

Longitudinal study of life events, well-being, emotional regulation and depressive symptomatology

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Abstract

Etiological research regarding adolescent depression is relatively recent and longitudinal studies are needed to explicate the mechanisms by which risk and protective factors impact the development of depression. This investigation evaluated the effects of time 1 predictors (e.g., depressive symptoms, life events, subjective well-being, and emotion regulation strategies) on depressive symptoms assessed at time 2 (one year later). The sample comprised 182 Portuguese adolescents, aged 12-16. Time 1 depressive symptoms and psychological well-being were significant predictors of time 2 depressive symptoms. Time 1 rumination predicted time 2 depressive symptoms, but time 2 brooding mediated this effect. Time 1 life events did not predict time 2 depressive symptomatology. This study identified several factors that seem to play an important role in the development of depressive symptomatology. Previous depressive symptomatology and well-being had a significant effect in this process and the relationship between rumination in time 1 and depressive symptoms at time 2 (one year later) was fully mediated by brooding at time 2. Given these results, implications for prevention and treatment of depression include addressing risk factors like early depressive symptoms, rumination and brooding, and increasing the protective effect of psychological well-being.

Keywords: depressive symptoms, rumination, brooding, subjective well-being, life events

Longitudinal study of life events, well-being, emotional regulation and depressive symptomatology

Across the life span, depressive disorders are heterogeneous in their expression, explained by multiple causes, and have mixed prognoses (Marujo, 2000). During the past two decades empirical evidence has shown an increasingly earlier onset of depression (Burke, Burke, Regier, & Rae, 1990; Kovacs, Akistal, Gatsonis, & Parrone, 1994; Lewinsohn, Clarke, Seeley, & Rohde, 1994; Zisook, et al., 2004). These findings have implications for the nature, symptomatic presentation, prevention, and treatment of depression; they also highlight the importance of the acquisition and development of age-appropriate protective skills or resilience.

Depressive episodes occurring first during adolescence follow a relatively stable course, typically becoming more frequent and more severe over time. Adolescent depression produces a high risk of recurrence, as 40% of adolescents experience a new depressive episode within 2 years after recovery and in 70% of cases depressive episodes reoccur within 5 years (Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013). It is also highly comorbid with other disorders that worsens the prognosis (Angold, Costello, & Erkanli, 1999; Cicchetti & Toth, 1998; Hankin, 2009), and it interferes with the overall functioning of the individual (Angold & Costello, 1993; Rohde et al., 2013; Rao, Hammen, & Daley, 1999).

The preceding data justify more extensive research on depressive disorders, and reinforce the urgency and need to develop and implement targeted interventions for adolescents with subclinical depressive symptoms. In regard to the development of prevention programs, it is imperative to identify protective and risk factors prior to the development of major depression.

Previous models of depression have identified several factors that render an individual “at risk” for developing depressive symptoms; these include the existence of prior depressive symptoms, negative life events, and some cognitive regulation strategies such as catastrophizing and rumination. For example, several authors (Ge, Conger, & Elder, 2001; Nolen-Hoeksema, Girgus, & Seligman, 1992; Petersen, Sirigiani, & Kennedy, 1991; Susman, Dorn, & Chrousos,

1991) have shown that higher levels of depressive symptomatology in pre-adolescence predict higher levels of depressive symptoms during ensuing years. Among samples of adolescents, Arnarson and Craighead (2011) and Verstraeten, Vasey, Raes, and Bijttebier (2009) found that depressive symptoms predicted depressive symptomatology 12 months later.

Negative life events have also predicted adolescent depression (Patton, Coffey, Posterino, Carlin, & Bowes, 2003). Longitudinal studies with adolescents have frequently shown that the experience of stressful life events precedes the development, recurrence, or exacerbation of depressive symptomatology. (e.g., Ge et al., 2001; Goodyer, Herbert, Tamplin, & Altham, 2000). In an adolescent sample, Carter, Garber, Ciesla, and Cole (2006) found that stressful life events predicted depressive symptoms one year later. Kercher, Rapee, and Schniering (2009) reported, however, that negative life events and negative thoughts mediate the frequently obtained relationship between prior depressive symptomatology and subsequent depressive symptoms.

It is important to note, however, that not all adolescents exposed to negative life events develop emotional problems. A growing number of studies have shown that stressful life events considered *alone* are not sufficient to predict emotional problems (Ingram, Miranda, & Segal, 1998). The coping strategies that adolescents use in face of a negative life event may influence the relationship between a life event and depressive symptomatology (Compas, Orosan, & Grant, 1993). The cognitive strategies falling under the rubric of “emotional regulation” have been emphasized as skills that facilitate adolescents’ coping with a stressful life event. Emotional regulation appears to be an essential component of mental health, and difficulties with emotional regulation are associated with various forms of psychopathology (Cicchetti, Ackerman, & Izard, 1995; Gross, 1998). In a study evaluating the emotional responses of adolescents in their daily lives, participants who showed a deficit in the regulation of negative emotions experienced high levels of depressive symptoms compared to those who possessed adaptive levels of emotional regulation (Silk, Steinberg, & Morris, 2003).

