

A Silent Pathway to Depression: Social Anxiety and Emotion Regulation as Predictors of Depressive Symptoms

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Abstract

Background: Social anxiety is the most common comorbid disorder in patients with major depressive disorder, almost always preceding it and aggravating its presentation and course. A possible mechanism to explain this relationship may well be the use of specific maladaptive emotion regulation strategies, common to depression and social anxiety.

Objectives: This study aimed to explore, in an adolescent sample, if depression could be predicted by social anxiety and if emotion regulation strategies would mediate this relationship.

Method: The sample included 527 adolescents from the general population (59.2% were girls; $M_{age} = 13.8$; $SD = 7.57$). Self-report scales measuring depression, social anxiety and cognitive emotion regulation were filled out.

Results: Depression and social anxiety showed significant, positive and moderate correlations with all cognitive emotion regulation strategies (self-blame, catastrophizing, and rumination), exception made for the correlation with other-blame, which was very low. The final mediation model explained 39% of depressive symptomatology, with social anxiety having both a direct and an indirect effect. The only significant mediation variable that accounted for this indirect effect was self-blame.

Conclusions: The results clearly point to the role of social anxiety in adolescents' depressive symptoms either directly or indirectly, through self-blame. These results call attention to the importance of discriminating social anxious and depressive symptomatology offering specific preventive or therapeutic approaches for both conditions or including different components in these approaches to address both depression and social anxiety. Furthermore, effective intervention should also target specific cognitive emotion strategies. Theoretical and clinical implications are considered.

Keywords: Depression, social anxiety, cognitive emotion regulation, adolescents

Introduction

Depression and social anxiety in adolescence

The transition into adolescence involves a number of biological, cognitive, and social changes, making adolescence a particularly demanding period due to the several challenging developmental tasks it includes. Entering adolescence means dealing with a changing body, often going into a new school, having new peers and having to make new friends. Adolescents have to develop a sense of identity and emotional independence. They start turning to peers instead of parents for emotional support and social acceptance becomes more and more important, bringing about the awareness of being social objects more so than during childhood (Petersen & Spiga, 1982; Rubin, Coplan, & Bowker, 2009; Sroufe & Rutter, 1984). In face of all these changes and challenges, it is not surprising that many adolescents experience increased negative affect, emotional reactivity, social anxiety and risk for internalizing symptoms (Arnet, 1999; Larson and Ham, 1993; Parker & Asher, 1987). Therefore, the successful negotiation of these tasks results in a healthier and more prepared system to face the demands of the next period whereas its inadequate resolution may result in maladaptive strategies that may be expressed in anxiety or depression problems.

In fact, adolescence is a probable period to develop major depressive disorder (MDD) and social anxiety disorder (SAD), two of the most prevalent mental disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Adolescents' depression is a highly prevalent and serious mental health condition due to its prevalence, comorbidity, tendency to chronicity and impact (Petersen, Compas, Brooks-Gunn, Stemmler, & Grant, 1993), with a peak of onset around 15 years of age (Fergusson, Horwood, Ridder, & Beautrais, 2005). A recent study (Avenevoli, Swendsen, Jian-Ping, Burstein, & Merikangas, 2015) points to a lifetime prevalence and 12-month prevalence of MDD of 11% and 7.5%, respectively.

On the other hand, social anxiety disorder, characterized by an intense fear of being scrutinized by other people resulting in marked fear and anxiety in social situations often coupled with the avoidance of such situations, has its onset in adolescence and tends to follow a chronic course (APA, 2013), with a high negative impact and functional impairment in the adolescents' life (see Salvador, 2009, for a review).

MDD and SAD are frequently comorbid conditions, both in youth (Beidel, et al, 2007; Chavira, Stein, Bailey, & Stein, 2004; Crawley, Beidas, Benjamin, Martin, & Kendall, 2008; Essau, Conrad, & Petermann, 1999; Ranta, Kaltiala, Rantanen, & Marttunen, 2009; Viana, Rabian, & Beidel, 2008; Wittchen, Stein, & Kessler, 1999) and in adults (Kessler, Stang, Wittchen, Stein, & Walters, 1999;

Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005), with SAD being considered the most common comorbid anxiety disorder in patients with MDD (Belzer & Schneier, 2004). In addition, the comorbidity between SAD and MDD is associated, in both disorders, with greater impaired functioning, more severe and chronic course, greater risk of relapse, and, in general, worse prognosis than when the disorders present independently (Brown, Schulberg, Madonia, Shear, & Houk, 1996; Dalrymple, & Zimmerman, 2007, 2011; DeWit, Ogborne, Offord, & MacDonald, 1999; Lecrubier, 1998; Lewinsohn, Rohde, & Seeley, 1995; Ruscio, et al, 2008; Stein, et al, 2001). Furthermore, most patients with SAD who seek psychiatric treatment, primarily seek treatment for MDD, leaving SAD undiagnosed and untreated (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Dalrymple & Zimmerman, 2007; Lecrubier, 1998; Zimmerman & Chelminski, 2003).

