

Alexithymia's Epiphenomenality as a Mediator Between Unsupportive Emotion Socialization and Later Psychopathology

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In accord with its conceptualization as a deficit, alexithymia has often been theorized and tested as a mediator of the association between childhood risk factors and later psychopathology. However, this is questioned by recent research which conceptualizes alexithymia as the result of negative perceptions of emotions, which by themselves could act as the real mediator of this association between childhood risk factors and later psychopathology. Cross-sectional data were collected through self-report questionnaires, from 193 undergraduate students. Results suggest that, in non-clinical samples, the association between an unsupportive emotion socialization alexithymia is completely mediated by avoidance-related negative attitudes towards sadness. Also, the association between emotion socialization and later psychopathology is not (in the case of borderline personality traits) or only minimally (in the case of depressive symptoms) mediated by alexithymia when these avoidance-related negative attitudes towards sadness are also included as a mediator. Theoretical and clinical implications are discussed.

Keywords: alexithymia, parental socialization of emotion, attitude toward sadness, borderline personality trait, depression

En accord avec sa conceptualisation en tant que déficit, l'alexithymie a souvent été étudiée comme un médiateur de l'association entre divers facteurs de risque durant l'enfance et la psychopathologie ultérieure. Or, cette approche est remise en question par des travaux qui conceptualisent plutôt l'alexithymie comme le résultat de perceptions négatives des émotions, lesquelles pourraient à elles seules agir comme le véritable médiateur de l'association entre facteurs de risque et psychopathologie ultérieure. Des données transversales ont été recueillies, au moyen de questionnaires auto-rapportés, auprès de 193 étudiants. Les résultats suggèrent que l'association entre socialisation émotionnelle et alexithymie est complètement médiatisée par les attitudes négatives face à la tristesse. De plus, l'association entre socialisation émotionnelle et psychopathologie n'est pas (dans le cas des traits de personnalité borderline) ou seulement minimalement (dans le cas des symptômes dépressifs) médiatisée par l'alexithymie lorsque ces attitudes négatives face à la tristesse sont également incluses comme médiateur.

Mots-clés : alexithymie, socialisation parentale des émotions, attitude envers la tristesse, trouble de personnalité limite, dépression

With a 12-month prevalence of 6.9%, depression is the third most frequent mental disorder, as well as the leading cause of death by suicide (Bachmann, 2018). It is the single most disabling of diseases, its associated burden in terms of healthy life years lost being even higher than that of dementias, alcohol use disorders and stroke (Wittchen et al., 2011). It is also associated with perturbed interpersonal patterns such

as avoidance of social situations, lack of assertiveness and being cold or distant (Barrett & Barber, 2007), As a result, it is often linked with a smaller social network as well as lower quality of relationships with family (Lewinsohn et al., 2003). Borderline personality disorder, a severe mental disorder which involves a pervasive pattern of instability in affect regulation, impulse control, interpersonal relationships, and self-image, is another mental disorder associated with severely affected psychosocial functioning (American Psychiatric Association, 2013). In terms of interpersonal problems, it is, for example, diversely associated with being vindictive, submissive, exploitable or socially avoidant (Salzer et al., 2013). While it is less prevalent than major depression, with an estimated 12-month prevalence of 1-2%, it also involves a dramatically increased suicide risk, with up to 10 % of patients committing suicide (Lieb et al., 2004).

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On a clinical level, a major focus in the study of these mental disorders lies in understanding how their early roots, for example childhood trauma, leave individuals with perturbed socio-cognitive contents or processes which end up acting as development and/or maintenance factors for these disorders; this is at the heart of the recently growing movement toward transdiagnostic approaches to psychopathology (e.g., Wilamowska et al., 2010). In other words, it is by specifying the present-moment factors which mediate the impact of distant risk factors (e.g., childhood trauma) on later mental disorders (e.g., depression, or borderline personality disorder) that one identifies potentially fruitful targets of clinical intervention.

In this regard, one of the childhood factors which appears to be a strong predictor of later psychopathology, like depression (Thomas et al., 2011) or symptoms of borderline personality disorder (Gill et al., 2018), lies in an unsupportive emotion socialization. Unsupportive emotion socialization involves parents perceiving negative emotions as harmful or aversive, and as such tending to be distressed by, to dismiss or even to punish their child's negative emotions (Denham et al., 2007; Eisenberg et al., 1998). One of the variables which has itself often been argued to mediate between an unsupportive emotion socialization and later psychopathology is alexithymia (e.g., De Panfilis et al., 2008; Thomas et al., 2011).

Alexithymia, meaning literally “lacking words for feelings”, is a multidimensional construct characterized by difficulties in identifying (DIF) and describing feelings (DDF), an externally oriented cognitive style (EOT), and a limited imaginal capacity (Nemiah et al., 1976; Taylor & Bagby, 2012) see Preece et al. (2017) for evidence concerning the exclusion of this last facet from the construct). However, alexithymia has for long been conceptualized either as a deficit or a defense, or more precisely, the result of emotion avoidance processes. In fact, it has been conceptualized with only the first view offering conceptual reasons to regard alexithymia as a mediator of the relation between an unsupportive emotion socialization and later psychopathology. In fact, the second view tends to situate emotion avoidance processes as the true mediator of this relation, seeing alexithymia as just another consequence of those emotion avoidance processes. Since the variables which that research situates as mediating variables of the association between early problematic experiences and later psychopathology tend to determine clinical targets and modes of intervention, it is of paramount importance to clarify the respective roles of alexithymia and emotion avoidance processes in this relation. To sum up what will later be more elaborately developed, the

view of alexithymia as a deficit, tends to emphasize teaching a patient to symbolize emotions (Taylor & Bagby, 2012), while the view of alexithymia as the result of emotional avoidance tends to emphasize the mobilization of individual (e.g., meditation) or interpersonal (e.g., individual therapy) practices by which the client can safely make contact with, and become more accepting of their emotions. This later focus, of note, being exemplified by acceptance or mindfulness based third-wave behavior therapies (see Edwards et al., 2017).

