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Maternal Symptoms of Depression and Paranoid Ideation can be Predictive of the Onset of Eating Disorders in Early Adolescents Offspring: A Nine-year Longitudinal Study

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Abstract

Studies in the Developmental Psychopathology framework have demonstrated the maladaptive influence of maternal depression on offspring emotional-behavioral functioning. Moreover, many Authors have suggested that the chronicity and timing of maternal depression can be crucial aspects predictive of specific maladaptive outcomes in children such as Eating Disorders in early adolescence. Longitudinal studies in the field have usually focused on small clinical samples and covering short periods of time, typically from early childhood to toddlerhood. The present longitudinal study was aimed at investigating the stability of children and mother's psychopathological symptoms across different ages (2, 5, 8 years) in a community sample, and also at evaluating the capacity of these psychopathological problems to identify individuals at risk for the onset of eating disorders in adolescence. Results showed that both children's and mother's symptoms, assessed at different ages, can contribute to identify adolescents at risk for the onset of eating disorders. Moreover, among mothers' symptoms, only depression and paranoid ideation, which showed a high stability over time, gave a significant contribution to the categorization of youth at risk for eating disorders. Our study adds to the literature because, not only we confirmed the predictive power of general psychopathological maternal risk on offspring mental health, but we also found the effect of two specific symptomatic configurations: depression and paranoid ideation.

Key words: depression, paranoid ideation, early adolescence, eating disorders.

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Novelty and Significance

What is already known about the topic?

Maternal depression is predictive of several offspring emotional-behavioral problems over time.
Eating disorders can have their onset in early adolescence.

What this paper adds?

 Maternal depressive symptoms are predictive of eating disorders in early adolescents only if they are stable over time.

Maternal stable paranoid ideation symptoms foster eating disorders in their early adolescents offspring.

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It is widely known that maternal psychopathology can be a crucial risk factor for offspring's psychological functioning predicting both internalizing and externalizing symptoms in their childhood and adolescence (Rutter, 1989). The influence of mothers' symptomatology on their children's psychological well-being can be supported by a number of underpinning mechanisms. For instance, mothers and children can share genetics predisposing to psychopathology, maternal symptoms can be caused by disrupted parenting, children can be exposed to maternal distorted cognitions and/or to stressful environments, the family as a whole can lack social support (for a seminal and comprehensive study see Garber & Dodge, 2006; Goodman & Gotlib, 1999). Research in this field has used both maternal clinical diagnoses and continuous measures of psychopathological risk, considering respectively clinical or general populations (Markon, Chmielewski, & Miller, 2011) focusing on specific problems (e.g., maternal depression or anxiety) or evaluating the potential negative impact of parental more complex difficulties (e.g. general psychological distress, outcomes of traumatic experiences, etc.). Some authors (Cummings & Davies, 1994; Harvey, Stoessel, & Herbert, 2011) have suggested that the maladaptive influence of mothers' specific diagnosis (e.g. maternal depression) is such that it can predict emotional-behavioral problems in children even controlling for other relevant variables (e.g. other parent's characteristics, marital discord, socio-economical status, etc.). Moreover, it has been shown that when a wider range of maternal psychopathology are considered, more useful information for the prevention and early intervention programs can be captured (Sprafkin, Gadow, Weiss, Schneider, & Nolan, 2007) and children of mothers with co-occurring psychopathologies face more severe symptom and poorer psychosocial functioning (Cerniglia *et alii*, 2015; Mineka, Watson, & Clark, 1998).

Following the Developmental Psychopathology standpoint (for a seminal work, see Sroufe, 2009), it has been demonstrated that the onset of emotional-behavioral problems in children may occur early in life (Egger & Angold, 2006), and a vast number of studies have therefore focused on preschool psychological difficulties, with the aim of identifying potentially problematic developmental paths and organizing possible interventions (Capobianco, Pizzuto, & Devescovi, 2017; Tambelli, Cerniglia, Cimino, & Ballarotto, 2015). However, especially in normative samples, assessing and recognizing symptoms in very young children can be particularly challenging, and isolating internalizing (e.g. depression and anxiety) from externalizing syndromes (e.g. aggression and hyperactivity) has emerged as a difficult practice (Achenbach & Rescorla 2000; Sterba *et alii*, 2007). Notwithstanding these difficulties, several authors investigated psychopathology continuity in the general population, finding high correlations for children behaviors across a 6-year period (Verhulst & Van der Ende, 1992).