Interestingly, several studies found that SAD precedes the onset of MDD (Beesdo, et al, 2007; Beidel, Turner, Morris, 1999; Chavira, et al, 2004; Dalrymple & Zimmerman, 2011; De Graaf, Bijl, Spijker, Beekman, & Vollebergh, 2003; Kessler, et al, 1999; Stein et al, 2001; Wittchen et al, 1999), indicating SAD as an important predictor of subsequent depression. As an example, Kessler and collaborators (1999), in a large adult sample, found that 68,5% of the patients with a mood disorder and SAD stated that SAD occurred at an earlier age. Also, in a study from Beesdo et al (2007), in a 10-year prospective longitudinal study with participants aged 14 to 24 years at baseline and 21 to 34 at follow up, the risk of depression was 2-fold in individuals with SAD compared to those without SAD and almost 3-fold compared to individuals with no anxiety disorder, regardless of age. Moreover, Aune & Stiles (2009) found that youth level of social anxiety symptoms was a significant predictor of initial depressive symptoms, while depression did not predict later social anxiety.

Emotion regulation in depression and social anxiety

Despite the marked changes posed by adolescence, many adolescents manage to cope with them fairly well, never developing any psychiatric disorder. This may be due to the strategies adolescents use to deal with their increased negative emotions, a process that is called emotion regulation (e.g., Tillfords & Van Zalk, 2015), defined as “extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals” (Thompson, 1994, pp.27-28). In general, emotion regulation strategies refer to processes that aim to monitor, evaluate and modify emotions, altering their intensity, duration and/or valence (Gross & John, 2003).

In fact, to overcome the constant adolescence psychological challenges, adolescents are required to develop emotion regulation strategies, namely through conscious cognitive processes. Luckily enough, it is also during this period that important cognitive abilities develop, giving way to the development of cognitive coping skills. Adolescents are able think in abstract terms and to monitor their own process of thinking. These abilities enable them to plan ahead or to find different/alternative explanations for a given event, thereby regulating their emotions and not getting overwhelmed by them during a stressful life event (Garnefski, Legerstee, Kraaij, Van den Kommer, & Teerds, 2002). Examples of cognitive coping strategies are self-blame (blaming yourself for what happened), blaming others (putting the blame of what was experienced on the environment or another person), rumination (thinking about the feelings and thoughts associated with the negative event), catastrophizing (thoughts emphasizing the terror of what was experienced), acceptance (thoughts of acceptance of what was experienced) or positive reappraisal (thoughts of creating a positive meaning to the event) (Garnefski, Kraaij, & Spinhoven, 2001; Garnefski & Kraaij, 2006).

Although these cognitive abilities are universal, there are large individual differences in the strategies used, the frequency with which they are used, and in the content of thoughts adolescents use to regulate their emotions (Garnefski et al, 2002), acting as risk or protective factors in the development of psychopathology (McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; Silk, Steinberg, & Morris, 2003). Therefore, some of these cognitive emotion regulation strategies may allow adolescents to better cope with negative life events, while some other emotion regulation strategies, if used systematically, are considered maladaptive, forming an important risk factor for psychopathology in adolescents and adults. Garnefski and collaborators (2001) have found that cognitive emotion regulation strategies such as self-blaming, catastrophizing and rumination played an important role in the relationship between the experience of negative life events and maladjustment.

More specifically, depression has been consistently and positively associated to self-blame, rumination, and catastrophizing and negatively associated to positive reappraisal (Garnefski et al., 2001; Garnefski, & Kraaij, 2006; Garnefski, Boon, & Kraaij, 2003; Garnefski, Rieffe, Jellesma, Terwogt, & Kraaij, 2007; Garnefski, Teerds, Kraaij, Legerstee, & Van den Kommer, 2004; Martin & Dahlen, 2005; Ongen, 2010), with rumination acting as the most frequent predictor of depressive symptoms (e.g., Broderick & Korteland, 2004; Burwell & Shirk, 2007; Hankin, 2008; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007) and of the onset of clinical depressive episodes (Abela & Hankin, 2011). Moreover, in a very recent study, self-blame has also found to be associated with the prodrome period before the onset of the first MDD episode in adolescents (Sheriff, McGorry, Cotton, & Yung, 2015).

