both models. For example, they suggest that alexithymia could result from a right hemisphere dysfunction that subsequently affects interhemispheric communication. Lumley & Sielky (2000), using the tactile finger localization task and a short-term memory task with a non-clinical sample, found support for both models among male participants. Issues of generalisibility and measurement limit the strength of the conclusions that can be drawn from experimental studies of cerebral laterality, particularly about the degree to which any deficit can be seen as specific to the processing of emotions. Further replication studies are needed using the TAS-20 among both clinical and non-clinical populations, with a focus on the processing and transfer of affect-laden information.

Physiological evidence for processing deficits in alexithymia

More precise evidence for the role of specific cerebral structures in alexithymia has been offered by studies that have employed a physiological focus in their design. Lane, Reiman, Axelrod, Lang-Sheng, Holmes and Schwartz (1998) found a relationship between the Levels of Emotional Awareness Scale, known to correlate inversely with the TAS-20 (Lane & Reidel, 1998), and increased activity in the anterior cingulate cortex (ACC) using positron topography imaging. Consequently, Lane, Ahern, Schwartz and Kaszniak (1997) suggested a link between alexithymia and reduced ACC activity during emotional arousal, drawing a comparison with the phenomena of blindsight, whereby individuals are able to respond to visual stimuli without conscious awareness that they are doing so. The authors also contended that the role of the ACC in orchestrating autonomic, neuroendocrine and behavioural responses to emotional stimuli provided a basis for explaining the observed features of alexithymia, such as a tendency to focus on somatic symptoms. This theory was supported by the findings of an fMRI study by Berthoz, Artiges, Van De Moortele, Poline, Rouquette, Consoli and Martinot (2002) which showed differential ACC activity in response to positively and negatively valenced emotional stimuli. In an analysis of EEG power and coherence among high and low alexithymics from a non-clinical population, Houtveen, Bermond and Elton (1997) found that alexithymia was characterised by reduced coherence between the right frontal lobe and the left hemisphere. They suggested that their results were consistent with previous findings concerning interhemispheric transfer deficit (e.g. Zeitlin et al., 1989) and that the reduced coherence reflected reduced structural

connectivity and corpus callosal function between the right and left hemispheres. This formulation of alexithymia echoes Bermond's (1995) conception of certain manifestations of alexithymia where individuals may have emotional experiences (a predominantly right hemisphere phenomenon) but lack the accompanying cognitions (a predominantly left hemisphere phenomenon). This study used the Amsterdam Alexithymia Scale as opposed to the TAS-20. Physiological studies of brain structure and function offer a useful adjunct to experimental analogs of affective processing although they are limited by the small sample sizes employed. More studies are needed if the preliminary findings noted above are to be generalised further.

A small number of studies have examined the association between alexithymia and autonomic activity using emotion arousing stimuli. Two studies of non-clinical populations found that alexithymics indicated hypoarousal on measures such as blood pressure, heart rate (Linden, Lenz & Stossel, 1996) and skin conductance (Roedema & Simons, 1999), although Infrasca (1997) found higher skin conductance among alexithymic participants who viewed an emotional film. Inconsistency in the findings between studies may be attributed to variation in the stimuli used (Taylor, 2000). Researchers have also examined alexithymia in the context of variations in rapid eye movement (REM) density noted during sleep. Bauermann, Parker & Smith (1999) suggested that reductions in REM density as observed in alexithymic individuals imply a reduced capacity to process emotional information through dreams. Such findings have been criticised by De Gennaro, Ferrara, Curcio, Cristiani, Lombardo and Bertini (2002) as being a function of the 'first night effect', whereby adaptation to a sleep

laboratory disturbs the patterns of normal sleep. In a study that controlled for this effect, De Gennaro et al. (2002) found no association between alexithymia and poor sleep quality.

Towards an integrated neurocognitive model of alexithymia: considerations for future research

Since Taylor (2000) included a brief resume of neurobiological studies in his review of research developments in alexithymia there have been a number of further contributions across each of the areas outlined above. Whilst these have built upon previous findings and in some cases strengthened theoretical arguments, as with Houtveen et al. (1997) and the interhemispheric transfer deficit model, the case for a single definitive model of alexithymia has advanced little. The current literature is characterised by a degree of theoretical and methodological divergence that limits meaningful comparisons between many studies. However, certain themes are recurrent, notably the idea that alexithymic individuals are not 'a-emotional' but rather their experience of emotion lacks conceptual elaboration (Kreitler, 2002) and the suggestion that observed processing deficits are not necessarily specific to emotions. Both of these points relate to the notion of dissociation or de-coupling between cerebral structures responsible for the generation of affect, such as components of the limbic system, and cortical areas that modulate emotions through cognitive processes. Empirical studies designed to test predictions based on this concept would seem a profitable area for future research. Differing emphasis between authors on

certain theoretical models and the precise structures involved (e.g. Zeitlin et al., 1988; Houtveen et al., 1997; Lane et al., 1997: Suslow, Junghanns, Donges & Arolt, 2001) may reflect limitations in current knowledge with reference to the broader context of neurological science.

