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**Development of alexithymic personality features**

Karukivi M *et al.* Development of alexithymic personality features

Max Karukivi, Simo Saarijärvi

**Max Karukivi,** Psychiatric Care Division, Satakunta Hospital District, FI-29200 Harjavalta, Finland

**Max Karukivi,** **Simo Saarijärvi,** Department of Adolescent Psychiatry, University of Turku, FI-20700 Turku, Finland

**Simo Saarijärvi,** Unit of Adolescent Psychiatry, Turku University Hospital, FI-20700 Turku, Finland

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**Correspondence to: Max Karukivi, MD, PhD,** Psychiatric Care Division, Satakunta Hospital District, Sairaalantie 14, FI-29200 Harjavalta, Finland. max.karukivi@utu.fi

**Telephone:** +358-2-6274760 **Fax:** +358-2-6274785

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**Abstract**

The purpose of this paper is to review the current literature regarding the development of alexithymic personality features. Modern brain imaging technologies provide interesting data on the associations of alexithymia with different aberrations in brain function related to emotion regulation; however, the development of these deviations is poorly understood. A notable amount of research covers the relation of alexithymia to different environmental factors. Many of these associations, for example, with low socio-economic status and general psychopathology in childhood, are well established. However, the retrospective and cross-sectional designs commonly used in these studies, as well as the use of self-report measures, hinder the ability to firmly establish causality. Certain individual developmental factors, such as lagging speech development and congenital cardiac malformations in childhood, have been associated with the development of alexithymia. Regarding the stability of alexithymia, a systematic review of the literature was conducted for this paper. In addition to being characterized as a personality feature in the general population, alexithymia also clearly has a state-like dimension that results in increases and decreases in alexithymic features in conjunction with mental disorder symptoms. An essential question is whether the alexithymic features in adulthood are, in fact, infantile features of a restricted ability to identify and describe emotions that simply persist in individuals through adolescence to adulthood. To firmly establish the roots of alexithymia development, longitudinal studies, particularly in younger populations, are needed. Furthermore, multifaceted study settings are encouraged.

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**Key words**: Alexithymia; Development; Emotion; Personality; Stability

**Core tip:** This review summarizes the current literature regarding the development of alexithymic personality features. The subject is covered from several perspectives: neurobiological, environmental, developmental, and the stability of the core alexithymic features. Regarding the stability of alexithymia, the paper includes a systematic review of the literature. On this basis, both essential issues regarding the development of alexithymia and directions for future studies are raised.

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**INTRODUCTION**

The concept of alexithymia was introduced 40 years ago and signifies a personality construct representing difficulties in identifying and expressing feelings, a scarce imagination, and an externally oriented way of thinking[1]. Although first observed in psychosomatic patients, a major factor that contributes to the keen interest in alexithymia is its association with both mental and somatic illnesses. Alexithymia has repeatedly been shown to be related to mental disorders, such as depression[2], anxiety disorders[3], eating disorders[4], and substance misuse[5]. It has also been related to somatic illnesses, including essential hypertension[6], diabetes mellitus[7], and psoriasis[8]. Alexithymic features are not limited to different patient populations; in contrast, alexithymia has been shown to be a relatively common personality characteristic in the general population. In studies conducted in the general population, the prevalence of clinically significant alexithymia in adults has been approximately 10%, and it is somewhat more common in males[9-11].

Although the introduction of the concept dates back four decades, prospective studies have been scarce. This scarcity is partly explained by the lack of reliable methods for measuring alexithymia. The current gold standard in the measurement of alexithymia is the 20-item Toronto Alexithymia Scale (TAS-20)[12,13]. This scale consists of three subscales that measure the different dimensions of alexithymia: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF) and Externally Oriented Thinking (EOT). The scale has been criticized for its lack of a limited imagination dimension and the lack of reliability of the EOT subscale; however, the wide use of the instrument supports its application. Because of these limitations, other instruments have also been developed; the most notable self-report measure is the Bermond-Vorst Alexithymia Questionnaire (BVAQ)[14]. This instrument aims to form a more complete picture of the individual’s alexithymic features, for example, regarding the emotional component of alexithymia lacked by the TAS-20. The BVAQ is a psychometrically reliable and valid instrument, that correlates reasonably well with the TAS-20 scale[15].

