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Original article

Spousal resemblance in psychopathology: A comparison of parents of children with and without psychopathology

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ABSTRACT

Background: Spouses resemble each other for psychopathology, but data regarding spousal resemblance in externalizing psychopathology, and data regarding spousal resemblance across different syndromes (e.g. anxiety in wives and attention deficit/hyperactivity disorder [ADHD] in husbands) are limited. Moreover, knowledge is lacking regarding spousal resemblance in parents of children with psychiatric disorders. We investigated and compared spousal resemblance within and across internalizing and externalizing symptom domains in parents of children with and without psychopathology.
Methods: Symptoms of depression, anxiety, avoidant personality, ADHD, and antisocial personality were assessed with the Adult Self Report in 728 mothers and 544 fathers of 778 children seen in child and adolescent psychiatric outpatient clinics and in 2075 mothers and 1623 fathers of 2784 children from a population-based sample. Differences in symptom scores and spousal correlations between the samples were tested.
Results: Parents in the clinical sample had higher symptom scores than in the population-based sample. In both samples, correlations within and across internalizing and externalizing domains of psychopathology were significant. Importantly, correlations were significantly higher in the clinical sample ($P = 0.03$). Correlations, within and across symptoms, ranged from 0.14 to 0.30 in the clinical sample and from 0.05 to 0.23 in the population-based sample.
Conclusions: This large study shows that spousal resemblance is not only present within but also across symptom domains. Especially in the clinical sample, ADHD symptoms in fathers and antisocial personality symptoms in mothers were correlated with a range of psychiatric symptoms in their spouses. Clinicians need to be alert of these multiple affected families.

1. Introduction

There are several important clinical implications of spousal resemblance in psychopathology, as already discussed by Galbaud du Fort, Bland [1]. Firstly, due to the heritability of psychiatric disorders, children of parents with psychiatric symptoms have a higher risk to develop psychopathology. Furthermore, the family environment might become more unfavorable for a child when both parents suffer from psychiatric symptoms. In addition, parental psychopathology may also negatively affect the course and outcome of the treatment for a child with psychiatric symptoms [2–5]. Having two parents with psychopathology, as opposed to one, might have an even greater impact on the child's

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treatment effects [6]. Still, knowledge is lacking regarding spousal resemblance within, but especially across internalizing and externalizing symptom domains, in parents of children that are evaluated at a child and adolescent psychiatry outpatient clinic.

So far, resemblance between spouses has been frequently observed for mood disorders, in particular depression or related traits [7–9]. Other psychiatric disorders or traits, such as anxiety disorders or symptoms, alcohol or substance use, and antisocial or borderline personality traits have been less extensively investigated, but showed similar results [10–19]. Spousal resemblance across disorders has also been reported, e.g. for major depression and alcohol abuse [1,8,17,19–21], as well as asymmetry with respect to sex. For example, maternal anxiety has recently been found to be associated with paternal attention deficit hyperactivity disorder (ADHD) [22], but paternal anxiety not with maternal ADHD. Furthermore, major depression in wives was associated with antisocial personality disorder in husbands but not vice versa [1,11]. However, most of the studies investigating spousal resemblance across psychiatric disorders in clinical samples were restricted to only two or three diagnoses. Also, ADHD has been understudied, since it has only recently been acknowledged that ADHD can persist into adulthood [16,23]. Moreover, in a very large study in the entire Danish population between 18 and 70 years (n = 3,204,633), the risk to get a psychiatric disorder was increased in all spouses of individuals with a psychiatric disorder, but incidence rate ratios were more than twice as large among individuals with schizophrenia compared to other more mild psychiatric disorders [19]. Studies in other population-based samples have reported spousal resemblance by estimating correlations between and across traits in husbands and wives and these tend to be around 0.1 to 0.2 [14–16,18,21]. In clinical samples, spousal correlations range between 0.2 and 0.4 [10,22,24]. Clinical studies, however, mostly report odds ratios to quantify resemblance, making a direct comparison of the results of clinical and population-based samples difficult. Still it is hypothesized that spousal resemblance might, in general, be more prevalent for the more severe psychiatric disorders [19]. This hypothesis, though, has not yet been statistically tested and confirmed.

In the current study, we investigated spousal resemblance for psychiatric symptoms in a large sample of parents of children with psychopathology and for the first time statistically compared spousal resemblance in this clinical population to spousal resemblance in a population-based sample. We investigated a broad range of symptom domains, including ADHD and antisocial personality problems, which have been less often investigated in spousal resemblance studies than mood disorders. We hypothesized that, for all symptom domains, spousal resemblance will be higher in the clinical population following the earlier observations of higher spousal resemblance in clinical samples or for the more severe psychiatric disorders [10,19,22,24]. In an at-risk clinical sample, i.e. parents of children with psychopathology, there are several reasons why spousal resemblance for psychiatric symptoms may be higher than in a population-based sample. First, spouses might both be exposed stress, such as having a child suffering from psychiatric symptoms or experiencing financial problems, which can increase the risk for psychiatric symptoms in parents. Another possibility is that due to the heritability of psychiatric symptoms, the parents of children with psychopathology are more vulnerable to develop psychiatric problems themselves. Finally, parents who suffer from psychiatric symptoms might more easily recognize these symptoms in their partner or children and seek treatment. As having two parents afflicted with psychopathology, as opposed to one, can have a greater impact on the child's treatment effects, it is important for clinicians to know the patterns of the, possibly higher, resemblance in psychiatric symptoms between the parents of the children they evaluate, so that they can timely offer an intervention targeted at the parents. This study informs clinical practice by providing information on the co-occurrence of internalizing and externalizing psychiatric symptoms in parents of children with psychopathology.