In fact, specific cognitive patterns (e.g., self-blame, rumination, and catastrophizing) characterizing emotional dysregulation may play an important role in the impact of negative life

events and the development of depressive symptomatology among adolescents (Garnefski, Kraaij, & Spinhoven, 2001, 2002). Various studies have reported that self-blame, rumination, and catastrophizing are directly and positively related to high depressive symptomatology, whereas responses of positive reappraisal are inversely related to depressive symptoms among children, adolescents, and adults (Garnefski, Boon, & Kraaij, 2003; Garnefski & Kraaij, 2006a, 2006b; Garnefski, Rieffe, Jellesma, Terwogt, & Kraaij, 2007; Garnefski, Teerds, Kraaij, Legerstee, & van den Kommer, 2004; Martin & Dahlen, 2005). Among the cognitive patterns related to depression, rumination has been the most comprehensively studied risk factor (Pearson, Watkins, & Mullan, 2010). Longitudinal studies with community samples have shown that the tendency to adopt a ruminative style, when distressed, is a stable characteristic of depressed individuals (Nolen-Hoeksema & Davis, 1999). Additionally, longitudinal studies with adolescents have shown that rumination is predictive of the development of depressive symptoms (Broderick & Korteland, 2004; Burwell & Shirk, 2007; Hankin, 2008b; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Schwartz & Koenig, 1996) and of the onset of clinical depressive episodes (Abela & Hankin, 2011; Nolen-Hoeksema et al., 2007). Cross-sectional studies have also shown the importance of rumination in the maintenance of depressive episodes in adolescents (for a review, see Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008 or Olatunji, Naragon-Gainey, & Wolitzky-Taylor, 2013). Rumination may also be a mediator of the relationship between certain risk factors (such as the adoption of a negative cognitive style, self-blame, and past history of depression) and current levels of depression (Spasojević & Alloy, 2001). For example, Jose and Brown (2008) and Grodewald, (2010) showed that rumination mediated the relationship between stress and depressive symptoms in childhood and adolescence.

Response Styles Theory (Nolen-Hoeksema, 1991) defined rumination as a passive and repetitive way of thinking about experienced depressive symptoms and the possible causes and consequences of these symptoms. Moreover, Treynor, Gonzalez, and Nolen-Hoeksema (2003) stressed the existence of two distinct factors in rumination: brooding – “a passive comparison of the current situation to a non-achieved standard” (p. 256) and reflection – “a purposeful turning inward

to engage in cognitive problem solving” (p. 256). The maladaptive component of rumination, brooding, was remarkable as the strongest predictor of depressive symptomatology among both adults and adolescents (Burwell & Shirk, 2007; Gortner, Rude, & Pennebaker, 2006; Grassia & Gibb, 2008; Joormann, Dkane, & Gotlib, 2006; Miranda & Nolen-Hoeksema, 2007; Pearson et al., 2010; Treynor et al., 2003). In contrast, reflection is an adaptive form of rumination and has been less predictive of and less strongly associated with depressive symptoms (Burwell & Shirk, 2007; Crane, Barnhofer, & Williams, 2007; Joormann et al., 2006; Treynor et al., 2003). Brooding has also been identified as a *mediator* of the relationship between several factors in the prediction of depression during adolescence. For example, brooding mediates the effects of neuroticism (Kuyken, Watkins, Holden, & Cook, 2006), negative attributional style (Lo, Ho, & Hollon, 2008), passive coping (Marroquín, Fontes, Scilletta, & Miranda, 2010), self-compassion (Raes, 2010), maladaptive perfectionism (Harris, Pepper, & Maack, 2008), and negative emotionality (Mezulis, Simonson, McCauley, & Stoep, 2011) in depressive symptomatology among adolescents.

Complementing the study of risk factors is the need for the identification of resiliency factors, factors that protect against the development of depression. The identification of protective factors and the subsequent development of effective strategies to enhance resilience factors (while simultaneously decreasing risk factors) may provide the key to success in preventing depression among adolescents (Shortt & Spence, 2006). Flourishing is one of the most extensively studied protective factors that may play a key role in preventing depression among youth. Keyes (2009) has identified the components of flourishing among teens. Studying 1,200 adolescents from 12-18 years of age, Keyes (2009) obtained three internally consistent latent factors of flourishing. The factors were: 1) emotional well-being (the perception of happiness and the level of life satisfaction), 2) social well-being (the quality of personal performance in social contexts), and 3) psychological well-being (efforts of individuals to promote functioning at fullest levels of satisfaction and successfully overcome life challenges).

Flourishing describes one end of a bipolar continuum with languishing at the opposite end (Keyes, 2002); the flourishing/languishing dimension is obliquely related to a mental health/mental

illness dimension. Among adults, resilient individuals are characterized by the absence of mental illness and the presence of high levels of flourishing (Doll, 2008; Greenspoon & Saklofske, 2001; Keyes, 2002, 2005, 2006a, 2006b, 2007; Keyes & Lopez, 2002; Suldo & Shaffer, 2008). Flourishing individuals reveal hedonic characteristics (i.e., seek pleasure and avoid what is unpleasant) and report positive feelings and adaptive functioning, whereas depressed individuals are characterized by the presence of symptoms such as loss of interest and pleasure, negative mood, and an inadequate and dysfunctional pattern of behavior. Thus, it is hypothesized that, among adolescents, the higher the flourishing levels experienced, the greater the tendency to have adaptive and healthy forms of behavior, with flourishing individuals revealing a lower probability of becoming depressed (Keyes, 2006a, 2006b; 2007; Keyes et al., 2008). Languishing individuals, in contrast, have a higher probability of becoming depressed (Keyes, 2006a, 2006b; Keyes, Dhingra, & Simões, 2010). Among the flourishing subscales, psychological well-being has been identified as most strongly and negatively associated with depressive symptomatology (André, 2009; Fernandes, 2011a; Matos et al., 2010). Wood and Joseph (2010) found that adults with low levels of well-being were over 7 times more likely to be depressed 10 years later; even after controlling for prior depression and demographic, personality and medical variables, people with low well-being were still over twice as likely to be depressed.