To summarize, the literature presents an empirical controversy, which bears important clinical implications concerning the relation of alexithymia to psychopathology and more precisely its role as a mediator of the association between early experiences such as unsupportive emotion socialization and later psychopathology. This empirical controversy is intimately linked to a long standing theoretical debate opposing the view of alexithymia as a deficit, and the view of alexithymia as the result of emotion avoidance – or the idea, as concluded by Panayiotou et al. (2015) that “alexithymic individuals may not lack the ability to experience emotion appropriately, but may have learned to avoid it”. To address this dual controversy, a short literature review contrasting the two conceptualizations of alexithymia will follow, ultimately leading to the proposition of an empirical test devised to compare these two conceptualizations of alexithymia, while clarifying its role as a mediator of the association between an unsupportive emotion socialization and later psychopathology.

Alexithymia as a deficit

The original conceptualization of alexithymia, which stemmed from the psychoanalytic observation of psychosomatic patients, emphasized the view of alexithymia as a deficit in the representation or symbolization of emotions. This is a view preserved more recently by the Toronto Group (Taylor & Bagby, 2012; Taylor et al., 1997) which sees alexithymia as reflecting a trait deficit in the cognitive-experiential processing of emotions. In this view, alexithymia fundamentally involves underdeveloped or dissociated emotional schemas (Bucci, 1997; Lane & Schwartz, 1987), which leave individuals with poorly represented and hence poorly regulated affects, increasing vulnerability to psychological illness.

As a consequence of these difficulties regulating (poorly represented) affects, individuals scoring high on alexithymia are thought to be at risk of developing mental disorders such as depression (see Li et al., 2015, for a meta-analysis); borderline personality disorder (De Panfilis et al., 2015) and personality disorders more generally (De Panfilis et al., 2008);

eating disorders (see Nowakowski et al., 2013, for a critical review); and substance use disorders (see Morie et al., 2016, for a review). Alexithymia has also often been considered as a consequence of childhood risk factors like an unsupportive emotion socialization (e.g., Aust et al., 2013; Le, Berenbaum, & Raghavan, 2002) and sexual abuse (e.g., Thomas et al., 2011), which have themselves been shown to predict psychopathology. It thus came naturally to researchers that alexithymia could be argued to act as a mediator between these childhood risk factors and later psychopathology, for example depression (Thomas et al., 2011) or personality disorders (De Panfilis et al., 2008). However, this idea is undermined by recent empirical evidence (Panayiotou et al., 2015), as well as long-standing theorizing which views alexithymia not as a deficit (in emotion representation) but as the result of emotion avoidance processes, a view to which we now turn.

Alexithymia as the Result of Emotion Avoidance Processes

It wasn't long after the delineation of the classical view of alexithymia by Nemiah et al. (1976) that other psychoanalytic theorists began to conceptualize alexithymia as the manifestation of ego defense mechanisms (e.g., denial and repression), in other words, they thought of alexithymia as a way to cope with trauma or excessive negative affect (Hogan, 1995; Knapp, 1981; McDougall, 1985). In the non-psychoanalytic literature, Bonnano and Singer (1995) and Lane et al. (2000) related alexithymia to the repressive-defensive coping style, which similarly taps into a tendency to inhibit the experience and expression of negative feelings and associated cognitions. In support of this view of alexithymia as resulting from emotion avoidance strategies are the many studies which have linked alexithymia to various emotional avoidance-related constructs, such as negative beliefs about negative emotions (Edwards et al., 2017), dissociation (Clayton, 2004), avoidant coping style (Bilotta et al., 2016), and experiential avoidance (Bilotta et al., 2016; Panayiotou et al., 2015).

This second view of alexithymia thwarts the efforts to see it as a mediator of the association between an unsupportive emotion socialization and later psychopathology. This becomes manifest when one considers recent evidence suggesting that alexithymia could itself be a consequence of the actual mediator of this association, that is, emotion avoidance processes, or the negative perceptions of emotions (i.e., as dangerous, uncontrollable, shameful or linked to negative social consequences) that both cause and result from these (Leahy, 2002). Indeed, recent studies show that avoidance-related emotional schemas, or the

experiential avoidance that they motivate, mediate the relation between childhood risk factors and alexithymia (Edwards et al., 2017), and are responsible for the link between alexithymia and symptomatology, for example somatic and depressive symptoms (Panayiotou et al., 2015). This suggests that alexithymia might not in fact mediate the association between childhood risk factors and later psychopathology, being itself a consequence of emotion avoidance schemas or processes which, by also being responsible for the association between alexithymia and psychopathology, could be the actual mediator of the association between childhood risk factors and psychopathology (see Krause et al., 2003).

In brief, previous studies on the association of alexithymia with psychopathology, and more precisely on its purported role as a mediator of the association between childhood risk factors and psychopathology, do not take into account the above reviewed theoretical and empirical support for the conceptualization of alexithymia as the result of emotion avoidance schemas or processes. To fill this gap, the present study proposes a new model, in which unsupportive emotion socialization leads to the development of negative attitudes toward emotions, which themselves, presumably through the emotional avoidance or suppression that they motivate (Krause et al., 2003; Leahy, 2002), act as development and/or maintenance factors for both psychopathology and alexithymia. In this model, alexithymia does not, by itself, constitute a causal factor in the development of psychopathology, as it results from the negative attitudes toward emotions which alone mediate most of the association between unsupportive emotion socialization and later psychopathology.

The Present Research

To sum up, the present study aims to clarify the respective roles of alexithymia and emotion avoidance schemas or processes as a mediator between unsupportive emotion socialization and later psychopathology, with coincidental implications concerning alexithymia's conceptualization as a defense or a deficit. More specifically, the reviewed literature compels us to propose that alexithymia no longer mediates the relationship between childhood risk factors and later psychopathology when its own antecedent, avoidance-related emotional schemas, is also considered as a mediator. Alexithymic individuals may have learned to avoid experiencing emotion, as opposed to simply lacking the ability to experience them appropriately (Panayiotou et al., 2015). Beyond these theoretical implications (e.g., the defense versus deficit debate concerning alexithymia), results also hold important clinical implications as they could help clarify the actual perturbed socio-cognitive contents or

processes which mediate between an unsupportive emotion socialization and later mental disorders. These processes end up acting as development and/or maintenance factors for these disorders, and should constitute transdiagnostic clinical targets (Wilamowska et al., 2010). As alexithymic patients are known to be particularly difficult to treat (Vanheule et al., 2011), the identification of the actual socio-cognitive contents or processes underlying alexithymia hold the clinical potential of optimizing therapy outcomes with this population by helping to gear interventions toward underlying socio-cognitive contents or processes underlying alexithymia.

