



Research paper

Cognitive-affective depression and somatic symptoms clusters are differentially associated with maternal parenting and coparenting

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ABSTRACT

Background: Both depressive and somatic symptoms are significant predictors of parenting and coparenting problems. However, despite clear evidence of their co-occurrence, no study to date has examined the association between depressive-somatic symptoms clusters and parenting and coparenting. The current research sought to identify and cross-validate clusters of cognitive-affective depressive symptoms and nonspecific somatic symptoms, as well as to test whether clusters would differ on parenting and coparenting problems across three independent samples of mothers.

Method: Participants in Studies 1 and 3 consisted of 409 and 652 community mothers, respectively. Participants in Study 2 consisted of 162 mothers exposed to intimate partner violence. All participants prospectively completed self-report measures of depressive and nonspecific somatic symptoms and parenting (Studies 1 and 2) or coparenting (Study 3).

Results: Across studies, three depression-somatic symptoms clusters were identified: no symptoms, high depression and low nonspecific somatic symptoms, and high depression and nonspecific somatic symptoms. The high depression-somatic symptoms cluster was associated with the highest levels of child physical maltreatment risk (Study 1) and overt-conflict coparenting (Study 3). No differences in perceived maternal competence (Study 2) and cooperative and undermining coparenting (Study 3) were found between the high depression and low somatic symptoms cluster and the high depression-somatic symptoms cluster.

Conclusions: The results provide novel evidence for the strong associations between clusters of depression and nonspecific somatic symptoms and specific parenting and coparenting problems. Cluster stability across three independent samples suggest that they may be generalizable. The results inform preventive approaches and evidence-based psychotherapeutic treatments.

1. Introduction

Depression involves the presence of cognitive, affective, and physical symptoms, including fatigue, appetite and weight changes, and sleep disturbance (American Psychiatric Association, 2013; Beck and Bredemeier, 2016). Individuals with depression may also present other somatic complaints beyond those established as diagnostic criteria for depressive disorders. These nonspecific somatic symptoms include, for example, dizziness, nausea, and pain such as headaches, stomach pain, chest pain, and poorly localized pain (Harshaw, 2015; Novick et al., 2013). Prior research has showed the clinical utility of examining nonspecific somatic symptoms in the assessment of depressive disorders. In particular, these symptoms are frequently the first and/or the main symptoms of depression presented in primary care settings (Kirmayer et al., 1993; Simon et al., 1999; Tylee and Gandhi,

2005), and they may also be more predictive of cognitive-affective depressive symptoms than specific somatic symptoms of depression (Novick et al., 2013).

This substantial overlap between cognitive-affective depressive symptoms and nonspecific somatic symptoms is also documented in community and clinical samples of mothers (Apter et al., 2013; Brown and Lumley, 2000; Eisenach et al., 2008; Giallo et al., 2016; Webb et al., 2008; Williamson et al., 2014). For example, a community study with pregnant women revealed that depressed women exhibited a higher accumulation of different nonspecific somatic symptoms than women with lower cognitive-affective depressive symptoms (Apter et al., 2013). In addition, a cross-sectional study with American mothers showed that mothers with more depressive symptoms reported higher levels of severity in headaches, nausea, and backaches than those with lower depressive symptoms during the first year postpartum (Webb

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et al., 2008).

Despite empirical evidence of the higher prevalence of physical health problems and somatic complaints in depressed mothers (Apter et al., 2013; Brown and Lumley, 2000; Giallo et al., 2016), no study to date has been specifically designed to explore how the co-occurrence of cognitive-affective depressive symptoms and nonspecific somatic symptoms may be associated with parenting and coparenting problems. The main aim of this research was to identify typologies of cognitive-affective symptoms of depression and nonspecific somatic symptoms in mothers from a community and in mothers exposed to intimate partner violence (IPV). Our second aim was to test whether those typologies would be related differentially to parenting and coparenting problems.

1.1. Comorbidity of cognitive-affective depressive and somatic symptoms

Psychological, psychiatric, and neurobiological theoretical hypotheses have been proposed to explain the comorbidity of cognitive-affective depressive symptoms and somatic symptoms. Psychological hypotheses suggest that depression reduces the pain threshold, amplifying somatic hypervigilance and, subsequently, increasing somatic symptoms (Geisser et al., 2003). In addition, somatic symptoms are suggested as an emotional strategy to communicate distress in response to psychosocial stress, alleviating perceived psychological pain by repressing affective symptoms of depression (Wearden et al., 2005). Alternatively, the social psychiatric hypothesis proposes that the overlap of cognitive-affective depressive and somatic symptoms is grounded in psychosocial factors related to gender roles, particularly in perceived gender inequality. On the other hand, pure depression (without somatic symptoms) is strongly associated with genetic factors (Silverstein et al., 2013; Silverstein and Levin, 2014). The emphasis on the link between perceived gender inequality and somatic depression is rooted in empirical data showing that, compared to men, women report higher somatic depression but not pure depression symptoms. Furthermore, women report lower effectiveness of biologically-based treatments in somatic depression when compared with pure depression (Silverstein and Levin, 2014).

In contrast with this hypothesis, neurobiological models propose that the overlap between depression and somatic symptoms is largely explained by biological underpinnings. This is because somatic depression has been associated with a specific genetically homogenous profile (Kendler et al., 2013) and distinct patterns of immuno-inflammatory and autonomic regulation (Penninx et al., 2013).

Beyond these theoretical hypotheses, a large body of literature has also documented a clinical distinction between depression with somatic symptoms and depression involving non-somatic symptoms (Bekhuis et al., 2015; Kendler et al., 2013). Significant interindividual variability has been found between these clinical phenotypes in psychosocial outcomes and depression prognosis (Silverstein and Levin, 2014). First, the co-occurrence of cognitive-affective depressive and somatic symptoms was associated with higher odds of physical health problems and multiple stressful relationships (Bohman et al., 2010; Hwang et al., 2015). In addition, several studies have shown that the number, diversity, and severity of somatic symptoms significantly predict depression severity (Bahk et al., 2011; Bekhuis et al., 2016; Novick et al., 2013). In particular, the presence of somatic symptoms is associated with worse prognosis in depression, including higher chronicity of depression (Stegenga et al., 2012), lower remission rates (Novick et al., 2013), higher odds of new depressive episodes in the future (Terre et al., 2003), and a lower response to treatment (Huijbregts et al., 2013).

Previous work has also identified clusters of cognitive-affective depressive and nonspecific somatic symptoms (Baldassin et al., 2013; Dodd et al., 2011; Illi et al., 2012; Maes et al., 2012; Novick et al., 2013). In particular, Illi et al. (2012) found three distinct clusters: a low depression and somatic pain symptoms cluster, a high depression and low nonspecific somatic symptoms cluster, and a high depression and

nonspecific somatic symptoms cluster. These occurred among 83%, 4.7%, and 12.3% of the sample, respectively. In that study, clusters were highly reliable to differentiate the severity of the sickness behavior in oncology patients. However, despite their contribution to investigating the interplay between cognitive-affective depressive and nonspecific somatic symptoms, that study, and all previous studies that filtered depression-somatic symptoms clusters, primarily tested their association with depression severity, physical health, and quality of life outcomes in clinical samples (Hawkins et al., 2014; Novick et al., 2013). Thus, it remains unclear whether these findings can generalize associations between maternal depressive-somatic symptoms and parenting and coparenting problems.