Despite the fact that emotion regulation was not a common research theme on SAD, interest in understanding emotion regulation in SAD is growing (e.g., Hofmann, 2010; Hofmann, Sawyer, Fang, & Asnaani, 2012; Turk, Heimberg, Luterek, Mennin & Fresco, 2005) and studies in this area are accumulating over recent years, all of them finding significant associations between social anxiety and emotion regulation difficulties (e.g., Eastabrook, Flynn, & Hollenstein, 2014; Farmer & Kashdan, 2012; Helbig-Lang, Rush, Winfried, & Lincoln, 2015; Jazaeroi, Morrison, Goldin, & Gross, 2015; O'Toole, Jensen, Fentz, Zachariae, & Hougaard, 2014). Several studies have suggested that SAD is also characterized by elevated rumination, either in anticipation of the feared situation (Vassilopoulos, 2005) or following the feared situation (e.g., Abbot & Rapee, 2004; Brozovich & Heimberg, 2008; Edwards, Rapee, & Franklin, 2003). Other emotion regulation strategies have also been studied and associated to SAD or to social anxiety symptoms, such as suppression (e.g., Kashdan & Breen, 2008; O'Toole et al, 2014), experiential avoidance (e.g., Kashdan et al, 2013), and reappraisal (e.g., Kashdan et al., 2013; O'Toole et al, 2014). Reappraisal was also found to be associated SAD treatment effects (Goldin et al, 2012; Moscovich et al., 2012). However, unlike in depression studies, where self-blame and catastrophizing are frequent variables to be studied, we found no study in which the relation between social anxiety and catastrophizing was approached and only one, using an adult sample, that studied and found a relationship between social anxiety and self-blame (Gilbert & Miles, 2000). This scarcity leaves room for further studies on these themes.

Some research is also investigating the relation between social anxiety, depression and emotion regulation strategies, exploring possible pathways between the two psychopathological conditions and trying to account for the enormous comorbidity they present. A recent study investigated the use of rumination for SAD and MDD patients (D'Avanzato, Joorman, Siemer, & Gotlib, 2013). Both disorders were found to be characterized by high levels of rumination but depression showed even higher levels of rumination. Another study (Eastabrook et al, 2014) suggested that adolescents' emotional regulation strategies (suppression and reappraisal) played an important role in determining depressive and social anxiety symptoms. On the other hand, social anxiety also played a role in emotion regulation, predicting higher levels of rumination over time (Jose, Wilkins, & Spindelov, 2012). Closer to our goal, two studies explored the mediation role of rumination and brooding between social anxiety and depression. In one of these studies (Drost, Van den Does, Van Hemert, Pennix, & Spinhoven, 2014), rumination and worry were found to mediate the association of baseline fear disorders, among which was SAD, with distress disorders, among which was MDD. In this study, changes in rumination and worry also mediated the relation between baseline fear disorders and changes in distress disorders. Consistent with this result, Grant and collaborators (2014) also found a mediation role of brooding (a particular form of rumination) between Time 1 social anxiety and Time 3 depression.

Aims

In line with the revised literature and given that the research on the association between social anxiety and depression in adolescent is still at its beginning, this study aimed to explore how social anxiety and emotion regulation may predict depression.

By using cognitive emotion regulation strategies, such as self-blame, rumination, catastrophizing and blame-others, adolescents may be more vulnerable to develop internalizing disorders, namely social anxiety and depression. Therefore, we sought to examine the direct effect of social anxiety on depressive symptomatology, as well as its indirect effect through self-blame, rumination, catastrophizing and other-blame. It was expected that levels of social anxiety would be associated with levels of depressive symptoms, and that social anxiety and maladaptive cognitive emotion regulation strategies would predict depression.

Thus, the two investigated hypotheses were (see Figure 1): a) Social anxiety is a predictor of depressive symptoms; b) The effect of social anxiety on depressive symptomatology is mediated (totally or partially) by self-blame, rumination, catastrophizing and other-blame.

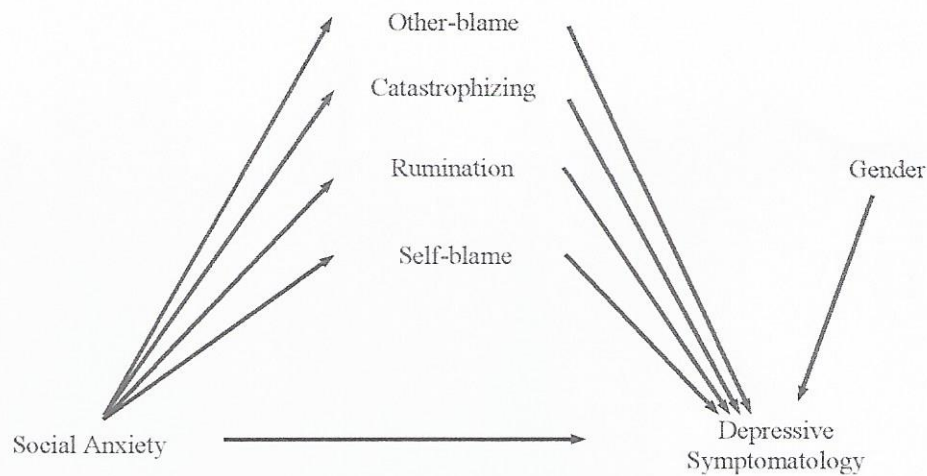


Figure 1. Theoretical model for mediation effects

Method

Participants

Participants for this study were recruited as a part of a Portuguese Project: “Prevention of depression in Portuguese adolescents: Study of the efficacy of an intervention with adolescents and parents”. The sample included 527 adolescents from the general population, in which 215 were boys (40.8%) and 312 were girls (59.2%), between from 13 and 15 years of age ($M = 13.84$; $SD = 7.57$), and attending the 8th and 9th grades in public schools. We did not find significant differences between genders on age ($t_{(525)} = -.638$, $p = .518$).