2. Methods

2.1. Participants

2.1.1. Clinical sample

Data have been collected in three child and adolescent outpatient clinics in The Netherlands (two in Amsterdam and one in Rotterdam), all mainly treating children with ADHD (35.2%), autism spectrum disorders (21.8%), behavioral disorders (9.7%), anxiety disorders (20%) and/or depressive disorders (8.2%). Note, due to co-morbidity the diagnoses are not mutually exclusive.

A total of 1272 parents (728 mother and 544 fathers) of 778 children participated in this study. In Amsterdam, first, a pilot study was carried out to examine how many parents are at risk for a psychiatric disorder at the moment of the first assessment of their child at a child and adolescent psychiatric outpatient clinic. Out of the 176 mothers and 122 fathers of 191 children that completed the Adult Self Report [25], 38.2% of the mothers and 31.5% of the fathers scored in the (sub)clinical range on at least one of the syndrome scales. Consequently, assessment of parental problems and offering further assessment and, if necessary, subsequent treatment became the standard procedure. The total Amsterdam sample consists of 395 mothers and 262 fathers from 425 families, after exclusion of 35 families who did not consent for the use of the data for research. In the Rotterdam outpatient clinic, data were collected as part of the standard clinical procedure from the start. Data were available for 333 mothers and 282 fathers from 353 families. In 70% of the families in Amsterdam and in 60% of the families in Rotterdam, at least one parent completed the survey. Age and educational achievement were significantly higher in the parents from Amsterdam than from Rotterdam and were included as covariates. This difference is in line with the already known regional differences in educational achievement in the Netherlands, with people in Rotterdam having on average a lower education than in Amsterdam [26]. The appendix provides a detailed description of the two study samples. The Amsterdam and Rotterdam studies were approved by the Central Ethics Committees of the participating institutions.

2.1.2. Population-based sample

Parents of twins registered with the Netherlands Twin Register (NTR) [27,28] completed the same questionnaires as the parents in the clinical sample to assess their psychiatric symptoms. Fathers and mothers in a similar age range as the parents in the clinical sample were included, i.e., fathers between 29 and 68 years and mothers between 27 and 60 years. The final NTR sample consisted of 2784 families with data available for 914 complete spouse pairs, 1161 mothers and 709 fathers. All data, i.e. from complete and incomplete spouse pairs, were analyzed.

The twin population from the NTR is representative of the general Dutch population regarding the presence of psychiatric disorders. Eight percent of the twins aged 14 to 16 years reported to have received treatment for psychiatric problems over the last four years. This is largely comparable to the general Dutch population in which 5% of the children aged 0 to 18 received treatment over the last year [29]. The higher percentage in the twins can be explained by the differences in age between the two populations, 14–16 years compared to 0 to 18 years.

2.2. Measures

Psychiatric symptoms in parents were measured with the Adult Self Report (ASR), belonging to the Achenbach System of Empirically Based Assessment (ASEBA) [30]. The ASR is a questionnaire for adults from 18 to 59 years and consists of 120 items that are rated on a three-point scale (0 to 2; not true, somewhat true, very true). The DSM-oriented scales [25] depressive problems, anxiety problems, avoidant personality problems, ADHD problems, and antisocial personality problems were analyzed. Several studies have reported the reliability and validity of the ASR [31,32]. The questionnaire was available for parents both on paper and online.

Educational achievement was defined in three categories: low (1 to 3; primary school, lower vocational schooling, lower secondary schooling), intermediate (4 to 5; intermediate vocational schooling, intermediate/higher secondary schooling) and high educational achievement (6 to 7; higher vocational schooling, university).

2.3. Statistical analyses

Since the distribution of the psychiatric symptom scales was skewed, a square root transformation was performed improving the normality distribution. To investigate differences in age and education between fathers and mothers and between the clinical and population-based sample, independent sample *t*-tests and Chi^2 tests were carried out in R (version 3.1.1). The effects of age and education on the ASR scores and differences in ASR scores between the clinical and population-based sample were tested in a regression analysis in SPSS (version 21) with sample, age and education included as fixed effects.

Spousal correlations were estimated and compared between the clinical and the population-based sample using structural equation modeling in OpenMx [33]. With the full information maximum likelihood option, all available information was analyzed, including data from incomplete spousal pairs. This provides better estimates than analyzing complete pairs only [34]. In the first most general model, means, standard deviations, and correlations were estimated separately for mothers and fathers in the clinical and the population-based sample. Age and education were included as fixed effects (covariates) on the scores. Next, more restricted models were tested against the most general model with a likelihood-ratio test, comparing the goodness of fit of the restricted model with the goodness of fit of the more general model. If the difference in fit is not statistically significant, the restricted model, which has fewer free parameters due to constraints, should be accepted. A significant test statistic (at $P < 0.05$ level) indicates that constraining the parameters results in a worsening of fit, thus that the more general model should be retained. First, the clinical and the population-based samples were compared. Differences between the two samples were tested for all scales simultaneously for:

- the standard deviations;
- the within person or "phenotypic" correlations among scales;
- and the spousal correlations between and across scales.