Many authors have evaluated the stability of emotional-behavioral difficulties in offspring by observing possible correlations between levels of symptoms measured at different assessment points over time (Campbell 1995; Fischer *et alii*, 1984). They found both homotypic (i.e. continuity of a certain clinical phenomenon over time without considerable phenotypical change) and heterotypic stability (i.e. manifestations of different clinical forms over time) (Mathiesen & Sanson 2000; Mesman *et alii*, 2001; Mian *et alii*, 2010). It is worth noting that the stability of some psychological problems, such as aggression or oppositional behaviors, can be clinically significant because during a normal development these difficulties usually tend to decrease, especially from 2 to 6 years of age (Fanti & Henrich 2010). The same reasoning can be applied to internalizing problems; for example separation anxiety can be normal at younger ages but constitute a cue of maladaptive development if they manifest when the child is older (Cerniglia, Cimino, Ballarotto, & Monniello, 2014; Gilliom & Shaw 2004; Pizzuto & Capobianco, 2008). In general, the stability of psychopathological symptoms from early childhood to early adolescence can be considered as a possible predictor of more structured mental disorders in adolescence, for instance Eating Disorders, Depression and others (Caspi, Moffit, Newman, & Silva, 1996; Monacis, de Palo, Griffiths, & Sinatra, 2017).

As for the continuity of children's psychopathological symptoms, maternal problems have shown stability over time in several important studies (e.g. Ghodsian, Fogelman, Lambert & Tibbenham, 1980; Rowe & Britt, 1991). This stability has been explained through two main mechanisms (Bartels, van den Oord, Hudziak, Rietveld, van Beijsterveldt, & Boosma, 2004): a transmission model, in which earlier experiences and vulnerabilities are transmitted to later points in time (even though the impact of earlier effects may not necessarily be limited to the same problematic phenotype), and a common factor model in which causal relations between subsequent time points are not assumed, but a stable underlying liability (e.g. a genetic factor) accounts for the clinically relevant problem. The Developmental Psychopathology framework, although considering genetic and biological correlates, mainly assumes the transmission model. The continuity of parental (and above all maternal) psychopathology can be conceptualized as a potential risk factor shared with offspring predicting the onset and persistence of emotional-behavioral problems in children (Campbell, 1995). Importantly, authoritative studies have demonstrated that even subclinical levels of psychopathological symptoms in mothers can predict maladaptive outcomes in their children if maternal problems are persistent and are present since the early infancy of their offspring (Connell & Goodman, 2002). For example, parental depressed mood and anxiety have been correlated with externalizing and withdrawn behaviors in children (Papp et alii, 2005). However, the vast majority of studies focusing on the transmission of psychopathological risk from mothers to children have selected samples of diagnosed mothers, leaving normative populations with subclinical samples relatively unexplored.

Several studies have demonstrated the maladaptive influence of maternal depression on offspring emotional-behavioral development (e.g. Downey & Coyne, 1990; Rutter, 1966). The underpinning mechanisms governing the homotypical or heterotypical transmission of psychopathology from depressed mothers to their children are essentially overlapping with those described in the previous paragraph with reference to psychopathology in general (i.e. shared genetics, disruption of parenting, exposure to maternal maladaptive cognitions, behavior and affects, exposure to stressful environments). However, more recent studies have suggested that the chronicity and timing of maternal depression (and its severity) can be key aspects to predict specific maladaptive outcomes in children (Hammen & Brennan, 2003). Importantly, several studies have shown that mothers with chronic depression have poorer interactions with their children, which can have long-lasting negative consequences on offspring development (Hammen, 2006). It is noteworthy that this literature typically focused on clinical samples in which depression severity and chronicity was usually high. However, it has been posited that depression chronicity in mothers, in itself, can predict emotional-behavioral problems in children, even controlling for severity and even in samples with sub-clinical levels of depression (Frankel & Harmon, 1996). With regards to the timing of onset of mothers' depression, most of research has suggested that earlier exposure of children to maternal symptoms can be particularly disruptive for their psychological well-being, although (as seen above) the vast majority of studies have been conducted on clinical samples that generally