Procedure

Permission to conduct the study was obtained from national entities that regulate scientific research. Schools were contacted in order to request their participation. After their approval, authorization was also obtained from students and their parents. Anonymity was ensured to the participants as well as confidentiality of the data. After obtaining all permissions required, the research protocol was applied in a classroom setting, before any psychological intervention. The exclusion criteria were: (a) incomplete fill of scales/missing index higher than 10% in each scale; (b) subjects below 13 and above 15 years of age (once the bigger project that included this study aims to prevent the first major depressive episode that usually happens around 15 years old).

Measures

Social Anxiety

The Multidimensional Anxiety Scale for Children (MASC, March, J. et al. 1997; Portuguese Version: Salvador, M. C. et al. 2015). The MASC is a self-report instrument that assesses anxiety symptoms in children and adolescents aged from 8 to 19 years old. It consists of 39 items, rated on a Likert scale of 4 points (from 0 = “never or almost never true” to 3 = “often true”). The original authors found four factors, three of which presented two sub-factors: a) *physical symptoms* (12 items), with sub-factors *tense/restless* (6 items) and *somatic* (6 items); b) *harm avoidance* (9 items), with sub-factors *perfectionism* (4 items) and *anxious coping* (5 items); c) *social anxiety* (9 items) that includes the sub-factors *humiliation* (5 items), *public performance* (4 items) and d) *separation anxiety* (9 items). March, J. and colleagues (2001) obtained alpha coefficients ranging from .68 to .83. The Portuguese version revealed that the internal consistency scores were acceptable for all factors of the MASC, excepting the sub-factors Perfectionism and Anxious Coping. The scale total, factors, and sub-factors showed

moderate to high temporal stability. In this research, we studied only the factor social anxiety. The alpha value will be presented in Table 1.

Emotion Regulation

Cognitive Emotion Regulation Questionnaire (CERQ, Garnefski, et al., 2001; Portuguese version: Matos & Serra, 2009). The CERQ is a self-report questionnaire that assesses specific cognitive emotional regulation strategies used by the adolescents when facing negative life events. Participants rated 36-statements on a 5-point Likert scale ranging from 1 (almost never) to 5 (almost always). To illustrate, some questions of this questionnaire: "I feel that I am the one to blame for it", "I think about the mistakes I have made in this matter". The CERQ has 9 subscales: self-blame, rumination, catastrophizing, other-blame, acceptance, positive reappraisal, refocus on planning, putting into perspective, positive refocusing. Garnefski and collaborators (2001) obtained alpha coefficients ranging from .68 to .83. In the Portuguese version (Matos & Serra, 2009) alpha coefficients ranged from .68 to .83 as well.

Depressive Symptomatology

Children's Depression Inventory (CDI, Kovacs, 1985; Portuguese version: Marujo, 1994). The CDI is a 27-item self-report measure depressive symptoms over the two previous weeks in 7 to 17 year-old children. It has three answering options that range from 0 (no problem) to 2 (severe problem). Kovacs (1985) found five factors with Cronbach's alpha coefficients ranging .83 to .94. In the Portuguese version (Marujo, 1994) a unifactorial structure was found, with an alpha coefficient of .80 for the total scale.

Results

Data analysis

Data was explored using SPSS (Statistical Package for the Social Sciences), version 20 (IBM Corp, Armonk, NY, USA) and AMOS version 20. Descriptive statistics were conducted to explore sample's characteristics. Gender differences were tested using independent sample t-tests, and two-tailed Pearson correlation coefficients were performed to explore the relationships between predictor variables, outcome variables, and mediators (Cohen, Cohen, West, & Aiken, 2003; Tabachnick & Fidell, 2007).

Cohen's guidelines (1988) were used for describing the effect sizes of reported correlations (i.e. small for correlations around .10, medium for those near .30, and large for correlations at .50 or higher). Significance was set at the .01 and .05 levels.

According to Kline (2005), path analysis 'involves the estimation of presumed causal relations among observed variables' (p. 93) and test theoretical relationships on the basis of covariation and correlations among variables. To test the mediator effect of self-blame, rumination, catastrophizing and other-blame in the relationship between social anxiety and depressive symptomatology a path analysis was conducted.

A maximum likelihood method was used to evaluate the significance of the model's path coefficients. Bootstrap resampling procedure (2000 cases) was conducted to analyze the significance of the effects. The results were considered significant at the .001 level if the 95% CI did not include the zero (Kline, 2005).

Multivariate outliers were screened using Mahalanobis squared distance (D^2) method, and univariate and multivariate normalities were assessed by skewness and kurtosis coefficients. There was no severe violation of normal distribution ($|Sk| < 3$ and $|Ku| < 8-10$) (Kline, 2005). Regarding multicollinearity or singularity amongst the variables, Variance Inflation Factor (VIF) values indicated the absence of β estimation problems ($VIF < 5$). Effects with $p < .01$ were considered statistically significant.

Descriptives

The means, standard deviations and Cronbach's alphas for all variables used are presented on Table 1. All scales showed reasonable to good internal consistencies (Pestana & Gageiro, 2005).

