Intergenerational Depression and Anxiety Disorders: A Network Analysis of Family Functioning and Depression/Anxiety Symptoms in Offspring

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Abstract

Introduction: Depression and anxiety are frequent disorders that commonly develop during youth. The offspring of parents with depression and/or anxiety present a high vulnerability to mental disorders. This study investigates the associations between family functioning and individual symptoms of depression/anxiety in offspring.

Method: Cross-sectional data were collected from 483 offspring (13 to 25 years) of parents treated for depression/anxiety. Family cohesion and flexibility were assessed with the Family Dimensions Scales, depression/anxiety symptoms in offspring were assessed with the DSM-IV questionnaire. A multiple linear regression examined associations of cohesion and flexibility with the number of symptoms in offspring, then logistic regressions were performed for each symptom. A mixed graphical network estimated the direct and indirect links between family functioning dimensions and individual symptoms.

Results: Cohesion was negatively related to total number of symptoms in offspring (ß = -0.13, $p = .007$), while flexibility was positively related to symptoms (ß = 0.33, $p < .001$). Flexibility also had significant individual associations with all symptoms. In the network structure, cohesion showed two negative (anhedonia and suicidality) and one positive link (insomnia), whereas flexibility showed two positive links (guilt and insomnia).

Discussion: High family cohesion has a protective role in offspring depression/anxiety, especially against anhedonia, sadness and worthlessness. Excessive family flexibility is detrimental to offspring, especially via guilt and sleeping problems that are further related to anxiety. Findings suggest that different family factors relate to distinct pathways of symptoms in the offspring and that family interventions should increase cohesion and reduce excessive flexibility.

Keywords: Family functioning, depression, anxiety, network analysis

Intergenerational Depression and Anxiety Disorders: A Network Analysis of Family Functioning and Depression/Anxiety Symptoms in Offspring

Depression and anxiety are frequent, chronic, and burdensome mental disorders (Baxter et al., 2014; Kessler & Bromet, 2013; Malhi & Mann, 2018). Both disorders are leading causes of impairment in mental health, accounting for considerable disruption of daily functioning, reduced quality of life, and high risk of suicide (Hawton et al., 2013; Lund et al., 2018; Malhi & Mann, 2018). The lifetime prevalence of depression and anxiety in Europe is estimated at about 15% and 14%, respectively (Alonso et al., 2004; Asselmann et al., 2019; Auerbach et al., 2018; Bromet et al., 2011), with comorbidity rates as high as 75% (Lamers et al., 2011). The development of depression and anxiety often occurs before or during early adulthood (Penninx et al., 2011), and thus young individuals are at a peak age of risk (Kessler et al., 2005; Moffitt et al., 2010). About 50% of people experience multiple episodes of depression over life or anxiety symptoms for several years, with high chances of recurrence (Kessler et al., 2012; Penninx et al., 2011; Spijker et al., 2002).

Despite extensive literature, much remains unclear about the complexity of mechanisms in depression and anxiety. Both disorders show high heterogeneity of symptoms, while models of psychopathology are oversimplified and unable to fully incorporate the clinical reality (Boschloo et al., 2015; Goodman, 2020). Simplistic models may also harm the efficacy of current prevention programs, which show suboptimal rates of 30% to 50% of disorder prevention in young people (Rasing et al., 2017; Stockings et al., 2016). The investigation of how different risk factors are related to specific symptoms of depression and anxiety is crucial for improving models and the efficacy of psychological interventions.

Intergenerational Depression and Anxiety

Parental history of depression and/or anxiety is a major risk factor for the development of mental disorders in offspring (Holzel et al., 2011). The offspring of parents

with depression/anxiety show two to three times higher incidence of disorders in comparison to the general population of young adults (Havinga, 2020; Rasic et al., 2014; Weissman et al., 2016). Most individuals exposed to parental disorder develop a psychiatric disorder themselves, with a cumulative incidence of 65% at the age of 35 years (Havinga et al., 2017). Nonetheless, prevention programs focused on children of parents with mood and anxiety disorders still show mixed results and short-lived effects (Havinga et al., 2021). Altogether, literature shows that the offspring of parents with depression or anxiety present a high vulnerability to developing mental health problems themselves, combined with suboptimal responses to interventions. While at an even higher risk during late adolescence and early adulthood, young offspring are a key population for research about family mechanisms of disorder maintenance across generations.

Family Functioning

Multiple pathways connect parental disorder to offspring maladaptation. Family environment is a major determinant of mental health through interrelated genetic, social and psychological mechanisms (Brennan et al., 2002; Stanton, 2009). Systemic approaches outline that depressed and anxious parents are less able to provide an organized environment (Timko et al., 2009), setting a cascade of psychosocial processes that contribute to both the onset and maintenance of offspring symptoms (Garbner & Cole, 2010). Indeed, the presence of parental depression and/or anxiety disrupts family functioning and harms the quality of parenthood (Krug et al., 2016). By mechanisms of negative cognitions, depressive affect, and impaired social interaction, parental disorder harms the ability to provide support and fulfill the emotional needs of children (Downey & Cowney, 1990; Goodman & Gotlib, 1999; Havinga, 2020). Moreover, depressed and anxious parents more often display dysfunctional behaviors towards their offspring, including low autonomy granting, low parental monitoring, and inconsistent discipline (Yap et al., 2014a). As the number of dysfunctional parental

behaviors increases, the prevalence of psychiatric disorders in offspring also increases (Johnson et al., 2001). Noteworthy, parental episodes of depression have enduring negative effects on the parental-offspring relationship that persist for many years beyond the remission of symptoms (Herr et al., 2007; Timko et al., 2002). Overall, behavioral and cognitive factors are intertwined in producing a chain of environmental family risks that mediate the negative effects of parental disorder on the mental health of their offspring (Havinga, 2020; Natsuaki et al., 2014).