1.2. Depressive-somatic symptoms, parenting, and coparenting

Depressive and somatic symptoms in women have been strongly associated with problems in parenting and coparenting (Lovejoy et al., 2000; Solmeyer and Feinberg, 2011; Wilson and Fales, 2015). Cognitive-affective depressive symptoms have predicted lower maternal competence and ineffective discipline practices, such as hostile, harsh, inconsistent, and abusive parenting behavior (Childs et al., 2014; Dix and Meunier, 2009; Leung and Slep, 2006). In addition, cognitive-affective depressive symptoms were related to unsupportive, undermining, and conflictual coparenting behaviors in cross-sectional and longitudinal studies (Lamela et al., 2016; Solmeyer and Feinberg, 2011; Tissot et al., 2016). Less studied is the relationship between maternal somatic symptoms and parenting and coparenting. However, some research suggests that both maternal specific and nonspecific somatic symptoms are associated with parenting problems (Evans et al., 2006; Giallo et al., 2011; Hiraoka et al., 2014). An American study of mothers with chronic pain, for example, demonstrated that severity of pain was associated with more overreactive discipline (Evans et al., 2006). Another study with a community sample of mothers and fathers revealed that higher pain sensitivity and pain intolerance were associated with higher risk of child physical maltreatment (Hiraoka et al., 2014).

Surprisingly, both cognitive-affective depressive and specific and nonspecific somatic symptoms have been traditionally tested separately as predictors of parenting and coparenting problems, despite clear evidence of an adverse cumulative effect of their co-occurrence on health and psychosocial outcomes. An empirical exception was conducted with an Australian community sample of mothers of young children. It found that the co-occurrence of cognitive-affective depressive symptoms and fatigue (a specific somatic symptom of depression) was associated with higher hostile parenting and lower perceived maternal competence (Wade et al., 2012).

However, since multiple cognitive-affective depressive and nonspecific somatic symptoms may be related to parenting and coparenting outcomes (Dix and Meunier, 2009; Evans et al., 2006), a further examination of the association between cognitive-affective depressive and nonspecific somatic symptoms clusters and parenting and coparenting problems may have potential significance and clinical utility. Insight into the prevalence and distribution in which depression-somatic symptoms clusters occur in mothers may also be valuable for prevention and the detection of subgroups at higher risk of specific parenting and coparenting problems.

The potential value in understanding these associations may also find support in previous theoretical frameworks. According to action-control conceptualizations of parental self-regulation, parenting and coparenting behaviors are thought to be determined by five cognitive-affective regulatory processes: goal processing, input processing, appraisal processing, emotional activation, and response processing (Dix and Meunier, 2009).

Under this framework, the presence of cognitive-affective depressive symptoms is postulated to negatively affect parenting and coparenting through impairing those regulatory processes. These impair-

ments subsequently activate motivational-cognitive biases and negative emotional arousal and dysregulation that ultimately disrupt parenting and coparenting (Crick and Dodge, 1994; Haskett et al., 2003). In particular, higher levels of child physical maltreatment, permissive parenting, and coparenting overt-conflict exhibited by depressive mothers might be theorized as behavioral consequences of impairments in three specific regulatory processes: parents' goal processing, emotional activation, and response processing (Dix et al., 2014; Dix and Meunier, 2009). Impairments in these processes reduce child-oriented goals, increase low-effort and short-term goals in family interactions, increase negative emotion, and lower effective responses to family stimuli (Callender et al., 2012; Lovejoy et al., 2000). As a result, these specific cognitive biases and emotional difficulties are likely to increase mothers' tendency of using more ineffective and disruptive behavioral strategies in their relationships with other family members, such as child physical maltreatment, permissive parenting, and overt-conflict in a coparenting relationship (Favez et al., 2016; Haskett et al., 2003).

Furthermore, cognitive-affective depression symptoms negatively affect parents' appraisal processes. This jeopardizes how mothers adaptively appraise the goals and content of their interactions, the psychological resources they need to respond to them, and positive attributions to family members' intentions and behaviors (Dix and Meunier, 2009). Disturbances in this process are empirically associated with lower perceived maternal competence (Michl et al., 2015). The dysfunctional appraisal processing of depressed mothers might also reduce cooperative coparenting and undermining coparenting by promoting negative evaluations of the other parent and negative attributions to other parent behaviors, as suggested by previous empirical work (Jia and Schoppe-Sullivan, 2011).

Despite being informative, initial formulations of this framework did not account for the overlapping of cognitive-affective depressive and specific and/or nonspecific somatic symptoms in those regulatory processes. However, a large body of research has shown strong associations between nonspecific somatic symptoms and negative emotional arousal and emotional dysregulation in women, including anger dysregulation (Estlander et al., 2008), higher irritability (Tikotzky, 2016), affective volatility (Dinges et al., 1997), lower threshold to frustration (Massey et al., 2009), and fear dysregulation (Seng et al., 2006). Drawing from these previous contributions, a plausible thesis is that the overlap of cognitive-affective depressive and nonspecific somatic symptoms might have a differential impact on the cognitive-affective regulatory processes that determine parenting and coparenting. The presence of these somatic symptoms might particularly impair emotional activation processes related to parenting and coparenting in mothers. Child physical maltreatment risk, permissive parenting, and overt-conflict coparenting are primarily regulated by goal processing, emotion activation, and response processing mechanisms; the presence of somatic symptoms would therefore increase the predisposition for these specific parenting and coparenting problems beyond the effect of cognitive-affective depressive symptoms.

In contrast, problems in perceived maternal competence and cooperative and undermining coparenting are thought to be primarily determined by impairments in mothers' appraisal processing, triggered by the cognitive and affective symptoms of depression. Thus, the overlap of nonspecific somatic-depressive symptoms would not account for higher levels of these parenting and coparenting problems beyond those depressive symptoms. In summary, the co-occurrence of cognitive-affective depressive and nonspecific somatic symptoms might have a detrimental cumulative effect in parenting and coparenting behaviors that are primarily determined by how mothers generate and evaluate goals and how they modulate emotion and select responses in interactions with other family members.

1.3. Aims of the current study

The potential differential effects of the co-occurrence of cognitive-

affective depressive and nonspecific somatic symptoms in specific parenting and coparenting problems have remained unexplored at an empirical level. In addition, the only research that explores the association between the depression-somatic symptoms overlap and parenting problems only tested a single specific somatic symptom (Wade et al., 2012). To address this gap, our research had two aims.

Firstly, we sought to identify cognitive-affective depressive symptoms and nonspecific somatic symptoms profiles through applying cluster analysis procedures. As a person-centered procedure, cluster analyses describe similarities and differences among individuals instead of relations among variables (Rupp, 2013). This approach focuses on classifying individuals into homogeneous subgroups characterized by a similar pattern of relationships among variables. By assuming population heterogeneity in the associations between variables, multiple configurations of cognitive-affective depression symptoms and nonspecific somatic symptoms could be potentially extracted from the data (Masyn, 2013). As a result, cluster analytic procedures are potentially more sensitive for the detection of less prevalent groups, when compared with the use of cutoff scores derived from non-gold standard self-reported measures (García et al., 2015). The utility of person-centered procedures to define and validate depression phenotypes in mothers was already established (Putnam et al., 2015).