Second, post hoc tests were carried out to investigate asymmetry with respect to sex in spousal resemblance. It was tested whether the correlation between the maternal score on scale X and the paternal score on scale Y could be constrained to be equal to the correlation between the maternal score on scale Y and the paternal score on scale X. These post hoc tests were based on visual inspections of the correlational patterns.

3. Results

3.1. Descriptives

Characteristics of the parents in the clinical ($n = 1272$) and population-based samples ($n = 3698$) are shown in Table 1. Both in the clinical and the population-based sample, the fathers were significantly older than the mothers ($t = 13.25$ (521), $P < 0.001$ and $t = -26.98$ (1065), $P < 0.001$, respectively) and significantly higher educated ($\chi^2_4 = 186.57$, $P = <0.001$ and $\chi^2_4 = 242.52$, $P < 0.001$). Comparing the clinical and the population-based samples showed that the mothers were higher educated in the clinical sample compared to the mothers in the population-based sample ($\chi^2_4 = 23.83$, $P < 0.001$). Fathers were similarly educated in the clinical and the population-based sample ($\chi^2_4 = 5.08$, $P = 0.08$). Further, parents in the population-sample were older than the parents in de clinical sample (mothers: $t = -33.61$ (2870), $P < 0.001$, fathers: $t = -32.34$ (2276), $P < 0.001$), but parental age at child birth hardly differed between the two samples (Table 1).

3.2. Means

Table 2 shows the mean psychiatric symptom scores for the parents in the clinical and population-based sample. While controlling for the effects of age and education, mothers in the clinical sample scored significantly higher on depression, anxiety, avoidant problems and ADHD than mothers in the population-based sample. Fathers in the clinical sample had significantly increased scores for depression, anxiety, ADHD and significantly decreased scores for antisocial personality problems compared to fathers in the population based sample. The regression analyses to test for the effects of age and education showed significant coefficients for education ranging from −0.12 to −0.06, confirming the protective effect of a higher education (Appendix A). Table 3

Table 1
Characteristics of parents in the clinical and population-based samples.

	Mothers		Fathers	
	Clinical sample ($n = 728$)	Population-based sample ($n = 2075$)	Clinical sample ($n = 544$)	Population-based sample ($n = 1623$)
Age, mean (SD)	41.80 (6.80)[a]	50.20 (5.50)[a]	44.69 (7.03)[a]	55.27 (6.61)[a]
Parent's age at childbirth, mean (SD)	30.38 (6.48)[a]	29.43 (3.72)[a]	33.61 (6.65)[a]	31.23 (3.99)[a]
Education achievement, n (%)				
Low	189 (25.5%)[a]	686 (32.9%)[a]	157 (26.2%)	443 (26.6%)
Intermediate	264 (35.6%)[a]	777 (37.2%)[a]	193 (32.2%)	460 (27.6%)
High	289 (38.9%)[a]	625 (29.9%)[a]	249 (41.6%)	764 (45.8%)

Age was a continuous variable measured in years. Educational achievement was a categorical variable with categories: low, intermediate and high.
[a] Significant difference ($P < 0.05$) between the clinical and population-based sample.

Table 2
Mean symptom scale scores (standard deviations) of the square root transformed scores in the clinical and population-based sample.

	Mothers		Fathers	
	Clinical sample ($n = 728$)	Population-based sample ($n = 2075$)	Clinical sample ($n = 544$)	Population-based sample ($n = 1623$)
Depression	1.92 (1.14)[b]	1.66 (1.03)[b]	1.48 (1.06)[b]	1.22 (0.97)[b]
Anxiety	1.92 (0.82)[b]	1.80 (0.80)[b]	1.60 (0.84)[a]	1.41 (0.87)[a]
Avoidant	1.38 (0.88)[a]	1.26 (0.85)[a]	1.18 (0.90)	1.14 (0.86)
ADHD	1.98 (1.03)[b]	1.75 (0.94)[b]	1.86 (1.05)[a]	1.71 (0.97)[a]
Antisocial	1.26 (0.82)	1.28 (0.78)	1.37 (0.90)[a]	1.50 (0.78)[a]

ADHD: attention deficit/hyperactivity disorder (ADHD).
[a] Significant difference ($P < 0.01$) in mean scores between the clinical and population-based sample.
[b] Significant difference ($P < 0.001$) in mean scores between the clinical and population-based sample.

shows the test statistics of the comparisons between the standard deviations between the clinical and the population-based sample. Comparing model 2, with the standard deviations constrained to be equal across the samples, to the most general model 1, revealed that the clinical sample had significantly higher standard deviations although the differences are small.

3.3. Correlations

Table 4 shows the within-person correlations for psychiatric symptoms in the clinical sample and in the population-based sample for mothers (below the diagonal) and fathers (above the diagonal). Within-person correlations for psychiatric symptoms varied between 0.37 and 0.70 in mothers and between 0.33 and 0.65 in fathers in the clinical sample and between 0.35 and 0.64 in mothers and between 0.33 and 0.58 in fathers in the population-based sample. Table 3 shows that the within-person correlations among the different psychiatric symptom scales in mothers were similar across the samples (model 3), while the within person correlations in fathers were significantly higher in the clinical than in the population-based sample (model 4).