The Circumplex Model (Olson, 1986) is a framework that conceptualizes dimensions of family functioning according to the quality of intrafamilial relationships. The model proposes that unbalanced levels of cohesion and flexibility, either too high or too low, correspond to problematic functioning (Olson, 2011). Cohesion refers to the emotional connection between family members, while flexibility refers to the internal rules, leadership roles and negotiations. Empirical studies show associations between the dimensions of family functioning and offspring adjustment. High family cohesion is related to psychological well-being (Manzi et al., 2006; Tramonti et al., 2020; Vandeleur et al., 2009), while low cohesion levels are reported in families of children with depression (Kashani et al., 1995). Particularly, high family flexibility combined with low cohesion levels may represent a maladaptive system of disengagement and low parental monitoring that can be highly harmful to adolescents (Everri et al., 2016). Mainly, unbalanced family functioning is more frequent in families of depressed or anxious parents (Nomura et al., 2002; Timko et al., 2002), thus suggesting family functioning as an important pathway that connects parental disorder to the presence of depression/anxiety symptoms in the offspring.

Networks of Symptoms

Research on family risk factors for symptoms of mental disorders is still restricted due to limitations in theory and methodology. It is not clear in the literature how parental disorder

represents differential susceptibility for specific symptoms in offspring (Natsuaki et al., 2014). In addition, the widespread “latent variable” approach to mental health fails to translate the clinical heterogeneity of disorders into classification systems (Fried et al., 2014). Depression is commonly assessed with summative scores on questionnaires that consider symptoms interchangeable or equivalent (Boschloo et al., 2015). Core symptoms (depressed mood and anhedonia) are then summed to other cognitive, behavioral, and neuropsychological alterations. Nonetheless, a pronounced variability in symptom presentation is observed in clinical practice, suggesting that depression is not a consistent unitary disorder (Fried & Nesse, 2015). There are hundreds of possible combinations of symptoms presentation and some of the symptoms are highly heterogeneous themselves, either being present in form of hyper or hypofunction (e.g., insomnia or hypersomnia; Buch & Liston, 2021). The same methodological issues are also observed in the assessment of anxiety disorders (Cramer et al., 2010). Current one-factor models seem unable to capture the complex nature of depression and anxiety and may ignore valuable information for mapping mechanisms of symptom development (Boschloo et al., 2015; Buch & Liston, 2021).

A novel conceptualization of mental disorders proposes a symptom-specific approach rather than the use of sum scores (Borsboom et al., 2018). Accordingly, network analysis addresses psychopathology as domains of causal interplay between symptoms that are unique and not interchangeable (Contreras et al., 2019). Network studies about depression and anxiety have already unfolded some associations between different risk factors and specific symptoms. For example, experiencing childhood stress, a traumatic event or a previous disorder episode predict different sets of depressive symptoms (Fried et al., 2014). One study of suicide motives by Shiratori and colleagues (2014) has identified depression as a central reason, which in turn was closely related to family dysfunction and conflicts. Findings illustrate the value of the network approach in showing multiple direct and indirect

connections between specific symptoms and risk factors, contributing to disentangling the complexity of mechanisms that lead to depression and anxiety.

Nonetheless, network analysis is still a novel approach and many subpopulations have not yet been investigated with this technique. Available network research of parental disorders so far has prioritized maternal postpartum depression (e.g., Phua et al., 2020; Zhang et al., 2021). Few network studies directly investigated family relationships and no network study was found including data from both parents and offspring. The current literature is thus insufficient to clarify the structure of connections between family factors and specific symptoms of depression/anxiety in offspring. Applying specified family risk factors to identify individuals at risk for developing particular symptoms of mental disorders can be a key strategy for improving early interventions and symptom-focused prevention programs.

The Current Study

This study aims to investigate the role of family functioning in relation to symptoms of depression and anxiety in the offspring participating in the baseline assessment of ARIADNE (Adolescents at Risk of Anxiety and Depression: Combined Neurobiological and Epidemiological approach). The study comprises 522 offspring of patients in treatment for depression/anxiety disorder in the Netherlands. Two hypotheses are tested: a) low family cohesion is associated with a higher number of symptoms in offspring, and b) high family flexibility is associated with a higher number of symptoms in offspring. In addition, a network is estimated to verify symptom-specific associations with family functioning.

