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Depression partially mediates the relationship between alexithymia and somatization in a sample of healthy children

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Abstract

A link between alexithymia and somatization has been widely established, yet little is known about different factors that may influence this relationship. Evidence supporting the idea of psychopathology as a mediator has been presented but not widely tested, particularly in children. The present study examined depressive symptoms as a mediator of alexithymia and somatization in a sample of healthy children in order to better understand the alexithymia-somatization link from a developmental perspective. Results indicated that depression significantly partially mediated this relationship, at least for two facets of alexithymia (difficulty identifying and describing feelings). Possible mechanisms, implications, and directions for future research are discussed.

Keywords

youth; depression; emotions; health psychology; mediator

Alexithymia, literally meaning “without words for emotions,” refers to difficulties in both identifying and expressing emotions (Sifneos, 1972, 1973). A strong body of evidence exists documenting the relationship between difficulties with emotion identification and expression and physical health problems (e.g., Lumley, Beyer, & Radcliffe, 2008), including adult patients with inflammatory bowel disease ($n = 108$) (Iglesias et al., 2010), rheumatoid arthritis ($n = 40$) (Fernandez, Sriram, Rajkumar, & Chandrasekar, 1989), and fibromyalgia ($n = 70$) and chronic low back pain ($n = 56$) (Tuzer et al., 2010). In adolescents, somatoform pain disorder ($n = 120$) (Burba et al., 2006) and tension headaches ($n = 32$) (Gratta et al., 2010) have been associated with higher levels of alexithymia as compared to healthy controls. Alexithymia has also been linked to somatization (e.g., De Gucht & Heiser, 2003), which is the attribution of the physiological sensations of emotions to physical illness rather than recognizing them as a component of one’s emotions and dysfunctional cognitive processes (Kooiman, Spinhoven, & Trijsburg, 2002). Yet, while the links among alexithymia, physical illness and somatization have been clearly established, little has been done to better understand the nature of these relationships, particularly from a developmental perspective, and what other factors may contribute to the connection between alexithymia and somatization (De Gucht & Heiser, 2003).

Although the exact mechanisms underlying these associations are not clear, some have hypothesized that shared neurobiological pathways for emotions and physical health may result in problems for both (Lumley, Stettner, & Wehmer, 1996). Another intriguing possibility is that alexithymic individuals fail to respond adaptively to emotional situations, thereby leading to chronic levels of negative emotions. Without emotion identification and subsequent appropriate action, individuals become mired in a pattern of negative emotions and maladaptive responses to both external and internal stimuli (e.g., pain) that have potential long-term consequences on physical and mental health (Rieffe et al., 2007). In this model, emotional distress and negative emotions may help explain some of the relationship between alexithymia and somatization.

Past research has demonstrated a strong link between alexithymia, somatization, and psychological constructs such as anxiety and depression (De Gucht & Heiser, 2003; Mattila et al., 2008). Somatization has also been shown to be associated with both alexithymia and with depressive disorders (Bailey & Henry, 2007; Garyfallos et al., 1999; Simon & VonKorff, 1991). The relationships between the constructs of alexithymia, somatization and psychopathology are complex, with some arguing that alexithymia predisposes a person to experience chronic negative mood states (Lumley, 2000), which could potentially lead to somatization. It remains to be determined whether the alexithymia-somatization connection can be fully explained by psychopathology, and at least some evidence suggests that anxiety and depression cannot fully account for this relationship (Mattila et al., 2008). However, in previous studies, only diagnoses of anxiety and depression (i.e., presence v. absence) were considered, and exploring the role of anxious and depressive symptoms might provide a clearer understanding of how psychopathology is related to alexithymia and somatization.

Lumley, Smith and Longo (2002) proposed that, with depression's link to alexithymia and somatization, depression could be a mediator of the relationship between alexithymia and somatization in chronic pain patients. These authors included both depression and alexithymia as predictors of the affective (i.e., "unpleasantness") component of pain in a hierarchical regression, and results indicated that only depressive symptoms significantly contributed to the variance in pain unpleasantness – evidence consistent with the idea of depression as a mediator of an alexithymia-pain association, although a mediational relationship was not directly tested. A direct test of depression as this proposed mediator between alexithymia and somatization is lacking, whether in clinical or healthy samples.

