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Patterns of Psychopathology in the Families of Children with Conduct Problems, Depression, and both Psychiatric Conditions

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Abstract Comorbid conduct problems (CPs) and depression are observed far more often than expected by chance, which is perplexing given minimal symptom overlap. In this study, relations between parental psychopathology and children's diagnostic status were evaluated to test competing theories of comorbidity. Participants included 180 families with an 8–12-year-old child diagnosed with CPs, depression, both conditions, or neither condition. Although no single theory of comorbidity was supported fully, evidence suggested that CPs and depression may be inherited separately. Paternal antisocial characteristics and maternal depression provided independent prediction of both child depression and CPs. However, paternal antisocial behavior moderated the effect of maternal depression on CPs. For children with antisocial fathers, CPs were observed regardless of maternal depression levels. In contrast, a strong relation was observed between CPs and maternal depression for children without antisocial fathers.

Keywords Family history · Comorbidity · Antisocial personality disorder · Conduct disorder · Depression

Comorbidity among common childhood psychiatric disorders occurs at rates significantly higher than expected by chance (Bird, Gould, & Staghezza, 1993). As a result, comorbidity has received increasing attention in the last decade, with researchers considering both theoretical and practical implications of the phenomenon (e.g., Angold, Costello, & Erkanli, 1999; Klein & Riso, 1993; Lilienfeld, Waldman, & Israel, 1994). Comorbidity rates exceeding chance occurrence may suggest common etiological substrates among disorders. However, elucidating such etiological relations is often complicated by the use of the term 'comorbidity' to describe several different patterns of diagnostic co-occurrence (Klein & Riso, 1993; Lilienfeld, 2003). Perhaps the most common use of the term is to indicate the presence of two assumedly distinct psychiatric conditions within an individual. However, rates of comorbidity may be inflated for reasons ranging from artifactual methodological issues such as rater bias or insufficient clinical assessments, to ontological issues such as inappropriate diagnostic splitting of single or closely related latent traits (Beauchaine & Marsh, 2006; Klein & Riso, 1993; Lilienfeld, 2003). Apparent comorbidity may also emerge when behavioral symptoms of etiologically distinct disorders overlap. Comorbidity that is inadequately characterized may misdirect therapeutic efforts and reduce the effectiveness of treatment.

Comorbidity rates among externalizing disorders such as ADHD, oppositional defiant disorder (ODD), conduct disorder (CD), antisocial personality disorder (ASPD), and substance abuse are especially high (Lewinsohn, Shankman, Gau, & Klein, 2004; Nadder, Rutter, Silberg, Maes, & Leaves, 2002). Similarly, internalizing disorders such as depression, dysthymia, and a multitude of anxiety disorders frequently co-occur among both children and adults (Angold & Costello, 1993; Brady & Kendall, 1992; Cloninger, 1990; Donaldson, Klein, Riso, & Schwartz, 1997). Comorbidity within the externalizing and internalizing spectra, often referred to as homotypic comorbidity, is not surprising given considerable symptom overlap among disorders. Moreover, many if not most disorders within these symptom domains share common genetic vulnerability. For example, Krueger et al. (2002) reported that 80% of the variance in ADHD, ODD, CD, and substance abuse symptoms was accounted for by a single latent impulsivity trait. Thus, impulsivity, as a core trait, may arise from genetic predispositions but may

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manifest as one or more of several symptom clusters depending on the moderating effects of such factors as environment, cognitive ability, and other personality predispositions across development. Accordingly, an afflicted individual could simultaneously or sequentially traverse multiple pathways of psychopathology. This offers an explanation of co-morbidity not as co-occurrence of individual diagnostic entities, but rather as co-variation of related syndromes stemming from a common latent genetic vulnerability.

In contrast to comorbidity within externalizing and internalizing symptom domains, comorbidity across domains (e.g., depression and CD) is somewhat more perplexing because symptom profiles overlap minimally (Angold, Costello, & Erkanli, 1999). Angold and colleagues term this occurrence heterotypic comorbidity because such disorders are often assumed to be of different origin. Indeed, depression includes symptoms of depressed mood, anhedonia, and feelings of guilt or worthlessness, and is often manifested in a withdrawn behavioral presentation. In contrast, CD is characterized by symptoms such as sensation seeking, lying, property destruction, aggression, and may include a pathological lack of empathy and restricted emotional range. Yet rates of comorbidity for CD and depression in childhood are far greater than expected by chance (Angold & Costello, 1993; Capaldi, 1991; Essau, 2003; Garber, Quiggle, Panak, & Dodge, 1991). To date, no clearly supported mechanism for this comorbidity has been identified, although several theories have been proposed (see Drabick, Beauchaine, Gadow, Carlson, & Bromet, 2006).