To advance our knowledge on these theoretical and clinical issues, and in accord with the network of associations outlined by the reviewed literature, childhood risk factors will be operationalized through retrospective reports of unsupportive parental socialization of emotions. Avoidance-related emotion schemas will be operationalized through the four avoidance-related negative attitudes toward sadness shown by Boucher et al. (2013) to mediate between unsupportive emotion socialization during childhood and adult depression (perception of sadness as a form of complaining, anger against the self if sad, fear of being rejected if sad, and fear of where sadness might lead). Finally, psychopathology will be operationalized through depressive symptoms and borderline personality traits since, as mentioned, they both have been related to unsupportive emotion socialization, avoidance-related emotion schemas, as well as alexithymia.

In light of the theoretical and empirical literature reviewed above, we hypothesize that:

1. Avoidance-related attitudes toward sadness will mediate the association between unsupportive emotion socialization and alexithymia.
2. Avoidance-related attitudes toward sadness, but not the alexithymia to which it leads (as of hypothesis #1), will alone mediate the associations between unsupportive emotion socialization on one side, and depressive symptoms as well as borderline personality traits on the other, when these two variables are included as mediators in a serial mediation model.

Method

Participants and Procedure

193 students from undergraduate psychology courses at the Université de Montréal (87% female, mean age 22.4 years, range 17-62 years) were

recruited via advertisement in class and by a recruitment platform offered by the university. The potential participants received an e-mail with descriptive information about the study and a link to the survey, which contained the consent form, demographic items, and a series of questionnaires. Online consent was obtained for every participant, under protocols approved by the Université de Montréal ethics committee.

Instruments

Toronto Alexithymia Scale (TAS-20). The TAS-20 (Bagby et al., 1994a; Bagby et al., 1994b) is the most commonly used self-report instrument for measuring alexithymia. It is a 20-item inventory which uses a 5-point Likert scale (1 = *strongly disagree* to 5 = *strongly agree*) to capture an individual's perception about their emotional experience and related phenomena. Previous research has shown it to have good internal consistency, test-retest reliability, and concurrent, convergent and discriminant validity, as well as a three-factor structure consisting of difficulty identifying feelings (e.g., "*I am often confused about what emotion I am feeling*"), difficulty describing feelings (e.g., "*It is difficult for me to find the right words for my feelings*") and externally oriented thinking (e.g., "*I prefer to analyze problems rather than just describe them*"). The present research used a French translated version with similar psychometric properties (Loas et al., 1996). Only TAS-20 total scores will be used in the present study, for which internal reliability was good ($\alpha = .85$).

Questionnaire de Socialisation des Émotions (QSE). The QSE (Lecours et al., 2009) is a self-report retrospective measure of perceived parental reactions to a participant's emotions when they were a child. It is a 20-item inventory which uses a 5-point Likert scale (1 = *never true* to 5 = *very often true*) to separately measure 5 subscales (prompt: "*When I was a child...*"):

- Parental indifference (e.g., "*my parents were not interested in how I reacted to important events*")
- Parental hostility (e.g., "*my parents punished me when I was really angry*")
- Parental distress (e.g., "*my parents panicked when I started to cry*")
- An attitude of fostering cognitive evaluation, which is reversely coded when the QSE total score is calculated (e.g., "*my parents tried to understand why I was angry*")

- And parental lack of emotional communication (e.g., “my parents did not talk about their emotions”)

The questionnaire form specifies that the expression “my parents” refers to one or both parents or to the principal caretaker(s) during childhood, and participants are asked to rate an item as true even if it only fits one of the parents or caretakers. As such, the questionnaire does not produce separate indices for the two parents/caretakers. The QSE total score, which indicates the level of perceived unsupportive parental emotion socialization, is coherently associated with childhood trauma, alexithymia and symptoms, and further information concerning the development of the scale and its initial psychometric properties can be found in Boucher et al. (2013). Only QSE total scores are used in the present study, for which internal reliability was excellent ($\alpha = .92$).

Questionnaire sur l'Attitude Face à l'Émotion de Tristesse (QAFET). The QAFET (Lecours & Philippe, 2010) is a 34-item self-report inventory which uses a 7-point Likert scale (1 = *not at all* to 7 = *a lot*) to capture typical attitudes toward sadness. The QAFET has nine subscales, but only the four following subscales were used in the present study (prompt; “*Usually, when I am in a situation that can cause sadness...*”):

- Perception of sadness as a complaint (3 items, e.g., “*I tell myself that I don't want to be pitied*”)
- Anger against the self if sad (4 items, e.g., “*I find myself stupid for being affected that way*”)
- Fear of being rejected if sad (4 items, e.g., “*I tell myself that if I express my sadness, someone is going to make fun of me*”)
- Fear of where sadness might lead (4 items, e.g., “*I am scared of not knowing where my sadness could take me if I let myself being sad*”)

These are the four negative attitudes toward sadness which were shown by Boucher et al. (2013) to mediate between an unsupportive emotion socialization and depressive symptoms, the authors explaining this relation by the fact that these four attitudes can all be seen as contributing to emotional inhibition. As such, and in order to preserve statistical power, these four negative attitudes toward sadness will be averaged into a single score, from now on referred to as “avoidance-related negative attitudes toward sadness” ($\alpha = .93$). The other QAFET subscales which were not used in the present study are replacement of sadness with a positive thought, perception of sadness as useful for self-growth, perception of sadness as being harmful, denial of

sadness, and lack of interest for sadness. The QAFET total score, which indicates typical negative attitudes toward sadness, was coherently associated with measures of alexithymia, depression, anxiety, well-being, and self-esteem. Further information concerning the development of the scale and its initial psychometric properties can be found in Boucherand and colleagues (2013). In the present sample, internal reliability was good for the total score ($\alpha = .89$).

Beck Depression Inventory – Version II (BDI). The BDI-II (Beck et al., 1996) is a self-report measure consisting of 21 items that assess cognitive, behavioral, affective and somatic symptoms of depression, where each item consists of four statements reflecting the degrees of symptom severity. Scores range from zero, indicating the absence of symptom, to three, indicating a severe symptom (e.g., 0 = *I do not feel sad*; 1 = *I feel sad much of the time*; 2 = *I feel sad all of the time*; 3 = *I am so sad or unhappy that I can't stand it*). Total scores thus range from zero to 63, with higher scores indicating higher severity of symptoms. The French version that was used in this study was established by Éditions du Centre de Psychologie Appliquée (1998) and has shown strong psychometric properties in both clinical (depressed) and nonclinical samples. In the present sample, the internal consistency was excellent ($\alpha = .91$).