show atypically severe symptoms and frequently provide non-generalizable results. A particularly informative study on normative sample demonstrated that children exposure even to mild maternal depression for a period of at least one year was predictive of psychopathological risk in offspring (Hammen & Brennan, 2003).

Importantly for the present study, a relatively recent branch of literature has proposed that maternal depression could determine eating disorders (ED) as well as disordered eating in early adolescents (Ravi, Forsberg, Fitzpatrick, & Lock, 2008; Stroberm Morrell, Burroughs, Salkin, & Jacobs, 1985). The prevalence of ED in the early adolescents general population (5,5% in girls and 1,5% in boys; Tafà, Cimino, Ballarotto, Bracaglia, Bottone, & Cerniglia, 2017) permits to study the possible relations between the most frequent and potentially impairing symptomatology in mothers (depression) and disordered eating in their adolescent offspring. Maternal negative affection and depressed mood are suggested to be statistically related to EDs in early adolescents (Munsch, Meyer, Quartier, Wilhelm, 2012; Striegel-Moore et alii, 2005), and several authors have linked the reduced capacity of affect regulation in children of depressed mothers to the their likability of manifesting an eating disorder in early adolescence (Haycraft et alii 2014; Lask and Bryant-Waugh 2013). Children of mothers with depression (especially if maternal symptoms were chronic and stable over time) frequently show difficulties in recognizing, communicating and coping with negative feelings (Nowakowski *et alii*, 2013), which are associated with emotional eating and bulimia nervosa. Moreover, depressed mothers often fail in restrain from negative comments on others' physical appearance, due to their individual emotional sufferance. Therefore, early adolescents with mothers with depression are liable to present idealization of thinness and body dissatisfaction (Levine, Striegel-Moore & Smolak, 2013). Further, Goodier et alii (2013) posited that relational patterns between mothers with depressive symptoms and their children are often incoherent, with low quality of interaction during offspring's infancy and toddlerhood (Cerniglia et alii, 2014; Paciello et alii, 2013). Specifically, Hayaki (2009) demonstrated that mothers with depression can show intrusiveness and withdrawal in their exchanges with their children (also due to an impairment in the brain reward system circuitry in depressed mothers, which could account for the lack of pleasure in their interactions with offspring; Belden, Irvin, Hajcak, Kappenman, Kelly, Karlow, Luby, & Barch, 2016) and can predict the onset of EDs during early adolescence. Finally, it has been widely demonstrated that exposure to chronic maternal depressive symptoms is associated to a reduced functionality in socio-cognitive processing in early adolescence, which in turn is a key feature of eating disorders (Pratt, Goldstein, Levy, & Feldman, 2017).

Notwithstanding the growing literature focusing on emotional-behavioral problems in offspring and their relations with maternal psychopathology, longitudinal studies have, so far, been relatively rare, concentrated on small clinical samples and covering short periods of time, typically from early childhood to toddlerhood (Bryant-Waugh, 2013).

The present work is part of a cohort study designed in 2007 in collaboration with a network of pediatricians and schools in Italy, aimed at identifying early predictors of internalizing/externalizing problems in children at 2, 5, 8 years and early adolescents at 11-12 years of age. Moreover, the possible factors fostering disordered eating in early adolescents were also assessed. We hypothesized that emotional-behavioral problems in children and maternal psychopathology (specifically mothers' depressive symptoms), would be associated with disordered eating in early adolescents.