Within the context of the preceding and relevant literatures, the present study had two aims (represented in Figure 1):

- 1) To explore the direct effect of variables measured at baseline [time 1 (t1)], on depressive symptoms measured 12 months later [time 2 (t2)]. The predictor variables included risk and protective factors: previous depressive symptoms, cognitive strategies of emotional regulation (self-blame, rumination, catastrophizing, and brooding), life events (family and parents, relationships, school and classes, and friends and social activities), and well being (emotional, social and psychological).
- 2) To test mediation (partial or total) between risk and protective factors measured at t1, and depressive symptomatology measured at t2. It was hypothesized that life events,

prior depressive symptoms, well-being, and emotional regulation cognitive strategies would have an indirect effect on depressive symptoms through emotional regulation strategies – brooding, self-blame, rumination and catastrophizing, measured at t2 (mediating variables).

INSERT FIGURE 1

Method

Sample and Participant Selection

This study sample comprised 182 adolescents in Portugal. The sample included 130 females (71.4%) and 52 males (28.6%) aged 12-16 ($M = 13.65$, $SD = 1.43$), with an educational level of grades 5-11 ($M = 8$, $SD = 1.50$). In terms of the family socioeconomic status, 52.3% belonged to a low socioeconomic level and 47.7% belonged to a middle or high socioeconomic level.

Prior to data collection, the legal authorities that regulate research and the schools' board of directors approved the project. Both parental and students were informed of the research aims and their written consents were obtained. In order to safeguard the anonymity and confidentiality of the data, an identification code corresponding to each adolescent was created to identify each protocol. The coded list with participants' names was kept locked in the office of the first author. Thus, only the code number for each student was associated with the actual data.

Data collection took place in six schools in central and northern Portugal, at two different times 12 months apart, between 2010 and 2011. The completion of the protocols occurred during January-February in the full classroom context and in the presence of both teachers and researchers. Exclusion criteria of participants were: (a) less than 12 years or more than 16 years of age; and (b) clear evidence of difficulties in understanding the assessment instructions (1% of the sample).

Assessment and Measures

Emotional regulation was assessed with two following self-report questionnaires. The **Cognitive Emotion Regulation Questionnaire** (CERQ, Garnefski et al., 2001; Portuguese version: Serra, 2009) measures functional and dysfunctional cognitive strategies for emotional

regulation. It is composed of 36 items rated from 1 (almost never) to 5 (almost always), and the CERQ yields nine distinct 4-item sub-scales: self-blame, rumination, catastrophizing, other blame, acceptance, positive refocusing, refocus on planning, positive reappraisal, and putting into perspective. The authors of the original study in a population between 13 to 15 years-of-age found that the internal consistency ranged between .68 and .83 for each sub-scale. In the Portuguese version standardization, the Cronbach alphas for each subscale also varied between .69 and .83. In the present study, we only used the scores of the negative regulation strategies for which Cronbach's alphas were: .63 for self-blame, .69 for rumination, and .76 for catastrophizing. The other blame sub-scale and the adaptive dimensions of the questionnaire were not used because in prior studies these factors have not been significantly associated with depressive symptoms in adolescents and adults (Garnefski et al., 2003, 2004; Garnefski & Kraaij, 2006b).

The second emotional regulation measure was the **Rumination Responses Scale-10** (RRS-10; Treynor et al., 2003; Portuguese version: Dinis, Pinto Gouveia, Duarte, & Castro, 2011), which consists of 10 items (short version of the 22-item Ruminative Responses Questionnaire), developed for the adult population. Grant et al. (2004) demonstrated that the RRS-10 can be completed by children and adolescents, and several studies with satisfactory psychometric results have been conducted with youth (Hilt, McLaughlin, & Nolen-Hoeksema, 2010; Jose & Brown, 2008; Morris, Blyma, & Rottenberg, 2009). This scale is designed to determine if individuals adopt a ruminative response style to deal with depressed mood. Subjects respond to each of the 10 items on a 4-point Likert rating scale (from 1 – almost never; 4 – almost always). The RRS-10 has two component scores: a) reflection, which suggests a purposeful turning inward to engage in cognitive problem solving to alleviate one's depressive symptoms and is therefore not predictive of depression; and b) brooding, which reflects a passive comparison of one's current situation with some unachieved standard. Both the original and the Portuguese versions have found satisfactory levels of internal consistency, ranging from .71 to .77. In the present study we have only used the brooding factor because it is a maladaptive variable predictive of depression; its Cronbach's alpha in this study was .76.

The Adolescent Life Events Questionnaire was used to assess life events. The **Adolescents Life Events Questionnaire** (ALEQ, Hankin & Abramson, 2002; Portuguese version: Fernandes, 2011b) assesses typical adolescent negative life events that include problems in school/performance, difficulties in friendships and romantic relationships, and family problems. The ALEQ comprises 57 different negative life events. Hankin and Abramson (2002) recommended assessing the events for the past three months. In the present study, adolescents were asked to remember events that had taken place over the preceding 12 months. This decision was because this questionnaire includes major life events that may have an impact at the time the participants completed the questionnaire, even if they occurred more than three months ago. Each item is rated from 0 (never) to 4 (always) according to the frequency each event occurred during the past year. These scores were converted into a dichotomous classification of the events (never = 0 and scores from 1 to 4 = 1) following the method used by Hankin (2008a). Higher scores indicated a greater number of negative life events experienced. In its original version, Cronbach's alpha for the total score of the scale was .94. In the present study the Cronbach's alpha was .96 for the total scale, .92 for family and parents, .92 for friends and social activities, .85 for the school and classes, and .83 for relationships.