Table 1.
Means (M), standard deviations (SD) and Cronbach's Alpha for all subjects (N=527)

Variables	M	SD	α
MASC			
Social Anxiety	1.36	0.66	.88
CDI			
Depressive Symptomatology	11.18	7.08	.77
CERQ			
Other-blame	1.94	0.71	.79
Catastrophizing	2.29	0.88	.79
Rumination	20.17	0.89	.79
Self-blame	2.53	0.82	.77

Note: MASC: Multidimensional Anxiety Scale for Children; CERQ: Cognitive Emotion Regulation Questionnaire; CDI: Children's Depression Inventory.

Gender differences were tested and significant differences were found on in the majority of variables (Table 3). Girls scored significantly higher than boys in depressive symptoms ($M_{girls} = 12.54, SD = 7.56; M_{boys} = 9.19, SD = 5.78$), social anxiety ($M_{girls} = 1.48, SD = .66; M_{boys} = 1.18, SD = .60$) and rumination ($M_{girls} = 3.02, SD = .87; M_{boys} = 2.59, SD = .85$). Boys scored higher than girls only in other-blame ($M_{girls} = 1.88, SD = .71; M_{boys} = 2.03, SD = .70$).

On the other hand, no significant differences were found on catastrophizing [$M_{girls} = 2.32, SD = .93; M_{boys} = 2.25, SD = .80; t(525) = .972, p = .331$].

Table 2.
Student's t-test differences between boys and girls for MASC, CERQ and CDI

	Boys (n = 215)		Girls (n = 312)		t	p
	M	SD	M	SD		
Social Anxiety (MASC)	1.18	0.60	1.48	0.66	-5.338	.000
Depressive Symptomatology (CDI)	9.19	5.78	12.54	7.56	-5.486	.000
Other-blame (CERQ)	2.03	0.70	1.88	0.71	2.358	.018
Catastrophizing (CERQ)	2.25	0.80	2.32	0.93	-0.972	.331
Rumination (CERQ)	2.59	0.85	3.02	0.87	-5.688	.000
Self-blame (CERQ)	2.35	0.74	2.51	0.86	-2.287	.023

Note: MASC: Multidimensional Anxiety Scale for Children; CERQ: Cognitive Emotion Regulation Questionnaire; CDI: Children's Depression Inventory.

Correlational analyses

Pearson correlation coefficients were performed to explore the association between social anxiety, depressive symptomatology, other-blame, catastrophizing, rumination and self-blame (Table 3). Significant, positive and very low to moderate correlations were obtained. Results indicated a moderate correlation between social anxiety and depression. Furthermore these two variables were associated with all emotion regulation variables, although other-blame showed very small correlations both with social anxiety ($r = .09, p < .05$) and with depression ($r = .12, p < .01$). Moreover, correlations between all emotion regulation variables were moderate to high.

Table 3.
Correlations (two-tailed Pearson *r*) between variables in study (*N* = 527)

	1	2	3	4	5	6
1. Social Anxiety (MASC)	–					
2. Depressive Symptomatology (CDI)	.52**	–				
3. Other-blame (CERQ)	.09*	.12**	–			
4. Catastrophizing (CERQ)	.33**	.47**	.48**	–		
5. Rumination (CERQ)	.34**	.38**	.31**	.58**	–	
6. Self-blame (CERQ)	.32**	.50**	.32**	.60**	.64**	–

Note: MASC: The Multidimensional Anxiety Scale for Children; CERQ: Cognitive Emotion Regulation Questionnaire; CDI: Children's Depression Inventory. ***p* < .01; **p* < .05

Mediation Analysis

Taken the previous results and our hypothesis, we tested a path model in which we investigated the mediation role of emotion regulation strategies (other-blame, catastrophizing, rumination, self-blame) in the relation between social anxiety and depression.

All the possible paths (direct and indirect paths between predictive, mediators and criterion variables) were not included in the final model (see Figure 1). The paths social anxiety → other-blame [*b* = 0.099; *SE_b* = 0.047; *Z* = 2.112; *p* = 0.035; β = 0.099]; rumination → depressive symptomatology [*b* = -0.802; *SE_b* = 0.279; *Z* = 2.881; *p* = 0.014; β = -0.101]; catastrophizing → depressive symptomatology [*b* = 1.304; *SE_b* = 0.117; *Z* = 11.181; *p* = 0.012; β = -0.173]; and gender → depressive symptomatology [*b* = 1.613; *SE_b* = 0.497; *Z* = 10.423; *p* = 0.016; β = -0.112] were not statistically significant at .01 level, and by that reason were removed. Since our aim was to test possible mediation effects of cognitive emotion regulation strategies between social anxiety and depression, once one of the path to or from the mediator variable was non-significant (trajectories *a* and *b*) we removed the related path (trajectories *b* and *a*).

The mediation model (Figure 2) was tested through a fully saturated model (i.e., zero degrees of freedom). Model fit indices are not reported since fully saturated models always produce a perfect fit to the data.