Correlations between spouses within the internalizing and externalizing symptom domains were between 0.22 and 0.28 in the clinical sample (Table 5, upper part). Correlations across symptoms ranged from 0.14 to 0.30. All correlations were significantly higher than 0 as shown by the confidence intervals. The spousal correlations for the five psychiatric symptom scales in the population-based sample ranged from 0.09 to 0.23 within scales (Table 5, lower part). Correlations across symptom scales ranged from 0.05 to 0.16. Again, all correlations were significant, with the exception of paternal anxiety with maternal ADHD. It becomes clear from Table 5 that the within and across symptom correlations in the population-based sample are lower, sometimes almost twice as low, than in the clinical sample.

In model 5, the correlations between the parents were constrained to be equal within and across the psychiatric symptoms in the clinical and in the population-based sample. Comparing model 5 to model 1 confirmed that the spousal correlations significantly differed between the clinical and the population-based sample (Table 3).

Next, we performed post hoc tests to investigate the asymmetry with respect to sex in spousal resemblance in the clinical sample. Based on visual inspection of the correlations, we tested whether:

- the correlations between paternal ADHD and all maternal scales were higher than the correlations between maternal ADHD and all paternal scales;
- and the correlations between maternal antisocial personality problems and all paternal scales were higher than the correlations between paternal antisocial personality problems and all maternal scales.

The two post hoc tests showed that paternal ADHD indeed was significantly higher correlated with all five maternal symptom clusters than vice versa ($\chi^2_4 = 10.21$, $P < 0.001$). The same held for antisocial personality problems in mothers and all five symptom clusters in fathers ($\chi^2_4 = 10.21$, $P = 0.04$). Asymmetry with respect to sex was non-significant in the population-based sample, neither for paternal ADHD with the five maternal symptom clusters ($\chi^2_4 = 3.73$, $P = 0.44$), nor for maternal antisocial personality problems with all five paternal symptom clusters ($\chi^2_4 = 2.90$, $P = 0.57$).

4. Discussion

In this study, we investigated spousal resemblance within and across internalizing and externalizing symptom domains and for the first time statistically compared resemblance in a large sample of parents of children with psychopathology with spousal resemblance in a population-based sample. Spousal correlations were generally significant within and across internalizing and externalizing symptom domains in both the clinical and population-based sample. Moreover, as hypothesized, spousal resemblance was clearly higher in the at-risk population of parents. We further observed significant asymmetry with respect to sex in the clinical and not in the population-based sample. Paternal ADHD correlated higher with maternal internalizing and externalizing symptom scores than maternal ADHD with paternal symptom scores and maternal antisocial personality problems correlated higher with paternal internalizing and

Table 3
Test statistics for the comparisons of the models to explore the differences between the clinical and population-based sample.

	Estimated parameters	−2LL	df	Compared to	χ^2	P-value
Saturated clinical and population-based sample	170	56,145.78	24,640	–	–	–
Equal standard deviations between the clinical and population-based sample	160	56,187	24,650	1	41.22 (10)	< 0.00
Equal correlation matrices within mothers between the clinical and population-based sample	160	56,159.29	24,650	1	13.51 (10)	0.20
Equal correlation matrices within fathers between the clinical and population-based sample	160	56,169.46	24,650	1	23.68 (10)	0.01
Equal correlation matrices across mothers and fathers between the clinical and population-based sample	145	56,185.61	24,665	1	39.83 (25)	0.03

All models are compared to model 1. Likelihood ratio tests are performed. The negative log-likelihoods (−2LL) of the models (2–5) are subtracted from the −2LL of the saturated model (1). The difference between the −2LL of the two models follows a χ^2 distribution with degrees of freedom (*df*) equal to the difference in the numbers of parameters in the two models.

Table 4
Within-person correlations [confidence intervals] for psychopathology in the clinical sample (upper part) and the population-based sample (lower part). Within-mother correlations below the diagonal, within-father correlations above the diagonal.

	Depression	Anxiety	Avoidant	ADHD	Antisocial
Clinical sample					
Depression		0.65 [0.60, 0.70]	0.58 [0.52, 0.63]	0.58 [0.52, 0.63]	0.40 [0.32, 0.46]
Anxiety	0.70 [0.66, 0.73]		0.50 [0.43, 0.56]	0.47 [0.40, 0.53]	0.33 [0.26, 0.41]
Avoidant	0.60 [0.56, 0.65]	0.55 [0.49, 0.60]		0.44 [0.37, 0.51]	0.33 [0.26, 0.41]
ADHD	0.57 [0.52, 0.62]	0.48 [0.42, 0.53]	0.47 [0.41, 0.52]		0.58 [0.52, 0.63]
Antisocial	0.45 [0.39, 0.50]	0.37 [0.30, 0.43]	0.40 [0.34, 0.46]	0.50 [0.44, 0.55]	
	Depression	Anxiety	Avoidant	ADHD	Antisocial
Population-based sample					
Depression		0.56 [0.53, 0.59]	0.58 [0.57, 0.61]	0.54 [0.53, 0.57]	0.41 [0.37, 0.44]
Anxiety	0.64 [0.62, 0.64]		0.45 [0.42, 0.49]	0.46 [0.42, 0.50]	0.33 [0.31, 0.37]
Avoidant	0.61 [0.61, 0.62]	0.50 [0.47, 0.53]		0.45 [0.41, 0.49]	0.36 [0.33, 0.40]
ADHD	0.54 [0.53, 0.57]	0.42 [0.39, 0.45]	0.45 [0.41, 0.48]		0.48 [0.44, 0.51]
Antisocial	0.40 [0.37, 0.43]	0.35 [0.31, 0.38]	0.36 [0.35, 0.39]	0.43 [0.40, 0.46]	

ADHD: attention deficit/hyperactivity disorder.

externalizing symptom scores than vice versa. Parents of children with psychopathology also had higher symptom scores than parents in the population-based sample, as expected and in line with previous studies [35–44].