Measures based on self-observation have obvious limitations. Because the labeling and describing of emotions is difficult for individuals with ample alexithymic features, it has been questioned if these individuals are able to correctly assess themselves using self-reporting instruments[16]. Furthermore, the divergent validity of the scales is easily limited. For example, alexithymic features and depressive symptoms appear to be intertwined at least to some extent[17]. As a result of these limitations, interview-based methods of assessment have also been developed, such as the Observer Alexithymia Scale (OAS)[18] and the Toronto Structured Interview for Alexithymia (TSIA)[19]. The TSIA is a semi-structured interview method; it appears to correlate well with the TAS-20 scale[19], and it is a psychometrically sound instrument[20]. However, compared with self-assessment methods, interviews are time consuming, which limits the use of this type of instrument.

The development of feasible measures has provided the opportunity for quantitative research and thus, a marked increase in alexithymia studies. However, similar to all personality characteristics, alexithymia is clearly a dimensional (not categorical) concept. A common finding in clinical settings is that individuals reaching equal TAS-20 scale scores are far from homogenous. Thus, it has been suggested that different subtypes of alexithymia exist. Based on the TAS-20 scores, four subtypes of alexithymia have been suggested: general-high alexithymia, which is characterized by high scores on all three dimensions, introvert-high alexithymia, which is characterized by high DIF and DDF scores and low EOT scores, extrovert-high alexithymia, which is characterized by high EOT scores and normal DIF and DDF scores, and non-alexithymia, which is characterized by low scores on all dimensions[21]. Furthermore, based on the BVAQ scores, two subtypes of alexithymia can be distinguished: type I alexithymia, which is characterized by both low emotional experience and, consequently, poorly developed cognitions that accompany the emotions, and type II alexithymia, which is characterized by low emotionality, but well-developed emotional cognitions[14]. Although providing interesting standpoints for research, the evidence that supports the existence of these subtypes has been so far limited and somewhat controversial[22].

Despite the extensive research on the associations of alexithymia with different variables, several questions regarding the development of alexithymia remain. In the present paper, we aim to comprehensively review the current scientific research on the development of alexithymic personality features. We further discuss the extent to which the current literature supports the perspective of alexithymia as a personality trait and raise several questions that concern the understanding of the development of these features.

**GENETIC BACKGROUND**

Heiberg and Heiberg[23] (1977) were the first investigators to suggest the inheritance of alexithymic characteristics. However, the method used to measure alexithymia in their twin study is not comparable with the current standards. Over two decades later, Valera and Berenbaum[24] (2001) published another twin study in which they demonstrated that the EOT dimension was associated with genetic factors; however, their study sample was rather small. Several years later, Jørgensen *et al*[25] (2007) conducted a similar study in a large twin sample (*n* = 8785) and confirmed the association of both the TAS-20 total score and all alexithymia dimensions with genetic factors.

Gene polymorphisms that are potentially associated with alexithymia have also been studied. Ham *et al*[26] (2005) suggested a connection between alexithymia and the catechol-O-methyltransferase (COMT) Val108/158 Met gene polymorphism, but this association was challenged in a subsequent study[27]. In separate studies, alexithymia has been associated with functional variants of the brain-derived neurotrophic factor (BDNF) and DRD2/ANKK1 gene polymorphism[28] as well as a polymorphism in the serotonin (5-hydroxytryptamine) transporter-linked promoter region (5-HTTLPR)[29].

**NEUROBIOLOGICAL FACTORS**

Regarding the neurobiological correlates of alexithymia, the primary foci have been regions of the central nervous system (CNS) that are vital for emotion regulation, such as the frontal lobe and limbic system. Soon after the introduction of the alexithymia concept, “split-brain” patients were observed to have alexithymic features[30]. “Split-brain” represents the outcome of cerebral commissurotomy, where the corpus callosum is either completely or partially cleaved, leading to reduced transfer between the two brain hemispheres. This led to a hypothesis that alexithymia is a manifestation of a defect in the interhemispheric transfer. However, commissurotomy has primarily been used to treat epilepsy, and the significance of this illness alone for alexithymia has not been evaluated. Furthermore, more recent studies disagree on this issue; observations that alexithymia is both related to facilitated transcallosal inhibition[31] and reduced transcallosal inhibition exist[32].