Method

Participants

In 2007, our research group initiated a screening program in collaboration with pediatricians, and public and private schools in Italy. The study protocols were approved by the Ethical Committee of Psychology Faculty of Sapienza-Universita di Roma, in accordance with the guidelines approved in Helsinki Declaration. Moreover, participating schools obtained ethical clearance through their respective institutional review bodies. The study was organized over four waves (T1 to T4), each of three years and included N=267 children with their mothers. N=35 mothers declined to participate in the study after their first acceptance. N=25 mothers or children were pursuing pharmacological and/or psychological treatment and were excluded from the study, as other N=18, due to severe organic or physical problems of the child. N=29 subjects were excluded from the study for incomplete or missing data at one or more assessment points. Therefore, the sample of this study is composed of N=160 children [mean age at T1= 2.46 (SD= 1.1); mean age at T2= 5.25 (SD= 0.9); mean age at T3= 8.1 (SD= 1.2); T4= 11.2 (SD= 1.2)] and their mothers.

At T1, T2, T3 and T4 mothers were administered SCL-90-R; they also filled-out the CBCL1/2–5 (at T1 and T2) and CBCL6-18 (at T3). At T4, with parents' written consent, early adolescents were asked to fill out a questionnaire designed to assess risk of eating disorder (see Measures section below).

Informed written consent was obtained from all subjects for the aims of the study, and thanks to the great effort of pediatricians and school workers, the sample was not affected by attrition.

Eighty-nine percent of children and mothers belonged to intact families (and the remaining number, although not married, had a partner living in the same house). All children attended kindergarten and (later on) school. Mothers had a middle socioeconomic status and an average educational level measured through and anamnestic questionnaire at T1 (Bornstein & Bradley, 2014).

Measures

- Symptom Check-List-90-90 (SCL-90-R; Derogatis, 1997). A 90-item self-report symptom inventory aimed to measure psychological symptoms and psychological distress. Its main symptom dimensions are Somatization, Obsessive-Compulsivity, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. All scales of the SCL-90-R have shown previously a good internal consistency (alpha= 0.70-0.96) in adolescents and adults (For the Italian version see Prunas, Sarno, Preti, Madeddu, & Perugini, 2011).
- *Child Behavior Check-List*_{1/2-5} (CBCL_{1/2-5}; Achenbach & Rescorla, 2001). A questionnaire filled out by parents and caregivers to assess a child's abilities and his/her specific behavioral/emotional characteristics. It is composed of 100 items. It is composed by an Internalizing Problems Scale, which consists in its turn of four subscales: Emotionally Reactive, Anxious/Depressed, Somatic Complaints, and Withdrawn. The Externalizing Problems Scale is composed of two subscales: Attention Problems and Aggressive Behavior. The CBCL_{1/2-5} has high test-retest reliability and high internal consistency (Achenbach & Rescorla, 2001). The CBCL₆₋₁₈ (Achenbach & Rescorla, 2001) is composed of 113 items. The Internalizing Problems Scale consists of three

subscales: Anxious/ Depressed, Withdrawn/Depressed, and Somatic Complaints. The Externalizing Problems Scale consists of two subscales: Rule-Breaking Behavior and Aggressive Behavior. The criterion-related validity of both versions of the CBCL is supported by the ability of the CBCL's quantitative scale scores to discriminate between demographically matched, referred and non-referred children (Kim *et alii*, 2012). In the present study, we used the Italian validated versions of the measure (LaFreniere *et alii*, 2002).

Eating Disorders Inventory-Referral Form (EDI-3-RF; Garner, 2004; see Giannini & Conti, 2008 for the Italian version). A 25-item brief self-report measure, derived from the EDI-3 (which contains 91 items) and designed to measure eating disorder risk. The EDI-3-RF can be administered in non-clinical or clinical settings and, as indicated by the scale's author and by other studies (e.g. Stice & Whitenton, 2002), the measure can be administered to subjects as young as eleven years old. The EDI-3-RF uses a 6-point likert scale (1= always, 6= never) forming the Drive for Thinness (e.g., "I think about dieting"), Bulimia (e.g., "I eat moderately in front of others and stuff myself when they're gone"), and Body Dissatisfaction subscales (e.g., "I think that my stomach is too big"). Internal consistency of EDI-3-RF was proved to be .80 (Cumelia, 2006).