One such theory suggests a common latent genetic vulnerability for both CD and depression, with symptom presentation driven largely by environmental influences. In a family history study of children with attention deficit disorder (ADD), Biederman, Faraone, Keenan, and Tsuang (1991) found that rates of affective disorders among the first-degree relatives of index cases were high in ADD groups both with and without comorbid affective disorders. This finding led the authors to suggest a common diathesis for internalizing and externalizing syndromes. Furthermore, in a twin/sibling behavioral genetics study, 45% of the covariation between depression and antisocial behaviors was accounted for by a common genetic liability (O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998). Such findings suggest that comorbidity of CD and depression may be in large part an artifact of splitting symptom profiles that are generated by a common latent trait. One candidate trait is negative emotionality, a temperamental construct that predisposes children to a variety of negative emotions including guilt, anxiety, and irritability (Lilienfeld, 2003). Indeed, irritability is considered to be an indicator of both depression and CD among children (DSM-IV-TR, 2000), which may in part explain the high rates of comorbidity for these disorders. However, other studies have failed to identify significant increases in internalizing disorders among family members of externalizing children (e.g., Frick et al., 1992).

A second explanation for comorbidity of CD and depression is that one disorder is primary, with symptoms of the other deriving from this principal condition. This hypothesis has been offered in both directions (i.e., with both depression and CD considered to be the primary diagnosis), and is usually accompanied by the supposition that proper identification of the primary disorder has important implications for treatment. Glaser (1967) theorized that CD may represent "masked depression", with externalizing symptoms of anger and aggression marking a behavioral expression of depressed mood. According to this formulation, depression is often mistaken for CD, inflating apparent rates of comorbidity. However, support for this hypothesis has been equivocal. In particular, success in treating comorbid CD symptoms with antidepressants has been limited (Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Puig-Antich, 1982).

The converse prediction, that CD is primary to depression, has also been offered (e.g., Capaldi, 1991). According to this theory, factors such as peer rejection, school failure, and social isolation develop as secondary consequences of aggressive and antisocial behavior (Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003). In a longitudinal study of clinic referred boys, initially high levels of CD predicted increases in depression at later assessments, even when controlling for initial levels of depression (Lahey, Loeber, Burke, Rathouz, & McBurnett, 2002). In contrast, initially high levels of depression did not predict later increases in CD symptoms.

Each of these alternative hypotheses suggests that comorbidity between CD and depression is largely artifactual, and does not indicate the presence of two distinct disorders. If depression develops secondarily to CD in comorbid children, the observed depressive symptoms may mark an underlying latent trait that is quite distinct from depression observed in the absence of preexisting CD. As noted above, this may have important implications for treatment. Standard approaches to treatment of CD and depression are quite different. Targeting interventions toward a primary disorder may potentiate treatment efficacy and reduce the latency to clinical improvement.

A final possibility is that the observed rates of comorbidity between CD and depression represent true comorbidity. Factors such as assortative mating may explain why two disparate disorders exist in individual children with such frequency. Cross-trait assortative mating between antisocial males and depressed females has been documented (e.g., Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). Although explanations for this assortment are speculative at this time, antisocial males may be more likely to seek out depressed women because they consider them to be more vulnerable to manipulation, or depressed women may be

more likely to remain in a relationship with an antisocial partner than non-depressed women.

One approach to clarifying the source of symptom overlap for comorbid disorders is to examine familial patterns of psychopathology among the relatives of offspring diagnosed with these disorders. Familial factors contributing to vulnerability for both CD and depression are well known. Increased rates of depression are observed among family members of depressed probands (Klein, Lewinsohn, Rhode, Seeley, & Shankman, 2003; Pfiffner et al., 1999), and increased rates of ASPD are observed among family members of CD probands (Barnow, Lucht, & Freyberger, 2005; Chronis et al., 2003; Frick et al., 1992; Pfiffner et al., 1999). A recent study of adolescents revealed that both maternal depression and paternal ASPD were significantly associated with both adolescent depression and CD (Marmorstein & Iacono, 2004). However, these investigators did not compare comorbid with non-comorbid participants to determine the exact pattern(s) of association.

Such comparisons could be quite informative. For example, if comorbidity is caused by a latent trait common to both CD and depression, then elevated rates of both ASPD and depression should be observed among parents of children with CD, depression, and both disorders. In contrast, if comorbidity results from true transmission of two distinct disorders, then both ASPD and depression would likely be elevated only in parents of children with both disorders. Furthermore, parents of children with only depression should exhibit elevated rates of depression and not ASPD, and parents of children with CD should exhibit elevated rates of ASPD and not depression. Thus, the question of how patterns of parental psychopathology differ for comorbid and non-comorbid children could inform our understanding of the intergenerational transmission of psychopathology.