A major limitation of the majority of the studies described earlier is that study populations have consisted primarily of adult samples. The complex nature of the association between alexithymia, somatization, and depression may be different as individuals get older, and there is a need for further examination of these relationships in younger populations. Biological (e.g., hormonal), social, and psychological factors may be different in children and adolescents, and developmental differences between children and adults may result in different relationships or pathways between these constructs. For example, Rieffe et al. (2010) explored the relationship between alexithymia, moods (i.e., happiness, anger, sadness, and fear), and internalizing symptoms (i.e., somatic symptoms and worry/rumination) in a sample of Iranian children ages 10–15. The authors found that mood states mediated the relationship between alexithymia and internalizing symptoms, but alexithymia also contributed to internalizing symptoms above the effects of mood, suggesting that an independent relationship with alexithymia and somatization also exists. Yet, in another recent study, Jellesma, Rieffe, Terwogt & Westenberg (2009) found that the children reporting many somatic symptoms did not have an impaired ability to identify emotions, as compared to children reporting few somatic symptoms. However, those reporting many somatic symptoms did have difficulty identifying and communicating negative internal states (i.e., moods) but were better able to identify multiple emotions as compared to

children reporting few somatic complaints. Given these findings, it appears as if the exact nature of the relationship between alexithymia, emotions, and somatization is quite complex in children and warrants further investigation.

The purpose of the current study was to investigate the association between alexithymia, depression, and somatization in a sample of healthy children. Exploring these relationships in children allows us to consider the developmental aspects of how alexithymia and somatization may be related, and whether this is different in younger populations as compared to adults. We hypothesized that measures of alexithymia, depression, and somatization would all be significantly correlated, as evidenced by past research demonstrating strong relationships between these constructs (e.g., De Gucht & Heiser, 2003; Lumley, 2000; Mattila et al., 2008). Consistent with prior research in adult populations suggesting the possible mediation of alexithymia and somatization (Lumley et al., 2002), the hypothesized model (see Figure 1) posited that difficulty identifying feelings (DIF) and difficulty describing feelings (DDF) would independently predict depression (Path a) and somatization (Path c), and depression would also predict somatization (Path b). Moreover, within the hypothesized model, depression was expected to mediate the association between DIF/DDF and somatization (Path c').

Method

Participants

The data from the current study were drawn from a larger study examining sex and pubertal differences in responses to laboratory pain tasks. Participants were recruited from mass mailing, posted advertisements, and classroom presentations. Initial eligibility was confirmed by telephone: 489 individuals were screened for eligibility by telephone, but 17 children (3.5% of those screened) were excluded due to acute or chronic illness, or use of medications that could affect study outcomes. Of the 472 (96.5%) invited to participate, 228 (48%) declined participation mainly because of parental lack of interest (54%) or not enough time (21%).

A total of 244 healthy children (124 females [50.8%]; mean [SD] age, 12.7 [3.0] years [range, 8–17 years]) participated in the study. The mean ages of girls and boys (13.0 years [3.1] and 12.4 years [2.9] years, respectively) were closely matched. Ethnic composition and pubertal status are displayed in Table 1. The Institutional Review Board (IRB) and the IRB recruitment sites approved all recruitment and study procedures.

Inclusion criteria were as follows: self-reported healthy children and adolescents, aged 8 to 17 years. Participants were excluded from study participation for any of these reasons: (1) acute or chronic illness at the time of study participation; (2) developmental delay or significant anatomic impairment that would preclude understanding of study procedures (e.g., developmental age of <8 years) assessed by parents and principal investigator, or participation in pain induction procedures (e.g., arm immersion in cold water); and (3) daily use of opioid medication. Although data on the use of medications in children and parents were not formally gathered, use of analgesic medication on the day of the study participation was prohibited. In the current study, the term “healthy” referred to children who, by parent- and self-report, denied the presence of any acute or chronic illness such as a heart condition or arthritis, recent surgery on, or an injury to any limb, history of frostbite, history of fainting spells, or developmental delay.