Based on previous research (Dodd et al., 2011; Illi et al., 2012), we hypothesized that three distinct clusters would be found: a low depressive and nonspecific somatic symptoms cluster, a high depressive and low nonspecific somatic symptoms cluster, and a high depressive and nonspecific somatic symptoms cluster. The three studies reported here directly examine this hypothesis and also tested differences on specific parenting and coparenting outcomes among the identified clusters. In Study 1, we tested whether clusters would differ on child physical maltreatment risk (CPM) in a community sample. In Study 2, we investigated whether the hypothesized cluster solution would be replicated in a high-risk sample of mothers who were exposed to IPV. We then examined whether clusters would differ in permissive parenting and perceived maternal competence. Finally, in Study 3, we investigated whether these clusters would differ on coparenting in a community sample. As stated above, in the three studies, we particularly focused on nonspecific somatic symptoms that are not drawn on as physical symptoms of depressive disorders, such as fatigue and sleep and appetite disturbances.

2. Study 1: clusters validation and risk of CPM

Study 1 aimed to identify and validate cognitive-affective depressive-nonspecific somatic symptoms clusters through cluster analysis procedures. We then examined whether those clusters would differ in mothers' risk of CPM. CPM risk is defined as parental predisposition to use coercive and harsh parenting strategies to control child behavior (Lamela and Figueiredo, 2015). The predictive association between mothers' depression symptoms and CPM has been consistently reported in the literature (Lovejoy et al., 2000).

In addition, previous work also demonstrates that somatic disturbance in depressed individuals is associated with higher neuroendocrine hyperreactivity (O'Connor et al., 2013) that, in turn, is related to higher coerciveness in parenting practices (Sturge-Apple et al., 2011). Consistent with these findings, it is possible that nonspecific somatic symptoms may be related to an increased overreactivity in parenting practices, beyond the effect of depression symptoms. Thus, we expected that mothers with high cognitive-affective depressive and nonspecific somatic symptoms would exhibit the highest risk of CPM.

2.1. Method

2.1.1. Participants

The participants were 409 mothers from a Portuguese community-based study. Their mean age was 38.31 years (SD = 5.99). Demographic

Table 1
Description of samples, n (%) for categorical variables and M (SD) for continuous variables.

	Study 1 (N = 409)		Study 2 (N = 162)		Study 3 (N = 652)	
	n	%	N	%	n	%
Marital Status						
Married/	365	89.2	97	59.9	477	73.2
Cohabiting						
Single/Divorced/	44	10.8	65	40.1	175	26.8
other						
Employment status						
Employed full/	377	92.2	39	24.1	558	85.6
part-time						
Unemployed/	32	7.8	123	75.9	94	14.4
other						
Education						
< 12 years	314	76.8	145	89.5	198	30.4
College degrees	95	23.2	17	10.5	454	69.6
Children gender						
(boys)	198	48.4	87	53.7	306	46.9
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	37.0	6.15	36.4	7.6	39.5	6.31
Number of	1.93	.96	2.57	1.28	1.76	.82
children						
Children age	7.50	1.12	7.22	1.94	6.53	5.05
BSI depression	3.13	3.89	11.29	5.78		
BSI somatization	3.39	3.89	7.72	5.91	2.66	3.71
PHQ9 depression					6.21	5.08
CAPI	22.68	12.67				
PSCS			3.29	1.26		
PPS			61.69	10.78		
Cooperative					4.41	1.47
coparenting						
Childcare labor					4.23	1.45
division						
Overt-conflict					.81	.92
coparenting						
Undermining					.97	1.25
coparenting						

data, means, and standard deviations for the study's measures of the total sample are summarized in Table 1.

2.1.2. Procedure and materials

Data from the National Study of Child Abuse and Neglect in Portugal was used. Participants were mothers of school-age children attending one of the five public elementary schools of Northern Portugal randomly selected from the national list of public schools. After receiving the ethical approval of all relevant institutional review boards (DREN, Direção Regional da Educação do Norte), mothers were contacted by the research team and teachers and were informed about research aims and ethical procedures. The assessment protocols and informed consent form were provided in sealed envelopes to the children who in turn handed them to their mothers. Mothers who agreed to participate in the survey completed and returned the assessment protocols in sealed envelopes to the research team via their children (response rate = 76%).

As a multivariate statistical technique, observations with missing data are excluded in the cluster analysis. Despite the availability of imputation methods to handle missing values, these imputation procedures might be particularly problematic and less reliable in cluster analysis (Cross, 2013; Wagstaff, 2004). Therefore, only participants with complete data were included in the current research, as suggested previously (Cross, 2013). Thirty-two participants were excluded. No statistical differences between retained and excluded participants were found in main sociodemographic variables, such as age, $t(2, 439)$

$= -.91, p = .36$, marital status, $\chi^2(1) = .84, p = .93$, education, $\chi^2(1) = 2.65, p = .26$, and number of children, $t(2, 439) = -1.97, p = .08$.

Cognitive-affective depressive symptoms were assessed by the depression scale of the Brief Symptom Inventory (BSI; Derogatis and Melisaratos, 1983). This 6-item scale measures individuals' depressed mood, sadness, loss of interest in life activities, unworthiness/worthlessness, hopelessness, loneliness, and vulnerability to criticism. Items were answered using a 5-point Likert-scale (from 0 'not at all' to 4 'extremely'), based on respondents' levels of distress over the previous two weeks. Higher scores reflect higher depression symptoms. The BSI depression scale reliability for screening depression symptoms was established previously (Callender et al., 2012). The Portuguese version of the depression scale of the BSI revealed good internal consistency (Canavarro, 1999). In the current sample, Cronbach's alpha was .83.

Nonspecific somatic symptoms were assessed by the BSI somatization scale (Derogatis and Melisaratos, 1983). This 7-item scale measures the severity of psychological distress arising from a perception of bodily dysfunction, focusing on four types of symptoms (cardiopulmonary, gastrointestinal, musculoskeletal, and general symptoms). This scale inquiry into seven symptoms: faintness or dizziness, pains in the heart or chest, nausea or upset stomach, trouble getting your breath, numbness or tingling in parts of the body, feeling weak in parts of the body, hot or cold spells. The scale's items are not restricted to medically unexplained symptoms but instead, they examine somatic symptoms in general. Mothers were asked to answer based on their level of distress over the previous two weeks, using a 5-point Likert-scale (from 0 'not at all' to 4 'extremely'). Higher scores reflect more somatic symptoms. Scale's high reliability to screening somatic symptoms was demonstrated elsewhere (Zijlema et al., 2013). The Portuguese version of the somatization scale of the BSI revealed good internal consistency (Canavarro, 1999). In the current study, Cronbach's alpha was .80.

The risk of CPM was measured using the abuse scale of the Child Abuse Potential Inventory (CAPI; Milner, 1986). This scale assesses parents' attitudes and practices regarding physical forms of discipline and abuse. The CAPI abuse scale examines six evidence-based risk indicators of CPM, including parental distress (e.g., 'I often feel very frustrated'), attitudinal rigidity in response to child behavior (e.g., 'Children should always be neat'), personal unhappiness, problems with the child and the self (e.g., 'I have a child who is slow'), problems with the family (e.g., 'My family fights a lot'), and general interpersonal difficulties in social relationships (e.g., 'Other people have made my life hard'). Comprising a forced-choice format (0 'no' or 1 'yes'), the 74-items of the current Portuguese version of the CAPI showed very good psychometric properties (Gomes, 2010). Higher scores reflect higher risk of CPM (range from 0 to 74). The validity of the CAPI to predict subsequent confirmed reports of abuse was established (Milner, 1994). In the current study, the CAPI showed high internal consistency (Cronbach's alpha = .91).