RESULTS

Table 1 illustrates means and standard deviations of CBCL sub-scales along with their Skewness and Kurtosis. A series of repeated measures ANOVA showed that means scores were not stable over time (p < .05). Notably, skewness and kurtosis were not ranging between ± 1 for many dimensions, suggesting that they significantly deviated from normal distribution. All scales showed adequate levels of internal consistency.

Descriptive statistics of mothers' scores on SCL-90-R sub-scales are illustrated in Table 2 along with Skewness and Kurtosis. Again, a series of repeated measures ANOVA

	-	-			
	CBCL Scales	М	SD	Skewness	Kurtosis
	Emotional Reactivity	4.57	3.74	1.16	1.01
	Anxiety and Depression	4.44	3.00	.78	08
	Somatization	4.59	3.81	1.03	.23
2 years	Withdrawal	4.03	3.59	1.01	.15
2 years	Problematic Attitudes	3.85	2.21	.54	33
	Aggressive Behaviours	11.74	6.56	.96	.26
	Sleep difficulties	4.43	2.90	1.08	.43
	Other Problems	16.12	1.14	1.47	1.27
	Emotional Reactivity	5.34	4.17	.67	31
	Anxiety and Depression	4.84	3.01	.47	35
	Somatization	6.09	4.38	.77	.02
5	Withdrawal	4.94	3.81	.68	18
5 years	Problematic Attitudes	4.32	2.51	.32	48
	Aggressive Behaviours	12.84	7.48	.59	15
	Sleep Difficulties	4.19	3.03	.49	22
	Other Problems	18.81	11.82	.86	.14
	Anxiety and Depression	7.24	5.76	02	-1.51
	Withdrawal with Depression	4.29	5.34	1.14	47
	Somatization	3.75	3.47	.98	.10
	Social Problems	4.33	3.73	.68	34
8 years	Thought Problems	4.81	4.20	.84	.59
	Attention Problems	3.98	3.07	.51	.24
	Rule Breaking Behaviours	4.89	4.67	1.40	2.61
	Aggressive Behaviours	6.86	6.16	1.21	1.66
	Other Problems	5.92	5.51	1.33	2.54

Table 1. Descriptive Statistics of Children Symptoms across different ages (2, 5 and 8 years).

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	SCL-90 Scales	М	SD	Skewness	Kurtosis
	Somatization	1.11	1.07	.66	-1.28
	Obsessive-Compulsive	.49	.41	.76	08
	Interpersonal	.40	.37	1.36	2.25
	Depression	1.62	1.24	.03	-1.76
2 years	Anxiety	.92	.91	.85	76
2 years	Hostility	.59	.52	1.00	.54
	Phobia	.38	.46	1.42	1.47
	Paranoia	1.46	1.07	.08	-1.44
	Psychoticism	.38	.39	1.36	1.88
	Sleep Difficulties	.50	.59	1.26	1.18
	Somatization	1.02	1.16	.76	-1.24
	Obsessive-Compulsive	.35	.41	1.34	1.03
	Interpersonal	.33	.35	1.59	3.04
	Depression	1.54	1.31	.02	-1.87
5 years	Anxiety	.77	.89	.91	79
	Hostility	.50	.62	1.43	1.35
	Phobia	.33	.40	1.56	1.92
	Paranoia	1.32	1.19	.28	-1.41
	Psychoticism	.35	.39	1.46	1.87
	Somatization	.86	1.20	1.12	64
	Obsessive-Compulsive	.24	.31	2.14	4.83
	Interpersonal	.24	.31	1.97	4.91
	Depression	1.42	1.32	.07	-1.90
8 years	Anxiety	.66	.91	1.30	.16
	Hostility	.46	.67	1.78	2.66
	Phobia	.28	.32	1.96	4.65
	Paranoia	1.21	1.17	.29	-1.57
	Psychoticism	.31	.35	2.04	4.43
	Sleep Difficulties	.27	.45	1.94	3.57

Table 2. Descriptive Statistics of Mothers' Symptoms across children's different ages (2, 5 and 8 years).

showed that the means scores were not stable over time (p < .05), except for psychoticism and phobia. Many of the SCL-90-R dimensions did not follow a normal distribution, as suggested by the high level of kurtosis and skewness. All scales showed adequate levels of internal consistency, except for hostility scale that showed an unsatisfactory Cronbach's Alpha estimation.