Procedure

In brief, an investigator informed parents and children about the laboratory assessment, which was described as a study about how children experience pain. Parents and children

signed consent and assent forms, respectively. Children received a \$30 video store gift certificate and a T-shirt for their participation. On the day of the laboratory session, participants and their parents were greeted by an investigator and escorted to separate rooms. There was no contact between parents and children until the session finished. Both parents and children each completed a packet of self-report questionnaires prior to the laboratory session that included the measures used in the current study. Total time to complete all study questionnaires took approximately 60 minutes.

Measures

Toronto Alexithymia Scale – 20 item version (modified) (TAS-20) (Bagby, Parker, & Taylor, 1994). The original Toronto Alexithymia Scale included 26 items and was designed to measure three dimensions of alexithymia: difficulty identifying feelings (DIF), difficulty describing feelings (DDF), and externally-oriented thinking (EOT) (Taylor, Ryan, & Bagby, 1985). Participants are asked to indicate the extent to which they agree with each statement (e.g., “I am often confused about what emotion I am feeling”) on a 5-point scale (*Strongly disagree to Strongly agree*). The TAS-20 is a revised version of the original 26-item TAS; the TAS-20 has shown good internal consistency (Cronbach’s $\alpha = .81$), test-retest reliability (0.77), as well as good convergent and discriminant validity (Bagby, Taylor, & Parker, 1994) and is the more widely used version of the measure. Originally developed in an adult population, the TAS-20 has since been validated in a Finnish adolescent sample (Sakkinen, Kaltiala-Heino, Ranta, Haataja, & Joukamaa, 2007) and a modified version has been validated in a sample of children (Rieffe, Oosterveld, & Terwogt, 2006).

In the current study, participants were administered the 26-item TAS; however, to be consistent with previous research, we aimed to evaluate the TAS-20 version, so the analyses reported in this paper only included items from the TAS-26 that also correspond to the TAS-20. As a result, we were able to retain the full DIF subscale, although the DDF subscale had one missing item (“it is difficult for me to reveal my innermost feelings even to close friends”), as this item was included for the TAS-20 version following the publication of the TAS-26. Higher scores indicate higher level of difficulty describing feelings or identifying feelings. In the current sample, reliability estimates were acceptable (Cronbach’s α for DIF and DDF subscales were 0.73 and 0.55, respectively). The EOT subscale was not included in the analyses, given evidence that this scale has considerably lower reliability in adolescents (Rieffe et al., 2006), inadequate internal validity, and has not been shown to be significantly related to somatization and measures of emotion and affect (Devine, Stewart, & Watt, 1999; Erni, Lotscher, & Modestin, 1997; Loas, Otmani, Verrier, Fremaux, & Marchand, 1996; Rieffe et al., 2006).

Children’s Somatization Inventory (CSI) (Garber, Walker, & Zeman, 1991; Walker & Garber, 2003). This measure assesses children’s perceptions of the nonspecific somatic symptoms, and higher scores on this measure reflect endorsement of higher rates and distress about somatic symptoms. Respondents rate how much they were bothered by each of 35 symptoms (e.g., headaches) during the last 2 weeks using a 5-point scale (*not at all to a whole lot*). Adequate reliability and validity have been established. In healthy samples, internal consistency for the CSI has been shown at .92 (Garber et al., 1991), and test-retest reliability at .66 ($p < .001$) (Walker, Garber, & Greene, 1991). In the current sample, internal consistency was high (Cronbach’s $\alpha = 0.88$).

Children’s Depression Inventory (CDI) (Kovacs, 1992). The CDI is a 27-item instrument that assesses symptoms of depression, ranging from 0 (absence of symptom) to 2 (definite symptom), with higher scores indicating greater endorsement of depressive symptoms. The CDI has been demonstrated good internal consistency, adequate test-retest reliability and validity (Craighead, Curry, & Ilardi, 1995; Kovacs, 1985; Mattison, Handford, Kales,

Goodman, & McLaughlin, 1990; Nelson & Politano, 1990; Saylor, Finch, Spirito, & Bennett, 1984). The total score was calculated for CDI and used for a measure of depressive symptoms in the current study. Internal consistency of this measure was high for the current study (Cronbach's $\alpha = 0.90$).