Another issue pertinent to future research concerns participant populations. With the ongoing debate over whether alexithymia is a primary personality trait or a state phenomenon secondary to other forms of psychological distress, many previous studies have chosen to study non-clinical populations although not all have taken care in controlling for the possible influence of psychological distress. Questions remain as to whether 'clinical' and 'non-clinical' alexithymia are the same phenomenon although there are obvious difficulties in interpreting data from clinical populations (e.g. Zeitlin et al., 1989). Findings suggesting that processing differences are more likely to be found only in non-clinical participants recording high TAS-20 scores (e.g. Berenbaum & Prince, 1994; Lundh and Simonsson-Sarnecki, 2002) reflect the importance of care in constituting experimental groups. The recruitment of participants for prospective studies is needed in future as current data comes almost exclusively from cross-sectional designs among adult populations. Such studies could allow further development of speculative theories linking alexithymia to attachment style (Taylor, Parker & Bagby, 1999) and the early formation of cognitive schema (Glucksman, 2000). It would seem that there is more to be discovered about how early emotional experiences influence subsequent neurocognitive development.

Experimental studies of alexithymia have typically involved measuring responses to affective stimuli. In its broadest sense, this basic paradigm is likely to underpin forthcoming enquiry although future researchers face the challenge of identifying which tasks or situations best illustrate alexithymic characteristics. Face recognition is a task that would seem to have high ecological validity but one cannot be certain that observed responses are specific to affect recognition rather than other processes that may occur concurrently. It can be seen from past research that variation in stimuli is at least in part responsible for differential findings (e.g. Suslow, Junghanns, Donges & Arolt, 2001) and future studies would benefit from a degree of methodological standardisation in this regard.

The theoretical and methodological variation among the studies reviewed here reflects the different levels of explanation that can be applied to the alexithymia construct. Single theories (e.g. Taylor et al., 1997; Lane et al., 1997) should perhaps be considered as preliminary in the light of limited supporting empirical evidence. Frawley and Smith's (2001) processing model represents an attempt to draw elements of existing theory and research together toward a logical overarching account of alexithymia, suggesting that it may represent failure at interfaces between a range of components within the emotional processing structures of the brain. As with other complex emotional presentations, new models and perspectives offer additional opportunities for theorising and hypothesis testing. However, akin to theories that have preceded it, further empirical testing is required before more weight can be added to Frawley and Smith's (2001) ideas. The absence of a clear, empirically supported understanding of a basis for alexithymia limits the advice that can be offered at

present to those involved in the clinical management of individuals with an alexithymic presentation. This issue has received a little attention from authors (e.g. Taylor et al., 1999; Glucksman, 2000) although further work needs to be done before firm recommendations can be made.

Conclusion

The past decade has seen a proliferation of research into alexithymia, stimulated in part by the development and widespread acceptance of a reliable tool for measurement, in the form of the TAS-20. Authors have developed an increasing interest in examining neurocognitive bases for the construct and evidence has been put forward for a number of theories that seek to explain clinical observations that alexithymic individuals seem to possess a range of deficits in processing and regulating affect. Chief amongst these are hypotheses concerning the influences of attention and cognitive bias at different levels of awareness, right hemisphere dysfunction and interhemispheric transfer deficit. Physiological theories implicating particular brain structures and differential levels of autonomic arousal are also receiving increasing attention. Whilst these research threads offer new opportunities for exploring the alexithymia construct, the divergence in theoretical starting points, models, methodology and participant populations raises some difficulties. The various theoretical developments are not necessarily mutually exclusive, although it is often not easy to meaningfully compare different studies. Consequently the current state of knowledge in the field cannot support a definitive model of the processes underlying an alexithymic

presentation. In addition, the ongoing debate over primary versus secondary alexithymia raises questions about conclusions from studies of clinical populations. In terms of informing clinical practice there is a need to understand more about the developmental history and function of alexithymia where it forms part of an individual's presentation. Despite these difficulties, neurocognitive lines of enquiry have contributed greatly to the knowledge base of the alexithymia construct and further developments seem probable as methodology improves and studies are replicated. As has been the case with the study of other affective disorders, further research evidence in this area has the potential to translate into more effective strategies for therapeutic intervention.