The **Children's Depression Inventory** (CDI, Kovacs, 1985, 1992; Portuguese version: Marujo, 1994), used in this study, is a self-report inventory of depressive symptoms for ages between 6 and 18. The CDI has 27 items with three alternative answers, and each item is rated with a score ranging from 0 (no problem) and 2 (severe problem). Kovacs (1985, 1992) and Smucker, Craighead, Craighead, and Green (1986) have found that this inventory has an excellent internal consistency (alpha coefficients of .83 to .94). The Portuguese version of this inventory showed high internal consistency with a Cronbach's alpha coefficient of .80 (Marujo, 1994). The factor structure of five factors found in the original study was not replicated in the Portuguese sample, where a one-factor structure proved to be more appropriate (Dias & Gonçalves, 1999). In the present study sample the Cronbach's alpha for this single factor score was .79.

Well-being was assessed with the Mental Health Continuum-Short Form. The **Mental Health Continuum-Short Form** (MHC-SF, Keyes, 2009; Portuguese version: Matos et al., 2010). The MHC-SF derives from the long form (MHC-LF), which consisted of 40 items most of them derived from Ryff's (1989) model of psychological well-being, Keyes' (1998) model of social well-being, and scales measuring emotional well-being (e.g., Bradburn, 1969). The MHC-SF consists of 14 items that were chosen as the most prototypical items representing the construct definition for each facet of well-being: a) *emotional well-being*; b) *social well-being*; and c) *psychological well-being*. The response option for the short form was established to measure the frequency with which respondents experienced each symptom of positive mental health. The items are rated as follows on a 5-point Likert scale: never (0), once or twice (1), about once a week (2), two or three times a week (3), almost every day (4), every day (5). The estimates of internal consistency reliability for each measure – emotional, psychological, and social well-being – in the MHC short and long forms have all been high (> .80; Keyes, 2005). In the Portuguese version (Matos et al., 2010), the values of Cronbach's alpha for the three factors were high: .85 for emotional well-being, .80 for social well-being, and .83 for psychological well-being. In the present study, the Cronbach's alpha values for the same factors were, respectively, .87, .80 and .77.

Procedure

The sample collection took place in schools in Northern and Central Portugal, between 2009 and 2011. After permissions and consents were obtained, the cooperation was sought and received from the schools that participated. A few members of the research team went to every school to explain the objectives of the study and to deliver and collect the questionnaires. All questionnaires were completed in a classroom setting, where only those that had permission and consented were present and participating under the supervision of a researcher investigator. One-half of the students completed the questionnaires in the following order: CDI, MHC-SF, CERQ, ALEQ, RRS. The other half completed the same questionnaires in the reverse order. Participants completed the same questionnaires one year later. To ensure anonymity and confidentiality of data, a code was created that corresponded to each adolescent and consequently to each research

protocol. All identifying materials were kept in locked file cabinets in the office of the lead investigator.

Results

Preliminary Data Analysis

T-tests and ANOVAs were performed for all Time 1 variables. Males and females did not differ on age [$t(180) = -.201, p = .841$] or educational level [$t(180) = -.171, p = .865$]. T-tests between genders showed that boys and girls did not differ on any of the variables except CERQ Rumination scores; girls ($M = 3.02, SD = .81$) scored slightly higher than boys ($M = 2.76, SD = .65$); $t(116) = -2.27, p = .025$). A one-way ANOVA was computed to explore possible differences between ages in all the variables under study. To achieve this aim, three groups were formed: 12 year old adolescents ($N = 60$), 13-14 year old adolescents ($N = 61$), and 15-16 year old adolescents ($N = 61$). Significant differences between these groups were found in three Life Events factors: family related events [$F(2, 179) = 5.129, p = .007$], school related events [$F(2, 179) = 8.946, p < .001$], and negative events related to friends [$F(2, 179) = 3.378, p = .036$]. Tukey post-hoc comparisons showed that the mean scores of the 15-16 year-old adolescents group were always higher than those of adolescents who were 12 or 13-14 years old, with the latter two groups not differing from each other in family related events [15-16 (Mean = .46, $SD = .21$), 13-14 (Mean = .34, $SD = .26$), and 12 (Mean = .34, $SD = .27$)], school events [15-16 (Mean = .60, $SD = .27$); 13-14 (Mean = .42, $SD = .33$); 12 (Mean = .39, $SD = .29$)], and events related to friends [15-16 (Mean = .45, $SD = .27$); 13-14 (Mean = .34, $SD = .31$); 12 (Mean = .32, $SD = .31$)].

Our sample's CDI scores were below cut off points usually used to identify depressive and non-depressive youths. A score of 19 is the most frequent cut-off point for screening depressed samples. However, a score of 17 has been identified as more sensitive and selective for discrimination of Major Depressive Disorder in adolescent clinical samples (Craighead, Curry, & Ilardi, 1995; Kendall, Stark, & Adam, 1990; Kovacs, 1992; Ollendick & Yule, 1990; Smucker, Craighead, Craighead, & Green, 1986). From the 182 adolescents, 23 (12.64%) had CDI scores above 17 and 16 (8.79%) had CDI scores above 19.

Means and standard deviations for all variables were also analyzed at Time 2 via t-tests and ANOVAs as appropriate. T-tests explored possible significant differences between genders and showed that boys and girls did not differ on any of the variables. An ANOVA revealed no differences among the three age groups. For the CDI scores at Time 2, 27 of the 182 (14.84%) scored above 17, and 18 adolescents scored above 19 (9.89%), again showing that the majority of our sample did not report significant depressive symptomatology and demonstrating the representativeness of this sample. We found that 12 adolescents (6.59%) were above 17 at both Time 1 and Time 2 assessments, and 9 (4.95%) were above 19 at both times. For adolescents with scores of 17 or higher at both assessments, the parents were informed and advised to seek mental health services for their children.