Results

Confirmatory factor analysis

As previously mentioned, we focused on two dimensions of alexithymia as measured by the TAS-20: DIF and DDF. We first examined the factor structure of the DIF and modified DDF subscales in our sample. Items assessing these two dimensions were subjected to confirmatory factor analysis (CFA) using EQS software. CFA revealed a good two factor model fit, with $\chi^2 = 53.97$, $df = 43$, $p = 0.12$, CFI = 0.97, NNFI = 0.96, and RMSEA = 0.032. The first factor corresponded to the DIF with seven items; the second factor corresponded to the DDF with four items.

Correlation and regression analyses

Descriptive statistics for all measures are provided in Table 2. Child age was significantly correlated with both CDI scores ($r = .178$, $p < .01$) and DIF scores ($r = -.199$, $p < .01$), so this variable was controlled for in all future analyses. There were no significant differences between boys and girls on any measure. Partial correlational analyses controlling for child age were conducted for the variables of interest: alexithymia, depression, and somatization. As shown in Table 2, both facets of alexithymia (DIF and DDF) were significantly correlated with CDI and CSI scores.

Mediational model

We followed Baron and Kenny's (1986) steps to test a mediational model (see Figures 1a and 1b). First, we evaluated whether each subscale (DIF and DDF) was a significant predictor of CDI scores (Path a). Significant models emerged for both the DIF and DDF subscales. Next, we explored the relationship of DIF/DDF and CSI scores, and both were significant predictors (Path c). We evaluated whether CDI scores were significant predictors of CSI scores, when DIF/DDF scores were included in the regression (path b), and this model was also significant for DIF and DDF. The direct effect of DIF/DDF scores on CSI scores (Path c') was lower than the total effect (Path c), suggesting that inclusion of CDI scores in the model accounted for some of the variance in the relationship between DIF/DDF and CSI scores. This suggests a partial mediational model, which was confirmed with a significant Sobel test for each DIF (Sobel's test = 1.983, $p < 0.05$) and DDF (Sobel's test = 2.091, $p = 0.05$) subscales (see Figure 1).

Discussion

Previous research has suggested that alexithymic individuals are more likely to experience health problems, however the exact mechanisms underlying this relationship are unclear. We aimed to specifically examine the relationship among these variables and hypothesized that depression would at least partially explain the relationship between alexithymia and somatization.

As expected, we found strong correlations among the two subscales of alexithymia (DIF and DDF) and measures of depression and somatization. Moreover, results from our test of a mediational model indicated that depression significantly partially mediated the relationship of DIF/DDF and somatization, even though the mediation effect remained somewhat small. This suggests that depression explains part of the relationship between alexithymia and

medically unexplained physical complaints. This finding is consistent with current models explaining alexithymia and physical illness. For example, Lumley, Stettner, and Wehmer (1996) described various pathways that may link alexithymia and physical illness, including physiological, behavioral, cognitive, and social pathways, as well as possible third variables such as sociocultural and neurological factors. A psychological pathway (i.e., depressive symptoms) is another potential route that apparently links emotional difficulties and somatization, and may also overlap with the pathways listed above. Regardless, it appears that it is important to consider the role of depression in models of alexithymia and health.

The results from the current study also offer support for the model proposed by Rieffe et al. (2007), where alexithymic individuals may fail to respond adaptively to emotional situations which leads to chronic negative emotions and further maladaptive responses. This cycle of failing to respond adaptively to external and internal cues (such as somatic sensations) can later lead to both physical and mental health problems. Although Rieffe et al. (2007) did not test a mediational model, their findings suggest that the connection between health problems and difficulty identifying subtle feelings or describing feelings may be related to the degree of negative emotions experienced. Our findings build on this model by demonstrating that negative emotions appear to partly explain this relationship, at least in children. Taken together, our data appear to suggest that these relationships in children mirror those found in adult populations, and highlight the potentially stable pathways that may be established early on linking emotional and physical health problems.