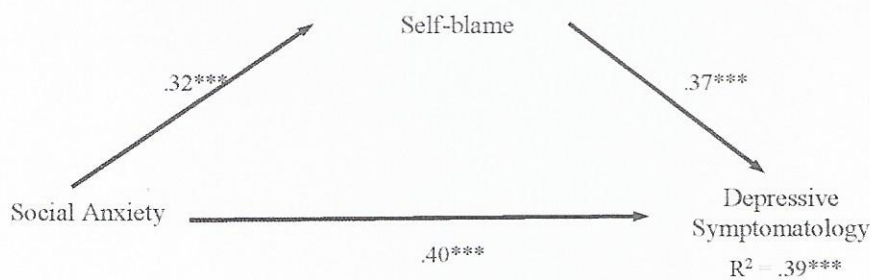


Figure 2. Mediation Model. Standardized path coefficients among variables are presented. ****p* < .001

The model accounted for 39% of the explained variance of depressive symptoms. A significant indirect effect of social anxiety on depression through self-blame [β = .119 (95% CI = 0.79; 1.62), *p* = .001] was found. However because the standardized direct effect from social anxiety on depression was also statistically significant [β = .400 (95% CI = .329; .474), *p* = .001] we can only consider the existence of a partial mediation. The total effect, that represents the sum of the standardized direct effect with the standardized indirect effect was [β = .519 (95% CI = 4.762; 6.472), *p* = .001]. Thus, the indirect effect mediated by self-blame consisted of 23% (.119 / .519) of the total effect of social anxiety on depression, suggesting that part of the effect of social anxiety on depressive symptoms was explained by self-blame.

Discussion

The transition into adolescence is characterized by important biological, cognitive, and psychosocial changes. It is a period when many typical social fears and worries emerge and vulnerability to emotional difficulties increase (Rapee & Spence, 2004). Recent reviews have shown that prevalence of mental health problems rises among adolescents, namely social anxiety and depression (Avenevoli et al. 2015, Burstein, He, Kattan, Albano, Avenevoli, & Merikangas, 2011).

Major depressive disorder (MDD) and social anxiety disorder (SAD) often co-occur in adolescence, with SAD preceding the onset of MDD (e.g., Beesdo et al., 2007). Many authors speculate about the mechanism(s) by which pre-existing SAD may increase the risk of subsequent depression. One possible explanation is that some features of SAD predispose to the development of MDD, namely through cognitive emotion regulation strategies, once difficulties in emotion regulation are proposed to be associated with a wide range of emotional disorders (e.g., Hofmann et al., 2012).

While depression has been consistently associated to dysfunctional emotion regulation strategies (e.g., Garnefski & Kraaij, 2006), and despite the fact that conceptualization models of SAD include difficulties in emotion regulation (Clark & Wells, 1995), only recently has clinical research focused on emotion regulation in SAD, confirming that this condition is associated with the use of particular emotion regulation strategies (e.g., Eastabrook et al., 2014).

Given the scarcity of research linking social anxiety, depression and emotion regulation strategies, especially in adolescence, the aim of our study was to explore the predictive power of social anxiety over depression and a possible mediation effect of maladaptive cognitive emotion regulation strategies in this relationship. Thus, we hypothesized that social anxiety was a predictor of depressive symptoms, and the effect of social anxiety on depressive symptomatology would be mediated by self-blame, rumination, catastrophizing and other-blame.

In accordance with our hypothesis, results showed that depressive symptoms were significantly, positively and highly correlated with social anxiety. These findings are in line with prior research demonstrating that social anxiety has been positively related with depressive symptoms (Stein, et al. 2001, Wittchen et al., 2003).

As expected, and consistent with other studies, correlation analyses showed that other-blame, catastrophizing, rumination and self-blame were significantly associated with depressive symptoms (Garnefski et al., 2001; Garnefski, et al., 2002; Garnefski, et al., 2003) and social anxiety (D'Avanzato et al., 2013; Eastabrook et al., 2014; Gilbert & Miles, 2000). In general, previous studies suggested that by using cognitive emotion regulation strategies such as self-blaming, rumination and catastrophizing adolescents may be more vulnerable to emotional problems (Garnefski et al., 2001).

However, we found that blaming others showed very small correlations both with social anxiety and with depression. Our results seem to be in line with Garnefski and colleagues (2001). On the other hand, the psychometric study of the Cognitive Emotion Regulation Questionnaire, as well as its prospective relationships with symptoms of depression and anxiety showed non correlation between blaming others, anxiety and depressive symptoms, in adults (Garnefski & Kraaij, 2007).

In the prediction of depressive symptoms, social anxiety had a strong effect on depression, either directly or indirectly, accounting for 39% of the variance explained.