Past studies assessing comorbid conditions using the family history method have typically approached the analysis from a categorical perspective, with parents classified as having or not having the disorder or disorders of interest according to traditional diagnostic thresholds. Yet symptom severity may be additionally informative, indicating the degree of genetic and/or environmental loading. A recent study of depression across three generations of families indicated that illness severity, as measured by impairment, conferred greater risk of transmission to later generations (Weissman et al., 2005). Functional impairment increases incrementally with symptom severity (Judd et al., 2005). More severe depression may mark biological liability, whereas less impairing depression may manifest for a variety of reasons, and may not necessarily increase depression risk for offspring. Very few studies have explored this question from both categorical and dimensional perspectives. Using both approaches may improve our understanding of the influences that contribute to comorbid expression of CD and depression. In the current study, we examine rates of maternal depression and paternal ASPD, as well as symptom severity, among parents of children diagnosed with CD alone, depression alone, comorbid CD and depression, and among control participants with neither disorder. Our intent was to evaluate the following predictions derived from the theories outlined above:

1. *Depression and CD result from a common latent trait*. This hypothesis suggests that familial transmission of these disorders demonstrates no specificity and that either depression or ASPD in the parental generation will increase risk for both depression and CPs in the offspring generation. Therefore, support for this theory would be provided if rates of both depression and ASPD were elevated among parents of all three diagnostic groups but were low in the control group.
2. *Depression is primary to CD in comorbid cases*. If the observed comorbidity between CD and depression is indeed artifactual, and CPs result from underlying depression, then maternal depression should be more predictive of comorbidity than paternal ASPD. Full support for this theory would be provided if (a) increased rates of depression were observed among mothers of children with depression, both with and without CD; (b) these rates of depression were higher in the depression and CD + depression groups than in the CD only and control groups; and (c) rates of paternal ASPD were elevated for the CD only group but not for the comorbid group.
3. *Conduct disorder is primary to depression in comorbid cases*. This is the converse of Prediction 2, and implies that paternal ASPD should be more predictive of comorbidity than maternal depression. Full support for this theory would be provided if (a) increased rates of ASPD were observed among fathers of children with CD with or without depression; (b) these rates of ASPD were elevated in comparison with the depressed only and the control groups; and (c) rates of maternal depression were elevated only for the depressed group but not for the comorbid group.
4. *Comorbidity represents two individual disorders*. This possibility suggests that comorbidity among children should predict elevated paternal ASPD and maternal depression. That is, both disorders occur in the child because both disorders occur in the child's parents. This interpretation would be supported if (a) rates of paternal ASPD were elevated for children with CD; (b) rates of parental depression were elevated for children with depression; and (c) rates of both paternal ASPD and parental depression were elevated for children who are comorbid.

Method

Participants

Participants included 180 children (121 male; 129 Caucasian, 25 African American, 2 American Indian, 2 Asian American, 10 Latino, and 12 unspecified) between the ages of 8 and 12, and one biological parent (the mother in all but 4 cases). Children were classified into one of four groups (control, CP, depressed, or comorbid) according to procedures detailed below. Choice of this age range followed from consideration of several factors. First, these ages encompass a transition period that marks rapid escalation of both depressive and delinquent behaviors (Loeber & Keenan, 1994). Second, depressive symptoms become more differentiated from other psychiatric symptoms across this period of development (Weiss & Nurcombe, 1992), thereby increasing reliability of assessments. The mean age of onset for childhood dysthymia is between 8.3 and 11.5 years (Kovacs et al., 1988, 1994; Lewinsohn et al., 1991), and as many as 80% of these children will go on to experience major depression. Third, significant sex differences in depression that are observed in adolescence are not expected to have emerged in this age group (Angold & Rutter, 1992; Hankin & Abramson, 1999; Nolen-Hoeksema & Girgus, 1994). Fourth, the presence of CP behaviors in this age range ensures that participants are of the childhood-onset subtype, as important etiological differences between the child- and adolescent-onset subtypes could affect interpretations (Moffitt, 1993).

Participants were recruited primarily from urban Seattle neighborhoods through advertisements placed in radio spots, newspapers, city busses, school newsletters, and flyers posted at clinics and community centers. Interested parents responded to the advertisements by telephoning the laboratory and completing a 20–30 min structured clinical interview to assess their child's appropriateness for the study. Qualified families were invited to the laboratory to participate in the study. A total of 445 interviews were conducted to recruit 180 participant families, each of whom was paid $75 for their initial visit.

Procedures

Families were enrolled in a larger longitudinal study consisting of four visits across three years. Only data from the first visit are presented here as information about family history was only obtained during this visit. All procedures were approved by the University of Washington Institutional Review Board. All participant parents signed informed consent documents and participant children signed assent forms. Data were protected by a Certificate of Confidentiality issued by the National Institute of Mental Health.