Method

Study Design and Participants

ARIADNE is a cohort study including 522 offspring (13-25 years; 57.3% girls) of patients (32-60 years; 68.8% women) who received treatment for depression or anxiety disorders in the previous ten years (Landman-Peeters et al., 2005). Parents were selected

from 16 psychiatric services in The Netherlands between 2000 and 2002. Based on medical record screening, inclusion criteria were a) being diagnosed and treated for at least one episode of depressive (90.8% of the sample) or anxiety (73.0% of the sample) disorder, b) having biological children aged between 13 and 25 years. Parents were interviewed for Major Depressive Disorder, Dysthymia, Panic Disorder and Agoraphobia with the Composite International Diagnostic Interview (CIDI; Kessler & Üstün, 2004) based on the Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). The CIDI is a structured interview that provides valid diagnoses and is well-accepted among Dutch psychiatric patients (de Graaf et al., 2004; Reed et al., 1998). Parents were excluded if they presented a diagnosis of schizophrenia or post-traumatic stress disorder. ARIADNE received approval from the Medical Ethical Committee of the University Medical Center Groningen. The current study was registered and exempted from full revision by the Ethical Review Board of the Faculty of Social and Behavioural Sciences of Utrecht University.

This study focuses on cross-sectional data from the baseline assessment of ARIADNE. Of the 522 offspring, 39 (7.5%) were excluded for missing data on key variables, resulting in a sample of 483 offspring for the current analyses. The mean age was 18.2 (*SD* = 3.2) years and 276 (57.1%) participants were girls.

Procedure

Parents were contacted at mental health services by local psychologists. An information letter presented the study goals and the measurement procedures. Those who agreed in participating were contacted by the research team and provided a term of consent at the first appointment, to be signed by parents and offspring. All participants filled in questionnaires and were interviewed individually by trained researchers at home or clinical facilities. Participants were allowed to withdraw from participating at any time, without any implications for their regular mental health care.

Materials

Family Functioning

Family functioning was measured with the Dutch Family Dimensions Scales (FDS; Buurmeyer & Hermans, 1985), in which the offspring participants reported perceptions about family relationships The FDS is adapted from the Family Adaptability and Cohesion Evaluation Scales (FACES; Olson, 1986). Answers to each item were recorded on a four-point scale, from one (*Never true*) to four (*Always true*). The Cohesion scale consists of 23 items assessing the degree of closeness and emotional connection between family members. The cohesion score is calculated by subtracting negatively worded items (e.g. "Our family members feel lonely at home") from positively worded items ("We prefer fun in the family") and then adding 75, resulting in a scale that ranges from 23 (minimal cohesion) to 92 (maximal cohesion). Scores are further classified as Disengaged (23 to 62), Individual (63 to 68), Connected (69 to 75), and Enmeshed (76 to 92). The Flexibility scale refers to the family's ability to adapt rules and power structures to circumstances, assessed with 13 items. Negatively worded items (e.g. "In our family the rules are constantly changing.") are subtracted from positively worded items (e.g. "Once someone takes a task, that remains their task.") and then ten is added to the sum. The flexibility score ranges from 13 (minimal flexibility) to 52 (maximal flexibility). Four classifications are possible: Rigid (13 to 19), Structured (20 to 24), Flexible (25 to 28), or Chaotic (29 to 52). The FDS showed Cronbach's alpha of .68 for the Cohesion scale and .72 for the Flexibility scale, values considered moderate but acceptable for internal consistency (Pallant, 2001).

Symptoms of Depression/Anxiety in the Offspring

Selected items of the DSM-IV questionnaire (Hartman, 2002; Hartman et al., 2001) were used to assess key symptoms of depression and anxiety in the offspring: feeling sad, feeling hopeless, feeling worthless, feeling guilty, lack of energy or tiredness, anhedonia,

retardation, suicidal thoughts, early night insomnia, middle night insomnia, sleep disturbance, excessive worry, feeling tense/nervous, panic, and indecisiveness – all experienced at the time of measurement. Examples of items are “I am often unhappy” and “I feel tired or low in energy for no reason”. Answers were provided on a four-point Likert scale and coded in 15 binary items representing the presence of each symptom. The answer *Not at all* was recoded as “not present”, while the answers *Several days*, *Half of the days* and *Nearly every day* were recorded as “present”. The total number of symptoms present was also calculated and showed a Cronbach’s alpha of .86, which represents very good internal consistency (Pallant, 2001).

Data Analysis

Analyses were performed in multiple steps with the software SPSS Statistics version 26 and R Studio. First, outliers were checked with boxplots and assumptions were verified for the statistical models of regression, applying tests for multicollinearity, linearity, normality, and homoscedasticity.

To assess the association between dimensions of family functioning and the number of symptoms in offspring, a multiple linear regression was performed including cohesion and flexibility scores (independent variables) and the number of symptoms in offspring (dependent variable). Considering the differences in both scales, standardized scores of cohesion and flexibility were calculated and applied for the analyses.

The next analyses zoomed in on the association of family functioning with the presence of individual symptoms. Logistic regression analyses were applied to examine the associations of cohesion and flexibility scores with each individual symptom in offspring, in a total of fifteen models. An interaction term between cohesion and flexibility was also included in further multivariable logistic regressions, to verify effect modifications among participants who simultaneously present both family dimensions with either low or high values. Crosstabulations of each individual symptom across the categorical levels of cohesion

(Disengaged, Individual, Connected or Enmeshed) and flexibility (Rigid, Structured, Flexible or Chaotic) were also included.

