Alexithymia, emotion dysregulation, impulsivity and aggression: A multiple mediation model

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A B S T R A C T

There is a need to better understand the antecedent of aggressive behaviors in order to tailor treatments and reduce the associated damage to the others and the self. Possible mechanisms underlying aggression are poor emotional awareness and emotion dysregulation, as well as impulsivity. Here, we examined the relationships among alexithymia, emotion dysregulation, impulsivity and aggression, comparing a mixed psychiatric sample (N=257) and a community sample (N=617). The clinical sample reported greater levels of alexithymia, emotion dysregulation, trait impulsivity and aggression, than the community sample. Furthermore, in the community sample, emotion dysregulation and impulsivity mediated the relationship (i.e., accounted for the shared variance) between alexithymia and aggression. In the clinical sample, only emotion dysregulation explained the alexithymia-aggression link. In particular, specific dimensions of the emotion dysregulation (i.e., Negative Urgency) and impulsivity constructs (i.e., cognitive and motor impulsivity) played a unique role in explaining these associations. Finally, controlling for depressive symptoms reduced some of the findings involving impulsivity to nonsignificant results. Overall, our findings add to the extant literature attesting to the relevance of alexithymia and emotion dysregulation for understanding aggression, and providing concrete recommendation for the treatment and prevention of aggressive tendencies.

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1. Introduction

Human aggression, rather than being inherently pathological, represents a natural predisposition that is vital for survival, and can be adaptively used in the everyday life as a form of genuine protest against adversities in life (Bushman and Anderson, 2001; Fonagy, 2003). However, when there is a failure in some of the processes that normally tame and canalize aggressive tendencies, this can pave the way for the occurrence of destructive behavior (Fonagy, 2003). Such behavior can vary in intensity and severity, spanning from chronic anger expression or frequent angry outbursts, to sexual or otherwise interpersonal violent acts. Therefore, understanding the psychological antecedents of aggressive behavior is a crucial step to increase the possibility to prevent reoffending, at least in some sub-populations. Mechanisms that are likely to predate maladaptive form of aggression include poor awareness of emotions and a diminished ability to think and talk about feelings, that is, alexithymia (Loas et al., 2015; Nemiah and Sifneos, 1970; Taylor et al., 1997), emotion dysregulation, and impulsivity. Individuals who are unaware of their feelings have more difficulties toning down emotional arousal, especially when they are under stress. When people perceive a possible danger in their relational environment, they can feel a tension, and if they are not able to recognize the source and the nature of this sensation, they may consequently remain deprived of adaptive strategies to solve possible interpersonal conflicts (e.g., adopting appropriate communication and negotiation skills). For example, being aware that one is feeling humiliated by comments from other people, can help him to explain them that he was offended by their comments and, for example invite them to plea excuse or to abstain in the future from making similar comments. In absence of a proper awareness of feeling humiliated, such a communication could not
happen. In that case, humiliation will just be felt as a “tension”, “pain”, or at best will be perceived as another emotion (e.g., anger), increasing the possibilities that attack will be considered as one of the few strategies available to protect oneself from the source of such pain (Elison et al., 2014).

As for the case of some hypotheses about the mechanisms underlying self-harm (Linhean, 1993), it is possible that poor emotional awareness hampers emotion regulation, and in turn emotion dysregulation may represent one of the proximal antecedents of aggression (Jenkins et al., 2014). There is more, though, to emotion dysregulation, than alexithymia. One of the most widely adopted conceptualizations describes emotion dysregulation as involving: lack of awareness for and understanding of emotions, nonacceptance of emotional responses, difficulties engaging in goal-directed behavior when upset, inability to refrain from impulsive reactions when experiencing negative emotions, and limited access to effective emotion regulation strategies (Gratz and Roemer, 2004). Recent research has shown that difficulties in these domains can increase the individual proneness to react with aggression to perceived threats or offenses (Garofalo et al., 2016). Indeed, even among people who are fully aware of their emotions, there is a certain variability in how they deal with and regulate them (e.g., reappraising the situation, or thinking about different kind of problem solving strategies). Therefore, dysregulation of emotions, and in particular of anger, is considered a possible mechanisms underlying aggression (Scott et al., 2014; Wahlstrom et al., 2015).