In addition to the previously described deficit in interhemispheric communication, there have been two central attempts to model the neurobiological correlates of alexithymia: hemispheric lateralization and dysfunction in specific regions of the CNS associated with emotional regulation, such as the prefrontal cortex and the amygdala. Regarding hemispheric lateralization, alexithymia has been associated with functional asymmetry and, in particular, left hemisphere dominance[33-35]. The hypothesis is largely based on the finding that many brain functions, such as the processing of verbal or emotional information, predominantly occur in only one hemisphere[36,37]. Traditionally, emotional processing has been located in the right hemisphere, whereas logical processing is, for the most part, located in the left hemisphere[37]. Therefore, left hemisphere dominance in alexithymia would be convenient. In contrast, increased activity in the right hemisphere has also been associated with alexithymia[38]. A core problem in this model is that recent research has identified little evidence for this type of crude distribution of hemispheric functions. The human brain is a plastic organ, and it is plausible that although some brain functions tend to occur in one side of the brain, individuals do not actually possess left- or right-sided brain networks[39].

The amygdala is a central part of the limbic system that has an essential role in the processing of emotional stimuli; thus, it is understandably a point of interest for alexithymia studies. It is also heavily involved in facial expression recognition; indeed, alexithymia has been associated with lower activity in the amygdala when processing facial emotion (in particular the DIF and DDF dimensions)[40,41]. The dysfunction of both the amygdala and fusiform gyrus, a structure that also plays a central role in the early stages of facial expression processing[42], may have a significant role in the deficits of emotional awareness and social function that are related to autism spectrum disorders (ASD)[43,44]. These findings also lead to a hypothesis that these dysfunctions are the potential link between the theory of mind deficits that are typical for ASD and associated with severe cases of alexithymia. The relationship between ASD and alexithymia is discussed further in the Developmental considerations section.

The anterior cingulate cortex (ACC) is a region in the corpus callosum that, in addition to regulating autonomic and endocrine functions, is very active in emotional functioning and goal-directed behaviors[45]. In several studies, abnormal ACC function has been observed in alexithymic individuals, for example, during the perception of facial expression or the stimulation of different emotional states[35,46,47]. An interesting finding in one particular study was that while the activation of the ACC was lower in alexithymic individuals when processing emotional stimuli, the motor and somatosensoric cortices were more active[47]. This finding may be related to the known liability of alexithymic individuals to somatization[47]. Interestingly, a recent study suggests that specifically the cognitive, but not affective, component of alexithymia is associated with deficits in emotional attention and recognition[48]. Thus, while several studies suggest that alexithymia alters the way individuals perceive emotions, the specific effects on emotion regulation remain uncertain[44,48].

**ENVIRONMENTAL FACTORS**

The association of alexithymia with sociodemographic and familial factors has been extensively studied. Of the sociodemographic factors, the relation of alexithymia with low educational level, low socio-economic status and living in a rural area have been firmly established[9,11,49]. Previous research also indicates that a lack of social support is associated with alexithymia, both in adults[50,51] and adolescents[52]. However, these studies are scarce, and the causality is difficult to establish. Low social support may promote the emergence of alexithymia; however, alexithymic features may also impede the ability to build supportive relationships or the ability to utilize them.

Familial factors, such as a mother’s low education level, parental divorce, or being an unwanted child, have been associated with alexithymia[49,53]. Maternal alexithymia and general psychopathology in the family while growing up have been associated with the development of alexithymic features[54]. Inadequate parenting and childhood adversities have been repeatedly shown to impair the development of emotion regulation and are thus likely to have a significant impact on the development of alexithymic features[5,55-57].However, it has been observed that even if one parent exhibits an optimal parenting style, this may very well prevent the development of alexithymia in the child[58].