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Appendix 1

Search strategies.

Date	Database	Keywords / phrases	Returned[1]	
14.11.2002	PsycINFO	"Alexithymia"	1191	
		+"Neuropsychology"	5	(5)
		+"Cognitive Processes"	86	(35)
		"Suslow"	20	(5)
06.12.2002	Science Direct	"Alexithymia"	138	(12)
20.03.2003	BIDS	"Alexithymia"	5	(2)
23.04.2003	PsycINFO	"Alexithymia"	2525	
	EMBASE	+"interhemispheric"	45	(25)
	MEDLINE			
	Journals@OVID			
23.04.2003	PsycINFO	"Alexithymia"	2525	
	EMBASE	+"lateral dominance"	14	(3)
	MEDLINE			
	Journals@OVID			
23.04.2003	PsycINFO	"Alexithymia"	2525	
	EMBASE	+"controlled"		
	MEDLINE	+"processing"	50	(4)
	Journals@OVID			

[1] Numbers in brackets indicate papers that were retained for consideration in this review.

Previously retained papers found in subsequent searches were not retained again.

23.04.2003	PsycINFO	“Alexithymia”	2525	
	EMBASE	+”automatic”		
	MEDLINE	+”processing”	20	(0)
	Journals@OVID			

Further papers were obtained from reference lists of those papers that had been returned in electronic searches.

Appendix 2

Instructions for authors.

Journal of Psychosomatic Research

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Guide for Authors

Papers must be written in English. They will be acknowledged on receipt, and then reviewed. The decision on acceptance will usually be conveyed to the authors within two months.

Full Length Papers. Full length research papers will not normally be more than 4000 words in length and will preferably be shorter. Submission of a paper to the **Journal of Psychosomatic Research** will be held to imply that it represents original research not previously published (except in the form of an abstract or preliminary report), that it is not being considered for publication elsewhere, and that if accepted by the **Journal of Psychosomatic Research** it will not be published elsewhere in the same form in any language without the consent of the Publisher. Major papers of topical content will be given priority in publication.

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Review Articles. Review papers are normally 4000-5000 words. Authors are advised to consult one of the Editors with an outline before submitting a review.

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Manuscript Requirements

Manuscripts should conform to the uniform requirements known as the 'Vancouver style' (International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. N Engl J Med 1997; 336:309-315). *The Editors and Referees attach considerable importance to a succinct and lucid prose style and well organized tables. Authors whose native language is not English are advised to seek help before submission. Statistical procedures should be clearly explained.*

Manuscripts should be typed with wide margins, double-spaced on one side of standard A4 or 8.5" x 11" paper. The format should be as follows:

Title page. This should contain (a) the **title** of the article; (b) a short **running head;** (c) name of **department** where the work was conducted; (d) **names of the each author** with highest academic degree; (e) name, address, phone and fax of **author responsible for correspondence** and to whom requests for reprints should be addressed; (f) up to six **keywords** should be listed in alphabetical order after the abstract. These terms should optimally characterize the paper.

Abstract. This should be subdivided under the headings *Objective, Methods, Results, Conclusion* and should not exceed 150 words.

Text. This should be divided into sections with main headings: Introduction, Method, Results and Discussion. Accepted papers will usually be between 2000 and 4000 words in length.

Acknowledgments. These must include mention of any source of funding outside the basic funding of the host institution.

References. These should be numbered consecutively in the text in the order in which they are first mentioned and be so denoted in the list. Their form should be that adopted by the US National Library of Medicine, as used in the Index Medicus and as recognized in Uniform Requirements:

1. Ingham JC, Miller P McC. Self-referral to primary care: symptoms and social factors. J Psychosomatic Res 1986;30:49-56.

2. Berkenbosch F. Corticotrophin-releasing factor and catecholamines: a study on their role in stress-induced immunomodulation. In: Schneiderman N, McCabe P, Baum, A, eds. Perspectives in behavioral medicine. Hillsdale, New Jersey: Erlbaum 1992:73-91.

Tables. Each should be on a separate sheet, numbered consecutively in Roman numerals.

Figures A glossy photograph or clear ink drawing of each should be sent. Each figure should be numbered on the back and the top should be marked. A photocopy should be attached to each copy of the manuscript. Captions should be on a separate sheet. The number of illustrations should be kept to a minimum. Color illustrations are not normally acceptable. Authors may be asked to support the costs of color reproduction.

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