Another possible antecedent of aggression is impulsivity (Bousardt et al., 2015). As described below, when an individual appraises a certain situation as a possible source of menace and pain, he or she can become negatively aroused. In such a situation, an individual with high levels of trait impulsivity might lack the cognitive resources necessary to inhibit aggressive reaction or refrain from acting out the fantasies of self-protective aggression or vendetta that he or she is experiencing. Therefore, once feeling to have a right to retaliate, action can rapidly follow.

To date, studies linking alexithymia, emotion dysregulation and impulsivity with aggression have produced mixed results. Emotion dysregulation has been found to predict aggression in offenders (Garofalo et al., 2016; Robertson et al., 2014), as well as in patients with borderline personality disorder (Scott et al., 2014). Further, in a community sample of women, emotion dysregulation mediated the relationship between trauma and both impulsive and premeditated aggression (Miles et al., 2015). Of note, some authors (e.g., Scott et al., 2014) considered impulsivity as an aspect of emotion dysregulation, although the two constructs do not completely overlap. Indeed, emotion dysregulation (as described above) refers to a wider set of difficulties that are not necessarily linked to impulsivity. On the other hand, impulsivity includes components, such as cognitive impulsivity and lack of planning, which are not necessarily related to emotion dysregulation. Moreover, within the emotion dysregulation construct (Gratz and Roemer, 2004), it is often included the state-like action-component of impulsivity, that is, an inability to inhibit impulsive behavior when emotionally aroused (also referred to as negative urgency). On the other hand, trait impulsivity represents a more general propensity toward rash actions and decisions, or unplanned reactions to internal or external stimuli with a lack of regard for the possible consequences of these actions (Moeller et al., 2001). Prior studies have reported that these two aspects of the impulsivity domain only partially overlap, and present differential associations with personality traits (Velotti and Garofalo, 2015).

As regards a direct impact of alexithymia, prior studies found that high levels of alexithymia in a sample of juvenile sexual offenders (Moriarty et al., 2001) and in violent offenders (Keltikangas-Jarvinen, 1982). In another investigation, alexithymia predicted impulsive aggression in a sample of veterans (Teten et al., 2008). As regards a poor ability to recognize and express feelings, both emotional avoidance (which can be considered a correlate of alexithymia), and emotional inexpressivity were uniquely related to self-reported aggression in a sample of adults with Post Traumatic Stress Disorder (PTSD; Tull et al., 2007). Furthermore, in a community sample of adolescents, alexithymia mediated the association between attachment insecurities and impulsive aggressiveness (Fossati et al., 2009).

In this study, we sought to confirm and extend prior research examining the mechanisms linking alexithymia, emotion dysregulation, impulsivity, and aggression in a clinical sample as well as in a community sample. First, we hypothesized that the clinical sample reported higher levels of alexithymia, emotion dysregulation, impulsivity, and aggression. Second, we expected to confirm the positive associations among alexithymia, emotion dysregulation, impulsivity, and aggression, in both samples. Finally, we hypothesized that the association between alexithymia and aggression was mediated by emotion dysregulation and impulsivity, in both samples.

We emphasize that although we use the terms ‘prediction’ and ‘mediation’ as they are commonly used also in cross-sectional studies (e.g., Hayes, 2000; Preacher and Hayes, 2008), we do not use them to imply causal or temporal relationships among the study variables (as it would not be possible in any correlational design). Rather, we refer to these terms as statistical mediation and predictions. As such, a significant predictor is an independent variable that in a multiple regression analyses is significantly associated with the dependent variable, showing a unique and independent contribution (i.e., controlling for the shared variance among predictors) to the model. Likewise, we carried out mediation analyses to examine whether the shared variance between an independent (here, alexithymia) and a dependent variables (here, aggression) was accounted for by a third intervening variable (i.e., the mediators; here, emotion dysregulation and impulsivity), at least partially. That is, we wanted to test whether the association between alexithymia and aggression was partly explained by emotion dysregulation and impulsivity. Including both mediators in the same model allowed us to statistically remove also the shared variance between the mediators, as well as the variance that the mediators shared with the independent variable (i.e., alexithymia). In doing so, we investigated the unique and independent contribution of each mediator to the model, testing whether they could explain a portion of the variance shared by alexithymia and aggression. Therefore, the term mediators define here those variables that are expected to account for the association between an independent and a dependent variables, without by any means implying that causal relationships among these variables can be drawn.