2.2. Results

2.2.1. Clustering cognitive-affective depressive and nonspecific somatic symptoms

The BSI depression and somatization scales were used to perform the cluster analysis. A Ward's agglomerative hierarchical cluster analysis (Standardized Euclidian Distance method used) was initially conducted in order to establish the number of clusters. The visual inspection of the hierarchical cluster analysis outputs (e.g., dendrogram analysis, Euclidian distance plot, and agglomeration scheme) suggested the adoption of three clusters as an optimal solution. To confirm this solution, cases were then clustered performing a K-means analysis with squared Euclidean distance as index of similarity (z-scores were employed). The comparison of the results of the K-means analysis with those obtained in the hierarchical cluster analysis revealed a substantial agreement ($\kappa = .84$).

Table 2
Symptoms cluster differences on clustering variables.

	Variable	NoS		DS		DSS		Cluster differences tests		
		M	SD	M	SD	M	SD	gl	F ^a	Cluster contrasts ^b
Study 1	BSI depression	1.00	1.24	8.01	3.24	8.65	4.19	2, 407	458.72	NoS < DS, DSS
	BSI somatization	1.29	1.59	3.27	2.05	11.15	2.45	2, 407	353.85	NoS < DS < DSS
Study 2	BSI depression	5.01	2.94	15.1	2.82	13.8	4.80	2, 160	128.8	NoS < DSS < DS
	BSI somatization	3.49	3.35	5.31	2.81	15.1	3.26	2, 160	205.4	NoS < DS < DSS
Study 3	PHQ9 depression	3.44	2.05	10.3	3.24	19.3	4.20	2, 650	931.02	NoS < DS < DSS
	BSI somatization	.69	.84	.95	.93	11.4	1.23	2, 650	450.01	NoS < DS < DSS

Note. ^a All ANOVA analyses were significant at $p < .001$. ^b Significant cluster differences at $p < .05$ using Tukey-Kramer test. NoS = No symptoms cluster. DS = Depression symptoms only cluster. DSS = Depression and somatic symptoms cluster. *** $p < .001$.

Stability of the three-cluster solution was also tested using a cross-validation procedure (Mandara, 2003). After randomly splitting the overall sample into two subsamples, a K-means analysis was conducted on both subsamples, and the agreement between the two solutions was computed. This procedure was replicated ten times (Hoeve et al., 2008). These analyses revealed a substantial agreement for the three-cluster solution ($\kappa = .78$, range: .61–.93). Stability of the cluster solution was additionally examined by performing a MANOVA on the depression and somatization scales using the clusters as a fixed factor. The MANOVA showed that clustering variables significantly differed between the clusters, Wilks's λ , $F(2, 407) = 254.13$, $p < .001$, $\eta^2 = .56$. Follow-up Bonferroni-corrected ANOVAs revealed that all clustering variables were significantly different across the three clusters (Table 2).

2.2.2. Defining profiles

The means and standard deviations for cognitive-affective depressive and nonspecific somatic symptoms variables for each cluster, as well as the cluster-by-cluster differences, are presented in Table 2. The three clusters were labeled based on the most salient dimensions. Cluster 1 (80.2% of the sample) reported the lowest scores on both depression and somatization scales. Therefore, this cluster was labeled as No Symptom cluster (NoS). Cluster 2 (14.4% of the sample) reported high average scores on the depression scale and low average scores on the somatization scale. Since the most salient dimension in this cluster was cognitive-affective depressive symptoms, it was labeled the Depression Symptoms only cluster (DS). Finally, Cluster 3 (5.4% of the sample) revealed high average scores on both depression and somatization scales. Based on these scores, this cluster was labeled as Depression and Somatic Symptoms cluster (DSS).

Tukey–Kramer post-hoc tests revealed that, when compared with the other clusters, the NoS cluster exhibited the lowest scores on both depression and somatization scales. The DSS cluster reported the highest levels of nonspecific somatic symptoms. The DS cluster showed higher levels of nonspecific somatic symptoms than NoS but lower levels than the DSS cluster. No significant differences were found between DS and DSS on cognitive-affective depressive symptoms assessed by the BSI depression scale.

2.2.3. Cluster differences in risk of CPM

ANOVA revealed significant cluster differences in risk of CPM, $F(2, 407) = 106.9$, $p < .001$. Tukey post-hoc comparisons showed that, when contrasted with the other two clusters, the NoS cluster reported the lowest risk of CPM. The DS cluster also significantly scored higher on the risk of CPM than NoS. The DS and DSS clusters also differed, in which the DSS cluster exhibited the highest scores of CPM (Fig. 1).

3. Study 2: symptom clusters and parenting in high-risk mothers

Study 2 sought to conceptually replicate and extend the findings from Study 1 in two ways. First, we tested the cluster solution with

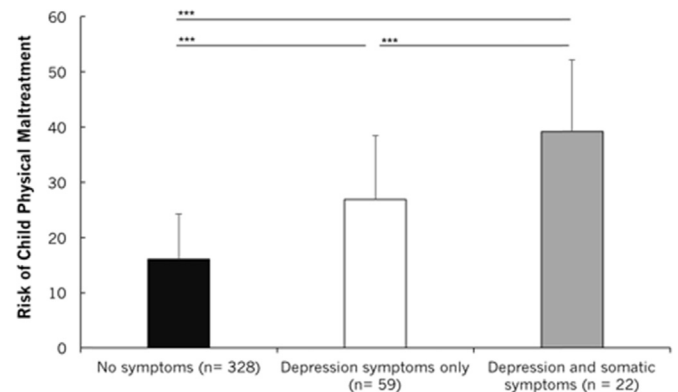


Fig. 1. Differences among clusters on child physical maltreatment risk. Bars show mean values and standard error of mean. Asterisks above error bars indicates statistically significant differences between the groups (***, $p < .001$).

mothers exposed to IPV. Women exposed to IPV report higher prevalence of both depression and somatic symptoms when compared with women with no IPV history (Eberhard-Gran et al., 2007). In addition, a greater comorbidity of these symptoms in battered women was also described previously. Therefore, at-risk participants could provide additional insight into clusters stability and their validity to detect problems in parenting across diverse samples. Second, Study 1 only tested the differences between clusters on a specific parenting behavior. A further question is to understand whether similar differences between clusters would be found in other parenting variables that are linked to different maternal self-regulatory processes. To address this question, Study 2 tested whether clusters would differ on permissive parenting and maternal perceived parenting competence.

Permissive parenting is defined by inconsistent parenting practices expressed in difficulties in controlling child misbehavior, low enforcement of rules, and low persistence in discipline strategies (Johnson and Kelley, 2011). Previous research has documented significant associations between depression symptoms and permissive parenting in IPV samples (e.g., Boeckel et al., 2014). Surprisingly, no studies to date examine the association between somatic symptoms and permissive parenting in battered women, and studies with community samples report contradictory findings. However, as individuals with several somatic symptoms tend to exhibit higher variation in daily pain intensity and more functional impairments (Bromley Milton et al., 2013), it is possible to expect these variations in pain intensity may be detrimental to parenting consistency. Therefore, we hypothesized that the DSS cluster would report the highest levels of permissive parenting, while the NoS cluster would exhibit the lowest levels of permissive discipline.

We next tested cluster differences on perceived parenting competence (PPC). PPC is defined as the parental perception of their own knowledge, attitudes, and skills to parent successfully (Johnston and

Mash, 1989). Previous work showed that maternal depression symptoms have longitudinally predicted lower PPC (Dix and Meunier, 2009). As PPC is linked with maternal motivational, cognitive, and affective self-regulatory processes, we expected that lower PPC would be significantly related only to high depression symptoms. In other words, we hypothesized that high somatic symptoms would not be associated with lower PPC, beyond depression symptoms.