Recent research has also highlighted the need to attempt to control for levels of psychopathology when examining how alexithymia and health are related, as it is possible that psychopathology could account for much of this relationship (e.g., Mattila et al., 2008). However, our data seem to indicate that, while symptoms of depression do account for some of this relationship, the path between alexithymia and somatization is not fully accounted for by depression. This is consistent with the findings from Rieffe et al. (2010), where the best fitting model of the relationship between alexithymia, mood, and internalizing symptoms (i.e., somatization and worry/rumination) suggested that somatization, although explained partly by mood, shared an independent relationship with alexithymia in a sample of 579 Iranian children ages 10 – 15. The findings from the current study go beyond those reported by Rieffe et al. (2010) by testing this model in a sample of American children and adolescents across a wider age range and including a specific, well-validated measure of depression, as opposed to mood states in Rieffe et al. (2010). However, other measures of psychopathology, cognition, or emotion regulation abilities that were not included in this or our study may help further explain this relationship by creating a larger mediation effect. In support of this, Bailey and Henry (2007) found that negative affect (defined as *both* symptoms of depression and anxiety) fully mediated alexithymia and somatization in an adult community sample ($n = 301$). It is conceivable that additional measures would provide a more complete model.

Several limitations to the current study should be noted. It is important to note that our sample population of the present study consisted of self-reported healthy children, so it is not clear whether this model would be applicable to populations with chronic physical illnesses or pain. Future research should explore whether our model holds true in a variety of clinical populations. Also, all data were self-report which could introduce bias to the extent that participants are aware of their deficits in identifying and describing feelings (Ross, 1989). Related to this, we included self-report measures of somatization and depression in a healthy sample, so there may have been a floor effect with regard to the number of symptoms endorsed. For example, Kovacs (1992) suggests a cutoff score of 19 or 20 on the CDI for nonclinical populations to indicate clinical levels of depression. Clearly, our sample mean on this measure was below this suggested cutoff (see Table 2). Additionally, there

may be some selection bias for study participation, as a large portion of the invited study sample declined to participate (48%), primarily due to lack of interest or time. This potential selection bias in the sample may have limited the generalizability of the findings. Finally, although we confirmed a meditational model, this model does not imply causation as this study was cross-sectional in nature. As mentioned previously, we tested a sample of healthy children in the current study, so it is not clear to what extent these data can be generalized to clinical populations.

These data may suggest that it may be particularly important to assess for and possibly treat symptoms of depression when working with children who have difficulty identifying and describing feelings and who also report a number of medically-unexplained symptoms. Given that the mediation effect was significant but small, other psychological factors such as anxiety and emotion regulation may also be important to consider. Nonetheless, depressive symptoms appear to play an important role in this relationship, and it is conceivable that treating symptoms of depression may help address emotional and physical symptoms.

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Figure 1. Mediation models of difficulty identifying feelings (DIF), difficulty describing feelings (DDF), depression, and somatization, controlling for child age. B = unstandardized regression coefficient; β = standardized regression coefficient. $^{**}p < .01$.

Table 1

Demographic information for study participants.

Characteristic	Total Sample (N = 244)
Male, no. (%)	120 (49.2)
Age, mean (SD), y	12.7 (3.0)
Ethnicity, no. (%)	
White	98 (40.2)
Hispanic	58 (23.8)
African American	34 (13.9)
Asian American	24 (9.8)
Other	30 (12.3)
Pubertal status, no. (%)	(N = 234) *
Early puberty	109 (44.7)
Late puberty	125 (51.2)

* Pubertal status data were unavailable for 10 participants.

Table 2
Descriptive statistics and partial correlations among alexithymia, depression, and somatization

	(2)	(3)	(4)	M	SD	Range
(1) Difficulty Identifying Feelings	0.511**	0.232**	0.313**	12.39	4.70	7 – 29
(2) Difficulty Describing Feelings		.213**	0.229**	9.82	3.34	4 – 17
(3) CDI total score			.211**	8.91	8.43	0 – 41
(4) CSI total score				12.34	11.35	0 – 98

Note: Correlations are controlling for child age. CDI = Childhood Depression Inventory; CSI = Children's Somatization Inventory.

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 $p < .01$.