To take into account the interrelatedness of symptoms, a network was estimated including the two dimensions of family functioning and all individual symptoms. The network was estimated with the mgm package (Halsbeck & Waldorp, 2020) including individual symptoms as binary items (0 = not present, 1 = present) and continuous cohesion and flexibility scores. The mgm package generates a mixed graphical model that combines generalized linear models and neighborhood selection; the model is visualized with the qgraph package (Epskamp et al., 2012). A fixed layout was applied displaying cohesion and flexibility on the left, directly connected symptoms as nodes in the center, and indirectly connected symptoms as nodes on the right. The link between one family functioning dimension and one individual symptom indicates a direct association that is independent of the other dimension and any of the other symptoms. The indirect links from family dimensions to symptoms via other specific symptom represent indirect association pathways. Lines displayed positive (green) and negative correlations (red) and different association strengths (thickness of the line between nodes).

Results

Sample Characteristics

The average cohesion score was 63.3 (*SD* = 8.4) and the average flexibility score was 23.3 (*SD* = 5.5). Disengaged was the most frequent classification in family cohesion (41.6%), while structured was the most frequent group in family flexibility (37.1%, see Table 1). Offspring participants reported an overall mean of 5.8 symptoms (*SD* = 4.1) of depression or anxiety. Prevalence rates differed substantially across symptoms, ranging from 15.9% for suicidal thoughts to 75.4% for excessive worry (see Table 2).

Table 1

Frequency of Groups According to Family Cohesion and Flexibility

	n	%
Cohesion		
Disengaged	201	41.6
Individual	152	31.5
Connected	104	21.5
Enmeshed	26	5.4
Flexibility		
Rigid	133	27.5
Structured	179	37.1
Flexible	87	18.0
Chaotic	84	17.4

Table 2

Frequency of Depression/Anxiety Symptoms in Offspring

	n	%
Excessive worry	364	75.4
Feeling guilty	282	58.4
Indecisiveness	250	51.8
Feeling sad	236	48.9
Lack of energy or tired	235	48.7
Early night insomnia	190	39.3
Retardation	176	36.4
Feeling tense / nervous	174	36.0
Feeling worthless	169	35.0
Sleep disturbance	162	33.5
Feeling hopeless	148	30.6
Anhedonia	134	27.7
Panic	122	25.3
Middle night insomnia	98	20.3
Suicidal thoughts	77	15.9

Prior to the main analyses, assumptions for regression models were tested. Cohesion and flexibility showed no multicollinearity (r = - .30, p < .001). Analysis of a scatterplot matrix indicated linearity between cohesion and number of symptoms, as well as between flexibility and number of symptoms. Random distribution of residuals around zero in the plot of standardized residuals indicated homoscedasticity. A positively skewed distribution was observed in the variable number of symptoms. The Shapiro-Wilk test showed non-normality (W(483) = .95, p < .001), with kurtosis of – 0.86 (*SE* = 0.22) and skewness of 0.37 (*SE* = 0.11). These values of kurtosis and skewness do not cross a threshold that represents a violation of normality, especially in large sample sizes (Hair et al., 2010). Thus, normality was assumed for the dependent variable. No outliers were identified in any of the variables. A posthoc power analysis indicated an achieved power of 100% for the observed effect size (f^2 = .20) in the multiple regression, given the current sample size.

Associations of Family Functioning with Number of Symptoms

A multiple linear regression was performed to examine whether family cohesion and flexibility scores were linearly related to the number of depression/anxiety symptoms. In line with the first hypothesis, cohesion was negatively related to the number of symptoms (t = - 2.70, ß = - 0.13, p = .007). In accord with the second hypothesis, flexibility was positively related to the number of symptoms (t = 6.72, ß = 0.33, p < .001). The model explained 16.0% of variance in number of symptoms.

Associations of Family Functioning with Individual Symptoms

Multivariable logistic regressions were performed to examine the individual associations of standardized scores of cohesion and flexibility with each symptom (see Table 3). Cohesion was negatively related to seven symptoms (p varying from < .001 to .048), namely anhedonia, suicidality, lack of energy, retardation, sadness, hopelessness and worry – the odds ratios varied from 0.50 for anhedonia to 0.77 for worry. Flexibility was positively

related to 14 symptoms (p varying from <.001 to .029), except for lack of energy (p = .125). The odds ratios differed substantially in the significant associations with flexibility, from 1.34 for excessive worry to 1.87 for early night insomnia.

Table 3

Association of Family Dimensions (Standardized) with the Presence of Individual Symptoms

	Cohesion		Flexibility	
	OR	sig	OR	sig
Excessive worry	0.77	.048*	1.34	.029*
Feeling guilty	0.90	.365	1.59	<.001**
Indecisiveness	0.99	.891	1.41	.002*
Feeling sad	0.75	.012*	1.64	<.001**
Lack of energy or tired	0.73	.004*	1.18	.125
Early night insomnia	1.20	.099	1.87	<.001**
Retardation	0.75	.010*	1.37	.005*
Feeling tense/nervous	1.00	.972	1.48	<.001**
Feeling worthless	0.81	.069	1.75	<.001**
Sleep disturbance	0.95	.637	1.67	<.001**
Feeling hopeless	0.76	.021*	1.64	<.001**
Anhedonia	0.50	<.001**	1.39	.006*
Panic	0.90	.380	1.54	<.001**
Middle night insomnia	1.11	.435	1.70	<.001**
Suicidal thoughts	0.65	.003*	1.47	.005*