In order to evaluate the stability of children and mothers' symptoms over time, product-moment correlations were computed among CBCL and SCL-90-R scores as assessed in the different assessment points (children's ages 2, 5, 8 years). Results showed that children's psychopathological symptoms were relatively stable over time (see Table 3), with significant and moderate-large correlations (rs > .30) across the different assessment points. Conversely, only mother's depression and paranoid ideation showed a high consistency over time, while the other mothers' symptoms revealed low correlation among the different measurement occasions (see Table 4). Overall, children's symptoms appeared to be relatively stable in a rather long period of time (6 years), while mothers' symptoms showed a high revealed of instability, except for depression and paranoid ideation that showed a high level of consistency over time.

Table 3. Correlations of a	children's sympto	oms across the 3	3 observations.
CBCL Scales	r (OL-OII)	r (OL-OIII)	r (OII-OIII)

CBCL Scales	r (OI-OII)	r (OI-OIII)	r (OII-OIII)
Anxiety-Depression	.47**	.39**	.46**
Withdrawal-Depression	.71**	.53**	.48**
Somatization	.72**	.57**	.60**
Problematic Attitudes	.65**	.57**	.55**
Aggressive Behaviors	.59**	.48**	.59**
Other Problems	.65**	.53**	.67**

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CBCL Scales	r (OI-OII)	r (OI-OIII)	r (OII-OIII)
Somatization	.32**	13	.41**
Obsessions-Compulsions	.05	16*	.11
Interpersonal Problems	.06	07	.25**
Depression	.91**	.92**	.94**
Anxiety	.32**	07	.43**
Hostility	.21**	.06	.21**
Phobia	.19*	.01	.16
Paranoia	.76**	.81**	.87**
Psychoticism	.25**	02	.22**
Sleep Problems	02	.07	.09

Table 4. Correlations of mothers' symptoms across the 3 observations.

In order to evaluate the discriminative power of mothers' and children's psychopathological profiles (as assessed at the different children's ages) for the accurate identification of children at risk of eating disorders in early adolescence, three discriminant analyses for each outcome were conducted (one for each measurement wave), including children's and mother's symptoms across the different waves as factors, and the occurrence of Body Dissatisfaction (BD), Drive to Thinness (DT) and Bulimia (B) at age 11 as grouping variables. Considering that many children's and mothers' symptoms showed considerable deviations from normality in our sample (see Table 1 and 2), a root square transformation was applied to CBCL and SCL-90-R scales for each measurement occasion. All the transformed variables showed distributions close to normal curve.

The analyzed discriminant functions showed large canonical correlations with all outcomes BD (rs > .50), DT and B memberships (rs > .80), supporting the adequate fit of the models tested. Moreover, as shown in Tables 5, 6 and 7 many children's symptoms

Body Dissatis	<i>Body Dissatisfaction</i> across three different childrens' age (2, 5, 8 years).				
	Body Dissatisfaction	OI	OII	OIII	
	Discriminant functions	.58	.54	.58	
	Anxiety-Depression	.18	.29	.69	
	Withdrawal-Depression	.54	.59	.49	
	Emotional Reactions	.52	.59	-	
	Thought Problems	-	-	.65	
	Attentional Problems	-	-	.59	
CBCL	Somatization	.41	.64	.47	
	Problematic Attitudes	.54	.55	-	
	Social problems	-	-	.53	
	Rule Breaking	-	-	.51	
	Aggressive Behaviors	.33	.40	.56	
	Sleep Behaviors	.33	.42	-	
	Other Problems	.39	.59	.54	
	Somatization	.41	.31	.47	
	Obsessions-Compulsions	07	12	.34	
	Interpersonal Problems	.03	.12	.40	
	Depression	.79	.86	.83	
	Anxiety	.53	.29	.44	
SCL-90	Hostility	.34	.19	.55	
	Phobia	.21	.26	.39	
	Paranoia	.59	.70	.79	
	Psychoticism	.30	.27	.33	
	Sleep Problems	01	.15	.40	