Effects in multiple mediator models

Given the high number of variables studied, we first conducted a stepwise regression analysis to check which of the 11 independent variables (at Time 1) had a predictive effect on the dependent variable (CDI) at Time 2. This overall model was significantly different from the null model ($F(3,178) = 36.755, p < .001$), explained a substantial percentage of variability (adjusted $R^2 = .372$), and did not present any problems of multicollinearity. Rumination (CERQrum.t1) ($\beta = .130, t = 2.182, p = .030$), previous depressive symptomatology (CDI t1) ($\beta = .534, t = 8.440, p < .001$) and psychological well-being (MHC-SFpsyWB t1) ($\beta = -.150, t = -2.360, p = .019$) significantly predicted Time 2 CDI scores.

Mediation analyses were performed using SPSS macro INDIRECT (Preacher & Hayes, 2008), which allows for the examination of mediation effects of a variable conditional on the effects of the other variables in the model. Bootstrapping with 95% confidence intervals (CI) was utilized to determine significance of mediation effects. This method does not assume that the sampling distribution of the indirect effects are normally distributed, and it is preferable to the products-of-coefficients approach in regard to Type I error rate, power, and hypothesis testing (Preacher & Hayes, 2008).

For theoretical reasons, from the three significant predictors found, rumination (CERQ t1) was selected as an independent variable, and previous depressive symptomatology (CDI t1) and psychological well-being (MHC-SFpsyWB t1) have been chosen as covariates. This decision about variable types was made for two reasons: because rumination has often proved to be a predictive of depression, and to control for the effects of previous depressive symptomatology and well-being. This allowed us to study the additional and unique effects of rumination as an emotional dysregulation variable.

We first confirmed that the independent variable rumination (CERQ rum.t1) had a significant direct effect on the dependent variable (CDI t2) ($\beta = .8570$, $t = 2.1816$, $p = .031$). Then, we checked the effect of the independent variable (CERQ rum.t1) on the mediator variables (Time 2 CERQ rumination, catastrophizing, and self-blame as well as RRS-brooding). All the relationships between the independent variable and mediator variables were statistically significant.

When analyzing the effects of mediator variables (CERQ rumination, self-blame and catastrophizing, and RRSbrooding, all at Time 2) on the dependent variable (CDI t2), the total indirect effect of the independent variable on the dependent variable through the four proposed mediators was not significant ($ab = .3481$, 95% CI: $[-.0148 - .7239]$), even when there were no problems related to multicollinearity ($VIF < 2$). Analysis of the indirect effect for each of the mediator variables indicated that only brooding (RRS brood t2; $\beta = .3828$, $t = 3.4046$, $p = .0008$) and self-blame (CERQ s.bla. t2; $\beta = 1.2833$, $t = 2.0009$, $p = .047$) had a significant mediation effect. Rumination (CERQ rum t2) and catastrophizing (CERQ cat t1) did not contribute to this mediation effect.

We therefore tested two models, including the two significant mediators and in each analysis one of the non-significant mediators. In both models, previous depressive symptomatology (CDI t1) and psychological well-being (MHCpsyWB t1) were controlled as covariates. In the first model (Fig. 2), which accounted for 51.7% of variance in CDI [$\text{Sum}(ab)/c = .4427 / .8570 = .517$], only brooding (RRS brood) was a significant mediator (95% CI $[.0878 - .7298]$). Control variables were statistically significant (CDI: $\beta = .4171$, $t = 7.3791$, $p < .001$; MHCpsyWB: $\beta = -.8968$, $t = -$

2.9584, $p = .004$). The total indirect effect of the independent variable on the dependent variable through the three proposed mediators was significant, ($ab = .4427$, 95% CI: [.1393-.8624]),

INSERT FIGURE 2

In the second model (Fig. 3), brooding (RRS-brood) and catastrophizing (CERQ cat.) were significant mediators (95% CI [.0993 - .7499] and 95%CI [.0997 - .5748], respectively), and accounted for 40.4 % of variance in CDI [Sum (ab)/c = $.3458 / .8570 = .404$]. None of the models presented problems with multicollinearity. Control variables were statistically significant (CDI: $\beta = .4097$, $t = 7.2609$, $p < .001$; MHCpsyWB: $\beta = -.8342$, $t = -2.7421$, $p = .007$). The total indirect effect of the independent variable on the dependent variable through the three proposed mediators was also significant, ($ab = .3458$, 95% CI: [.0023 - .7741]),

INSERT FIGURE 3

Once both models were significant, we chose the first model, both for its parsimony and for the higher percentage of total variance explained. In this model, only brooding mediated the relationship between Rumination at Time 1 (assessed by CERQ) and depressive symptomatology at Time 2. When both independent and mediator variables were included as predictors of the dependent variable, the mediator variable remained significant whereas the independent variable did not; thus, by conventional standards, we can say that the mediation found was a full mediation.

Correlations between the model variables

Tables 1, 2, and 3 present the correlations between the variables entered in the various models tested. Table 1 shows the correlations between all the variables at Time 1 that entered the initial model. Only the rather small negative correlation between psychological well-being and depressive symptomatology was found significant.

INSERT TABLE 1

Table 2 includes the correlations between variables at Time1 and variables at Time 2. The highest correlation was a moderate one between CDI at Time 1 and CDI at Time 2. Other correlations were either non-significant or significant but very low.

INSERT TABLE 2

Finally, table 3 presents the correlations among variables at Time 2. It is noteworthy that the correlation between the two rumination variables (CERQ rumination and RRS brooding) was only moderate ($r = .461$).

INSERT TABLE 3

Discussion

The current research study evaluated whether adolescent risk and protective factors measured at an initial assessment (T1) significantly predicted the presence of depressive symptoms measured one year later (T2). The study also explored whether the predicted relationships were mediated by emotional regulation.