For clinical practice, these findings imply that parents have a significantly higher risk for psychopathology when their child is seen in a child and adolescent psychiatric outpatient, and that, if one parent suffers from psychiatric symptoms, chances are increased that their spouse also suffers from psychiatric symptoms, either in a similar or different symptom domain. Especially in the clinical sample, spousal correlations across symptom domains were of similar magnitude as the spousal correlations within symptom domains. Clinicians should be aware of this great variety in spousal resemblance for psychopathology between parents. Besides the negative effects of psychiatric symptoms on the parent's own daily functioning, the course and outcome of the treatment for a child can be negatively influenced when both parents are afflicted with psychopathology [6].

Spousal resemblance can be due to different processes, including phenotypic assortment, social homogamy and marital interaction [13]. Assortative mating implies the tendency for people to start long-term relationships with those who are more similar to themselves than with those who are not [45]. When this co-occurrence is due to phenotypic assortment, partner selection is based directly on the partner's phenotype. Social homogamy refers to the tendency for individuals to have partners with similar social backgrounds [46]. Marital interaction refers to the mutual influences or the sharing of the same environmental factors between spouses living together [47]. In this study we cannot differentiate between phenotypic assortment, social homogamy and marital interaction as the cause of spousal resemblance, but for psychiatric disorders phenotypic assortative mating has been indicated as causes for spousal resemblance [8,13,20].

As discussed in the introduction, several mechanisms may underlie the higher spousal correlations within and across internalizing and externalizing symptom domains in the clinical population. As we observed significant correlations across the symptom domains, we tested whether co-morbidity could underlie the higher spousal correlations observed in the clinical sample. If individuals with higher scores on depression also report higher anxiety scores, it follows that, in case of a significant spousal correlation for depression, spousal correlations are probably also significant between depression and anxiety [48]. If co-morbidity is higher in the clinical sample than in the population-based sample, this can result in higher across-symptom domain spousal correlations. We have explored this mechanism by comparing the within-person correlations across the internalizing and externalizing scales, between the two samples. These correlations

Table 5
Spousal correlations [confidence intervals] for psychopathology in the clinical sample (upper part, $n = 494$ for complete spouse pairs) and the population-based sample (lower part, $n = 914$ for complete spouse pairs).

	Mothers clinical sample ($n = 728$)				
	Depression	Anxiety	Avoidant	ADHD	Antisocial
Fathers clinical sample ($n = 544$)					
Depression	0.26 [0.17, 0.34]	0.19 [0.10, 0.27]	0.24 [0.16, 0.32]	0.18 [0.09, 0.27]	0.30 [0.22, 0.38]
Anxiety	0.23 [0.14, 0.31]	0.24 [0.15, 0.32]	0.18 [0.10, 0.27]	0.13 [0.04, 0.22]	0.20 [0.11, 0.29]
Avoidant	0.20 [0.12, 0.28]	0.18 [0.10, 0.26]	0.22 [0.14, 0.30]	0.16 [0.07, 0.24]	0.22 [0.13, 0.30]
ADHD	0.30 [0.22, 0.38]	0.24 [0.16, 0.32]	0.30 [0.22, 0.38]	0.25 [0.16, 0.33]	0.29 [0.21, 0.37]
Antisocial	0.22 [0.13, 0.30]	0.20 [0.12, 0.28]	0.16 [0.07, 0.25]	0.14 [0.05, 0.23]	0.28 [0.20, 0.36]
	Mothers population-based sample ($n = 2075$)				
	Depression	Anxiety	Avoidant	ADHD	Antisocial
Fathers population-based sample ($n = 1623$)					
Depression	0.16 [0.11, 0.23]	0.14 [0.08, 0.20]	0.14 [0.12, 0.21]	0.08 [0.01, 0.14]	0.09 [0.02, 0.15]
Anxiety	0.15 [0.09, 0.21]	0.23 [0.17, 0.29]	0.07 [0.05, 0.13]	0.07 [0.00, 0.13]	0.05 [−0.01, 0.12]
Avoidant	0.13 [0.07, 0.18]	0.16 [0.10, 0.22]	0.14 [0.07, 0.20]	0.09 [0.03, 0.15]	0.10 [0.04, 0.15]
ADHD	0.12 [0.06, 0.18]	0.15 [0.09, 0.21]	0.13 [0.07, 0.18]	0.09 [0.02, 0.15]	0.12 [0.06, 0.18]
Antisocial	0.09 [0.02, 0.15]	0.09 [0.03, 0.15]	0.08 [0.03, 0.14]	0.09 [0.03, 0.15]	0.10 [0.04, 0.16]

ADHD: attention deficit/hyperactivity disorder.

were only significantly different for fathers, and not for mothers, and the differences were small. This indicates that co-morbidity does not play an important role in explaining the higher correlations.

Another explanation for the higher spousal resemblance, as mentioned in the introduction, could be that parents in the at-risk sample are more exposed to stress. Living with and caring for a person with psychiatric symptoms is known to be a stressful experience and stress might evoke the development of psychiatric symptoms [17,49,50]. In this sample, both parents are exposed to the stress of a child suffering from psychiatric symptoms. A recent study by van Steijn et al. [51] highlighted the increased burden of raising a child with autism spectrum disorder and/or ADHD and its relationship with parental autism spectrum disorder, ADHD, depressive symptoms and levels of stress. The other possibility is that the family members are all exposed to the same stressors, such as financial problems, which can increase the risk for psychiatric symptoms in parents as well as in children.