2. Method

2.1. Participants and procedure

A large community sample was enrolled by means of a snowball technique and comprised 617 participants ($M_{age}=36.88$, $SD=13.11$; 54.2% males). The snowball sampling method allowed us to obtain a wide sample of adults of different ages, status and education strata. Indeed, each research assistants involved in data collection started with a known group of people, asking them to recruit further participants among their acquaintances. As such, each contact leads to another, in turn expanding the pool of potential participants (Sadler et al., 2010). Males ($M_{age}=37.93$, $SD=12.20$) were significantly, albeit slightly, older than females.
dysregulation, and confirmed its good psychometric properties and its construct and predictive validity in the Italian adaptation of the scale (Giromini et al., 2012). The DERS assesses difficulties in six clinically relevant dimensions of emotion regulation through 36 items rated on a 1 to 5 Likert scale: nonacceptance of emotional responses (Nonacceptance, ($\alpha = 0.87$)); lack of awareness for and difficulties in acknowledging emotions (Awareness, $\alpha = 0.70$); lack of emotional clarity (Clarity, $\alpha = 0.82$); ability to rely on adaptive emotion regulation strategies (Strategies, $\alpha = 0.90$); capacity to pursue desired goals when upset (Goals, $\alpha = 0.84$); and ability to refrain from impulsive behavior when emotionally distressed (Negative Urgency, $\alpha = 0.85$). Greater scores on any of these scales are indicative of greater difficulties in each emotion regulation dimension, and the DERS total score is a valid indicator of overall levels of emotion dysregulation (Garofalo and Velotti, 2015).

In the present study, the DERS total score yielded a Cronbach's alpha of 0.94.

2.2.3. Impulsivity

Trait impulsivity was measured using the Barratt Impulsiveness Scale-11 (BIS-11; Patton et al., 1995), which consists of 30 items on a 4-point Likert scale. The BIS-11 assesses three distinct, albeit related, dimensions: motor impulsivity (i.e., the tendency to act on the spur of the moment; $\alpha = 0.61$); cognitive impulsivity (inability to focus on a task at hand and tendency to be easily distracted; $\alpha = 0.67$); and lack of planning (decision making without a careful forethought and poor self-control; $\alpha = 0.64$). The relatively low (albeit still considered acceptable; Schmitt, 1996) internal consistency coefficients of the BIS-11 subscales are in line with previous studies with the Italian version of the scale (Fossati et al., 2001). The reliability and validity of the BIS-11 total score as a measure of overall trait impulsiveness has been confirmed in the Italian adaptation of the scale (Fossati et al., 2001). In the current study, Cronbach's alpha was 0.81.

2.2.4. Aggression

An Italian version of the Aggression Questionnaire (AQ; Buss & Perry, 1992) was used to assess the propensity toward aggression. The AQ is a 29-item, Likert type, self-report rating scale, which captures four components of aggression: physical and verbal aggression; anger; and hostility. These four dimensions are summed to produce a composite aggression score. Previous research evidenced that the AQ – and its Italian translation – have adequate psychometric properties (Buss and Perry, 1992; Fossati et al.; 2003). The internal consistency reliability of the AQ total score in the present study was 0.90.

2.2.5. Depression

In order to control for the potential confounding effect of depressive symptoms, the Depression subscale of the Brief Symptom Inventory (BSI) was used. The BSI (Derogatis, 1975; Derogatis & Melisaratos, 1983) is a 53-item short-form version of the well-known and widely-used Symptom Checklist–90-R (Derogatis, 2011). The Depression scale of the BSI asks respondents to indicate the frequency of the occurrence of depressive symptoms in the past month, and comprises 6 items rated on a Likert scale ranging from 0 (not at all) to 4 (extremely), with higher scores indicating greater severity. The BSI Depression scale was the most reliable scale of the BSI in the original validation study ($\alpha = .85$), and confirmed its good psychometric properties in recent studies with the Italian version (Zavattini et al., 2015). In the present study, the internal consistency coefficient of the BSI Depression scale was 0.90.