Child psychopathology

Children were placed into specific symptom groups based on parental reports of their child's behavior completed during the initial structured phone interview, which was administered by a trained research assistant. Parents completed the ADHD (inattentive and hyperactive subscales), oppositional defiant disorder (ODD), conduct disorder (CD), major depressive disorder (MDD), and dysthymia (DYS) subscales of the Child Symptom Inventory (CSI), a dimensionalized checklist of DSM-IV-TR (2000) criteria, which was used to establish a probable diagnosis (see Gadow & Sprafkin, 1997). Sensitivity and specificity of the CSI scales are as follows: ADHD (.80, .74), ODD (.69, .75), CD (Not Available, .83), MDD/DYS (.81, .73). Because of the age range of participants, some were quite young to reach full criteria for CD or major depression. Accordingly, children were accepted into the CP groups if they met criteria for CD and/or ODD, and children were accepted into the depressed groups if they met criteria for dysthymia and/or major depression.

Based on results of this interview, children were placed into one of the following four groups: *control* (CTR; $n = 63$; 51% male) could not meet CSI criteria for ODD, CD, MDD or DYS; *conduct problems* (CPs; $n = 36$; 78% male) met CSI criteria for CD ($n = 15$) and/or ODD ($n = 21$), did not meet CSI criteria for DYS or MDD; *depression/dysthymia* (DEP; $n = 24$; 63% male) met CSI criteria for MDD ($n = 4$) or DYS ($n = 20$) and failed to meet CSI criteria for CD and ODD; *comorbid* (CMB; $n = 57$; 81% male) met inclusion criteria for both the DEP and the CP groups (all participants in this group met criteria for dysthymia and all but 1 met criteria for ODD. In addition, 20 met criteria for CD and 14 met criteria for MDD). Groups did not differ on racial minority status (Caucasian/Non-Caucasian) χ^2 (3, $n = 179$) = 2.6, $p = .46$. Additional details about the sample, including psychopathology scores by group, appear in Table 1.

Parent psychopathology

During the initial lab visit the accompanying parent was administered the Structured Clinical Interview for DSM-IV, non-patient version (SCID-NP; Spitzer, Williams, Gibbon, & First, 1992) assessing Axis I disorders, by a trained graduate interviewer. After the SCID was complete, the parent was administered the Family Interview for Genetics Studies (FIGS; Maxwell, 1992) to assess for psychopathology in the child's first degree relatives. The FIGS interview assesses depression, mania, substance use, and schizophrenia on AXIS I, and schizoid, schizotypal, paranoid, and antisocial personality disorders on AXIS II. The parent was asked to report only on biological relatives of the participant child including and limited to the father (or mother in the four cases where the father was the accompanying parent),

Table 1 Descriptive characteristics of sample by group

Variable	CTR ($n = 63$)	CP ($n = 36$)	DEP ($n = 24$)	CMB ($n = 57$)	Test Statistic	p
Race	79% Caucasian	66% Caucasian	67% Caucasian	68% Caucasian	$\chi^2_{(1)} = 2.9$	.41
Age	9.8 (1.5)	9.5 (1.5)	10.3 (1.5)	9.9 (1.5)	$F_{(3,179)} = 1.2$	.30
CD symptoms	1.1 (1.7)	8.0 (4.4)	1.8 (2.2)	7.9 (5.5)	$F_{(3,179)} = 44.5$	<.01
ODD symptoms	4.8 (3.4)	16.8 (3.4)	7.6 (3.3)	18.7 (3.7)	$F_{(3,179)} = 195.2$	<.01
MDD symptoms	1.5 (2.1)	4.6 (3.2)	8.6 (4.3)	11.0 (5.2)	$F_{(3,179)} = 67.1$	<.01
Dysthymia symptoms	1.8 (1.9)	4.2 (2.2)	8.8 (2.8)	10.8 (3.8)	$F_{(3,179)} = 118.5$	< .01

Notes. Parenthesized values are standard deviations. Conduct disorder (CD), major depressive disorder (MDD), oppositional defiant disorder (ODD) and dysthymia scores were computed by summing the values of each as reported on the Child Symptom Inventory (Gadow & Sprafkin, 1997). CTR = control; CP = conduct problems; DEP = depressive; CMB = comorbid.

paternal grandparents, maternal grandparents, and both full and half siblings. Because only one informant was interviewed, data were often difficult to obtain about more distant relatives (for instance, some mothers had no knowledge of the biological father's parents). Therefore, only data pertaining to participant children's two biological parents were analyzed.

The decision to interview only one parent was driven by practical considerations. In addition to the added inconvenience and potential reduction in participation from requiring two parents, 52% of participant families did not have the father living at home with the child at the time of the interview, as is typical among low income families of children with behavior problems. Thus, this percentage was higher for the diagnostic groups, particularly children in the CP groups. Research on this topic has revealed that antisocial characteristics of offspring are higher in families where the father is not present in the home and cannot be recruited for research participation (Pfiffner, McBurnett, & Rathouz, 2001). Therefore, requiring the participation of both biological parents would likely bias the sample toward lower symptom severity. Accordingly, only one parent was required to participate. Because the mother was unlikely to have detailed knowledge of the father's childhood behavior, the ASPD criterion for evidence of CD was not enforced. These procedures have been used in several family history studies (e.g., Barnow et al., 2005; Chronis et al., 2003; Pfiffner et al., 1999). Using these criteria, 53 fathers met criteria for ASPD according to mother report, and 91 mothers met lifetime criteria for depression or dysthymia according to self-report.