Maclean Screening Inventory for Borderline Personality Disorder (MSI-BPD). The MSI-BPD (Zanarini et al., 2003) is a true-false 10-item screening scale for BPD that is based on the DSM-IV criteria. There is an item for each of the first eight DSM criteria (e.g., “*Have any of your closest relationships been troubled by a lot of arguments or repeated breakups*”), and two for the paranoia/dissociation criterion. True items are summed to form a total score. Higher scores thus indicating more self-reported symptoms. Its validity has been established in multiple studies predicting from a cut-off score (seven in the original validation study) the presence of a BPD diagnostic as established through structured diagnostic interview, both in the general population (Gardner & Qualter, 2009) and in subjects with treatment histories (Zanarini et al., 2003). The French version that was used in this study was developed by the second author and is still awaiting a formal validation study. In the present sample, the internal consistency was acceptable ($\alpha = .73$).

Statistical Analyses

Statistical analyses were run using with SPSS 25.0. Descriptive and correlational analyses were first conducted. Next, Haye's (2018) PROCESS v.3.1 macro was used to conduct mediation analyses. First, a simple mediation determining if the association between an unsupportive emotion socialization (X)

and alexithymia (Y) is mediated by avoidance-related negative attitudes toward sadness (M1), and second two serial mediations (see Figure 1) determining if the association between an unsupportive emotion socialization (X) and psychopathology (Y; depressive symptomatology and borderline personality traits) is mediated by avoidance-related negative attitudes toward sadness (M1) and/or alexithymia (M2).

The PROCESS macro yields the total effect (c: effect of X on Y without the mediators), the direct effect (c': effect of X on Y controlling for all mediators), and percentile bootstrap confidence intervals (5000 samples) for the indirect effects. In the simple mediation, this is achieved by regressing: Y on X; M1 on X; and Y on X and M1 (as entered in a same block). In the serial mediations, this is achieved by regressing: Y on X; M1 on X; M2 on M1 and X; and Y on M2, M1 and X. These regressions produce individual paths' coefficients (e.g., a1,a2,b1,b2 and d21 in a serial mediation), and specific indirect effects are calculated by multiplying corresponding path coefficients. In the simple mediation model predicting alexithymia (see Figure 1a), there is one indirect effect, via avoidance-related negative attitudes toward sadness (ab). In the two serial mediation models (see Figure 1b and 1c), there are three indirect effects: via avoidance-related negative attitudes toward sadness only (a1b1), via avoidance-related negative attitudes toward sadness and then alexithymia (a1d21b2), and via alexithymia only (a2b2). An indirect effect is said to be positive to a statistically significant degree if the bootstrap CI interval does not cross zero, an approach that is now preferred (Hayes, 2009, 2018) to the traditional causal steps approach (Baron & Kenny, 1986).

Also, although the first mediation model is a replication of what Edwards et al. (2017) conceptually justified and empirically supported, tenants of the traditional vision of alexithymia as a deficit explain the association between alexithymia and defensive processes the other way around, arguing that it is alexithymia which, by leaving individuals with poorly represented affects, constrains individuals to resort to so-called primitive defenses or avoidant emotion regulation strategies (Taylor & Bagby, 2012). As such, a corresponding "reverse mediation" model will also be tested, in which alexithymia acts as a mediator of the association between an unsupportive socialization of emotions and avoidance-related negative attitudes toward sadness. This is in line with researchers' suggestion of testing such reverse mediation models and comparing the size and/or significance of the indirect effect in these alternative models to strengthen conclusions taken from mediation analyses (Gelfand et al., 2009; for an actual implementation of the strategy see for example Shrum

et al., 2011; and see Lemmer & Gollwitzer, 2017, as well as Thoemmes, 2015).

Results

Descriptive and Bivariate Analyses

An evaluation of missing data using Little's MCAR test (Little, 1988) revealed that the data may be assumed missing completely at random, $\chi^2(10) = 12.84, p > .05$. Moreover, none of the variables had more than 1% missing data. As justified by Hayes (2021), the PROCESS macro excludes missing data listwise when conducting mediation analyses.

Demographic and clinical characteristics of the sample are summarized in Table 1. On average, participants' reported levels of alexithymia ($M = 45.34, SD = 12.17, \text{range} = 20-79$) are similar to what has been observed elsewhere in college students (e.g., Edwards et al., 2017). Using the recommended cutoff score of 60 or greater (Bagby & Taylor, 1999), 9.4% of the participants have reported significant levels of alexithymia. On average, participants' reported levels of borderline personality traits ($M = 3.60, SD = 1.35, \text{range} = 0-10$) are similar to what has been observed in non-clinical samples (e.g., Gardner & Qualter, 2009). Using the MSI-BPD's developers' cutoff score of seven (Zanarini et al., 2003), 13.1% of the sample have reported clinical levels of borderline personality traits, which is similar to what has been observed elsewhere in undergraduate psychology students (Glenn & Klonsky, 2009). On average, participants' reported levels of unsupportive emotion socialization by parents ($M = 51.20, SD = 14.87, \text{range} = 23-91$) are similar to what has been observed elsewhere in college students (Boucher et al., 2013). On average, participants' reported levels of depressive symptoms ($M = 14.78, SD = 10.87, \text{range} = 0-61$) were somewhat higher than the norms (Whisman & Richardson, 2015) for both college students in general ($M = 9.14$) and female white college students in particular (which mainly compose the present sample; $M = 9.09$). Using Beck and colleagues' recommended cutoff scores (1996), 19.4% of participants reported moderate depressive symptoms (total score of 20-28) while 10.5% reported severe depressive symptoms (total score of 29-63). Also, descriptive statistics revealed BDI-II scores to be characterized by a significant positive skewness ($1.18, SE = .16$) and a significant positive kurtosis ($1.75, SE = .32$). Similarly to what has been done elsewhere (Roelofs et al., 2013; Thomas et al., 2011), BDI-II scores were thus transformed, with a square root transformation emerging as the most effective transformation (skewness = .05, $SE = .16$; kurtosis = -.07, $SE = .32$). These square roots transformed BDI-II were then used for remaining analyses. Pearson's correlations among the variables of interest are presented in Table 2.