2.3. Data analytic plan

Descriptive statistics were computed for all study variables and...
all sociodemographic information available. An independent sample t-test and a Chi-square test were used to examine group differences in terms of age and gender distribution, respectively. Univariate and multivariate analysis of covariance (ANCOVA or MANCOVA designs, controlling for age, gender, and other possible covariates as detailed below) were carried out to test for group differences on all study variables, as appropriate. In these analyses, Partial Eta Square was used as effect size measure. Partial correlation were conducted to examine the bivariate associations among study variables in each sample, while controlling for age and gender.

Normal-theory significance tests were used to evaluate each path coefficient involved in the proposed mediation model, whereas a bootstrapping approach (Preacher and Hayes, 2008) was used to test the indirect effect of alexithymia on aggression through the mediation of emotion dysregulation and impulsivity. Mediation analyses were carried out separately on the two groups. This nonparametric approach is preferable as it allows simultaneously to test multiple mediator models without inflating the risk for family-wise error. This method is also more powerful in detecting the direct effect of an x variable on a y variable while controlling for several mediators, as well as the specific indirect effects of different mediators. Importantly, this bootstrapping technique does not impose questionable distributional assumptions on the data and easily allows to control for the effects of covariates. To detect the specific nature of the mediation, Preacher and Hayes (2008) approach generates point estimates and bias-corrected confidence intervals (CIs) for each of the proposed indirect effects, as well as a point estimate of the remaining direct effect. For the indirect effects tests, CIs that do not include zero suggest significant mediation. In the present study, an SPSS Macro (i.e., PROCESS; Hayes, 2013) was used to carry out the mediation analyses, including age and gender as covariates and resampling data 5000 times to construct 95% CIs for the indirect effects. In particular, we examined in both samples whether impulsivity and emotion dysregulation mediated the association between alexithymia and aggression (a graphical depiction of this model is reported in Fig. 1). Notably, the bootstrapping method allows to compare the relative strength of the single indirect effects involved in the multiple mediation model, estimating the statistical significance of the point estimate for the difference between pairs of mediators with associated 95% bias-corrected CIs. To test the strength of the mediation effects, the effect proportion mediated measure \( \Delta R^2 \) (Shrout and Bolger, 2002) was computed. The \( \Delta R^2 \) is an estimate of the proportion of the association between the independent and the dependent variables that is explained by the mediator(s). The associated effect size for the indirect effects was inspected by means of the completely standardized indirect effect \( ab_{std} \) (Preacher and Kelley, 2011).

Throughout all analyses, missing data were treated excluding cases pairwise. The Variance Inflation Factors was analyzed to test whether multicollinearity among independent variables could bias regression analyses.

### 3. Results

Preliminary analyses revealed that the two samples significantly differed in terms of age, \( t(865) = -7.064, p < 0.001 \), but not in gender distribution, \( \chi^2 (1, N=875) = 1.17, p > .05 \). Furthermore, both age- and gender-related differences occurred on several study variables in the two samples. As a result, gender (dummy-coded) and age were controlled for by entering them as covariates in all subsequent analyses. Descriptive statistics and group comparisons for all study variables are detailed in Table 1.

As expected, psychiatric patients reported significantly higher levels of alexithymia, aggression, overall emotion dysregulation, and trait impulsivity. The magnitude of the differences in the means was moderate to large (according to Cohen’s (1988) benchmark for interpreting Partial Eta square indices of effect size).

![Fig. 1](https://example.com/fig1.png)

**Fig. 1.** An illustration of the proposed mediation model in the community (A) and clinical sample (B). In both cases, Path a represents the direct effect of alexithymia on the mediators, and Path b the effect of the mediators on aggression, with the predictor held constant. Path c’ is the direct effect of alexithymia on aggression controlling for the variance accounted for by the mediators. Gender and age were used as covariates in all paths. Bolded paths are significant. The total effect of alexithymia on aggression (i.e., Path c) is not represented since it is no longer necessary to test for mediation (Preacher and Hayes, 2008).
Table 1
Means, Standard Deviations (SD), and group comparisons for all study variables.