In a recent study, the degree of alexithymia was significantly associated with early neglect[59]. Although this study was also based on self-assessment, the association was very strong; thus, the authors suggested that alexithymia could be categorized into “neglect” and “non-neglect” subtypes. For alexithymic individuals with a history of emotional neglect, there appeared to be a lower acceptance for one’s own emotions and problems in the regulation of emotional states. In two other recent studies, previous traumatic experiences were significantly associated with alexithymia[60,61]. However, the cross-sectional design in all of these studies limits the generalization of the results. Overall, parental care during childhood, or rather, the lack of it, has also been associated with alexithymic features. In two separate studies, low experienced parental care has been associated with difficulties in the identification and verbalization of emotions[62,63]. However, some studies found no direct relation between reported parental care and the development of alexithymia[52,64]. In addition, there are studies that indicate that parental, in particular maternal, overprotection may have an effect on later alexithymia[52,65]. In both of these studies, maternal overprotection was associated with the dimensions of difficulties in identifying and describing feelings. This finding leads to a hypothesis that an overprotective, and hence restrictive and intrusive, mother denies psychological autonomy, which may lead to difficulties in sharing feelings with others.

However, the assessment of childhood experiences, such as traumatic experiences and parental attitudes, is difficult in cross-sectional settings because of the subjective nature and because the assessment often concerns experiences that date back several decades. As Kooiman *et al*[64] (1998) note, it is questionable whether the victims of parental neglect and abuse are suitable subjects for studies that use self-reporting instruments because they are prone to use primitive defense mechanisms.

**DEVELOPMENTAL CONSIDERATIONS**

Individual developmental factors associated with alexithymia have scarcely been studied. In one particular study, congenital cardiac malformations have been associated with a risk for alexithymic features[66]. This is due to the relationship between congenital cardiac malformations and neurodevelopmental morbidities that affect social cognition. In longitudinal studies, both Kokkonen *et al*[67] (2003) and Karukivi *et al*[68] (2012) reported the associations of alexithymia with lagging speech development in childhood[67,68]. Kokkonen *et al*[67] (2003) demonstrated that the ability to speak at the age of one year was negatively associated with adult alexithymia, in particular, with the dimension of externally oriented thinking[67]. Karukivi *et al*[69] (2012) studied the association between various developmental factors assessed at the age of five and alexithymia in late adolescence. They determined that in males, deficit in speech development at 5 years was associated with later alexithymia[68].

Previous research indicates that children with impaired speech development often have difficulties in various social situations and face problems in creating gratifying peer relationships, because of their lack of communication and regulation skills[69-71]. A central hypothesis is that these children struggle with interpreting both vocal and facial emotional cues[72,73], and thus, the association is similar to that of adults with alexithymia. It has been suggested that alexithymic individuals have adequate vocabulary to depict their feelings, but because these feelings are poorly differentiated, it is difficult for these individuals to itemize and verbalize them[74,75]. The hypothesis that children lacking language skills would have a higher risk of developing alexithymia because of their struggles in social situations is suggestible, but the limited research does not enable firm conclusions. Nevertheless, this association would only explain alexithymia in adults to a small extent.

An interesting standpoint is the connection between alexithymia and ASD. Because impaired recognition and expression of feelings are intrinsic features of ASD, it is not surprising that an association between alexithymia and autistic syndromes has been hypothesized. Indeed, an overlap of some extent between alexithymia and Asperger syndrome[76,77] has been observed. It has also been reported that clinically significant alexithymic features in the parents of children with ASD are more common than on average[78]. One hypothesis for this overlap is similar deficits in the theory of mind in both ASD and alexithymia[79]. However, the overall prevalence of ASD is approximately 1%-2%[80]; thus, a vast majority of individuals who present clinically significant alexithymic features do not fall in this category. Therefore, although the theory of mind deficit is an attractive hypothesis, it only explains alexithymia to a limited extent. Indeed, alexithymia and ASD are considered to be different constructs, but alexithymic features appear to be an idiosyncratic trait in many individuals with ASD[81].