Mediation Analyses

Figure 1 presents the three mediation models. Using the recommendations of Cohen et al. (2003), potentially errant cases in each model were investigated through three characteristics: leverage, influence and discrepancy. Examination of potentially problematic cases resulted in the exclusion of one participant, for cause of highly socially desirable and inconsistent responding. Another participant had answered the survey on two occasions, so one of her entries was randomly selected and deleted.

The first mediation model (see Figure 1a) tested whether avoidance-related negative attitudes toward sadness (ANAS) mediate the association between an unsupportive emotion socialization (QSE) and alexithymia (TAS-20). Results indicate that higher levels of QSE predict significantly higher levels of ANAS (path a), $b = .03$, $SE = .006$, 95% CI [0.02, 0.04]. In turn, higher levels of ANAS predict significantly higher levels of TAS-20 (path b), $b = 5.26$, $SE = .55$, 95% CI [4.17, 6.35]. The total effect (path c) of QSE on TAS-20 is significant ($b = .22$, $SE = .06$, 95% CI [0.11, .33]), while the direct effect (path c') is not significant ($b = .06$, $SE = .05$, 95% CI [-.04, .16]), indicating that ANAS fully mediate the association between QSE and TAS-20. The indirect effect (path ab) is significant ($b = .16$, $SE = .04$, 95% CI [.09, .24]), which supports the purported mediation. This indirect effect represents 73.39% of the total effect. Together, QSE and ANAS account for 37.31% of the variance in TAS-20 scores, $F(2, 188) = 55.95$, $p < .001$.

In the reversed model, a significant indirect effect via TAS-20 (path ab) was found for the association between QSE and ANAS ($b = .01$, $SE = .004$, 95% CI [0.006, 0.02]). Together, QSE and TAS-20 accounted for 40.12% of the variance in ANAS, $F(2, 188) = 62.99$, $p < .001$. However, in this reversed model, and by contrast with the theorized model, the direct effect (path c') remained significant ($b = .02$, $SE = .005$, 95% CI [.006, 0.02]), suggesting only partial mediation, and the ratio of indirect effect to total effect (44.26%) was lower than the one in the above theorized model (73.39%). While comparison of alternative mediation models should be done cautiously when data are cross-sectional (Lemmer & Gollwitzer, 2017; Thoemmes, 2015), this tentatively supports the causal ordering of the variables that transpired from the reviewed literature, with mutual interactions between alexithymia and negative attitudes toward sadness nevertheless remaining probable until further appropriate experimental research is conducted.

The second mediation model (see Figure 1b) investigates whether the association between QSE and depressive symptoms (BDI-II) is mediated by ANAS and/or TAS-20. The first part of the model is exactly the same as the single mediation which was tested first (compare Figure 1a and 1b). Then, higher levels of ANAS predict significantly higher levels of BDI-II (path b1), $b = .57$, $SE = .08$, 95% CI [.42, .72]), and higher levels of TAS-20 also predict significantly higher levels of BDI-II (path b2), $b = 0.02$, $SE = .008$, 95% CI [0.002, 0.3]). The total effect (path c) of QSE on BDI-II is significant ($b = .03$, $SE = .007$, 95% CI [.02, .05]), while the direct effect (path c') is not significant ($b = .01$, $SE = .006$, 95% CI [-0.0002, .02]), indicating that ANAS and TAS-20 fully mediate the association between QSE and BDI-II. Examination of the indirect effects reveals two significant indirect effects (see Table 1 for a summary). The first one runs through ANAS only (path a1b1), $b = .018$, $SE = .004$, 95% CI [.0010, .026]. The second runs through ANAS and TAS-20 (path a1d21b2), $b = .003$, $SE = .002$, 95% CI [.0004, .006]. By contrast, the indirect effect going through TAS-20 only (path a2b2) is not significant, $b = .001$, $SE = .001$, 95% CI [-.001, .004]. Comparison of the two significant indirect effects to each other reveals that the indirect effect through ANAS only (85.71% of total indirect effect) is significantly larger than the indirect effect through ANAS and TAS-20 (14.29% of total indirect effect), $b = .01$, $SE = .004$, 95% CI [.007, .02]. Together, QSE, ANAS and TAS-20 account for 45.51% of the variance in depressive symptoms scores, $F(3, 187) = 52.06$, $p < .001$.

The third mediation model (see Figure 1c) investigates whether the association between a QSE and borderline personality traits (MSI-BPD) is mediated by ANAS and/or TAS-20. The first part of the model is exactly the same as the single mediation which was tested first (compare Figure 1a and 1c). Then, higher levels of ANAS predict significantly higher levels of MSI-BPD (path b1), $b = .74$, $SE = .14$, 95% CI [.46, 1.02]), while higher levels of TAS-20 do not (path b2), $b = .02$, $SE = .02$, 95% CI [-.01, .05]). The total effect (path c) of QSE on MSI-BPD is significant ($b = .06$, $SE = .01$, 95% CI [.04, .09]), and the direct effect (path c') is still significant ($b = .04$, $SE = .01$, 95% CI [-.02, .06]), indicating only partial mediation. In congruence with the individual paths, examination of the indirect effects reveals only one significant indirect effect (see Table 1 for a summary), which goes through ANAS only (path a1b1), $b = .022$, $SE = .006$, 95% CI [.01, .04]). That is, the indirect effect through ANAS and TAS-20 (path a1d21b2), is not significant, $b = .003$, $SE = .003$, 95% CI [-.002, .01]). And neither is the indirect effect going through TAS-20 only (path a2b2), $b = .001$, $SE = .002$, 95% CI [-.001, .005]. Together, QSE, ANAS and TAS-20

account for 33.19% of the variance in MSI-BPD, $F(3, 187) = 30.97, p < .001$.

Results could thus be summed up as follows. In support of hypothesis #1, the first mediation model (see Figure 1a) revealed a full mediation by avoidance-related negative attitudes toward sadness of the association between unsupportive emotion socialization and alexithymia, while the reversed model revealed only a partial mediation by alexithymia of the association between unsupportive emotion socialization and avoidance-related negative attitudes toward sadness. And in support of hypothesis #2, the second and third mediation models revealed that the association between unsupportive emotion socialization and later psychopathology is only (in the case of borderline personality traits) or mostly (in the case of depressive symptoms) mediated by alexithymia's own antecedent, avoidance-related negative attitudes toward sadness, with alexithymia playing no or only a small further mediating role.