<table>
<thead>
<tr>
<th></th>
<th>Clinical sample</th>
<th>Community sample</th>
<th>F</th>
<th>ηp²</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAS-20 total score</td>
<td>N  229</td>
<td>Mean (SD) 54.53 (13.62)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AQ total score</td>
<td>N  254</td>
<td>Mean (SD) 76.18 (19.68)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS total score</td>
<td>N  234</td>
<td>Mean (SD) 97.93 (27.36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Nonacceptance a</td>
<td>N  227</td>
<td>Mean (SD) 17.92 (6.70)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Goals b</td>
<td>N  227</td>
<td>Mean (SD) 16.31 (5.16)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Negative Urgency c</td>
<td>N  227</td>
<td>Mean (SD) 15.40 (6.24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Awareness d</td>
<td>N  227</td>
<td>Mean (SD) 14.12 (4.75)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Strategies e</td>
<td>N  227</td>
<td>Mean (SD) 22.04 (8.65)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Clarity f</td>
<td>N  227</td>
<td>Mean (SD) 11.93 (4.81)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 total score</td>
<td>N  245</td>
<td>Mean (SD) 66.55 (11.77)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 Motor Impulsivity g</td>
<td>N  242</td>
<td>Mean (SD) 22.89 (5.10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 Cognitive Impulsivity h</td>
<td>N  242</td>
<td>Mean (SD) 17.38 (4.57)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 Lack of Planning i</td>
<td>N  242</td>
<td>Mean (SD) 26.38 (5.63)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AQ total score</td>
<td>N  583</td>
<td>Mean (SD) 43.23 (11.16)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS total score</td>
<td>N  615</td>
<td>Mean (SD) 65.49 (16.69)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Nonacceptance a</td>
<td>N  584</td>
<td>Mean (SD) 75.20 (20.10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Goals b</td>
<td>N  576</td>
<td>Mean (SD) 12.39 (4.92)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Negative Urgency c</td>
<td>N  576</td>
<td>Mean (SD) 12.86 (4.48)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Awareness d</td>
<td>N  576</td>
<td>Mean (SD) 11.13 (4.33)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Strategies e</td>
<td>N  576</td>
<td>Mean (SD) 13.82 (4.11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS Clarity f</td>
<td>N  576</td>
<td>Mean (SD) 15.40 (6.26)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 total score</td>
<td>N  615</td>
<td>Mean (SD) 9.57 (3.76)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 Motor Impulsivity g</td>
<td>N  608</td>
<td>Mean (SD) 59.68 (9.83)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 Cognitive Impulsivity h</td>
<td>N  608</td>
<td>Mean (SD) 14.91 (3.58)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS-11 Lack of Planning i</td>
<td>N  608</td>
<td>Mean (SD) 24.34 (4.73)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: TAS-20 = Toronto Alexithymia Scale. AQ = Aggression Questionnaire. DERS = Difficulties in Emotion Regulation Scale. BIS-11 = Barratt Impulsiveness Scale. F = statistics based on one-way ANCOVAs (controlling for gender and age). ηp² = Partial Eta squared, effect size (0.01 = small effect; 0.06 = medium effect; 0.13 = large effect; Cohen, 1988).

For the sake of parsimony, these results are not reported here, but are available upon request from the first author.

* Because alexithymia, at least as assessed with the TAS-20, usually shows moderate associations with depression (e.g., Li et al., 2015; see also Loas, 2012; Nicolò et al., 2012), we repeated all ANCOVA/MANCOVA analyses adding as a covariate the depression scale from the Brief Symptom Inventory (BSI; Derogatis, 1983), which assesses the presence of depressive symptoms over the last month. All group differences remained significant, albeit with reduced effect size, with the exception of the BIS-11 total score, and the BIS-11 Lack of planning scale.

For 57% of the variance in the AQ total score, R² = 0.57, F(5, 561) = 54.00, p < .001, indicating large effect size (Cohen’s f² = 1.33; Cohen, 1988). A PM of .72 evidenced that roughly the 72% of variance in aggression accounted for by alexithymia was explained by emotion dysregulation (PM = .54) and impulsivity (PM = .19). The overall indirect effect model showed a large effect size (ab = .28, 95% CI: 0.22, 0.36). In particular, the difference between the two indirect effects was significant (∆ = 0.20, 95% CI: 0.08, 0.34) evidencing that the strength of the indirect effect through the DERS was larger than the one through the BIS-11.