An initial stepwise regression analysis revealed that rumination, previous depressive symptomatology, and psychological well-being were variables that significantly predicted T2 depressive symptoms. Further mediational analyses produced two different models, when emotion regulation strategies at T2 were considered mediator variables and T1 depressive symptomatology and well-being were controlled. The most parsimonious model and also the one that explained the higher percentage of variance was selected. This selected model explained 51.7% of the variance of T2 depressive symptoms and the effect of rumination was mediated by brooding; however, given the small sample size this result must be interpreted with care.

In contrast to the findings of some prior research investigations, the current study found that life events were not statistically significant predictors of T2 depressive symptomatology. Previous studies had found that life events predicted the subsequent development of depressive symptoms (Ge et al., 2001; Goodyer et al., 2000; Patton et al., 2003), and longitudinal studies with adolescents have shown that sometimes the experiencing of stress-inducing life events precedes the development, recurrence, or exacerbation of depressive symptomatology (e.g., O'Connor, Rasmussen, & Hawton, 2010). The discordant results of the prior findings and the current study may in part have been because the instrument used to assess life events (ALEQ) in the current study only measured whether the event had occurred, and it did not include an assessment of the impact of life events. In a separate Portuguese cross-sectional study (Salvador et al., 2015) that used a questionnaire evaluating the impact of life events (DHMS; Daily Hassles Microsystem Scale; Seidman, et al., 1995), the authors found that several areas of life events had a direct or indirect effect on depressive symptoms. Furthermore, Horesh, Klomek, and Apter (2008) found that the individual meaning assigned to the event and the meaning life events played key roles in the onset of depression. These results point to the need to use life events assessment tools that include measurement of the impact of life events and the personal meaning assigned to them. It may also be essential to investigate possible mechanisms (such as brooding) which interact with life events to have an impact on depressive symptomatology.

Previous depressive symptomatology (T1) has consistently predicted depressive symptomatology one year later (T2). In other words, the current data illustrate that individuals with some depressive symptoms are “at risk” for future clinical depression. The current results were consistent with this previous research (Arnarson & Craighead, 2011; Ge et al., 2001; Nolen-Hoeksema et al., 1992; Petersen et al., 1991; Susman et al., 1991; Verstraeten et al., 2009) highlighting the predictive power of previous depressive symptoms on the subsequent levels of depressive symptomatology. This finding points to the need for preventive intervention once some symptoms of depression have been identified, underscoring the importance of both early

intervention for depressive symptoms and the need to develop, implement, and evaluate programs to prevent clinical depression among adolescents at risk for the disorder.

Keyes (2002, 2005, 2006b) posited that flourishing is a protective factor against the development of psychiatric disorders. He conceptualized flourishing as a combination of high levels of well-being subtypes (emotional, psychological, and social). In the current data, only the psychological well-being component revealed a statistically significant effect on depressive symptoms at time 2, thereby indicating that psychological well-being has a protective effect on the later development of depressive symptoms. This conclusion matches results of several other studies (André, 2009; Fernandes, 2011a; Matos et al., 2010) that have found negative associations between psychological well-being and depressive symptomatology. However, the correlations found between T1 well-being and T1 and T2 depressive symptomatology are low to moderate, indicating that well-being is not merely the inverse of depression.

According to Keyes (2002, 2005, 2006b), psychological well-being includes a positive self-evaluation, having goals, a sense of meaning and direction in life, and warm and intimate relationships. It is suggested, therefore, that preventive interventions for at risk adolescents might also focus on the development of psychological well-being. It is recommended that programs for prevention of depressive symptoms in adolescents promote and emphasize protective factors as much as the management and neutralization of risk factors.

This study also evaluated the effect of emotional regulation strategies (self-blame, rumination, catastrophizing, and brooding) as possible predictor and mediating variables of future development of depressive symptoms. Only rumination (measured by T1 CERQ) had a direct effect predicting T2 depressive symptomatology, but as noted below this effect was mediated by brooding. Studies by several authors have demonstrated that rumination, self-blame, and catastrophizing are directly related to elevated subsequent depressive symptomatology among adolescents and adults (Garnefski, 2006a, 2006b; Kraaij et al., 2003; Kraaij, Garnefski, & Vlietstra, 2008; Martin & Dahlen, 2005; Omran, 2011). Prior longitudinal studies among adolescents indicated that rumination predicted depressive symptoms (Burwell & Shirk, 2007; Hankin, 2008b;

Nolen-Hoeksema et al., 2007; Schwartz & Koenig, 1996) and the onset of depression episodes (Abela & Hankin, 2011; Nolen-Hoeksema et al., 2007). In previous research among adults, catastrophizing was related to the existence of depressive symptoms two years later (Kraaij et al., 2008).

When analyzing the mediator effects of the emotional regulation variables (rumination, self-blame, catastrophizing and brooding) at T2 on the relationship between rumination measured at T1 and depressive symptomatology at T2, the total indirect effect of the four proposed mediators was not significant. When examining the indirect effect for each of the mediator variables, brooding and self-blame were the only significant predictors (rumination and catastrophizing did not contribute to the mediation effect). Thus, two other models were tested, both including the two significant mediators (brooding and self-blame) and then entering a third mediator. In the first model with brooding, self-blame, and rumination entered as mediators, only brooding was found to be a mediator: the effect of rumination at T1 on subsequent depressive symptomatology (at T2) was fully mediated by brooding at T2. In the second model, brooding and self-blame were again entered as mediators, along with catastrophizing. In this second model, both brooding and catastrophizing were significant mediators. Since both models were significant, the first one was chosen, due to its parsimony and to the higher percentage of total variance explained.