Table 5. Canonical correlations of CBCL and SCL-90 scales with respect to

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	Drive to Thinness	OI	OII	OIII
	Discriminant functions	.88	.88	.91
	Anxiety-Depression	.20	.27	.60
	Withdrawal-Depression	.47	.53	.32
	Emotional Reactions	.42	.54	-
	Thought Problems	-	-	.57
	Attentional Problems	-	-	.46
CBCL	Somatization	.41	.57	.47
	Problematic Attitudes	.43	.44	-
	Social problems	-	-	.47
	Rule Breaking	-	-	.52
	Aggressive Behaviors	.28	.36	.50
	Sleep Behaviors	.30	.39	-
	Other Problems	.33	.53	.47
	Somatization	.29	.32	.26
	Obsessions-Compulsions	05	.01	.13
	Interpersonal Problems	.09	.12	.20
	Depression	.88	.84	.88
SCL 00	Anxiety	.31	.29	.25
SCL-90	Hostility	.19	.18	.25
	Phobia	.25	.21	.14
	Paranoia	.63	.70	.70
	Psychoticism	.26	.24	.13
	Sleep Problems	.06	.09	.19

Table 6. Canonical correlations of CBCL and SCL-90 scales with respect to *Drive to Thinness* across three different childrens' age (3, 6 and 8 years).

Table 7. Canonical correlations and standardized structural
coefficients (in parentheses) of CBCL and SCL-90 scales with respect
to Bulimia across three different childrens' are (3, 6 and 8 years)

to <i>Bulimia</i> across three different childrens' age (3, 6 and 8 years).				
	Bulimia	OI	OII	OIII
	Discriminant functions	.89	.89	.91
	Anxiety-Depression	.17	.26	.66
	Withdrawal-Depression	.42	.53	.33
	Emotional Reactions	.37	.56	-
	Thought Problems	-	-	.59
	Attentional Problems	-	-	.51
CBCL	Somatization	.37	.51	.48
	Problematic Attitudes	.41	.45	-
	Social problems	-	-	.55
	Rule Breaking	-	-	.54
	Aggressive Behaviors	.29	.36	54
	Sleep Behaviors	.30	.39	-
	Other Problems	.31	.51	.53
	Somatization	.28	.29	.27
	Obsessions-Compulsions	05	.01	.13
	Interpersonal Problems	.07	.12	.22
	Depression	.86	.84	.86
SCL-90	Anxiety	.30	.28	.26
3CL-90	Hostility	.18	.18	.24
	Phobia	.24	.21	.15
	Paranoia	.66	.67	.74
	Psychoticism	.26	.24	.12
	Sleep Problems	.06	.1	.20

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(as assessed with the CBCL scales) revealed considerable relations with BD, DT and B occurrence, with robust canonical correlations that tended to be consistent across different children's ages. In contrast, not all mothers' symptoms showed robust relations with the occurrence of BD, DT and B. More specifically, among the SCL-90-R sub-scales the greater and robust canonical correlations emerged for mothers' depression and paranoid ideation, which were also the more stable symptoms (see Table 5).

DISCUSSION

The present longitudinal study was aimed at investigating the stability of children and mother's psychopathological symptoms across different ages (2, 5, 8 years) in a community sample, and also at evaluating the capacity of these psychopathological problems to identify individuals at risk for the onset of eating disorders in early adolescence (i.e., 11 years old).