Because rates of maternal ASPD are typically too low to subject to statistical analyses (e.g., Barnow et al., 2005), only paternal ASPD was considered. Findings from previous studies suggest that this is unlikely to bias results. For example, in a study of parental psychopathology among children with ADHD with and without disruptive behavior disorders, paternal but not maternal antisocial behaviors were related to CPs of participants (Chronis et al., 2003). Similarly, only maternal depression status was considered because it has been shown most frequently to predict depressive and CD diagnoses among children (Boyle & Pickles, 1997; Kim-Cohen et al., 2005). Furthermore, assessing depression for both parents would have weighted it more heavily than ASPD, which was only assessed among fathers. This approach is supported by a recent study of depression and CD among adolescents in which neither paternal depression nor maternal ASPD were related to offspring psychopathology. In contrast, both paternal ASPD and maternal depression were (Marmorstein & Iacono, 2004).

Analyses

Patterns of psychopathology among parents of participants were compared across groups by first juxtaposing lifetime incidence rates of depression/dysthymia for mothers and rates of ASPD for fathers. Thus, the number of parents within each group meeting DSM-IV criteria for each disorder were compared using χ^2 tests of association, which are appropriate for categorical measures. Data were missing in some cases where the mother was not familiar enough with the biological father to report reliably on his psychological functioning. In four cases, the father was the accompanying parent and was not administered the antisocial interview, resulting in missing data. Given a significant χ^2 statistic for the distribution of a diagnostic category, cell frequencies were examined for the disorder across groups. Follow up χ^2 tests of association contrasting each of the groups were performed in order to determine if the increased rate of ASPD among fathers of CP children was statistically higher than that among fathers of depressed children, comorbid children, and controls. Parallel analyses were conducted for maternal depression/dysthymia.

In addition to these categorical analyses, child diagnostic groups were also compared on continuous symptom measures of psychopathology among their parents. A dimensional index of depression was computed by summing the value for each depressive symptom for each participant mother. Scores ranged from 9 (equivalent to a 1, or "not present" score for each item on the SCID) to 27 (equivalent to a 3 or "threshold" on each symptom on the SCID). The greater value between the current and the lifetime

Table 2 Observed and (expected) cell frequencies and percentages for χ^2 analyses of paternal antisocial and maternal depression status by child diagnostic group

	CTR	CP	DEP	CMB	Overall $\chi^2_{(3)}$	Sig. Contrasts
Paternal ASPD	9 (17.5) 14.5%	12 (9.3) 36.4%	7 (6.8) 29.2%	21 (15.5) 38.2%	9.6*	CP > CTR*, CMB > CTR**
Maternal Depression	28 (32.0) 44.4%	14 (18.3) 42.4%	12 (11.7) 52.2%	37 (29.0) 64.3%	7.6	CMB > CTR* CMB > CP*

Note. $^{*}p < .05$, $^{**}p < .01$.

assessments was used. A dimensional index of paternal ASPD was computed by summing the number of symptoms endorsed positively by the mother. Scores ranged from 0 to 8. Distributions of both variables were within acceptable limits with skewness and kurtosis values between -1 and 1.

Results

Paternal antisocial personality disorder

The omnibus χ^2 statistic comparing groups on paternal ASPD was significant. Follow up contrasts indicated that children in the CP group were more likely than children in the CTR group to have a father with ASPD, χ^2 (1, $n = 95$) $= 6.0$, $p = .02$. Children in the CMB group were also more likely than CTRs to have a father with ASPD, χ^2 (1, $n = 117$) $= 8.6$, $p = .003$. Additional contrasts were not significant. These results are summarized in Table 2.

ANOVAs conducted on continuous symptom severity scores for paternal ASPD indicated a significant main effect, $F(3,173) = 3.2$, $p = .02$. Post hoc analyses contrasting groups using the Tukey LSD test indicated that fathers of CMB children ($M = 2.5$, $SD = 2.9$) exhibited more antisocial symptoms than fathers of CTR children ($M = 1.1$, $SD = 2.2$), $p = .003$, $d = .55$. Fathers of CMB children did not differ from fathers of CP children on ASPD symptoms ($M = 2.2$, $SD = 2.7$), $p = .51$, $d = .14$. However, the difference in ASPD symptoms among fathers of CP children and fathers of CTR children failed to reach significance, $p = .06$, $d = .40$. In addition, fathers of CMB children did not differ from fathers of DEP children ($M = 1.7, SD = 2.8$), $p = .18$, $d = .33$, who did not differ from fathers of either CTR children, $p = .35$, $d = .22$, or CP children, $p = .49$, $d = .18$. These results are summarized in the top panel of Fig. 1.