Substantial empirical evidence supports this hypothesis. First, lower PPC was linked with depressive cognitive biases, including rumination and negative mother-centered attributions of child behavior (Dix and Meunier, 2009; Leung and Slep, 2006). Second, the effect of cognitive-affective depression symptoms on lower PPC may be partially explained by the negative interference of these depression symptoms on specific cognitive processes, such as executive function and memory (Pio de Almeida et al., 2012). Third, affective depression symptoms have been associated with mothers' heightened sensitivity to aversive inputs from children and difficulty in regulating their negative emotions which, in turn, have been linked with lower maternal competence (Dix et al., 2014). Finally, no empirical testing of the association between somatic symptoms and maternal PPC was found in literature, suggesting that these two constructs may be conceptually uncorrelated. Consistent with this reasoning, we predicted that clusters with high cognitive-affective depressive symptoms would show the lowest levels of PCC, independent of the level of nonspecific somatic symptoms.

3.1. Method

3.1.1. Participants

Participants were 162 mothers who were IPV victims. The mean age of participants was 33.8 years ($SD = 11.76$). Demographic data, means, and standard deviations for the study's measures of the total sample are described in Table 1.

3.1.2. Procedures and materials

Eligible participants should have experienced police or child protection services-reported male-perpetrated IPV, should be over 18 years old, and should have at least one child aged between 4 and 10 years. IPV was measured by two sources: at least one documented IPV-related police or child protection services incident and by mothers' scores on the Conflict Tactics Scale (CTS2; Straus et al., 1996). The CTS2 is a self-report to assess violent behaviors that occurred in the past 12 months in the context of an intimate relationship. Both original and Portuguese versions of the scale have been previously demonstrated to have good test-retest reliability, high alpha coefficients of internal consistency, and good construct validity (Paiva and Figueiredo, 2006; Straus et al., 1996). All participants (100%) reported being a victim of at least one physical violent behavior from their current intimate partner in the CTS2. Participants were recruited in Child Protective Services and shelter residences from north to south of Portugal. Professionals of these institutions firstly approached potential participants, and information about research aims and ethical procedures were provided.

In total, 352 mothers were contacted by the professionals, and 162 mothers agreed to participate. Mothers who consented to participate completed the assessment protocol in Child Protective Services or shelters facilities. In the case of having more than one child between 4 and 10 years old, participants were asked to complete the parenting measures regarding their youngest child. A member of the research team was available to clarify any difficulty in the completion of the assessment protocol. As a consequence of this recruitment procedure, no missing values were found in the data.

Cognitive-affective depressive and nonspecific somatic symptoms were assessed using the BSI depression and somatization subscales described in Study 1. Internal consistency of the BSI depression and somatization scales in this sample was .82 and .80 respectively.

Perceived parenting competence was measured with the Parenting

Sense of Competence Scale (PSCS; Johnston and Mash, 1989). This 17-item scale defines perceived Parenting competence as a sense of satisfaction and efficacy with the parenting role. Each item is rated on a 6-point Likert scale (from 1 'strongly agree' to 6 'strongly disagree'). Lower scores reflect higher perceived parenting competence. The Portuguese version of this scale showed good psychometric properties (Seabra-Santos et al., 2015). Internal consistency coefficients for the PSCS scale in this sample was good ($\alpha = .79$).

Permissive parenting was assessed with the laxness subscale of the Parenting Scale (PPS; Arnold et al., 1993). This subscale measures parents' inconsistent parenting practices defined by difficulties in control child misbehavior, low enforcement of rules and authority and low persistence in discipline strategies. The subscale consists of eleven items assessing the extent to which parents engaged in specific permissive discipline responses that are rated on a 7-point scale (from 0 'never' to 6 'always'). Higher scores reflect higher permissive parenting. Previous work showed that Parenting Scale scores were significantly associated with observational measures of dysfunctional discipline (Arnold et al., 1993). Good psychometric properties were found in the PPS Portuguese version (Cruz et al., 2011). For the current sample, Cronbach's alpha was .77.

3.2. Results

3.2.1. Clustering cognitive-affective depressive and nonspecific somatic symptoms

K-means clustering analysis with squared Euclidean distance as index of similarity (z -scores were employed) with an *a priori* defined $k=3$ was used to parcellate depression and somatic symptoms into three distinct symptom clusters based on the results of Study 1. The cluster solution found in Study 1 was confirmed in this study. The stability of the cluster solution in Study 2 was only tested by conducting a MANOVA on the depression and somatic symptoms using the clusters as a fixed factor, in contrast with Study 1. The MANOVA showed that clustering variables significantly differed between the clusters, Wilks's λ , $F(2, 160) = 154.4$, $p < .001$. Follow-up Bonferroni-corrected ANOVAs showed that all clustering variables significantly differed across the three clusters (Table 2). As a result of the sample size, the split-sample cross-validation procedure used in Study 1 to test the three-cluster solution stability was not conducted in this study, as recommended previously (Masyn, 2013; Tibshirani and Walther, 2005). Splitting the sample would compromise the detection of less prevalent clusters and, subsequently, the validity of the cluster-solution stability analysis within each of the subsamples for inadequate subsamples size (Masyn, 2013).

The means and standard deviations for cognitive-affective depressive and nonspecific somatic symptoms variables for each cluster and the cluster-by-cluster differences are displayed in Table 2. Results of Tukey–Kramer post-hoc tests on the differences between clusters on depression and somatization scales were comparable to those obtained in Study 1 (Table 2). Fifty-five mothers were assigned to the NoS cluster (34% of the sample), 57 participants to the DS cluster, and 50 to the DSS cluster (31% of the sample).

3.2.2. Cluster differences in permissive parenting and PCC

One-way analyses of variance analyses followed by Tukey–Kramer post-hoc tests were conducted to examine differences between clusters on permissive parenting and perceived parenting competence. For the permissive parenting scores, the ANOVA revealed significant cluster differences, $F(2, 161) = 4.73$, $p < .001$. As indicated in Fig. 2A, the DS cluster had higher permissive parenting scores than both NoS and DSS clusters. No cluster differences were unexpectedly found between the NoS and DSS cluster. Consistent with our hypothesis, significant differences between the clusters were found in perceived parenting competence, $F(2, 161) = 12.44$, $p < .001$. The results in Fig. 2B indicate that mothers in the NoS cluster reported significantly higher