Overall, in accordance with our hypothesis, results showed that: (1) both children's and mother's symptoms, assessed at different ages, can contribute to identify adolescents at risk for the onset of eating disorders; (2) among mothers' symptoms, only depression and paranoid ideation, which showed a high stability over time, gave a significant contribution to the categorization of youth at risk for eating disorders. Considering these results in a transactional standpoint (Cicchetti & Toth, 1997), we hypothesize that the stability of mothers and children's symptoms formed a general problematic familial configuration reciprocally reinforcing, eventually resulting in youths' eating disorder in part of the study population. As Sroufe (2009) posited the psychopathology in children can be conceptualized as a deviation over time that occurred when child's development diverged markedly from its normal path due to child's failures in adaptation to new environmental pressures (e.g., in early adolescence which brings physical and neurobiological maturation and requests an adjustment to new and challenging relational and social demands).

Our study provides a significant contribution to the literature in this specific field because, not only we confirmed the predictive power of general psychopathological maternal risk on offspring mental health, but we also found the effect of two specific symptomatic configurations: depression and paranoid ideation. One of them, the depressive symptoms of the mother, had already been proved to foster emotional-behavioral problems in children and adolescents, but depressive symptoms may vary in length from days, week or even years and can be episodic or persistent and recurrent (Beardslee, Versage, & Gladstone, 1997), and longitudinal studies from early childhood to early adolescence are still scarce. Our results bring new evidence on the specific effect of chronic and stable depression in a four-assessment points study.

Whist the role of stable maternal depression in predicting offspring's psychopathology was expected (Middeldorp, Wesseldijk, Hudziak, Verhulst, Lindauer, & Dieleman, 2016), our results captured a second, interesting and unexpected result. In fact, our data also showed a robust relation between mothers' stable symptoms of paranoid ideation and high levels of Body Dissatisfaction, Drive for Thinness and Bulimia in early adolescents. Although further studies will be needed to clarify the mechanism linking maternal paranoid ideation and eating disorders in youths, we can propose a hypothesis: some of the items composing the paranoid ideation of the SCL-90-R could have captured some sub-clinical forms of maladaptive cognitions and behaviors typical of subjects

with problems with their body image. For example, the item "Feeling that you are watched by others", could be particularly connected to one of the primary symptoms of body dissatisfaction or dysmorphobia (Phillips *et alii*, 1995). Thus, mothers with these symptoms could in fact suffer difficulties with their body image, possibly resulting in preoccupation for their weight or appearance. These difficulties could have been intergenerationally been passed from mothers to their offspring and, in youths, they could have assumed the form of an eating disorder.

However, the present study shows also a series of limits that it is important to discuss. First of all, it was conducted exclusively on a community sample, without a comparison with a clinical group. Considering that the present study was focused on the role of children and mothers psychopathology for the development of eating disorders, a future natural extension may be carrying out other investigations recruiting also participants with more marked problems of mental health.

Second, mothers' symptoms have been assessed only with self-report measures, with a high risk of social desirability biases, considering that they were recruited in a community sample, and thus with a natural tendency to minimize eventual subclinical psychopathological characteristics. Furthermore, several studies have suggested that depression in mothers can lead to under- or over-estimating their children's psychological problems (Fergusson & Horwood, 1987; Breslau, Davis, & Prabucki, 1988).

A third limitation regards the use of Pearson's correlations to estimate the stability of children and mothers' symptoms over time. Indeed, in the last decades more refined data analyses techniques (e.g., *Latent State Trait Models*; Steyer, & Schmitt, 1990) have been proposed that permit, using longitudinal data, to distinguish stability, occasion specificity and error variance of a measure. In future studies, it may be important to apply one or more of these new statistical procedures to provide a more accurate estimate of occasion specificity and stability of children and mothers' symptoms.

Finally, we did not considered fathers and their possible maladaptive characteristics, possibly having a role in the onset of emotional-behavioral symptoms in their offspring. Indeed, it has been proved that fathers' symptoms can have an impact on their children's behavioral problems, also through assortative mating or contagion/interaction effects, with one parent's symptomatology interacting and mediating or moderating the other's (Jensen, Traylor, Xenakis, & Davis, 1988; Phares, Compas, & Howell, 1989).

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