Alexithymia has also been associated with certain irregularities in the autonomic nervous and immune systems. For example, aberrant immune responses have led Guilbaud *et al*[82] (2003) to suggest that individuals with significant alexithymic features may suffer from unnoticed chronic stress. Furthermore, dysregulation of the autonomic nervous system has been hypothesized to be related to alexithymia[83,84]. Particularly, the affective component of alexithymia has been suggested to be of importance in the regulation of sympathetic responses[85]. However, the overall results regarding the associations with the autonomic nervous system are discordant[86,87]. Taking into account that, for example, depression has been associated with aberrations in the immune system[88], this is one possibility how alexithymia is connected to mental disturbances. Additionally, it has been suggested that alexithymia may be linked to somatic illnesses through an over-activation of the hypothalamic-pituitary-adrenal (HPA) axis[89]. Although an interesting issue to debate, the current evidence for the causality between these aberrations and the development of alexithymia is practically non-existent.

As previously discussed, the prevalence of alexithymia in adults is around 10%[9-11]. From a developmental standpoint, the most interesting questions concern the age at which the prevalence settles at this level. In studies conducted in adolescent populations, the prevalence of alexithymia has varied from 7.3%[90] to 23.5%[49]. On average, the prevalence is approximately the same as in adults[91-93]. One interesting finding is that to date, in contrast to adults, no gender difference regarding the prevalence has been identified in adolescents. In two studies, different age groups of adolescents were compared, and the prevalence of alexithymia was significantly higher in younger adolescents[90,93]. However, Parker *et al*[94] (2010) have questioned the measurement of alexithymia using the TAS-20 scale in adolescents, particularly in younger age groups, because of readability issues.

Previous research has shown that owing to their lack of cognitive capacity and adequate emotion regulation skills, children typically present psychosomatic symptoms when they face anxiety-provoking circumstances[95]. Therefore, it can be hypothesized that alexithymic characteristics are normal in childhood, at least to some extent. This would explain the finding that younger adolescents appear to be somewhat more alexithymic than older adolescents. The later development of emotion regulation skills facilitates the proper identification and verbalization of emotions. Thus, it is likely that developmental stages have a significant impact on the prevalence of alexithymic features.

The major question is whether alexithymia simply persists in alexithymic individuals from childhood or if it actually develops *de novo* in later phases. Soon after the concept of alexithymia was introduced, Freyberger[96] (1977) introduced the concepts of primary and secondary alexithymia, of which the first was defined as a disposition factor, and the latter as a defense mechanism. Several subsequent studies suggested that alexithymia might develop in response to overwhelming stress to avoid experiencing agonizing and unbearable emotions[97,98]. During the previous two decades, this “state or trait” issue has been assessed in several studies discussed in the next section. Overall, the contemporary view is that alexithymia is a multifaceted construct that often includes trait and state components alike.

**STABILITY OF ALEXITHYMIC FEATURES**

To comprehensively assess the current scientific evidence for the stability of alexithymia as a personality trait, a systematic review of the literature was conducted. The inclusion criteria for the studies were as follows: (1) a longitudinal design with a non-clinical or patient sample; (2) the assessment of the stability of alexithymia as a personality trait (absolute and/or relative stability); (3) the use of a validated instrument to measure alexithymia; (4) written in English; and (5) not a review.

A systematic search was conducted using the PubMed database. The period ranged from September 1, 1995, to September 1, 2014. Using the search terms [(“alexithymia” OR “alexithymic”) AND (“stability” OR “trait” OR “reliability”)], 304 papers were identified. In the next phase, the search term was complemented by adding [“longitudinal” OR “prospective” OR “follow” OR “test-retest” OR “change”], which resulted in 87 studies. The studies were inspected manually, and 34 papers met the inclusion criteria. The studies are presented in Table 1.

In terms of stability, it is vital to differentiate the absolute and relative stabilities of alexithymia. Absolute stability refers to potential changes in individual alexithymia scores over time, whereas relative stability refers to potential relative differences among individuals. Overall, a vast majority of the studies have been conducted in patient populations, and only a few studies have assessed the stability of alexithymia in non-clinical populations. Regardless of the studied sample, most studies indicate a rather high relative stability for alexithymia, which is typical for a personality-like feature. However, as some authors have noted, there are unanswered questions regarding the relative stability of alexithymia in patient populations[103,128]. Marchesi *et al*[103] (2014), for example, state that because alexithymic features decrease simultaneously with mental disorder symptoms, we do not know if they return to the level that preceded the mental disorder.