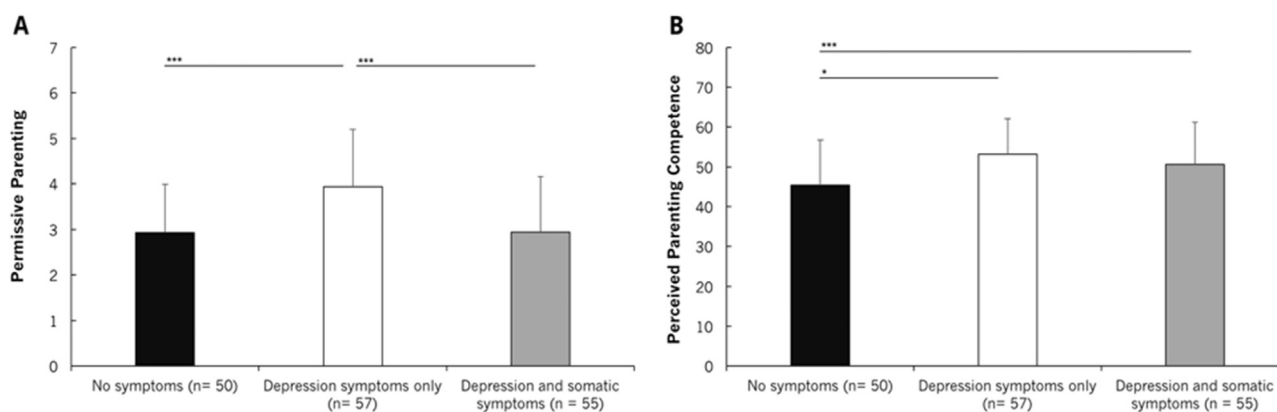


Fig. 2. Differences among clusters on permissive parenting (2A) and PPC (2B). Bars show mean values and standard error of mean. Asterisks above error bars indicate statistically significant differences between the groups (* $p < .05$; *** $p < .001$).

levels of perceived maternal competence than did the mothers of DS and DSS clusters. DS and DSS clusters scores were not significantly different from each other.

4. Study 3: symptom clusters and coparenting

In Studies 1 and 2, we showed that clusters were differentially associated with different parenting variables. In Study 3, we sought to extend these findings by testing the relationship between cognitive-affective depressive-nonspecific somatic clusters and coparenting dimensions. Coparenting broadly refers to the ways that parents relate to each other in the role of parent, how they support each other in childrearing, and how they solve childrearing-related conflicts (Feinberg, 2003; Lamela et al., 2016). Empirical work suggests coparenting as a pivot family process strongly associated with subsequent family members' mental health and parenting outcomes (Lamela et al., 2016; Teubert and Pinquart, 2010). However, empirical research linking parental psychopathology and coparenting behavior is limited.

To our knowledge, no research has attempted to systematically examine whether the co-occurrence of specific psychopathological symptoms would be differentially associated with different coparenting dimensions. To address this gap, in Study 3, we are particularly interested in investigating the relationship between the co-occurrence of cognitive-affective depressive and nonspecific somatic symptoms and three specific coparenting dimensions: cooperative, undermining, and overt-conflict coparenting.

Consistent with coparenting models (Feinberg, 2003; McHale, 1997), we expected differential associations between clusters and coparenting dimensions. First, we predicted that no differences between the DS cluster and the DSS cluster would be found in cooperative and undermining coparenting. Cooperative coparenting is defined as parents' perception of agreement in childrearing values and practices and perceived support in caregiving (Feinberg, 2003). It is a cognitive-motivational dimension of coparenting behavior that may be negatively affected by cognitive and affective disturbances related to depression symptoms but not by behavioral and functional disturbances related to nonspecific somatic symptoms. Using the same reasoning, we expected undermining coparenting (i.e., cognitive-emotional childrearing-focused conflict strategies such as hostility, criticism, disparagement, blame, and competition (Feinberg, 2003) to not differ between those clusters.

In contrast, we predicted that the DSS cluster would exhibit the greatest levels of overt-conflict coparenting, defined as behavioral overreactive conflict strategies, including anger, irritability, and verbal and physical attacks to manage childrearing disagreements (Feinberg, 2003; Lamela et al., 2016). As nonspecific somatic symptoms were previously associated with higher behavioral overreactivity (Study 1), we expected that nonspecific somatic symptoms would contribute to an

increased overreactivity in conflict, beyond cognitive-affective depressive symptoms. In all three coparenting dimensions, we expected that the NoS cluster would report the highest levels of cooperative coparenting and the lowest levels of undermining and overt-conflict coparenting.

4.1. Method

4.1.1. Participants

The participants were 652 mothers from a Portuguese community-based study. Their mean age was 39.5 years ($SD = 6.31$). Demographic data, means, and standard deviations for the study's measures of the total sample are summarized in Table 1.

4.1.2. Procedure and materials

Data for the current study was derived from an online survey designed to collect information regarding mental health, sexual behavior, and family functioning in Portuguese women. The survey was available on a Portuguese website hosted on a university server from March to July 2015. Participants were recruited via online forums, social media websites, and e-mails to institutional public entities' web accounts. The survey took 20–25 min to complete. To be included in this study, respondents had to be at least 18 years old and be residents of Portugal. No financial compensation was provided. To guarantee data quality, standard methodological and ethical guidelines for internet-based research were followed, such as the application of informed consent procedures, the design of a parsimonious plan for participants' recruitment, and application of procedures for potentially biased samples (Kraut et al., 2004).

Prior to statistical analysis, data cleaning procedures were performed in the dataset as described by Funk and Rogge (2007). Firstly, 31 respondents (3.9%) of the initial 801 respondents were deleted for failing to complete 70% of the entire survey. Finally, another 21 were omitted for leaving more than one item blank on the depression and somatic symptoms measures. No significant demographic differences were found between the excluded and retained participants, including age, $t(2, 768) = -1.33, p = .18$, marital status, $\chi^2(1) = 2.20, p = .53$, education, $\chi^2(1) = 6.35, p = .09$, and number of children, $t(2, 768) = -.91, p = .36$. The final database was comprised of 749 participants, but only participants with children were included ($N = 652$).

Cognitive-affective depressive symptoms were assessed using 6 items of the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001). The PHQ-9 measures the frequency of cognitive, affective, and somatic depressive symptoms experienced in the past two weeks as defined in DSM (American Psychiatric Association, 2013). As we were focused on the cognitive-affective depressive symptoms, we did not include three items of PHQ-9 that measure specific somatic symptoms of depression ("Trouble falling or staying asleep, or sleeping too much",

'Feeling tired or having little energy', and 'Poor appetite or overeating'). Each item is answered on a 4-point Likert scale (from 0 'not at all' to 3 'nearly every day'). The total score was computed by summing the six items. Higher scores reflect higher cognitive-affective depressive symptoms. Internal consistency of the PHQ-9 scale in this sample was .88. Nonspecific somatic symptoms were assessed with the BSI somatization scale described in Study 1. In the current sample, Cronbach's alpha was .81.

Cooperative coparenting was measured with the Portuguese short-form of the Parenting Alliance Measure (PAM; [Abidin and Brunner, 1995](#)). This 6-item scale measures how parents cooperate and communicate in childrearing and how they are compromised in a joint education. Each item is answered using a 5-point Likert scale (from 1 'totally disagree' to 5 'totally agree'). Higher scores suggest higher cooperative coparenting. The Portuguese version shows excellent psychometric properties ([Lamela et al., 2013](#)). In the current sample, internal consistency was very good (Cronbach's $\alpha = .89$).

Undermining and overt-conflict coparenting were measured through two subscales of the Coparenting Relationship Scale (CRS; [Feinberg et al., 2012](#)): undermining coparenting was measured with a 6-item undermining subscale, and overt-conflict coparenting by the 6-item exposure to conflict subscale. Each item is answered on a 7-point scale (from 0 'not true of us' to 6 'very true of us'). The total score of each subscale was calculated by taking the mean of all items. Higher scores on each subscale reflect greater undermining and overt-conflict coparenting. The Portuguese version of the CRS revealed excellent psychometric properties ([Morais, 2015](#)). Internal consistency of the undermining coparenting and the exposure to conflict subscales in this sample were .83 and .79, respectively.