Note. * $p < .05$, ** $p <.001$

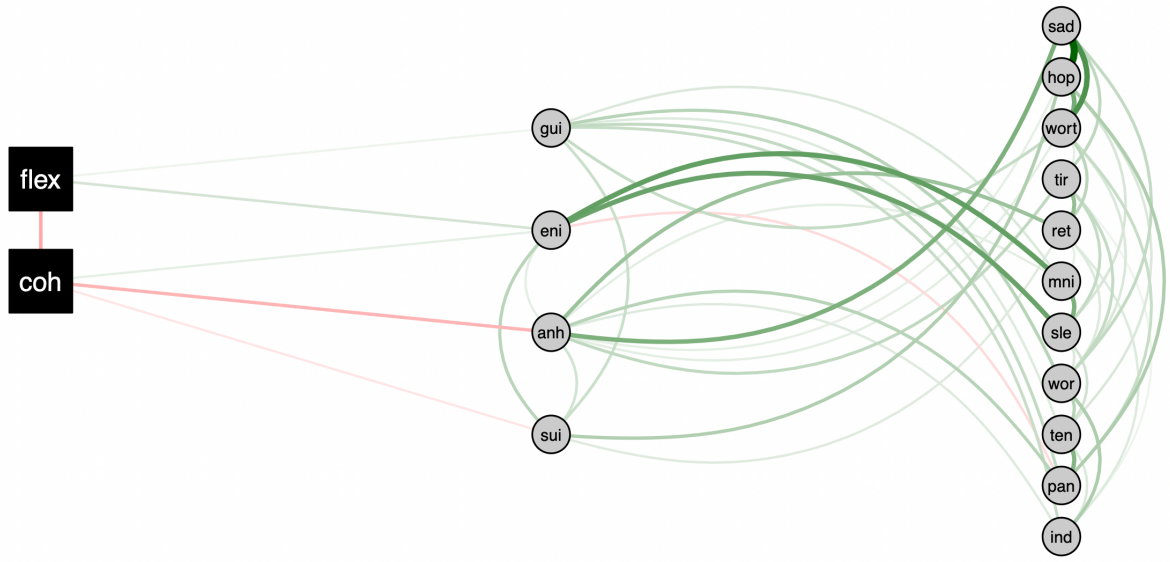
A set of sensitivity analyses were performed to investigate the individual associations with symptoms across the norm groups of family functioning (see Supplemental Table S1). Linear trends were found for all symptoms. In cohesion groups, disengaged families showed the highest prevalence of all symptoms. For flexibility, chaotic families had the highest prevalence of 13 symptoms. Additionally, no significant results were found for an interaction between cohesion and flexibility in relation to the total number of symptoms (t = 1.01, ß = 0.24, p = .312) or any of the individual symptoms (all $p \geqq 0.90$; see Supplemental Table S2).

Network Structure Estimation

The network structure of family cohesion and flexibility with all individual depression and anxiety symptoms in the offspring is displayed in Figure 1. The dimensions of cohesion and flexibility were negatively associated, as also observed in the previous analyses. Mainly, the network structure showed that high cohesion related to having fewer symptoms whereas high flexibility related to having more symptoms; that is, cohesion showed two negative and one positive connection, whereas flexibility showed two positive connections with individual symptoms. All nodes were interconnected, directly or indirectly, showing positive associations across all symptoms with the only exception of panic and early night insomnia.

Figure 1

Network Estimation of the Structure of Family Functioning and Symptoms in Offspring



Note. Green lines indicate positive associations, while red lines indicate negative associations. Bold lines indicate stronger connections. Symptoms of anxiety and depression are feeling sad (sad), feeling hopeless (hop), feeling worthless (wor), feeling guilty (gui), lack of energy or tired (tir), anhedonia (anh), retardation (ret), suicidal thoughts (sui), early night insomnia (eni), middle night insomnia (mni), sleep disturbance (sle), excessive worry (wor), feeling tense/nervous (ten), panic (pan), indecisiveness (ind).

Potential pathways through which cohesion and flexibility relate to individual symptoms were also analyzed. Cohesion showed the strongest direct negative connection with anhedonia, which in turn was positively associated with feeling sad, retardation, feeling tired, panic, suicidal thoughts, middle night insomnia, indecisiveness, feeling hopeless, and feeling worthless. In addition, cohesion showed a weaker negative connection with suicidality, which in turn had a positive association with feeling worthless, retardation, feeling guilt, and anhedonia. In contrast, cohesion showed a weak positive connection with early night insomnia, which in turn had a positive association with middle night insomnia and sleep problems, and a negative association with panic (see also Supplemental Figure S1).

Flexibility was positively related to guilt, which in turn was positively associated with worry, panic, indecisiveness, feeling worthless, suicidal thoughts, and tension. Moreover, flexibility had a weak positive association with early night insomnia, which in turn had a positive association with middle night insomnia and sleep problems, and a negative association with panic (see also Supplemental Figure S2).

Discussion

Main Findings

The purpose of this study was to investigate family functioning and symptoms of depression/anxiety in the offspring of parents treated for depression and/or anxiety. Analyses indicated four key findings. First, family cohesion had a negative linear relationship with symptoms of depression/anxiety in offspring – as initially predicted. Second, family flexibility was positively and linearly related to symptoms – in line with the initial hypothesis – and this association was even stronger than the one of family cohesion. Third, the symptom-specific analyses indicated positive associations between family flexibility and almost all symptoms in offspring, whereas cohesion was related to half of the symptoms. Fourth, the network structure of symptoms showed that the two dimensions of family

functioning were linked to all symptoms in offspring, through distinct pathways. There were positive associations, directly or indirectly, across almost all symptoms, which represents that having one type of symptom increases the chances of experiencing also other symptoms.