The few studies conducted with non-clinical populations also indicate high absolute stability, whereas in clinical samples, significant sample-wise changes in the scores over time weaken the absolute stability. It is plausible that this effect is related to an association between the alexithymic features and mental disorder symptoms. Factors beyond mental disorders can weaken the absolute stability of alexithymia: the studies conducted in populations with, for example, previous myocardial infarction[126], breast cancer[114] or service as peacekeepers[107] indicate that other stressful events may cause significant changes in absolute stability over time. Overall, understanding the stability of personality features over time is difficult. For example, personality disorders have typically been perceived as chronic and treatment-resistant; however, recent longitudinal studies indicate that they remit more often and faster than typically assumed[131]. Thus, personality features may be more flexible than we originally assumed.

Although other methods of alexithymia assessment exist, the studies that assess the stability of alexithymia have been conducted almost invariably with the TAS-20 scale. Regarding the TAS-20 scale, the stability of alexithymia appears to vary depending on the studied dimension. In previous studies, EOT has been reported to be the most constant subscale in the TAS-20[113,119]. It has been suggested that this stability reflects the developmental nature of this particular subscale. It is suggested that the DIF and DDF subscales fluctuate more with mood. However, there appears to be some variation in the reported findings, and in some studies, DDF has been suggested as the most stable subscale[100,114]. Nevertheless, the differences are small, and it is difficult to draw firm conclusions. Additionally, regarding the different dimensions of alexithymia, the TAS-20 does not enable the assessment of the stability of limited imagination.

**CONCLUSION**

Despite the substantial amount of research regarding alexithymia, how and why alexithymic features develop in an individual remain inadequately understood. Although several distinct theories have been suggested from different standpoints, no comprehensive understanding has been established.

One core difficulty in research on the development of alexithymic characteristics is the heterogeneity of the determinants. The current evidence indicates that alexithymia is a personality feature in the general population that is characterized by a variety of emphases in the symptom dimensions, and it is not a categorical concept confined to a group of “alexithymics”; thus, alexithymic features are prevalent. Furthermore, to some extent, alexithymic features appear to intertwine with psychiatric symptoms, such as depression[17], or other personality variables, such as negative affectivity[132]. These findings reflect the measurement of alexithymia, where it is difficult to achieve strong divergent validity with feasible self-report measures. Despite these limitations, the extensive alexithymia research strives to systematically identify additional details of this process.

Data from the few twin studies that have been conducted suggest that alexithymia is, to some extent, inherited, and certain genetic aberrations have been reported. Modern brain imaging technologies have led to an increasing amount of data on the neurobiological correlates of alexithymia. During recent years, for example, the abnormal function of the anterior cingulate cortex in alexithymia has been identified and a notable number of studies point to alterations in the perception of emotions on the neurobiological level. However, the research evidence has been obtained, for the most part, from studies based on comparisons of brain function between alexithymic and non-alexithymic individuals in specific situations. Thus, these studies provide only limited information on the development of these aberrant dysfunctions and their clinical manifestations.

Regarding the development of alexithymic features, the most substantial amount of research covers the relation of alexithymia to different environmental factors. Many of these associations, for example, with low socio-economic status and general psychopathology in childhood, are quite well established. However, the retrospective and cross-sectional designs commonly used in these studies, as well as the use of self-report measures, hinder the ability to firmly establish causality. Aside from the methodological problems, one central issue is the relevance of these potential risk factors to alexithymia. The factors are very similar to the factors that affect common mental disorders and other psychosocial problems; thus, specific risk factors for alexithymia are difficult to break down.

Specific individual developmental factors, such as lagging speech development and congenital cardiac malformations in childhood, have been associated with later alexithymia. However, to more profoundly understand the development of alexithymia, further research in this area is needed. Several studies have assessed the stability of alexithymia. Overall, in addition to its characterization as a personality feature in the general population, alexithymia clearly also has a state-like dimension that results in increases and decreases in alexithymic features in conjunction with, for example, mental disorder symptoms. The divergent stability of the different dimensions of alexithymia must also be considered.