A different explanation for the higher spousal resemblance in the clinical sample is the influence of genetic factors, if the parents are biological parents. Excluding the data from 29 non-biological mothers, 30 non-biological fathers in the clinical sample (n = 40 pairs) and 5 non-biological mothers and 7 non-biological fathers in the population-based sample (n = 11 pairs) led to similar spousal correlations in both samples, with a maximum difference of 0.01. This suggests that both genetic and environmental influences seem to underlie the higher spousal resemblance in psychopathology, as the exclusion of non-biological parents did not lead to an increase in the spousal correlations, especially in the clinical sample, within and across the symptom domains. The above described stressful environment as a consequence of the problems of the child might pull the trigger of these more genetically vulnerable parents, resulting in higher spousal correlations in parents of clinically referred children in comparison to parents of the children in the population-based sample. Vice versa, children of parents who both have a psychiatric disorder also have the highest genetic risk to suffer from psychiatric disorders themselves [52]. This could also result in an overrepresentation of these families in a clinical sample. Finally, parents who suffer from psychiatric symptoms might more easily recognize these symptoms in their children and seek treatment.

Longitudinal studies can partly unravel the role of these mechanisms. Earlier studies have, for example, already indicated that the course of psychiatric symptoms in children and parents are associated [2–5], suggesting that family members influence each other's symptoms.

We also showed asymmetry with respect to sex in the spousal correlations in the clinical sample, which was not apparent in the population-based sample. These results confirmed and extended the findings of Segenreich et al. [22] as paternal ADHD problems were more strongly associated with all five psychiatric symptom scales in mothers than the other way around. Contrary to the findings of Galbaud du Fort et al. [1] we reported paternal depressive problems to be significantly more strongly associated with maternal antisocial personality problems (0.30), than vice versa (0.22). An explanation for this difference might be the low prevalence of mothers with antisocial personality problems in the study sample of Galbaud du Fort et al. [1], who gave sex specific population prevalence rates for psychiatric symptoms as an explanation for their finding. This asymmetry with respect to sex together with the high correlations found across the five psychiatric symptom scales between parents can influence the risk for co-morbidity of psychiatric symptoms in the offspring of children with two affected parents.

There are several limitations and strengths of this study. First, even though we reached a family-response rate around 65% in the clinical samples, there are still parents whom we did not reach. Middeldorp et al. [53] conducted a non-response analysis in the clinical sample and found that families in which only mothers completed the questionnaire were exposed to less favorable circumstances than families in which both parents, or only father completed the questionnaires. This may point to non-response bias with fathers who are more at risk for psychiatric symptoms being less likely to participate. However, part of the families in which only the mother completed the questionnaires are single-parent families (without a father), which are known to be at a higher risk for unfavorable circumstances [54]. Secondly, the parents of children in the population-based sample were significantly older than the parents in the clinical sample, which might have influenced the spousal correlations due to effects of marital interaction. However, van Grootheest et al. [13] examined the effects of length of the relationship on spousal resemblance for obsessive-compulsive, anxious, and depressive symptoms and did not find any evidence for higher spousal resemblance in longer relationships. A major strength of this study is that we have reached a large number of fathers for the screening procedure for psychiatric symptoms.

To summarize, this study shows that parents whose children are evaluated at a child and adolescent outpatient clinic have an increased risk to both suffer from a variety of psychiatric symptoms, with correlations across symptom domains of similar magnitude as the correlations within symptom domains. Therefore, it is important to encourage mothers as well as fathers to visit the outpatient clinic along with the child and to offer screening of parental symptoms and treatment in case of psychiatric complaints.

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Disclosure of interest

The authors declare that they have no competing interest.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.eurpsy.2016.01.2423.

References

[1] Galbaud du Fort G, Bland RC, Newman SC, Boothroyd LJ. Spouse similarity for lifetime psychiatric history in the general population. Psychol Med 1998;28(4): 789–802.
[2] Cobham VE, Dadds MR, Spence SH. The role of parental anxiety in the treatment of childhood anxiety. J Consult Clin Psychol 1998;66(6):893–905.
[3] Owens EB, Hinshaw SP, Kraemer HC, Arnold LE, Abikoff HB, Cantwell DP, et al. Which treatment for whom for ADHD? Moderators of treatment response in the MTA. J Consult Clin Psychol 2003;71(3):540–52.