Family Cohesion as a Protective Factor

The results support the hypothesis that lower levels of family cohesion are associated with a higher number of depression/anxiety symptoms in offspring. The data replicate findings from the literature that family disengagement is associated with negative mental health outcomes. High family cohesion has been reported as a factor that increases psychological well-being (Manzi et al., 2006; Tramonti et al., 2020; Vandeleur et al., 2009) and facilitates the ability of parents to provide emotional support to children (Farrell & Barnes, 1993; Havinga, 2020). A possible explanation for such effects is the connection between family cohesion and the quality of parental attachment, from which the offspring can derive a sense of emotional security (Cummings et al., 2002). Noteworthy, parental depression/anxiety impairs affective interactions with the offspring. Such dysfunctional settings increase parent-offspring relational stress and thus affect the mental health of offspring (Landman-Peters, 2007).

Disengaged families showed the highest prevalence of all individual symptoms across the cohesion categories. Low cohesion was related to an increased risk of presenting anhedonia, suicidality, lack of energy, retardation, sadness, hopelessness, and worry. Network findings showed that family cohesion had a strong negative connection with anhedonia and suicidal thoughts, suggesting a protective role of family cohesion against important depressive symptoms. As previously noted, family cohesion is important for secure attachment (Cummings et al., 2002), which allows a sense of safety in future interpersonal relationships (Bartholomew & Horowitz, 1991). Via emotional connectedness, high family cohesion can reduce the transition from suicide ideation to a suicide attempt in adolescents

(Sun et al., 2020). A poor parental attachment, instead, impairs the fulfillment of interpersonal needs, contributing to both anhedonia and suicidal ideation in adolescents (Guo et al., 2021). Furthermore, the estimated network showed indirect links, via anhedonia and suicidality, between low cohesion and important depressive symptoms such as sadness, hopelessness, and worthlessness. The current findings endorse that low support from family correlates with such dysfunctional emotional responses and maladaptive schemas (Cohen & Wills, 1985; Langhinrichsen-Rohling et al., 2017) and further suggest that indirect symptoms likely occur by means of suicidal thoughts and decreased pleasure in life.

Family Flexibility as a Risk Factor

The results also supported the hypothesis that high family flexibility is associated with more symptoms in the offspring. The finding is in line with reviewed literature, which reported harmful effects of excessive family flexibility (Kerr et al., 2012). Theories about the role of family structure in increasing the levels of well-being focus on the importance of parental monitoring and parental knowledge, which are highly protective factors during youth (Everri et al., 2016). Parents with depression/anxiety frequently display the opposite: low parental monitoring in a disorganized family environment (Konac et al., 2021; Timko et al., 2009). A weak family structure also contributes to family instability, which in turn increases insecurity and psychological maladjustment in the offspring (Gregory & Sadeh, 2016).

Chaotic families had the highest prevalence in 13 of the 15 depression/anxiety symptoms and high flexibility was related to an increased risk of presenting all symptoms individually, except for lack of energy. In the network analysis, family flexibility had positive connections to insomnia and guilt. The association with insomnia likely reflects inconsistent enforcement of rules that is common in families with high flexibility (Yap et al., 2014a). A parental set of bedtime rules is an important factor that predicts the duration of sleep among adolescents, which in turn affects psychological arousal and behavioral control (Khor et al.,

2021). Families with excessive flexibility seem less able to establish a healthy sleep routine. In parallel, feelings of guilt may follow detachment from family. Adolescents who experience parental neglect frequently have ambivalent feelings toward their caregivers, which leads to self-blame, shame and guilt (Sekowski et al., 2020). Insomnia and guilt were further associated with worry and tension, suggesting a risk for anxiety in families with excessive flexibility that occurs via sleeping problems and guilt. Most recent reviews indicate robust evidence for sleep problems as precursors to anxiety in adolescence (Kelly & El-Sheikh, 2014; Willis & Gregory, 2015), due to disrupted emotional memory consolidation in sleep (McMakin & Alfano, 2015). Moreover, guilt increases anxiety for those with an exaggerated feeling of responsibility (Cândea & Szentagotai-Tătar, 2018), which may likely occur in the absence of a solid family structure. These findings highlight the importance of establishing adequate family rules to reduce feelings of worry in offspring (Yap et al., 2014b), and thus reduce the risk of mental disorders.

Theoretical and Practical Considerations

Whereas most findings are in line with reviewed literature, some results imply inconsistencies in theories of family functioning. Linear associations were found between dimensions of family functioning and symptoms in offspring, while the Circumplex Model proposes that maladjustment occurs in a U-shaped relationship (Olson, 2011). Previous empirical studies also did not find an increased risk in either low family flexibility or high family cohesion (Manzi et al., 2006; Vandeleur et al., 2009; Tramonti et al., 2020). Although the observed low rates of symptoms in rigid families seem initially controversial, the term rigidity (i.e., low flexibility) is arguably ambiguous and may be unfitting for a scale that correlates with family satisfaction (Everri et al., 2016). The wording of items in the FDS partially suggests that high flexibility is a detrimental feature (e.g., “In our family the rules are constantly changing.”, “Our house looks like a ship without a captain.”). Instead, the term

"family structure" seems more fitting for the measured constructs. Models of family functioning must be constantly revised to correctly depict family dynamics and accommodate shifts in cultural values (Hamilton & Carr, 2016), thus current remarks can contribute to advancing the Circumplex Model and its dimensions.