4.2. Results

4.2.1. Clustering cognitive-affective depressive and nonspecific somatic symptoms

The same statistical procedure to perform the cluster analysis described in Study 1 was replicated in the current dataset. In Study 3, PHQ-9 and BSI somatization scales were used. Firstly, the examination of Ward's hierarchical cluster analysis outputs suggested that three clusters should be retained as an optimal solution. This solution was then compared with the *K*-means analysis with an *a priori* defined $k = 3$. The comparison between the results obtained in the *K*-means analysis and in the hierarchical cluster analysis showed a substantial agreement ($\kappa = .88$). The MANOVA revealed that clustering variables significantly differed between the clusters (fixed factor), Wilks's λ , $F(2, 650) = 450.01$, $p < .001$, $\eta^2 = .58$.

Sixty-seven percent of mothers were assigned to the NoS cluster ($n = 434$), 28% ($n = 182$) to the DS cluster, and 5% to the DSS cluster ($n = 36$). Follow-up Bonferroni-corrected ANOVAs also indicated that all clustering variables significantly differed across the three clusters ([Table 2](#)). Follow-up Bonferroni-corrected ANOVAs showed that all clustering variables significantly differed across the three clusters ([Table 2](#)). Results of Tukey–Kramer post-hoc tests on the differences between clusters on the PHQ-9 score revealed that, when compared with the other two clusters, the NoS cluster exhibited the lowest levels of cognitive-affective depressive symptoms. The DSS cluster reported significantly higher values of cognitive-affective depressive symptoms than the DS cluster. In comparison with the other two clusters, the DSS cluster also exhibited the highest score on the somatization scale, while the NoS cluster showed the lowest levels of nonspecific somatic symptoms ([Table 2](#)). Sixty-seven percent of mothers were assigned to the NoS cluster ($n = 434$), 28% ($n = 182$) to the DS cluster, and 5% to the DSS cluster ($n = 36$).

4.2.2. Cluster differences in coparenting dimensions

As displayed in [Table 3](#), ANOVA analyses demonstrated significant overall differences between the three clusters on cooperative coparent-

ing, undermining coparenting, $F(2, 650) = 11.01$, and overt-conflict coparenting, $F(2, 650) = 16.08$ (all $p < .001$). Tukey–Kramer post-hoc comparisons showed, when contrasted with the other two clusters, the NoS cluster reported significantly higher levels of cooperative coparenting and lower levels of undermining coparenting ([Table 3](#)). There were no significant differences between DS and DSS clusters on these variables. For overt-conflict coparenting scores, the Tukey–Kramer post-hoc test revealed that the DSS cluster had higher levels of overt-conflict coparenting than both DS and NoS clusters. The DS cluster also had higher scores than the NoS cluster ([Table 3](#)).

The three clusters were labeled based on the most salient dimensions. Cluster 1 (80.2% of the sample) reported the lowest scores on both depression and somatization scales. Therefore, this cluster was labeled as No Symptom cluster (NoS). Cluster 2 (14.4% of the sample) reported high average scores on the depression scale and low average scores on the somatization scale. Since the most salient dimension in this cluster was cognitive-affective depressive symptoms, it was labeled the Depression Symptoms only cluster (DS). Finally, Cluster 3 (5.4% of the sample) revealed high average scores on both depression and somatization scales. Based on these scores, this cluster was labeled as Depression and Somatic Symptoms cluster (DSS).

5. Discussion

Our primary goal was to generate and cross-validate clusters of cognitive-affective depressive and nonspecific somatic symptoms in three independent samples of mothers. As hypothesized, three clusters were found in Study 1 and replicated in Studies 2 and 3: a cluster with high levels of cognitive-affective depressive symptoms and nonspecific somatic symptoms labeled as DSS; a cluster with high levels of cognitive-affective depressive symptoms and low levels of nonspecific somatic symptoms labeled as DS; and a cluster with low levels of cognitive-affective depressive symptoms and nonspecific somatic symptoms labeled as NoS.

Study 2 was particularly important in clusters validation since empirical evidence for clusters replicability in a high-risk sample was found. In addition, in Studies 1 and 3, we found a similar distribution of participants per cluster as identified in previous research ([Illi et al., 2012](#)). As expected, a higher prevalence of cognitive-affective depression and nonspecific somatic symptoms was identified in the high-risk mothers (Study 2). Taken together, these results suggest that the cluster-solution was stable and reliable to examine potential differences in parenting and coparenting dimensions across samples. Moreover, these findings emerged in demographically diverse samples of mothers of children in early and middle childhood periods, largely ranged in age, education, marital status, and risk level, suggesting that these clusters may be generalizable.

The second aim of this research was to test whether these clusters would differ in a range of parenting and coparenting dimensions. In the current research, our results across the three studies indicate different associations between cognitive-affective depression and nonspecific somatic symptoms and specific parenting and coparenting dimensions. Particularly, we found that high co-occurrence of cognitive-affective depressive and nonspecific somatic symptoms in the DSS cluster were associated with the highest levels of CPM risk and overt-conflict coparenting. However, no differences in maternal perceived parenting competence and cooperative and undermining coparenting were found between DS and DSS clusters. We also found an unexpected result in permissive parenting, in which the DS cluster reported the highest levels of permissive parenting and no differences between the other clusters were identified.

These findings bring into question why the co-occurrence of cognitive-affective depressive and nonspecific somatic symptoms were only associated with parenting and coparenting behaviors that reflect overreactive and anger behavioral strategies (i.e., harsh parenting and overt-conflict coparenting). In line with the action-control framework

Table 3
Clusters differences on coparenting dimensions.

Variable	NoS (n = 434)		DS (n = 182)		DSS (n = 36)		Cluster differences tests	
	M	SD	M	SD	M	SD	F (2, 650)	Cluster contrasts ^a
Cooperative coparenting	4.61	1.41	4.08	1.59	3.70	1.46	12.59***	NoS > DS, DSS
Undermining coparenting	.77	1.13	1.24	1.38	1.28	1.03	11.01***	NoS < DS, DSS
Overt-conflict coparenting	.69	.84	.95	.93	1.48	1.23	16.08***	NoS < DS < DSS

Note.
^a Significant cluster differences at $p < .05$ using Tukey-Kramer test. NoS = No symptoms cluster. DS = Depression symptoms only clusters. DSS = Depression and somatic symptoms cluster coparenting.

*** $p < .001$.

of parental self-regulation (Dix and Meunier, 2009), we hypothesize that co-occurrence of cognitive-affective depression and nonspecific somatic symptoms in the DSS cluster are linked with overreactive interpersonal interactions through its association with additional explanatory mechanisms, such as negative emotional arousal and dysregulation. Maternal anger hyperactivity may particularly operate as a key explanatory mechanism of these associations (Estlander et al., 2008). Conceptual frameworks suggest anger as a primary emotional reaction to physical and affective pain (Bruehl et al., 2006). As somatic symptoms are related to higher perceived pain severity and pain hypersensitivity, lower thresholds of somatic-related pain are needed to elicit anger arousals (Koh et al., 2005). These arousals, associated with hostile distortions and high sensibility to aversive stimulation found in depressed individuals (Dix et al., 2014; Smith et al., 2016), may predispose mothers with high depressive-somatic comorbidity in the DSS cluster to even greater levels of dysregulated behavioral anger expression in parenting and coparenting interactions (Callender et al., 2012; Du Rocher Schudlich and Cummings, 2007; Lamela and Figueiredo, 2013). Though deductively logical, this interplay between depressive-somatic comorbidity, pain, anger, and family interactions should be examined in future research.