Brooding as a mediator of the relationship between general rumination and depression has been previously noted (Harris et al., 2008; Kuyken et al., 2006; Lo et al., 2008; Marroquín et al., 2010; Mezulis et al., 2011; Raes, 2010). The data seemed to suggest that rumination, understood as “thinking about thoughts and feelings associated with the negative event” (Garnefski et al., 2001, p. 1315), may be different from brooding, conceptualized as “a passive comparison of the current situation to a non-achieved standard” (Marroquín et al., 2010; Treynor et al., 2003, p. 256). The moderate correlation found between rumination and brooding also seems to indicate that, although related, these constructs are measuring different mechanisms.

Individuals who tend to brood may activate their underlying depressive schemas (Teasdale, 1999). Brooding may be a type of activating agent that triggers the cognitive reactivity

typically found in depressed patients. It is hypothesized that once brooding plays a mediational role in the development of depression, it is important to directly address brooding in the treatment of depression. Effective treatment techniques suggest that it may be more effective to disengage from the brooding at times of depressed mood, rather than using distraction to offset the negative effects of rumination broadly defined (Barber & DeRubeis, 1989; Teasdale, 1999). In classic CBT (Beck, 1995), it would be important to use cognitive techniques such as identifying and challenging cognitive distortions that are aimed at interrupting the vicious cycle between brooding and increased depressive symptomatology. Other treatment strategies might include behavioral activation, which views brooding as a cue to the patient to engage in behavioral activities (Addis & Martell, 2004), or attentional training to increase the metacognitive control (Papageorgiou & Wells, 2004; Wells, 2000) and thereby reduce brooding and depressive symptomatology (Papageorgiou & Wells, 2000). From yet another perspective, Watkins and colleagues (2007) developed a form of cognitive therapy focused on brooding to diminish residual levels of depression.

Cognitive therapy for relapse prevention also has implications for treatment of brooding associated with depression (Segal, Williams, & Teasdale, 2002). Mindfulness teaches patients who have remitted from depression to engage in mindful emotional processing which, in turn, reduces emerging dysphoria (Ma & Teasdale, 2004; Ramel, Goldin, Carmona, & McQuaid, 2004; Segal et al., 2002). Mindfulness has proven to be more effective than distraction in reducing depressive symptoms (Broderick, 2005). Broderick and Korteland (2004) have suggested exploring the effectiveness of mindfulness in the treatment of brooding in children and adolescents.

There are some study limitations that include: the exclusive use of self-report measures, and the use of a largely female and non-clinical sample. Thus, appropriate caution must be applied in generalizing the results of the current study. The larger proportion of girls did not seem to affect the *predictive* results in the study when gender was taken into account but the sample of males was small.

There were also strengths of the study including its longitudinal design, the inclusion of risk and protective predictors, the consideration of direct and mediational effects and their analysis through structural equations models.

Future studies might employ larger samples, more balanced in terms of gender, and might also employ additional assessment methodologies (e.g., interviews to assess diagnosis and life events). It will also be important to conduct longitudinal studies with depressed clinical samples because variables such as emotional regulation and negative life events might have a more prominent effect on depressive symptoms within a clinically depressed sample compared to their effects in a general community sample.

In conclusion, the results of this study have identified factors that play an important role in the longitudinal development of depressive symptomatology. Specifically, we found an effect of prior depressive symptomatology and psychological well-being. There was an indirect effect of rumination (T1) mediated by T2 brooding on T2 depressive symptoms.

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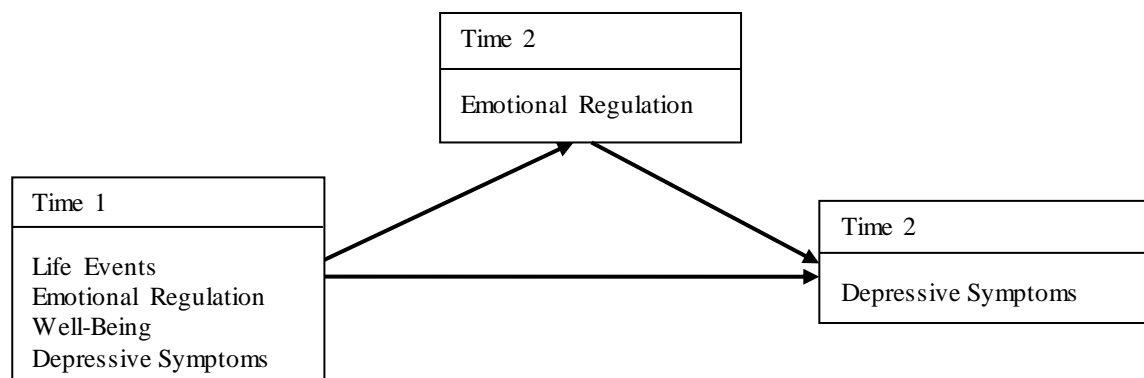


Figure 1. Proposed model for the mediation of emotional regulation (time 2) on depressive symptoms (time 2), having as predictors life events, emotional regulation strategies, subjective well-being, and depressive symptoms (time 1).