Likewise, in the clinical sample, the total effect of the TAS-20 on the AQ was significant. Further, after controlling for age and gender, the TAS-20 had a significant, positive effect on both the DERS and the BIS-11. Then, the indirect effect of TAS-20 on AQ through both mediators was significant (∆ = 0.41, 95% CI: 0.25 to 0.59). However, only the indirect effect through the DERS was significant, whereas the indirect effect through the BIS-11 was not significant. Thus, emotion dysregulation uniquely and independently accounted for the variance shared by alexithymia and aggression. Notably, the direct effect of TAS-20 on AQ became trivial and nonsignificant when the mediators where added to the model, indicating full mediation. Overall, the mediation model significantly explained roughly the 50% of variance in AQ, R² = .50, F(5, 212) = 13.82, p < .001, showing a large dimension of the effect (Cohen’s f² = 1.00). Specifically, the magnitude of the indirect effect was large (total ab = .30, 95% CI: 0.19, 0.42; total PM = .92; DERS-only PM = .72; BIS-11-only PM = .20, ns). It is worth noting that the relative contributions of the DERS and the BIS-11 did not differ significantly (∆ = 0.23, 95% CI: -0.02, 0.45), indicating that although the indirect effect through the DERS was significant and the indirect effect through the BIS-11 was not statistically significant, the difference in magnitude between the two indirect effects was not significant. In other words, although the BIS-11 did not play a mediating role independently, its contribution to the overall mediation model is not negligible.

As it was done for group comparison tests (see Footnote 4), we repeated all mediation analyses controlling for depressive symptoms by adding the depression scale from the BSI (Derogatis, 1994) as a covariate. Results remained largely unaltered, with the only exception that also in the community sample, only emotion dysregulation negatively and independently associated with AQ.
dysregulation significantly accounted for the association between alexithymia and aggression, whereas the contribution of impulsivity to the model dropped to nonsignificance. Furthermore, follow-up analyses were carried out to examine the mediating role of specific components of emotion dysregulation and impulsivity, entering all DERS and BIS-11 subscales simultaneously as mediators in the relationships between the TAS-20 and the AQ total scores. In the community sample, the DERS Negative Urgency (point estimate = 1.60, 95% CI: 1.20, 2.00), the BIS-11 Motor Impulsivity (point estimate = 0.45, 95% CI: 0.12, 0.78) and the BIS-11 Cognitive Impulsivity scales (point estimate = 0.81, 95% CI: 0.42, 1.21) emerged as significant (partial) mediators. Finally, in the clinical sample, only the effects of the DERS Negative Urgency (point estimate = 0.91, 95% CI: 0.31, 1.51) and the BIS-11 Cognitive Impulsivity scales (point estimate = 0.84, 95% CI: 0.12, 1.55) were statistically significant in a model which fully accounted for the indirect effect of alexithymia on aggression. Throughout all regression analyses, VIF values below 10 ensured that multicollinearity among predictors did not bias our results.

4. Discussion

Difficulties in naming one’s own feelings (i.e., alexithymia), emotion dysregulation and impulsivity are all possible candidates to predict elevated levels of aggression, in both clinical and community samples. People with poor abilities to understand their feelings and regulate them with conscious effort, as well as a marked tendency to act impulsively, might have more difficulties in controlling their aggressive tendencies. According to previous observations, we hypothesized that alexithymia, emotion dysregulation and impulsivity were connected to each other. We then considered that alexithymia could have both a direct effect on self-reported aggressiveness and an indirect effect mediated by impairments in emotion regulation and heightened impulsivity. Results were largely consistent with our predictions. First, participants in the clinical sample had more problems in recognizing their feelings and regulating them, as well as a greater tendency toward impulsivity, than the community sample. Self-reported aggression was also higher in the clinical sample than in the community sample. All these variables were significantly related to each other in both samples, supporting the existence of a system with partially overlapping domains devoted to emotional awareness and regulation of affects and behavior.