An alternative interpretation of these results is that, for depressed individuals, nonspecific somatic symptoms and overreactivity in interpersonal relationships are possibly physical and psychological correlates of the same biological patterns, therefore showing high covariance. This hypothetical explanation is consistent with emerging integrative psychobiological models that stress the interaction of psychological and neuroendocrine systems in the prediction of somatic dysregulation in depression and its multiple negative outcomes (Harshaw, 2015; Northoff et al., 2011). Specifically, the dysregulation of the hypothalamic-pituitary-adrenal (HPA)-axis is proposed as a main distal etiological path for the emergence of somatic complaints in depression (Penninx et al., 2013). A similar pattern of the HPA-axis dysregulation has been also associated with harsh parenting and anger-conflictual interparental relationships (Mills-Koonce et al., 2009; Sturge-Apple et al., 2011). Thus, our results raise the possibility that the association between high depression-somatic symptoms found in the DSS cluster and overreactive behavioral strategies in interpersonal interactions is related to HPA-axis dysregulation.

As expected, DS and DSS clusters did not differ in the levels of perceived parenting competence and cooperative and undermining coparenting. An explanation of this result may be derived from the psychological models of depression. According to these models, cognitive-affective depression symptoms are a product of maladaptive patterns of cognitive and emotional information-processing. This includes biased attention, biased emotional processing, biased thoughts and rumination, biased memory, and dysfunctional attributions. Therefore, as these specific dimensions are described as cognitive and affective components of parenting and coparenting behavior (Crandall et al., 2015; Krishnakumar and Buehler, 2000), we hypothesize that cognitive and emotional distortions in depression would be reflected in the parenting and coparenting dimensions that are regulated by

cognitive and affective mechanisms.

In contrast with our hypothesis in Study 2, DSS and NoS clusters reported the lowest scores of permissive parenting. This finding is contradictory with previous empirical work (Oyserman et al., 2005). Although still speculative at this time, some explanations of this finding might be advanced. Lower permissive parenting in the DSS cluster may be explained by a higher prevalence of harsh parenting styles in mothers with high comorbidity of psychopathology symptoms, as suggested previously (Harvey et al., 2011). Alternatively, this finding may reflect a variation of the compartmentalization hypothesis. In family systems models, the compartmentalization hypothesis posits that mothers exposed to high levels of marital conflict may be able to provide more responsive caregiving to their children as a compensatory mechanism of children's exposure to family violence (Sturge-Apple et al., 2014). Thus, based on the same family processes, mothers exposed to IPV with high levels of depressive-somatic comorbidity (DSS cluster) may also make an effort to limit the impact of their psychological distress on children by reducing permissiveness in parenting practices. Future research should replicate this finding in IPV-victims and community populations and examine these potential explanatory mechanisms.

Taken together, the convergence of findings across the studies may suggest that women with comorbid cognitive-affective depressive and nonspecific somatic symptoms are more likely to have poorer physical health. Additionally, the combined impairments caused by depression and a compromised physical health status may negatively interfere with mothers' abilities to efficiently employ their cognitive and emotional resources to manage parenting and coparenting challenges. This puts them at higher risk of ineffective parenting and coparenting strategies, such as harsher parenting practices and coparenting hostility. In addition, mothers with a high comorbidity of cognitive-affective depression and nonspecific somatic symptoms do not show higher risk of a lower sense of competence in parenting, higher undermining coparenting, and more problems in cooperative coparenting, when compared with mothers with only cognitive-affective depression symptoms; this suggests that these specific parenting and coparenting problems are not primarily linked with maternal physical health status and somatic complaints but instead with cognitive and affective symptoms of depression.

Several limitations warrant discussion. First, all constructs were only assessed using self-report measures. Despite that all measures used in the current research have demonstrated high and significant associations with interviewing and observational measures, multi-informant and multimethod procedures could have contributed to a higher accuracy of measurement and also decreased possible shared method variance. Second, changes in severity of depression and somatic symptoms over time might be expected, as suggested by longitudinal studies. However, due to the cross-sectional design of the current study, the potential differential impact of these changes over time on parenting and coparenting variables was not examined. Third, our results are specific to children in early and middle childhood periods and may not generalize to infants and adolescents. Finally, the current

research was only conducted with women. While this relative homogeneity in gender augments statistical confidence in the associations found, this limited variability restrains the generalization of these findings to fathers.

In summary, to our knowledge, this was the first study to examine the association between cognitive-affective depressive and nonspecific somatic clusters and parenting and coparenting. Our findings may have one theoretical implication. Across the three studies, the prevalence of comorbidity of cognitive-affective depression and nonspecific somatic symptoms, when compared to only cognitive-affective depression symptoms, was particularly higher in Study 2 composed of women exposed to IPV. As IPV victimization is an ultimate form of gender inequality, this finding may provide partial evidence for the social psychiatric hypothesis of the social nature of somatic depression (Silverstein and Levin, 2014). However, our results might also provide support for the recent psycho-neurobiological models that posit that somatic symptoms in depression may result from biological phenotypic and behavioral susceptibility to adverse social environmental conditions (Harshaw, 2015). To further draw more conclusive evidence, future research should include biological and social measures to examine sociobiological patterns among the clusters.

5.1. Clinical implications

Our results may also have four major clinical implications. First, by showing differential associations between specific parenting and coparenting problems and maternal somatic complaints, mental health professionals should not only assess depressive symptoms, but also routinely include measures of somatic symptoms in their assessment protocols. In addition, as depressive and nonspecific somatic symptoms were associated with different domains of parenting and coparenting, our findings suggest that clinical assessment may benefit from the inclusion of cognitive (e.g., parenting beliefs and perceived competence), emotional (e.g., parenting stress), and behavioral (e.g., parenting practices) measures of parenting and coparenting, rather than a unidimensional approach to assessment of these constructs. Multidimensional assessment procedures may be required for the translation of our findings into more effective clinical interventions in parenting and coparenting.

Second, by identifying specific subgroups reporting a higher risk of specific problems in parenting and coparenting, our findings may raise the necessity of selective preventive interventions. In primary care settings, clinicians should particularly monitor parenting and coparenting problems in mothers with high levels of cognitive-affective depressive symptoms (with or without somatic complaints comorbidity). In particular, mental health professionals could identify mothers with both high cognitive-affective depression symptoms and somatic complaints for early support and intervention to prevent child physical maltreatment and overt-conflict coparenting.

Third, to maximize effective intervention for parenting and coparenting problems in specific clinical profiles, maternal somatic symptoms should be targeted as part of treatment. Current evidence-based intervention programs already account for the effect of depression symptoms on parenting and coparenting behavior and for treatment efficacy and effectiveness (Feinberg et al., 2016; Reuben et al., 2015). However, our results might recommend that interventions on child physical maltreatment and overt-conflict coparenting include an additional component of psychological strategies to manage and reduce somatic symptoms in their curricula.

Finally, our research may also suggest that biological-medical interventions for pain management and treatment of physical health problems may partially contribute to the reduction of mothers' risk of child physical maltreatment and overt-conflict coparenting. Our findings showed that these specific parenting and coparenting problems were associated with high levels of maternal cognitive-affective depressive symptoms and somatic complaints; consequently, interven-

tions may benefit from a higher cooperation between mental health professionals and health professionals. Therefore, multidisciplinary teams may be recommended to increase treatment's effectiveness for both depressive and somatic symptoms, as well as for specific parenting and coparenting problems.

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