Discussion

The main goal of the present study was to examine the possibility that alexithymia, by contrast with what has often been tested, does not, in fact, mediate the association between childhood risk factors and later psychopathology, being itself a consequence of emotion avoidance schemas or strategies which act as the actual mediator of this association between childhood risk factors and psychopathology (see Krause et al., 2003).

Findings first revealed, in replication of Edwards et al. (2017), that avoidance-related negative attitudes toward sadness (i.e., perception of sadness as a form of complaining, anger against the self if sad, fear of being rejected if sad, and fear of where sadness might lead) completely mediate the association between unsupportive emotion socialization and alexithymia. Of note, Edwards et al. (2017) broader measure of negative perceptions of feelings at large (Leahy, 2015) did correlate more intimately with TAS-20 scores ($r = .73$ and $.74$, depending on the sample) than the four avoidance-related negative attitudes toward sadness which were used in the present study ($r = .59$). However the present results suggest that negative perceptions of sadness specifically are similarly enough to completely mediate the association between unsupportive emotion socialization and alexithymia.

Also, going further than Edwards et al. (2017), the corresponding reversed mediation was tested, in order to investigate the alternative directionality rather emphasized by the traditional view of alexithymia as a deficit (Taylor & Bagby, 2012). This reversed mediation model, in which alexithymia is tested as a mediator of the association between an unsupportive

emotion socialization and avoidance-related negative attitudes toward sadness, revealed only partial mediation, and a ratio of indirect effect to total effect which was substantially lower than that of the hypothesized model. Although only experimental designs will truly establish causal ordering of the variables, this tentatively supports the directionality which transpires from the literature on alexithymia as a learned emotional avoidance strategy, and which, as such, was tested by Edwards and colleagues (2017).

In other words, findings indicate that an unsupportive emotion socialization, in which a child's negative emotions are met with indifference, hostility or distress for example, leads to the development of negative attitudes toward negative emotions. In turn, these negative emotional schemas or meta-emotions, presumably through the emotional avoidance or suppression that they motivate (Leahy, 2002; Krause et al., 2003; Mitmansgruber et al., 2009), lead to a state in which feelings, being inhibited, are hard to identify or describe. Results thus suggest, in the present sample at least (see discussion below), that it is not so much that alexithymia constrains individuals to resort to so-called immature defenses or avoidant emotion regulation strategies, as claimed by the traditional view of alexithymia as a deficit (Taylor & Bagby, 2012), but rather that alexithymia itself results from a learned emotional avoidance strategy. As stated by Panayiotou et al. (2015), "alexithymic individuals may not lack the ability to experience emotion appropriately, but may have learned to avoid it". This learned emotional avoidance strategy, importantly, could not only have resulted from a more benign internalization of the parents' own maladaptive emotional schemas which guide their emotion socialization practices (Gottman et al., 1997), but could also have been more directly adaptive in certain harsher childhood contexts. For example, it could have allowed dissociating from unavoidable physical or emotional pain resulting from an abusive caregiver (Foa & Hearst-Ikeda, 1996), and/or more instrumentally allowed, as a matter of survival, to preserve the relationship with a caregiver who reacts with hostility, rejection or distress to a child's negative emotions (Bowlby, 1969, 1973, 1980). This view of alexithymia is in accord with recent results from Bilotta et al. (2016), who report first a negative interaction between avoidant coping and negative affect in the prediction of alexithymia levels, and second the mediation of this interaction effect by experiential avoidance. In other words, alexithymia levels in their sample were driven by an experiential avoidance which reflected either a more ingrained preference for avoidant coping or a more situational defense against negative affect. Such results are also in line with studies suggesting a lack of independence between alexithymia and the PTSD symptom cluster

of numbing/avoidance (Badura, 2003; Eichhorn et al., 2014).

This view of alexithymia as reflecting a learned emotional avoidance strategy is also supported by the second set of findings, which pertain more directly to the main goal of the present study. Results revealed that the association between unsupportive emotion socialization and later psychopathology is only or mostly mediated by alexithymia's own antecedent, avoidance-related negative attitudes toward sadness, with alexithymia playing no or only a small further mediating role. In the serial mediation model predicting borderline personality traits, only the indirect effect through avoidance-related negative attitudes toward sadness appeared to be significant. And in the serial mediation model predicting depressive symptoms, both the indirect effect through avoidance-related negative attitudes toward sadness only and the indirect effect going serially through avoidance-related negative attitudes toward sadness and alexithymia appeared to be significant, with the single mediation nevertheless being significantly larger than the serial mediation (85.71% vs. 14.29% of total indirect effect, respectively).

Taken together, this suggests that unsupportive emotion socialization leads to the development of negative attitudes towards sadness. In turn these negative emotional schemas or meta-emotions, presumably through the emotional avoidance or suppression that they motivate (Leahy, 2002; Krause et al., 2003; Mitmansgruber et al., 2009), constitute a psychological vulnerability for the development of depressive symptoms and borderline personality traits, while also leading to a state in which feelings, being inhibited, are hard to identify or describe. Alexithymia then itself does not predict much variance in the intensity of depressive symptoms or borderline personality traits, as it results from the negative attitudes towards sadness which alone mediates most of the association between unsupportive emotion socialization and later psychopathology.

With the important limitation that the present study measured negative attitudes toward sadness, rather than negative attitudes toward feelings in general (Leahy, 2002) or the emotional inhibition which presumably results from them, the present findings seem to be in line with past research which has argued experiential avoidance to constitute a generalized psychological vulnerability for various disorders (Kashdan et al., 2006; Masuda & Tully, 2011; Panayiotou et al., 2015). Again, with the same limitation in mind, results also seem to be in line with past research which has proposed seeing in alexithymia the result of emotional avoidance

strategies (Berrocal et al., 2009; Bilotta et al., 2016; Panayiotou et al., 2015; Venta et al., 2012).