According to the current knowledge, the true path for the development of alexithymia remains an unanswered question. The central question that arises is to what extent infantile features of restricted ability to identify and describe emotions simply persist in alexithymic individuals throughout adolescence and adulthood. Because the roots of developmental alexithymia appear to lie in the stages that precede adulthood, more studies that assess the development of alexithymia in younger age groups are needed. Additional longitudinal studies that prospectively assess the mechanisms of the development of alexithymia and the subsequent predisposition to mental and somatic illnesses are also needed. Studies that combine different standpoints, such as neurobiological and familial factors, are potentially fruitful.

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**Table 1 Studies that assessed the stability of alexithymia in patient and non-clinical populations**

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Ref.** | **Country** | **Sample** | ***n*** | **Age group and gender** | **Measure** | **Type of stability assessed** | **Follow-up period** | **Outcome** |
| Misterska *et al*[99] 2014  | Poland | Patients with idiopathic scoliosis undergoing brace treatment | 36 | Adolescent females | TAS-26 | Absolute and relative | 12 mo | Low absolute stability, high relative stability |
| Karukivi *et al*[100] 2014  | Finland | Non-clinical | 315 | Adolescent males and females | TAS-20 | Absolute and relative | 4 yr | High absolute stability, high relative stability |
| de Haan *et al*[101] 2014  | The Netherlands | Patients with substance use disorders | 101 | Adult males and females | TAS-20 | Absolute and relative | 3 wk | Low absolute stability, moderate to high relative stability |
| Zunhammer *et al*[102]2013  | Germany | Non-clinical | 142 | Adult males and females | TAS-20 | Absolute and relative | Not available  | Moderate to high absolute stability, high relative stability |
| Marchesi *et al*[103] 2014  | Italy | Pregnant panic disorder patients (*n* = 21) and healthy controls (*n* = 256) | 277 | Adult females | TAS-20 | Absolute and relative | Not available | Low absolute stability, moderate relative stability |
| de Haan *et al*[104] 2012  | The Netherlands | Patients with substance use disorders | 187 | Adult males and females | TAS-20 | Absolute and relative | 3 mo | Low absolute stability, moderate relative stability  |
| Porcelli *et al*[105]2011  | Italy | Patients with cancer (half received a psychological intervention) | 104 | Adult males and females | TAS-20 | Absolute and relative | 6 mo | Low absolute stability, high relative stability |
| Tolmunen *et al*[106]2011  | Finland | Non-clinical | 755 | Adult males | TAS-26 | Absolute and relative | 11 yr | High absolute stability, high relative stability |
| Larsson *et al*[107] 2010  | Sweden | Non-clinical peacekeepers | 104 | Adult males | TAS-20 | Absolute and relative | 6 mo | Low absolute stability, moderate relative stability |
| Meganck *et al*[108]2010  | Belgium | Patient sample (*n* = 201) and non-clinical (*n* = 264) | 465 | Adult males and females | OAS | Relative | 2 wk | High relative stability |
| Seo *et al*[109]2009  | South Korea | Non-clinical | 22 | Adolescent males and females | TAS-20 | Relative | 4 wk | High relative stability |
| Spek *et al*[110] 2008  | The Netherlands | Patients with sub-threshold depression | 119 | Adult males and females | TAS-20 | Absolute | 12 mo | Low absolute stability |
| Marchesi *et al*[111] 2008  | Italy | Patients with major depression (*n* = 16), sub-threshold depression (*n* = 21) and without depression (*n* = 112)  | 149 | Adult females | TAS-20 | Absolute and relative | Not available | Low absolute stability, high relative stability |
| Grabe *et al*[112] 2008  | Germany | Patients admitted to psychotherapeutic treatment | 297 | Adult males and females | TAS-20 | Absolute and relative | 8-12 wk | Low absolute stability, high relative stability |