[4] Kennard BD, Hughes JL, Stewart SM, Mayes T, Nightingale-Teresi J, Tao R, et al. Maternal depressive symptoms in pediatric major depressive disorder: relationship to acute treatment outcome. J Am Acad Child Adolesc Psychiatry 2008;47(6):694–9.
[5] Pilowsky DJ, Wickramaratne P, Poh E, Hernandez M, Batten LA, Flament MF, et al. Psychopathology and functioning among children of treated depressed fathers and mothers. J Affect Disord 2014;164:107–11.
[6] Dierker LC, Merikangas KR, Szatmari P. Influence of parental concordance for psychiatric disorders on psychopathology in offspring. J Am Acad Child Adolesc Psychiatry 1999;38(3):280–8.
[7] Merikangas KR. Assortative mating for psychiatric disorders and psychological traits. Arch Gen Psychiatry 1982;39(10):1173–80.
[8] Mathews CA, Reus VI. Assortative mating in the affective disorders: a systematic review and meta-analysis. Compr Psychiatry 2001;42(4):257–62.
[9] Meyler D, Stimpson JP, Peek MK. Health concordance within couples: a systematic review. Soc Sci Med 2007;64(11):2297–310.
[10] Sakai JT, Stallings MC, Mikulich-Gilbertson SK, Corley RP, Young SE, Hopfer CJ, et al. Mate similarity for substance dependence and antisocial personality disorder symptoms among parents of patients and controls. Drug Alcohol Depend 2004;75(2):165–75.
[11] Kim-Cohen J, Moffitt TE, Taylor A, Pawlby SJ, Caspi A. Maternal depression and children's antisocial behavior: nature and nurture effects. Arch Gen Psychiatry 2005;62(2):173–81.
[12] Rhule-Louie DM, McMahon RJ. Problem behavior and romantic relationships: assortative mating, behavior contagion, and desistance. Clin Child Fam Psychol Rev 2007;10(1):53–100.
[13] van Grootheest DS, van den Berg SM, Cath DC, Willemsen G, Boomsma DI. Marital resemblance for obsessive-compulsive, anxious and depressive symptoms in a population-based sample. Psychol Med 2008;38(12):1731–40.
[14] Distel MA, Rebollo-Mesa I, Willemsen G, Derom CA, Trull TJ, Martin NG, et al. Familial resemblance of borderline personality disorder features: genetic or cultural transmission? PLoS One 2009;4(4):e5334.
[15] Distel MA, Rebollo-Mesa I, Abdellaoui A, Derom CA, Willemsen G, Cacioppo JT, et al. Familial resemblance for loneliness. Behav Genet 2010;40(4):480–94.
[16] Boomsma DI, Saviouk V, Hottenga JJ, Distel MA, de Moor MH, Vink JM, et al. Genetic epidemiology of attention deficit hyperactivity disorder (ADHD index) in adults. PLoS One 2010;5(5):e10621.
[17] Joutsenniemi K, Moustgaard H, Koskinen S, Ripatti S, Martikainen P. Psychiatric comorbidity in couples: a longitudinal study of 202,959 married and cohabiting individuals. Soc Psychiatry Psychiatr Epidemiol 2011;46(7):623–633.
[18] Ask H, Idstad M, Engdahl B, Tambs K. Non-random mating and convergence over time for mental health, life satisfaction, and personality: the Nord-Trondelag Health Study. Behav Genet 2013;43(2):108–19.
[19] Thomsen AF, Olsbjerg M, Andersen PK, Kessing LV. Cohabitation patterns among patients with severe psychiatric disorders in the entire Danish population. Psychol Med 2013;43(5):1013–21.
[20] Merikangas KR, Spiker DG. Assortative mating among in-patients with primary affective disorder. Psychol Med 1982;12(4):753–64.
[21] Maes HH, Neale MC, Kendler KS, Hewitt JK, Silberg JL, Foley DL, et al. Assortative mating for major psychiatric diagnoses in two population-based samples. Psychol Med 1998;28(6):1389–401.
[22] Segenreich D, Paez MS, Regalla MA, Fortes D, Faraone SV, Sergeant J, et al. Multilevel analysis of ADHD, anxiety and depression symptoms aggregation in families. Eur Child Adolesc Psychiatry 2015;24(5):525–36. http://dx.doi.org/10.1007/s00787-014-0604-1 [Epub 2014 Aug 26].
[23] Faraone SV, Antshel KM. Diagnosing and treating attention-deficit/hyperactivity disorder in adults. World Psychiatry 2008;7(3):131–6.
[24] Reich T, Van Eerdewegh P, Rice J, Mullaney J, Endicott J, Klerman GL. The familial transmission of primary major depressive disorder. J Psychiatr Res 1987;21(4):613–24.
[25] Achenbach, Rescorla L. Manual for the ASEBA adult forms & profiles: for ages 18–59: adult self-report and adult behavior checklist. Burlington, VT: ASEBA; 2003 [xii, 216 p].
[26] CBS. Inkomen, arbeidsdeelname, opleidingsniveau, vermogen en sociale samenhang naar regio. Centraal Bureau voor de Statistiek [CBS]; 2014, http://www.cbs.nl/nl-NL/menu/themas/dossiers/nederland-regionaal/cijfers/incidenteel/maatwerk/2014-arbeid-inkomensbron-vermogen-opleiding-sociale-samenhang-mw.htm.
[27] Boomsma DI, de Geus EJ, Vink JM, Stubbe JH, Distel MA, Hottenga JJ, et al. Netherlands Twin Register: from twins to twin families. Twin Res Hum Genet 2006;9(6):849–57.
[28] Willemsen G, Vink JM, Abdellaoui A, den Braber A, van Beek JH, Draisma HH, et al. The Adult Netherlands Twin Register: twenty-five years of survey and biological data collection. Twin Res Hum Genet 2013;16(1):271–81.