At first sight, the present results would seem to contradict previous studies which have argued alexithymia to causally mediate between childhood risk factors and later psychopathology, for example depression (Thomas et al., 2011), psychological distress (e.g., Hébert et al., 2018), or personality disorders (De Panfilis et al., 2008). That is, alexithymia's apparent mediating effect could have constituted what Hayes (2018) refers to as an "epiphenomenal association": a variable seems to mediate between X and Y, but only because it correlates with the (non-measured) actual mediating variable (negative attitudes towards sadness, in the present case). However, it should be stressed that the present study was conducted in a mostly non-clinical sample of university students, and that it investigated an unsupportive emotion socialization as a childhood risk factor. By contrast, two of the above-mentioned studies (Thomas et al., 2011; De Panfilis et al., 2008) were conducted in mostly clinical samples, and two investigated child sexual abuse (Hébert et al., 2018; Thomas et al., 2011), a probably more traumatic childhood risk factor than an unsupportive emotion socialization. As such, it remains possible that alexithymia could tend to reflect emotional avoidance in non-clinical and well-functioning individuals such as university students, while still reflecting a true deficit in clinical samples of lower functioning, more traumatized individuals, for example in the psychosomatic patients whose highly concrete thought inspired the construct's development (Nemiah & Sifneos, 1970). That is, alexithymia might be characterized by the concept of equifinality (Cicchetti & Rogosch, 1996), with different processes leading to the same end state. Another example suggesting that the concept of equifinality might characterize alexithymia resides in the fact that alexithymia has been related to both attachment avoidance/deactivating strategies (e.g., O'Loughlin et al., 2018), and attachment anxiety/hyperactivating strategies (e.g., Riem et al., 2018), but for seemingly different reasons (Shaver & Mikulincer, 2014). Indeed, while attachment avoidance might be associated with alexithymia because of the ingrained inattention to feelings by which it is characterized, the association between attachment anxiety and alexithymia might reflect not so much this lack of emotional awareness, but more specifically the difficulties in differentiating between different emotions (Stevens, 2014) which result from a chaotic emotion structure, in which the activation of focal emotions spreads to non-focal emotions (Shaver & Mikulincer, 2014). In brief, different underlying processes might manifest as the same end state, namely similar levels of alexithymia,

at least as self-reported through measures like the TAS-20.

Another interesting finding from the present results lies in the fact that avoidance-related negative attitudes toward sadness and alexithymia, while completely mediating the association between unsupportive emotion socialization and depression, only partially mediated the association between unsupportive emotion socialization and borderline personality traits. This could be explained by the more interpersonally-relevant constructs that have been proposed to act as crucial mediators between childhood risk factors and borderline personality. Two examples of such constructs are mentalization (Fonagy & Luyten, 2009), which as opposed to alexithymia concerns the inferences about not only one's own but also others' mental states, or more recently epistemic mistrust (Fonagy et al., 2015), a mistrust of social information which could have originally been adaptive in the context of maltreatment, but which now prevents one from changing through interpersonal experience. As such, it would be interesting to replicate the present study and see if the inclusion of one of these constructs as an additional mediator, for example with the Reflective Functioning Questionnaire (Fonagy et al., 2016), allows to reach complete mediation of the association between unsupportive emotion socialization and borderline personality traits.

Still keeping in mind the considerations presented above about the possible diversity of alexithymic processes depending on the studied population, the present findings also hold clinical implications. Results suggest avoidance-related negative attitudes toward sadness are an etiological or maintaining factor in alexithymia, depressive symptoms and borderline personality, and as such the targeting of these attitudes appears as a good therapeutic target for all these three conditions. However, the following discussion will concentrate on alexithymia, because of the known difficulty of psychotherapeutic interventions with such patients. Indeed, possibly in part because the paucity and concreteness of alexithymics' affective communications tend to evoke negative reactions of frustration and boredom in therapists (Ogrodniczuk et al., 2011), patients with high alexithymia have long been recognized to be particularly difficult to treat (Grabe et al., 2008; Vanheule et al., 2011). Interventions guided by the view of alexithymia as a deficit (i.e., as involving underdeveloped emotion schemas) tend to emphasize teaching a patient to symbolize emotions, through the linking of bodily sensations and action tendencies to images and words (Taylor & Bagby, 2012). Although this more educational type of work would seem appropriate for extremely alexithymic patients like the ones in

psychosomatic units which prompted the original elaboration of the construct (Nemiah & Sifneos, 1970), the view of alexithymia suggested in the present sample rather emphasizes addressing the negative attitudes toward sadness which drive alexithymia levels. More precisely, the emphasis shifts from teaching a patient to symbolize his emotions, to creating a therapeutic context in which the patient can safely make contact with and become more accepting of their emotions. That is, the focus shifts to having the patient expand their "windows of affect tolerance" (Schore, 2012), so as to abandon the generalized reliance on avoidant/"disengaging" emotion regulation strategies which, however adaptive they might have been and still could be in certain situations (Bonanno & Burton, 2013; Sheppes et al., 2014), ultimately perturb long-term adaptation when they are inflexibly used.

As mentioned by Edwards et al. (2017), this refocusing on negative attitudes toward emotions mostly points in the direction of third-wave behavior therapies, or even just individual meditative practices, which through acceptance, mindfulness and/or experiential strategies explicitly target dysfunctional attitudes toward emotions and the associated experiential avoidance. Examples of such third-wave behavior therapies are Emotional Schema Therapy (Leahy, 2015), Acceptance and Commitment Therapy (Hayes et al., 2012) and Mindfulness-based cognitive therapy for depression (Segal et al., 2012). Importantly, alexithymia levels have been shown to decrease in non-clinical samples following mindfulness or other contemplative training interventions (Bornemann & Singer, 2017; de la Fuente Arias et al., 2010; Santarnecchi et al., 2014), which interventions include not so much the educational work suggested by the view of alexithymia as a deficit (i.e., teaching a patient how to symbolize emotions) but, more fundamentally, the cultivation of an intentional and accepting, non-judgmental awareness of bodily sensations, feelings and thoughts. This highlights how, at least in non-clinical samples like those in the three-interventions studies just mentioned, the main factor in alexithymia, and thus also therapeutic target, could reside in negative attitudes toward emotions and experiential avoidance, more than in underdeveloped emotion schemas.