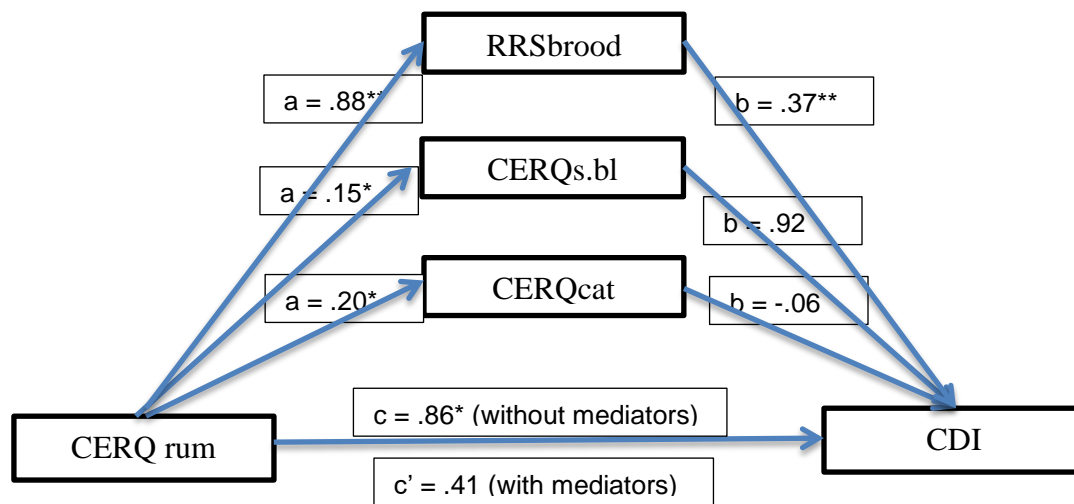


Fig 2. Mediating effect of brooding, self-blame and catastrophizing (at time 2) on the association between rumination at time 1 and depressive symptomatology at time 2. $*p < .05$, $**p < .01$, $***p < .001$. Covariates: CDI at time 1 and psychological well-being at time 1.

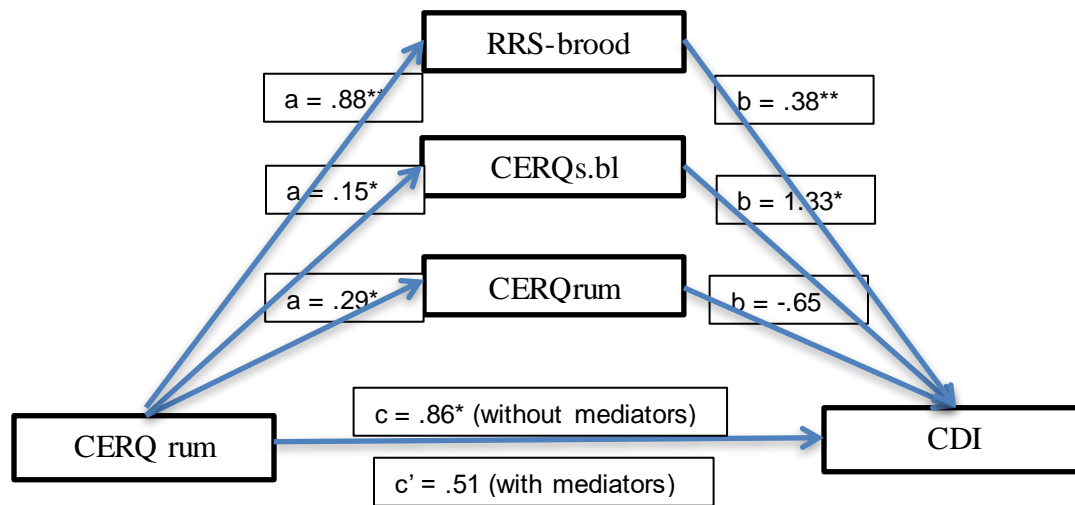


Fig 3. Mediating effect of brooding, self-blame and rumination (at time 2) on the association between rumination at time 1 and depressive symptomatology at time 2. * $p < .05$, ** $p < .01$, *** $p < .001$. Covariates: CDI at time 1 and psychological well-being at time 1.

Table 1

Correlations between independent variables (Time 1)

Measure	M(SD)	1	2	3
1. CDI t1	11.37 (5.57)	1		
2. CERQ Rum. t1	2.94 (.78)	.050	1	
3. MHC:psyWB t1	2.99 (1.02)	-.35	.13	1

Correlations in bold significant at $p < 0.01$. CDI = Children's Depressive Inventory; CERQ Rum. = Cognitive Emotion Regulation Questionnaire – Rumination factor; MHC:psyWB = Mental Health Continuum-short form – psychological well-being factor; t1 = Measure at time 1

Table 2

Correlations between independent variables (Time 1) and dependent and mediator variables (Time 2)

Measure	CDI t2(as DV)	CERQ s-bl.t2	CERQcat.t2	CERQ rum.t2	RRSbrood.t2
CDI t1	.59	.20	.13	.03	.22
CERQ Rum. t1	.14	.21	.21	.30	.23
Psy. WB t1	-.32	.10	.10	-.21	.01

Correlations in bold significant at $p < 0.01$. CDI = Children's Depressive Inventory; CERQ Rum. = Cognitive Emotion Regulation Scale – Rumination; CERQ s-bl = Cognitive Emotion Regulation Questionnaire – Self-Blame; Cognitive Emotion Regulation Questionnaire – catastrophizing; MHC.psyWB = Mental Health Continuum-short form – psychological well-being factor; RRSbrood = Rumination Responses Questionnaire – brooding; t1 = Measure at time 1; t2 = Measure at time 2.

Table 3

Correlations between mediator variables (Time 2) and between mediator variables and dependent variable (Time 2)

Measure	M (SD)	1	2	3	4	5
1. CERQ s-bl t2	2.47 (.65)	1				
2. CERQ cat. t2	2.39 (.81)	.60	1			
3. CERQ rum. t2	2.90 (.85)	.63	.56	1		
4. RRSbrood. t2	12.4 (3.18)	.54	.50	.46	1	
5. CDI t2 (as DV)	11.79 (5.10)	.32	.23	.11	.40	1

Correlations in bold significant at $p < 0.05$. CDI = Children's Depressive Inventory; CERQ s-bl = Cognitive Emotion Regulation Questionnaire – Self-Blame; Cognitive Emotion Regulation Questionnaire – catastrophizing; CERQ Rum. = Cognitive Emotion Regulation Scale – Rumination; RRSbrood = Rumination Responses Questionnaire – brooding; t2 = Measure at time 2.