Maternal depression

For maternal depression, the omnibus χ^2 statistic failed to reach significance χ^2 (3, $n = 179$) $= 7.6$, $p = .06$, $w = 2.76$. Given the effect size, however, follow-up analyses were conducted. Mothers of CMB children reported more depression than mothers of both CTRs, χ^2 (1, $n = 120$) $= 5.1$, $p < .05$ and mothers of CP children, χ^2 (1, $n = 93$) $= 6.0$ Remaining contrasts were not significant. These results are summarized in Table 2.

The ANOVA analyzing continuous maternal depression scores across groups was significant, $F(3,170) = 5.7$, $p = .01$. Post hoc analyses using the Tukey LSD test indicated that mothers of CMB children ($M = 20.6$, $SD = 5.7$) reported more depressive symptoms than mothers of CTRs ($M = 16.6$, $SD = 6.3$), $p = .001$, $d = .62$, and mothers of CP children ($M = 17.9$, $SD = 6.7$), $p < .05$, $d = .42$. Mothers of CMB children, however, reported no difference in depressive symptoms compared with mothers of DEP children ($M = 21.5$, $SD = 7.0$), $p = .57$, $d = .13$. Mothers of children in the DEP group did report more depressive symptoms than mothers of both CTRs, $p = .001$, $d = .75$,

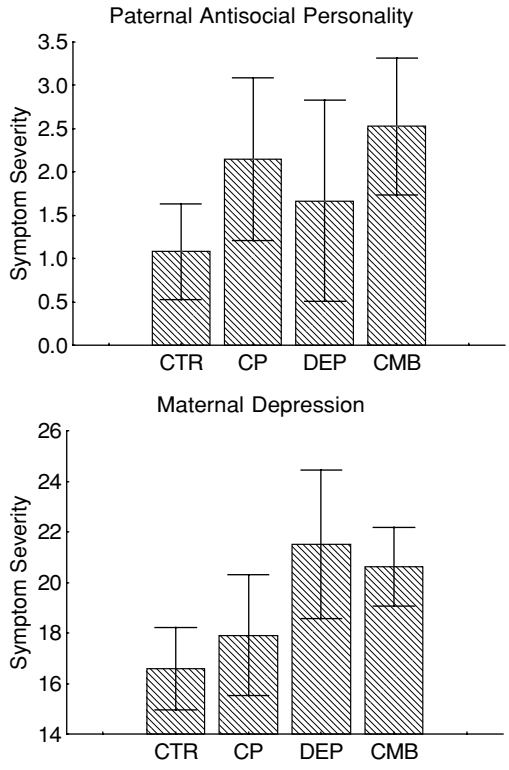

Fig. 1 Rates of paternal antisocial personality symptoms (top panel) and maternal depressive symptoms (bottom panel) among the parents of control children (CTR), children with conduct problems (CP), children with depression (DEP), and children with comorbid conduct problems and depression (CMB)

Table 3 Observed and (expected) cell frequencies and percentages of parental diagnoses by child diagnostic status

Group	Parental psychopathology status		
	No parental diagnosis	One parental diagnosis	Two parental diagnoses
Control	32 (22.9) 51.6%	24 (29.0) 38.7%	6 (10.0) 9.7%
Conduct Problem	10 (12.2) 30.3%	20 (15.5) 60.6%	3 (5.3) 9.1%
Depressed	8 (8.5) 34.8%	11 (10.8) 47.8%	4 (3.7) 17.4%
Comorbid	14 (20.3) 25.5%	26 (25.8) 47.3%	15 (8.9) 27.3%

Note. $\chi^2_{(6)} = 15.1, p < .05$.

and mothers of CP children, $p < .04$, $d = .55$ who did not differ from one another, $p = .33$, $d = .20$. These results are summarized in the bottom panel of Fig. 1.

Paternal ASPD and maternal depression

A final set of analyses was conducted examining relations between child diagnostic status and having both a father with ASPD and a mother with depression. Analyses using categorical diagnostic outcomes were conducted by comparing observed frequencies of no parental diagnosis, one parental diagnosis (paternal ASPD or maternal depression), and both parental diagnoses across child diagnostic groups. Paternal ASPD and maternal depression were not considered separately in this analysis because several cell sizes were too small. Chi-square results indicated a significant effect, χ^2 $(6, n = 173) = 15.1, p = .02$. Expected and observed cell frequencies are presented in Table 3. Although results should be interpreted with caution given small cell sizes for both CP and depressed children with two diagnosed parents, observed frequencies for CTR children whose mothers reported both paternal ASPD and maternal depression were lower than expected, and observed cell frequencies for CMB children whose mother reported both paternal ASPD and maternal depression were higher than expected.