From the four cognitive emotion regulation strategies that entered as mediators (self-blame, other-blame, catastrophizing, and rumination), only self-blame was shown to be a significant partial mediator in the relationship between social anxiety and depression, accounting for 23% of the total variance explained. This finding consists of an extension to the existing literature in adolescents and may be related to several factors. In fact, individuals with SAD (or with high social anxiety) are known for interpreting aversive social outcomes, such as criticism or rejection, as due to their own inadequacy, blaming themselves, and not the others, for these shortcomings (Clark & Wells, 1995). This characteristic may be responsible for other-blame not being found as a significant mediator and for self-blame being the only significant mediator. Since there are no other studies that have explore these relationships, we cannot compare our results with those of other studies, recommending the replication of this finding in future studies.

Unlike both studies that found that rumination (Drost et al., 2014) and brooding (Grant et al., 2014) mediated the relation between social anxiety and depression, our study did not confirm such a role for rumination. Drost and collaborators (2014) found that rumination mediated the relation between fear disorders, including SAD, and distress disorders, including MDD, in our study rumination was not

found to be a mediator of the relationship between social anxiety and depression. One possible reason for this fact may relate to the different instruments used in both studies. In our study, the Rumination factor from CERQ (Garnefski, et al., 2001) was used. This instrument asks subjects to rate the frequency of certain thoughts when a negative life event occurs. Rumination items refer to thinking about feelings and thoughts associated with the event. An example of an item is "I am preoccupied with what I think and feel about what I have experienced". On the other hand, Drost and collaborators have used the Leiden Index of Depression Sensitivity (LEIDS-R; Van der Does, 2002), which measures cognitive reactivity to sad mood. An example of an item is "When in a sad mood, I more often think about how my life could have been different". This instrument, unlike ours, assesses how people react to an already existent sad mood, thereby setting the way to an overlap between social anxiety and depressive symptoms, which can explain the mediation found. Instead, CERQ does not assume that sad mood is present, assessing how people react to a negative event.

A similar situation happened in the Grant and collaborators' study (2014). In this study, the instrument to measure brooding was the Brooding Subscale from the Ruminative Response Scale (RRS; Nolen-Hoeksema and Morrow 1991; Treynor et al., 2003) that assesses the degree to which an individual focuses on his/hers depressive symptoms and their consequences. An item example is "What am I doing to deserve this?". Therefore, the same hypothesis put forward in the previous case may also apply.

Another possible explanation for this discrepancy among studies may be related to the samples used. In fact, the sample used in Drost and collaborators' study was a clinical adult sample, and the sample from Grant and colleagues (2014), although being a community sample, was also an adult sample. Conversely, our sample was a community adolescent sample. It is possible that these rumination processes are not yet as developed in adolescents as they are in adults, particularly in clinical samples.

One final explanatory hypothesis for these different findings may underlie the fact that neither rumination nor catastrophizing were found to be mediators between social anxiety and depression. As assessed by CERQ, catastrophizing refers to thoughts that emphasize the terror of what was experienced. An example is "I continually think how horrible the situation has been". Thus defined, both catastrophizing and rumination (defined above) could, indeed, take place during post event processing, when individuals with high social anxiety review the social situation in detail (Clark & Wells, 1995), focusing on perceived shortcomings, negative images of oneself or flaws in one's performance. However, post-event processing seems less likely to include thoughts about thoughts and feelings related to the event (rumination) or thoughts about how horrible the situation was, in general (catastrophizing). Instead it includes a detailed revision of the interaction, dominated by negative perceptions of the self and of social performance (Wells & Clark, 1997), where individuals assume the responsibility for social flaws, in a mainly self-critical process, closer to self-blame, which is also a factor frequently associated with depression (e.g., Garnefski et al., 2002). In line with this, Seabra e Salvador (2015) have found that self-criticism was a significant predictor of post-event processing in a sample of SAD adult patients. If catastrophizing would be prospective, i.e., related to thinking about thoughts and feelings the individual believes will be associated to a forthcoming event, as it happens in the anticipatory processing so characteristic of SAD (Clark & Wells, 1995), we hypothesize that stronger associations would be found. However, we would still not expect this variable to mediate the effect of social anxiety on depression, given that depressive individuals are more prone to ruminate about past events and not so much catastrophizing about future events.

The finding that self-blame mediated the relation between social anxiety and depression may be related to the impact of self-blame in the decrease of mood and interference with problem solving, self-blame probably resulting from post-event processing where the individuals assume the responsibility for what has happened and criticize themselves for that. Moreover, assuming the blame for past negative events also fits the well-known internal, stable and global attributional style for negative events (Abramson, Seligman, & Teasdale, 1978), typical of depression. Therefore self-blaming in response to social mishaps (as it happens in SAD) may make the adolescents more vulnerable to develop depressive symptoms and, eventually, a full blown major depressive episode.

Beyond this indirect effect, social anxiety did not lose its predictive power, also presenting a substantial direct effect over depression. There are a variety of mechanisms through which social anxiety can have an impact on depression. For instance, social withdrawal characteristic of SAD may lead to have fewer friends, peer rejection, feelings of loneliness, sadness, and low self-worth or self-esteem (Beidel et al., 1999; Beidel et al., 2007; Gazelle & Ladd, 2003), also characteristic of depression.