[29] GGZ Nederland. Factsheet specialistische jeugd-ggz; 2013 [Available from: http://www.ggznederland.nl/uploads/assets/Factsheet%20GGZ%20Nederland%20-%20Jeugd-ggz%20juli%202013.pdf].
[30] Achenbach TM, Dumenci L, Rescorla LA. DSM-oriented and empirically based approaches to constructing scales from the same item pools. J Clin Child Adolesc Psychol 2003;32(3):328–40.
[31] Achenbach TM, Dumenci L. Ratings of relations between DSM-IV diagnostic categories and items of the Adult Self-Report (ASR) and Adult Behavior Checklist (ABCL); 2003 [Available from: http://www.aseba.org/research/dsm-adultratings.pdf].
[32] Achenbach TM, Krukowski RA, Dumenci L, Ivanova MY. Assessment of adult psychopathology: meta-analyses and implications of cross-informant correlations. Psychol Bull 2005;131:361–82.
[33] Boker S, Neale M, Maes H, Wilde M, Spiegel M, Brick T, et al. OpenMx: an open source extended structural equation modeling framework. Psychometrika 2011;76(2):306–17.
[34] Enders CK, Bandalos DL. The relative performance of full information maximum likelihood estimation for missing data in structural equation models. Struct Equ Modeling 2001;8(3):430–57.
[35] Last CG, Hersen M, Kazdin AE, Francis G, Grubb HJ. Psychiatric illness in the mothers of anxious children. Am J Psychiatry 1987;144(12):1580–3.
[36] Brown RT, Borden KA, Clingerman SR, Jenkins P. Depression in attention deficit-disordered and normal children and their parents. Child Psychiatry Hum Dev 1988;18(3):119–32.
[37] Lahey BB, Piacentini JC, McBurnett K, Stone P, Hartdagen S, Hynd G. Psychopathology in the parents of children with conduct disorder and hyperactivity. J Am Acad Child Adolesc Psychiatry 1988;27(2):163–70.
[38] Barkley RA, Anastopoulos AD, Guevremont DC, Fletcher KE. Adolescents with attention deficit hyperactivity disorder: mother-adolescent interactions, family beliefs and conflicts, and maternal psychopathology. J Abnorm Child Psychol 1992;20(3):263–88.
[39] Ferro T, Verdeli H, Pierre F, Weissman MM. Screening for depression in mothers bringing their offspring for evaluation or treatment of depression. Am J Psychiatry 2000;157(3):375–9.
[40] Swartz HA, Shear MK, Wren FJ, Greeno CG, Sales E, Sullivan BK, et al. Depression and anxiety among mothers who bring their children to a pediatric mental health clinic. Psychiatr Serv 2005;56(9):1077–83.
[41] Tan S, Rey J. Depression in the young, parental depression and parenting stress. Australas Psychiatry 2005;13(1):76–9.
[42] Cooper PJ, Fearn V, Willetts L, Seabrook H, Parkinson M. Affective disorder in the parents of a clinic sample of children with anxiety disorders. J Affect Disord 2006;93(1–3):205–12.
[43] Rishel CW, Greeno CG, Marcus SC, Anderson C. Effect of maternal mental health problems on child treatment response in community-based services. Psychiatr Serv 2006;57(5):716–9.
[44] Vidair HB, Reyes JA, Shen S, Parrilla-Escobar MA, Heleniak CM, Hollin IL, et al. Screening parents during child evaluations: exploring parent and child psychopathology in the same clinic. J Am Acad Child Adolesc Psychiatry 2011;50(5):441–50.
[45] Byrne PS, Freeman J, M'Comisky JG. General practitioners observed: a study of personality, intellectual factors, and group behaviour. Br J Med Educ 1970;4(3):176–84.
[46] Reynolds CA, Baker LA, Pedersen NL. Multivariate models of mixed assortment: phenotypic assortment and social homogamy for education and fluid ability. Behav Genet 2000;30(6):455–76.
[47] Penrose LS. Mental illness in husband and wife: a contribution to the study of assortative mating in man. Psychiatr Q 1944;18(1):161–6.
[48] Phillips K, Fulker DW, Carey G, Nagoshi CT. Direct marital assortment for cognitive and personality variables. Behav Genet 1988;18(3):347–56.
[49] Cronkite RC, Moos RH. The role of predisposing and moderating factors in the stress-illness relationship. J Health Soc Behav 1984;25(4):372–93.
[50] Eagles JM, Beattie JA, Blackwood GW, Restall DB, Ashcroft GW. The mental health of elderly couples. I. The effects of a cognitively impaired spouse. Br J Psychiatry 1987;150:299–303.
[51] van Steijn DJ, Oerlemans AM, de Ruiter SW, van Aken MA, Buitelaar JK, Rommelse NN. Are parental autism spectrum disorder and/or attention-deficit/Hyperactivity disorder symptoms related to parenting styles in families with ASD (+ADHD) affected children? Eur Child Adolesc Psychiatry 2013;22(11):671–81.
[52] Dean K, Stevens H, Mortensen PB, Murray RM, Walsh E, Pedersen CB. Full spectrum of psychiatric outcomes among offspring with parental history of mental disorder. Arch Gen Psychiatry 2010;67(8):822–9.
[53] Middeldorp CM, Wesseldijk LW, Hudziak JJ, Verhulst FC, Lindauer RJ, Dieleman GC. Parents of children with psychopathology: psychiatric problems and the association with their child's problems. Eur Child Adolesc Psychiatry 2016. http://dx.doi.org/10.1007/s00787-015-0813-2 [Epub ahead of print].
[54] McLanahan S, Sandefur GD. Growing up with a single parent: what hurts, what helps. Cambridge, Mass: Harvard University Press; 1994 [viii, 196 p].