In the community sample, the effect of alexithymia on aggression was partially mediated by both emotion dysregulation and impulsivity. Indeed, alexithymia maintained a direct effect on aggression, with emotional dysregulation and impulsivity partially accounting for their association. Conversely, in the clinical sample, the indirect effect of alexithymia on aggression through emotion dysregulation was significant, whereas impulsivity did not significantly mediate the association between alexithymia and aggression.

Table 2

Bivariate correlations among study variable, after partialing out the effects of age and gender, for the clinical (N=257; above the diagonal) and the community sample (N=617; below the diagonal).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Sample</th>
<th>Effect of IV on M (a)</th>
<th>Effect of M on DV (b)</th>
<th>Total effect (c)</th>
<th>Direct effect (c')</th>
<th>Indirect effect (bias corrected intervals) (a)(b): 95% CI</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Emotion dysregulation</td>
<td>Clinical</td>
<td>1.26***</td>
<td>0.26***</td>
<td>0.45***</td>
<td>0.05</td>
<td>0.32: 0.16 to 0.49</td>
<td>0.23</td>
</tr>
<tr>
<td>Community Emotion dysregulation</td>
<td>Community</td>
<td>1.09**</td>
<td>0.28</td>
<td>0.58***</td>
<td>0.16*</td>
<td>0.31: 0.21 to 0.43</td>
<td>0.21</td>
</tr>
<tr>
<td>Clinical Impulsivity</td>
<td>Clinical</td>
<td>0.44**</td>
<td>0.21</td>
<td>0.45***</td>
<td>0.05</td>
<td>0.09: -0.02 to 0.22</td>
<td>0.07</td>
</tr>
<tr>
<td>Community Impulsivity</td>
<td>Community</td>
<td>0.37**</td>
<td>0.29***</td>
<td>0.58***</td>
<td>0.16</td>
<td>0.06: 0.05 to 0.17</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Note: Emotion dysregulation (i.e., the Difficulties in Emotion Regulation Scale total score) and Impulsivity (i.e., the Barratt Impulsiveness Scale-11 total score) were entered as simultaneous mediators in multiple mediator designs. All paths were estimated holding constant the effects of age and gender. abes completely standardized indirect effect, measure of the effect size of the indirect effect (.01 = small effect size; .05 = medium effect size; .25 = large effect size; Preacher and Kelley, 2011).

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aggression. The absence of a significant contribution of impulsivity could suggest that, when taking into account the influence of emotion dysregulation, trait impulsivity do not significantly account for an additional portion of the variance shared by alexithymia and aggression. However, it should be noted that this difference that appeared when comparing the community and the clinical sample (i.e., multiple versus single mediator), could be due to differences in the sample size, therefore next studies should investigate larger clinical samples in order to verify whether the indirect effect of alexithymia on aggression through impulsivity could represent a meaningful alternative. Of note, note, emotion dysregulation fully mediated the link between alexithymia and aggression, suggesting that their association was completely explained by emotion dysregulation.

Overall, our results are consistent with findings obtained when examining selected psychiatric disorders (such as PTSD; Tull et al., 2007) and in populations with prominent aggressive tendencies (Garofalo et al., 2016). To the best of our knowledge, earlier studies did not analyze the impact, both direct and indirect, of alexithymia on aggression, through dysregulation and impulsivity. Mostly, former studies have focused either on the association between emotion dysregulation and impulsive aggression (Wahlstrom et al., 2015), or on the link between alexithymia and impulsive aggression (Fossati et al., 2009; Teten et al., 2007). Conversely, after replicating in our samples that these variables are reasonably intertwined, we tried to disentangle all variables and to consider them together (i.e., both alexithymia and emotion dysregulation on the one hand, and both impulsivity and aggression on the other hand). This allowed us to investigate the unique and independent associations among these constructs, taking into account their reciprocal associations – that is, removing the variance shared among them (e.g., the overlap between alexithymia and emotion dysregulation, or between emotion dysregulation and impulsivity, or between impulsivity and aggression).

With replication, these results may shed further light on the antecedents of aggression. Though our study was cross-sectional and did not allow for inferences on causality, it is reasonable to speculate that when one is unable to recognize and name own emotions, under stressful conditions he or she may be prone to inhibit impulsive action. However, it should be emphasized that longitudinal studies are warranted to test the validity of such speculations.