Moreover, SAD patients tend to make negative inferences about the meaning of adverse social events for their future and their self-worth (Stopa & Clark, 2000), which may, in turn, function as a vulnerability to develop MDD (Abramson, Metalsky & Alloy, 1989). Further studies are needed to explore possible mechanisms responsible for this effect.

Regardless of these mechanisms, our results are in line with other studies that considered social anxiety as a risk factor for the development of depression (Beesdo et al., 2007; Dalrymple & Zimmerman, 2011; Kessler et al., 1999).

At the same time, this result also points to the probably high percentage of adolescents with depression that may have a primary SAD diagnosis. SAD patients, particularly adolescents, rarely seek treatment for their condition. In addition, since SAD is a “silent” disorder, they are also rarely referred to therapy primarily for SAD. Instead, other more acute disorder is the reason for seeking treatment or being referred to therapy, namely MDD (Lecrubier, 1998). This is probably one of the reasons why, despite its high prevalence and impact, SAD often goes under-recognized and under-treated (Brown et al., 2001; Dalrymple & Zimmerman, 2007; Lecrubier, 1998; Zimmerman & Chelminski, 2003). This is particularly problematic due to SAD’s chronic course (APA, 2013), and its increased persistence and severity related to its early onset (Kessler, 2003). On the other hand, MDD with SAD as a comorbid disorder is associated with higher levels of severity, chronicity and relapse (e.g., Dalrymple, & Zimmerman, 2007, 2011; DeWit et al., 1999; Lewinsohn et al., 1995; Stein, et al, 2001). This may mean that, if left unrecognized and untreated in prevention and intervention programs for depression, SAD may function as a risk factor for relapse, undermining the benefits of these interventions. In fact, evidence suggests that untreated comorbid SAD may affect the treatment outcome of MDD (DeRubeis et al., 2005; Isometsä, Holma, Holma, Melartin, & Rytysala, 2008).

Clinical Implications

In terms of clinical implications, the results of this study point to several important clinical implications. Since social anxiety was found to be a predictive factor of depression, an important target for preventive intervention of depression may therefore be to prevent or intervene in social anxiety symptoms. This may imply to screen adolescents with depressive symptoms for social anxiety symptoms and either redirect them for a specific program or to include a specific component to address such symptoms in programs primarily aimed at preventing or treating depression.

Secondly, the important role played by self-blame in the relationship between social anxiety and depression points to the importance of including a component to target it in prevention and intervention programs, either addressing depressive symptoms/MDD or addressing social anxiety/SAD. In general, these interventions should aim to reduce the use of maladaptive cognitive emotion regulation strategies, preventing them from turning into long established patterns of dealing with negative emotions, and to develop and improve the use of more adaptive strategies. In line with this suggestion, third wave therapies and interventions, like Acceptance and Commitment Therapy (Hayes, Strosahl, Wilson, 1999), Mindfulness (Kabat-Zinn, 1994) or Compassion Focused Therapy (Gilbert, 2005), could be of use. Acceptance of uncomfortable thoughts, feelings and sensations, conscious attention and letting go of old negative cognitive patterns, and compassionate acceptance of personal flaws or shortcomings may counteract the deleterious effect of self-blame and self-criticism.

Also, since these results were obtained in a considerable young sample (adolescents between 13 and 15 years old), our findings confirm the introduction of prevention programs at an early age.

Limitations

The current study has some limitations that should be mentioned. The research involves a sample of the general population of specific geographical areas of Portugal, being relevant that, in the future, the study should be replicated in broader samples of the community, representative of the Portuguese population, in order to allow generalization of results. In addition, the use of a clinical sample in future studies should also ensure more robust results.

The exclusive use of self-report questionnaires is also a limitation of this study. Using structured interviews and other informers (e.g., parents) should be included in future studies.

Other variables that were not controlled may have influenced subjects’ answers, such as social desirability.

The cross-sectional and correlational nature of this study should also be taken into consideration. In this sense, causal relations between variables cannot be established, only interpretations based on theoretical literature. Causal relations between variables should be analysed within a longitudinal design.

Given the aim of our study was to explore the predictive power of social anxiety over depression and a possible mediation effect of maladaptive cognitive emotion regulation strategies in this relationship, adaptive cognitive emotion regulation strategies have not been included. Further studies should include adaptive emotion regulation strategies. Additionally, in order to explore other mechanisms underlying social anxiety and depression, variables such as suppression, acceptance or experiential avoidance should be explored in future studies, relating social anxiety and depression.

In conclusion, this study clearly adds further evidence to the existing literature, showing the impact social anxiety may have in the development of depressive symptoms, either directly or indirectly, through self-blame. Subsequently, the use of maladaptive cognitive emotion regulation strategies, in this case, self-blame, can play an important role in the beginning and exacerbation of depressive symptoms. It is therefore reasonable to suggest that social anxiety and depression have a cause-and-effect relationship, at least in some cases.

The results suggest that having a better understanding of shared factors between SAD and MDD may be relevant both in theoretical terms and to develop more effective therapeutic interventions. In this sequence, tailoring interventions to include strategies addressing social anxiety and aimed at improving emotion regulation may have a positive impact on the treatment outcome.

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