To examine multivariate relations between child and parent symptoms using continuous measures, *z*-scores were first computed for both maternal depression and paternal ASPD symptoms, and for both child depression (summed MDD and DYS symptoms) and child CPs (summed CD and ODD symptoms). This placed symptoms for each construct on the same metric, ensuring that variables with larger ranges would not pull inordinate weight in subsequent regression analyses. Next, two multivariate linear regressions (MLRs) were run in which (a) child depression scores were regressed on maternal depression and paternal ASPD, and (b) child conduct problem scores were regressed on maternal depression and paternal ASPD. The first MLR ($R^2 = .11$, $p < .001$) revealed independent prediction from paternal ASPD to child depression, $\beta = .21, t(164) = 2.9, p = .005$, and from maternal depression to child depression, $\beta = .25, t(164) = 3.3$, $p = .001$. Similarly, the second MLR ($R^2 = .076, p = .001$) indicated independent prediction from paternal ASPD to child CPs, $\beta = .23$, $t(164) = 3.0$, $p = .003$, and from maternal depression to child CPs, $\beta = .15$, $t(164) = 2.0$, $p = .05$.

In addition to assessing independent relations between parent and child psychopathology, we were interested in testing the combined effects of maternal depression and paternal ASPD in predicting children's CP and depression scores. To accomplish this, a product vector was computed by multiplying maternal depression and paternal ASPD *z*-scores by one another. This interaction term was then added to the regression analyses described above to determine whether having both a father with ASPD symptoms and a mother with depressive symptoms conferred increased risk on participant children, over-and-above the risk associated with one set of parental symptoms or the other (see Aiken & West, 1991). When predicting child depression, the regression coefficient for this interaction was not significant, $\beta = -.03$, $t(163) = 0.35$, $\Delta R^2 = .002$, $p = .73$. In contrast, the father ASPD $\times$ maternal depression interaction was a significant predictor of child CPs, $\beta = -.18$, $t(163) = 2.36$, $\Delta R^2 = .031$, $p = .019$. The nature of this interaction appears in Fig. 2, which depicts the relation between maternal depression and child CPs when fathers scored above vs. below the sample median on ASPD symptoms. As the figure reveals, there was a significant positive relation between maternal depression and child CPs only when fathers scored below the sample median on ASPD symptoms, $\beta = .25$, $t = 2.7, p = .01$. In contrast, when fathers scored above the sample median on ASPD symptoms, children's CP scores were high *regardless* of their mother's level of depression, $\beta = -.02, t = 0.1, p = .88$.

Discussion

Maternal patterns of depression and paternal patterns of ASPD were evaluated among the parents of children with corresponding disorders to examine how familial psychopathology might influence observed comorbidity in children. Patterns of psychopathology among parents were analyzed using both categorical and dimensional measures. Results from these analyses did not provide unequivocal support for any single theory of comorbidity. Categorical

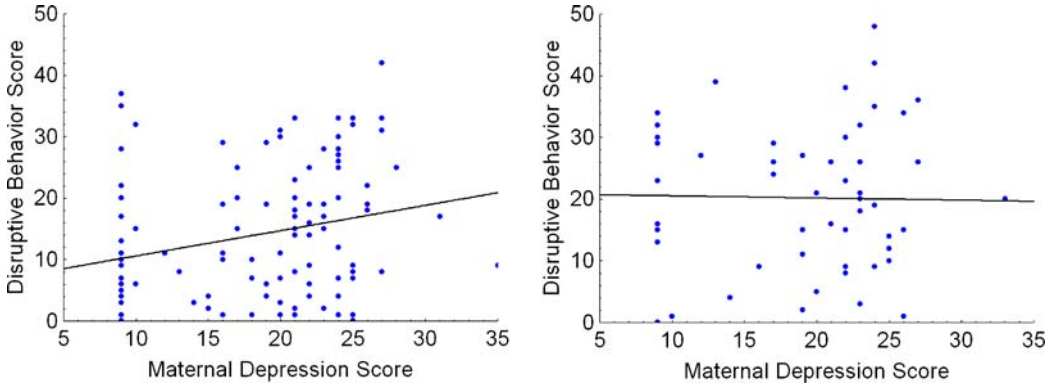

Fig. 2 Relations between continuous measures maternal depression and child disruptive behavior in families with fathers scoring below (left panel) and above (right panel) the sample median on antisocial personality disorder symptoms. Although analyses were conducted using *z*-scores, raw scores are plotted to aid in interpretation

analyses of paternal antisocial status yielded some evidence for Hypothesis 3, that CD is primary to depression, by demonstrating that both groups with CPs had fathers with significantly higher rates of ASPD than controls. However, paternal rates of ASPD for the CP groups (CP, CMB) did not exceed those for the DEP group, even though rates of ASPD did not differ among fathers of children in the DEP and CTR groups. These results may suggest that a father with ASPD confers general rather than specific risk for psychopathology, an interpretation that is more consistent with Hypothesis 1, that CD and depression share a common liability. This pattern was confirmed in the dimensional analyses of symptom counts for paternal ASPD. Thus, results using both analytic methods were partially consistent with Hypotheses 1 and 3 for paternal ASPD.