4.1. Limitation and future directions

There are limitations to our study, which invite to cautious interpretations of the findings and their generalizability. The main limitation is that we investigated a mixed psychiatric sample, limiting our possibilities to understand whether some of the mechanisms reported are different across specific disorders. Thus, future research should explore larger samples with enough participants to examine and compare these mechanisms across different diagnostic groups. At the same time, this could allow to control for the possible confounding role of selected disorders in which the presence of emotional problems and impulsivity is more prominent. For example, it is well-established that borderline personality disorder is strongly associated with emotion dysregulation and impulsivity (e.g., Linehan, 1993), and that borderline traits are also present in other psychiatric conditions. As such, it would be interesting to examine whether the greater levels of alexithymia, emotion dysregulation, and impulsivity among psychiatric patients, as well as their association with aggression, could be captured by the presence of borderline traits. Furthermore, other variables have strong associations with alexithymia and impulsivity (e.g., anhedonia, locus of control; Marechal et al., 2009), thus, replication of this study including additional covariates would strengthen the present findings. Another limitation regarding the lack of a structured diagnostic procedure that was consistent for all participants in the clinical sample. Although the diagnoses were always made by senior clinicians after staff meetings and clinical interviews and were on the DSM-IV-TR nomenclature (American Psychiatric Association, 2000), the recruitment of participants took place in different sites. This leaves open the possibility that slightly different procedures had been followed in different psychiatric institutions. Furthermore, because we only had access to the final diagnosis that was reported in clinical records, but we did not have access to item ratings (either single- or multi-coder) from structured clinical interviews (when they were applied) we could not calculate internal and inter-rater reliability for such diagnoses. However, we trust that the thorough clinical experience, as well as the full compliance with a mainstream psychiatric nosology (i.e., DSM-based), can ensure a reasonable level of reliability and validity of these procedures. A third limitation is the reliance on self-report measures only. There is evidence that all variables examined here may yield different profiles and association with symptoms and problems when analyzed with self-report, interviews and in the laboratory. For example, both emotion dysregulation and impulsivity encompass dimensions that are differently captured by self-report questionnaires and measures of bodily arousal (Sharma et al., 2014). Future studies should replicate the present findings using a multi-method approach. Moreover, we explored samples where no specific stories of aggression were reported, which leaves unanswered the question of whether these results could be similar in individuals with a history of aggression. Related to this, it should be noted that several other aspects could underlie and explain aggressive behavior. For instance, psychopathic traits such as lack of empathy and a pattern of callous interpersonal manipulation of others, might be associated with intact emotion regulation abilities, but still predict aggression and violent behavior. Although recent empirical evidence suggest emotion dysregulation is also linked to psychopathic traits (Donahue et al., 2014; Long et al., 2014), future studies should examine the potential role of other psychopathic traits (e.g., lack of empathy, callousness) in explaining additional variance in aggressive tendencies. Moreover, in this study we did not include a measure of premeditated aggression. Although there is a substantial overlap between instrumental/ premeditated and hostile/reactive aggression (Bushman and Anderson, 2001), it is reasonable to expect that different factors may contribute to aggressive behavior that are relatively more premeditated in nature (Bo et al., 2013). Because psychopathic traits could also play an important role in psychiatric patients with other disorders (Abu-Akel et al., 2015), it is of the utmost importance to consider different paths leading to different forms of aggression to get a more nuanced insight into the underlying mechanisms of violent behavior. Finally, as mentioned above, we emphasize that the cross-sectional nature of our study did not allow to draw conclusions about the direction of the relationships among the variables investigated. Therefore, longitudinal experimental studies are encouraged to explore whether the associations reported here actually resemble predictions and mediations in prospective designs in which causal relationships and temporal ordering of variables can be statistically detected.

With these limitations considered, our results may possibly
expand previous knowledge and add to the idea that alexithymia, emotion dysregulation and impulsivity are predictors of aggression. As a consequence, psychological treatments aimed at promoting emotional awareness as well as affective and behavioral regulation (Bateman et al., 2013; Cameron et al., 2014; Dimaggio and Lysaker, 2015; Linehan, 1993; Yakeley and Williams, 2014) are likely candidate to reduce aggression where it is a prominent problem.

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References