Some limitations warrant acknowledgment. First, the use of cross-sectional data does not allow causal conclusions. Whereas dimensions of family functioning were related to symptoms of anxiety/depression in offspring, it is plausible that the presence of such symptoms reciprocally affects the quality of family relationships. Alternative explanations also cannot be ruled out. Follow-up studies can expand current analysis with a longitudinal investigation of the cohort to shed light on the directionality and potential long-term effects on mental health outcomes in late adulthood. A replication of the study with the offspring of current participants in a few years would be particularly relevant for analyzing the presence of symptoms across three generations. Second, despite the strengths provided by network analyses, the symptom-specific approach to mental disorders is still a largely data-driven non-confirmatory model of psychopathology. While allowing more sophisticated statistical fitness for empirical data, the lack of experimental evidence impairs the translation of network findings into generative theories of mental disorders. Therefore, experimental manipulations of cohesion and flexibility levels, such as pretest-treatment-posttest family intervention compared to controls, are recommended to expand network models.

Despite the limitations, current findings have relevant implications for prevention and clinical practices. The suggested pathways of symptoms can be applied to improve prevention programs for youth by addressing key family risk factors, namely low cohesion and excessive flexibility, before the development of full-blown episodes of disorders. Since depression and anxiety often develop during youth and follow a chronic pattern (Penninx et al., 2021; Buckman et al., 2018), it is essential to address dysfunctional family environments

in early offspring ages to break the cycle of transgenerational transmission before the disorder onset. In clinical interventions, a specific risk and symptom approach can advance family treatment for adolescents and young adults with depression and anxiety. Families with low cohesion can benefit from therapeutic techniques focused on improving relationship warmth, affection expression, and parent-offspring involvement. In turn, families with excessive flexibility can receive psychoeducation and skills training for establishing adequate discipline and good sleep habits (Khor et al., 2021; Restifo & Bögels, 2009; Yap et al., 2014b). Future research can investigate whether prevention strategies targeting family functioning would reduce symptoms in the offspring with research designs that allow observing such "cascade" effects. This can be achieved, for example, with weekly monitoring of each symptom during family interventions that aim to increase cohesion and reduce excessive flexibility.

Conclusions

In conclusion, this study suggests a protective role of high family cohesion for depression/anxiety symptoms in offspring, while high family flexibility displays the opposite association. Findings showed that differential family risk factors are related to different pathways of symptoms in offspring. The evidence contributes to a growing body of network analyses that map how specific symptoms connect and interplay in mental disorders. Implications can be translated into prevention and treatment programs that explicitly target family cohesion and flexibility to improve the mental health of offspring.

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Appendix A

Supplemental Table S1

Presence of Symptoms in Offspring Across the Family Functioning Groups

	Cohesion				Flexibility			
	1	2	3	4	1	2	3	4
	Disengaged	Individ.	Connected	Enmeshed	Rigid	Structured	Flexib.	Chaotic
Worry	**83.1%**	72.4%	68.3%	61.5%	66.9%	73.7%	81.6%	**85.7%**
Feeling guilty	**65.7%**	50.7%	56.7%	53.8%	42.1%	60.3%	62.1%	**76.2%**
Indecisiveness	**57.7%**	46.7%	48.1%	50.0%	45.9%	45.3%	60.9%	**65.5%**
Feeling sad	**57.7%**	47.4%	36.5%	38.5%	30.1%	51.4%	50.6%	**71.4%**
Lack of energy	**57.2%**	49.3%	34.6%	34.6%	38.3%	48.0%	54.0%	**60.7%**
Early insomnia	**43.8%**	36.2%	38.5%	26.9%	27.8%	34.6%	48.3%	**58.3%**
Retardation	**45.8%**	34.2%	25.0%	23.1%	24.8%	31.3%	**52.9%**	48.8%
Feeling tense	**38.8%**	34.2%	36.5%	23.1%	24.8%	34.1%	43.7%	**50.0%**
Worthlessness	**39.3%**	28.3%	18.3%	26.9%	21.8%	30.2%	42.5%	**58.3%**
Sleep disturb.	**39.3%**	29.4%	30.8%	23.1%	22.6%	27.9%	43.7%	**52.4%**
Hopelessness	**39.3%**	28.3%	18.3%	26.9%	15.8%	29.1%	34.5%	**53.6%**
Anhedonia	**42.3%**	23.0%	10.6%	11.5%	13.5%	23.5%	40.2%	**46.4%**
Panic	**28.4%**	26.3%	19.2%	19.2%	18.0%	19.0%	32.2%	**42.9%**
Mid. insomnia	**23.9%**	18.4%	17.3%	15.4%	11.3%	17.9%	**31.0%**	28.6%
Suicidality	**24.9%**	11.8%	7.7%	3.8%	6.8%	13.4%	24.1%	**27.4%**

Note. In **bold**: group with the highest prevalence for each symptom.