| de Timary e*t al*[113]2008  | Belgium | Patients with alcohol-dependence undergoing treatment | 70 | Adult males and females | TAS-20 | Absolute and relative | 14-18 d | Moderate absolute stability, high relative stability |
| Luminet *et al*[114] 2007  | France | Breast cancer patients | 122 | Adult females | TAS-20 | Absolute and relative | 6 mo | Low absolute stability, high relative stability |
| Moriguchi e*t al*[115]2007  | Japan | Non-clinical | 196 | Adult females | TAS-20 | Relative | 11 wk | Moderate relative stability |
| Säkkinen *et al*[93]2007  | Finland | Non-clinical | 769 | Adolescent males and females | TAS-20 | Relative | 5 wk | High relative stability |
| Rufer *et al*[116] 2006  | Switzerland | Patients with obsessive-compulsive disorder | 42 | Adult males and females | TAS-20 | Absolute and relative | 6 yr | Low absolute stability, high relative stability |
| de Vente *et al*[117] *2*006  | The Netherlands | Patients with work-related stress (*n* = 69) and a non-clinical sample (*n* = 62) | 131 | Adult males and females | TAS-20 | Absolute and relative | 16 wk | Low to moderate absolute stability, moderate relative stability |
| Salminen *et al*[118] 2006  | Finland | Non-clinical | 901 | Adult males and females | TAS-20 | Absolute and relative | 5 yr | High absolute stability, moderate to high relative stability |
| Saarijärvi *et al*[119] 2006  | Finland | Patients with major depression | 116 | Adult males and females | TAS-20 | Absolute and relative | 5 yr | Low absolute stability, high relative stability |
| Picardi *et al*[57]2005  | Italy | Non-clinical | 115 | Adult males and females | TAS-20 | Absolute and relative | 1 mo | High absolute stability, high relative stability |
| Berthoz *et al*[120] 2005  | United Kingdom | Patients with autism spectrum disorder (*n* = 19) and healthy controls (*n* = 29) | 48 | Adult males and females | TAS-20BVAQ-B | Relative | 4-12 mo | TAS-20: high relative stabilityBVAQ-B: moderate relative stability |
| Yao e*t al*[121]2005  | China | Non-clinical | 34 | Adult females and males | OAS | Relative | 2 wk | High relative stability |
| De Gucht *et al*[122]2004  | The Netherlands | Patients with medically unexplained symptoms (*n* = 313) and a non-clinical sample (*n* = 698) | 1011 | Adult males and females | TAS-20 | Relative | 6 mo | High relative stability |
| Rufer *et al*[123] 2004  | Germany | Patients with obsessive-compulsive disorder | 42 | Adult males and females | TAS-20 | Absolute and relative | Not available | High absolute stability, high relative stability |
| De Gucht[124] 2003  | The Netherlands | Patients with somatization | 318 | Adult males and females | TAS-20 | Absolute and relative | 6 mo | High absolute stability, high relative stability |
| Porcelli *et al*[125] 2003  | Italy | Patients with functional gastrointestinal disorders | 112 | Adult males and females | TAS-20 | Absolute and relative | 6 mo | High absolute stability, high relative stability |
| Kojima *et al*[126] 2001  | Canada | Patients with previous myocardial infarction | 1443 | Adult males and females | TAS-20 | Absolute and relative | 3-6 mo | Moderate absolute stability, low relative stability  |
| Luminet *et al*[127] 2001  | Belgium | Patients with major depression | 46 | Adult males and females | TAS-20 | Absolute and relative | 14 wk | Low absolute stability, high relative stability |
| Honkalampi *et al*[128] 2001  | Finland | Non-clinical | 1584 | Adult males and females | TAS-20 | Absolute and relative | 12 mo | Low absolute stability, high relative stability  |
| Honkalampi *et al*[129] 2000  | Finland | Patients with major depression | 169 | Adult males and females | TAS-20 | Absolute | 6 mo | Low absolute stability |
| Bressi *et al*[130]1996  | Italy | Non-clinical | 180 | Adult males and females | TAS-20 | Relative | 2 wk | High relative stability |

N: Number of subjects; TAS-20: 20-item Toronto Alexithymia Scale; TAS-26: 26-item Toronto Alexithymia Scale; OAS: Observer Alexithymia Scale; BVAQ-B: Bermond-Vorst Alexithymia Questionnaire, version B.