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Appendix

Table 1

Demographic and clinical characteristics

Measure	<i>N (%)</i>	<i>M (SD)</i>
Gender (female)	87	
Age		
<20	22.5	
20-24	59.7	
25-29	11.0	
≥30	6.8	
Ethnicity		
Caucasian/White	77.5	
Asian	7.9	
Arab	5.8	
Black/African	3.1	
Hispanic	2.6	
Other	3.1	
TAS-20		45.34 (12.17)
BDI-II		14.78 (10.87)
MSI-BPD		3.60 (2.50)
ANAS		3.42 (1.35)
QSE		51.20 (14.87)

Note. *SD* : standard deviation; TAS-20: *Toronto Alexithymia Scale*; BDI-2: *Beck Depression Inventory-II*; MSI-BPD: *Maclean Screening Inventory for Borderline Personality Disorder*; ANAS: avoidance-related negative attitudes toward sadness on the *Questionnaire sur l'Attitude Face à l'Émotion de Tristesse*; QSE: *Questionnaire de Socialization des Émotions*.

Table 2*Pearson correlations of study variables*

	1	2	3	4	5	6	7	8	9
1. QSE	—								
2. ANAS	.34***	—							
3. REJ	.32***	.83***	—						
4. FEAR	.29***	.83***	.56***	—					
5. ANGER	.22**	.88***	.64***	.70***	—				
6. COMP	.31***	.84***	.60***	.58***	.64***	—			
7. TAS-20	.27***	.59***	.52***	.47***	.52***	.49***	—		
8. BDI-II sqrt	.33***	.65***	.51***	.59***	.58***	.55***	.51***	—	
9. MSI-BPD	.38***	.53***	.41***	.50***	.47***	.42***	.39***	.58***	—

Note. QSE: *Questionnaire de Socialization des Émotions*; ANAS: index of avoidance-related negative attitudes toward sadness on the *Questionnaire sur l'Attitude Face à l'Émotion de Tristesse* (QAFET); REJ: fear of being rejected if sad on the QAFET; FEAR: fear of where sadness might lead on the QAFET; ANGER: anger against the self if sad on the QAFET; COMP: perception of sadness as a complaint on the QAFET; TAS-20: *Toronto Alexithymia Scale*; BDI-II sqrt: square root of total scores on the *Beck Depression Inventory-II*; MSI-BPD: *Maclean Screening Inventory for Borderline Personality Disorder*. * $p < .05$; ** $p < .01$; *** $p < .001$

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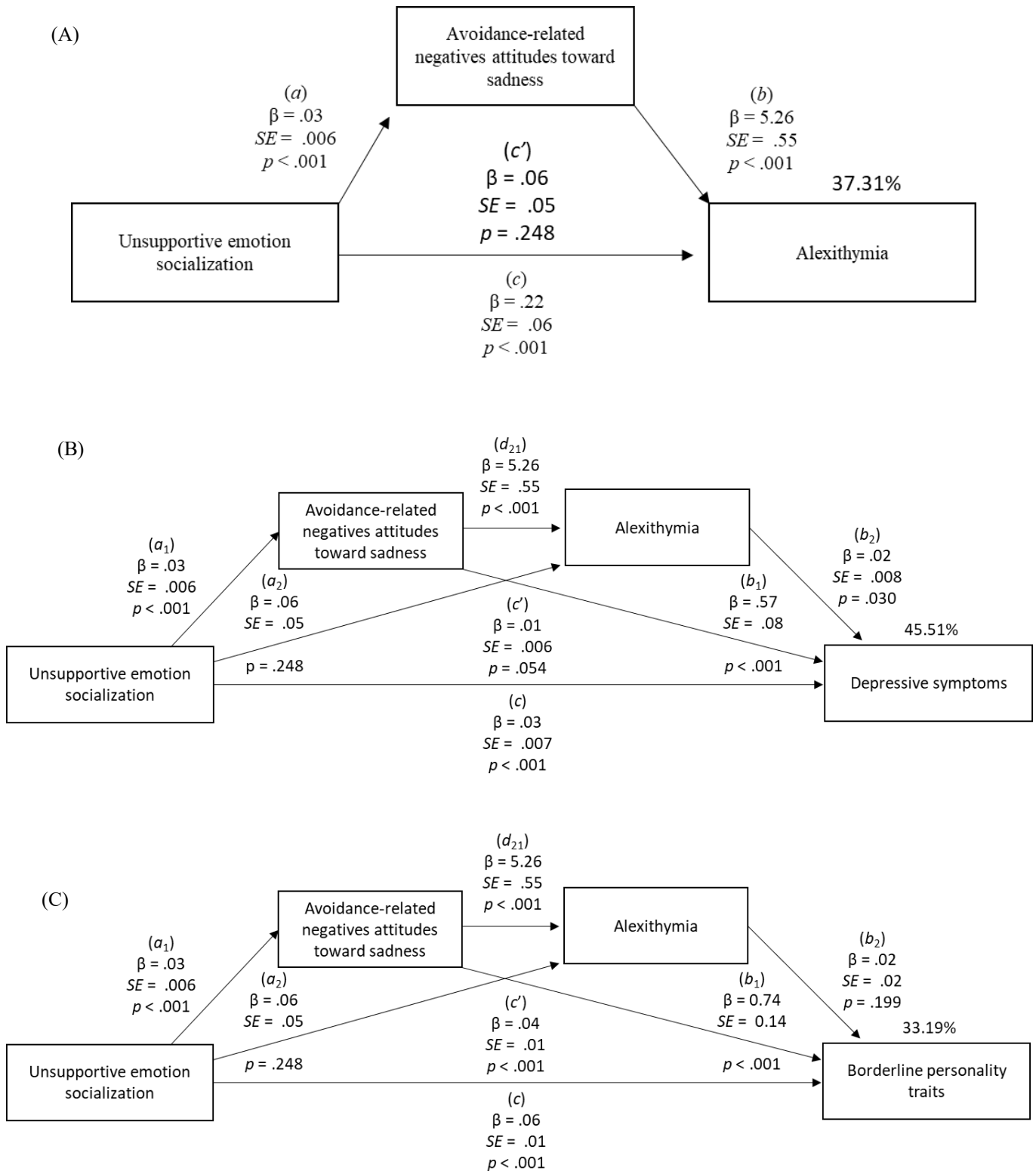


Figure 1. Mediation models. Direct effects are presented above the horizontal line, while total effect are presented below the horizontal line.