Appendix B

Supplemental Table S2

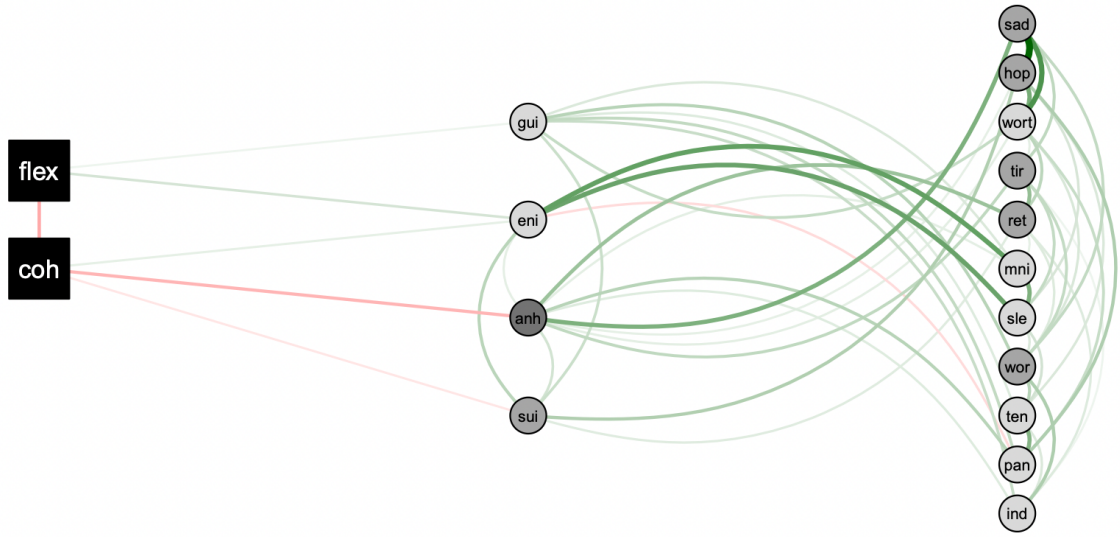
Effect Modification (Standardized) for Interaction Between Cohesion and Flexibility by Symptom

	Interaction between family cohesion and flexibility	
	OR	sig
Excessive worry	1.11	.313
Feeling guilty	0.95	.614
Indecisiveness	1.12	.185
Feeling sad	0.98	.900
Lack of energy or tired	1.16	.081
Early night insomnia	1.07	.428
Retardation	1.12	.217
Feeling tense/nervous	1.14	.150
Feeling worthless	1.01	.958
Sleep disturbance	1.05	.584
Feeling hopeless	1.06	.958
Anhedonia	1.03	.584
Panic	1.02	.842
Middle night insomnia	1.18	.128
Suicidal thoughts	1.21	.090

Appendix C

Supplemental Figure S1

Network Estimation Displaying the Strength of Association between Symptoms and Cohesion

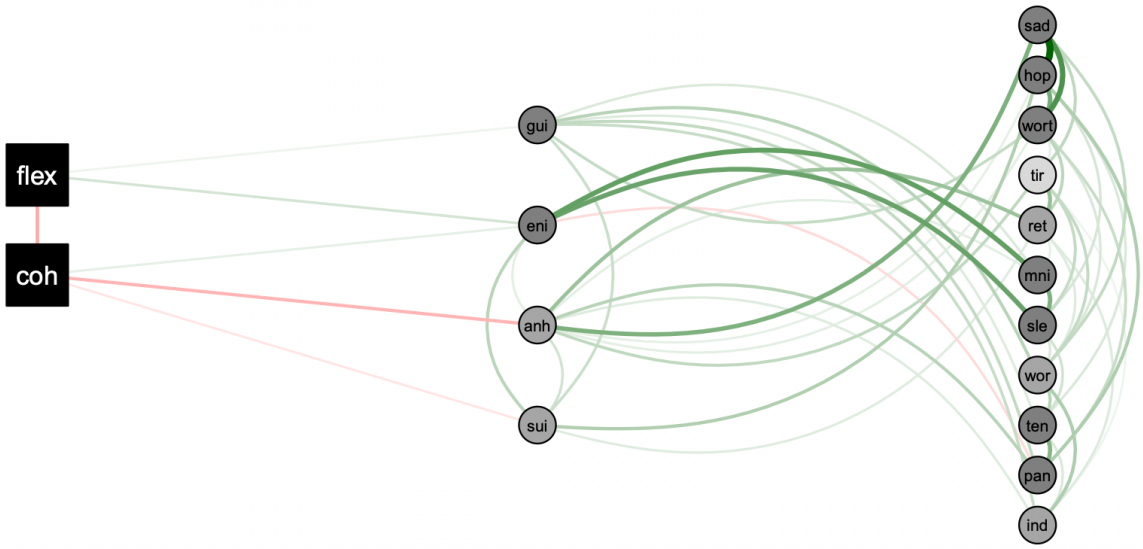


*Note***.** Grayscale in the nodes indicates the strength of the association of family cohesion with each symptom (darker = stronger connection).

Appendix D

Supplemental Figure S2

Network Estimation Showing the Strength of Association between Symptoms and Flexibility



*Note***.** Grayscale in the nodes indicates the strength of the association of family flexibility with each symptom (darker = stronger connection).