With regard to maternal depression, categorical analyses provided somewhat weaker support for Hypothesis 1, that depression and CPs share a common liability. Although mothers of children in the depressive groups (DEP, CMB) did not differ on rates of depression, each exhibited different patterns in relation to the other groups. Mothers of CMB children showed higher rates of depression diagnoses than mothers of CTRs and mothers of CP children. In contrast, mothers of DEP children did not. The diagnostic similarity between mothers of DEP children and mothers of CP children suggests some evidence for a common liability for depression and CPs, but the increased rates of maternal depression for CMB children suggests a potentially different, albeit unspecified, mechanism.

Analyses of depressive symptom severity presented a somewhat clearer picture of the relationship between maternal symptoms and child psychopathology. Evaluation of symptom scores indicated that all four groups had maternal depression means that fell in the clinical range. A score of 15 or higher (a rating of 3 on the SCID for 5 or more variables) indicates the potential for a diagnosis. Although a rating of 2 on the SCID for more than 5 variables may also lead to scores in this range without reaching diagnostic threshold, the finding that categorical variables did not clearly differentiate between the depressed and control groups suggests that diagnoses of maternal depression were generally high for control children as well. This is not surprising given the high prevalence of lifetime depressive episodes for women (Kessler, 2003). However, women may also differ in other important aspects of depression, including severity, frequency, and/or chronicity of episodes. When analyses addressed the severity of depression, a significant difference between the depression groups (DEP, CMB) and the non-depressive groups (CP, CTR) was observed. This suggests that some degree of depression among mothers is normative and may not necessarily increase risk in offspring. However, as depression severity increased, so did the apparent risk for transmission to children.

Taken together, the analyses of maternal depression and paternal ASPD suggest an additional interpretation. Children in the CMB group had fathers who scored higher on ASPD and mothers who scored higher on depression, regardless of whether a discrete or dimensional approach to analysis was used. Further analyses of parental comorbidity status indicated that comorbid children were much more likely than expected to have both a depressed mother and an antisocial father. This suggests that children of such parents are at especially high risk for psychopathology, both of internalizing and externalizing natures. These results appear most consistent with Hypothesis 4, that comorbidity of CD and depression in children represents true co-occurrence of different disorders.

On the other hand, the lack of differentiation of the CP and DEP groups on measures of parental psychopathology suggests that when these disorders present alone, they may be influenced by multiple factors, and appear with or without evidence of a family history of the disorder. In addition, the inability of categorical measures of maternal depression to distinguish between child DEP and CP groups could

result from heterogeneity in maternal depression. Mothers of children with behavior problems may develop depression in response to continuous conflict with the child. Because the current study did not restrict the time course of the depressive symptoms, the possibility that mothers of CP children experience a more reactionary than endogenous depression can not be eliminated.

With respect to the presence of childhood CPs, the regression analysis uncovering a significant maternal depression × paternal antisocial interaction was unexpected. These results indicate that antisocial characteristics among fathers confer considerable risk for CPs, regardless of whether mothers are depressed or not. This contrasts with previously posed theories that the relationship between child antisocial behavior and maternal depression is mediated through environmental mechanisms whereby children with antisocial tendencies show exacerbated symptoms in the presence of poor and inconsistent discipline (Brennan et al., 2000; Snyder, Cramer, Afrank, & Patterson, 2005). In support of this theory, a recent study of the relation between maternal depression and childhood CPs indicated that only depression experienced by the mother after the child's birth affected antisocial outcomes. This suggests that it may be exposure to, and not biological transmission of, the mother's depression that influences symptomotology in the crossover to CPs (Kim-Cohen et al., 2005). This distinction was not made in the current study, and it is possible that failure to find a relation between maternal depression and CPs for children with antisocial fathers was obscured by the inclusion of lifetime depressive episodes that have no current bearing on the mother's parenting behaviors.

Other research, however, is more consistent with our findings. Studies of both ODD and CD have revealed that when using paternal ASPD and maternal parenting variables, paternal antisocial behavior is the only significant predictor of child CPs (Frick et al., 1992). These findings are troubling because they suggest that impaired maternal functioning resulting from depression increases children's antisocial behavior, yet positive maternal functioning, at least in this domain, provides no buffering effect of the impact an antisocial father. Thus, the presence of antisocial tendencies in fathers appears to be a very potent risk factor for childhood antisocial outcomes. Given the finding that children in the two CP groups had high rates of absentee fathers, it is likely that this relationship is indicative of a genetically transmitted trait and not simply an effect of social modeling.

Our results also indicate that children in the DEP group showed unexpectedly high rates of paternal antisocial symptoms, suggesting that paternal ASPD may present a general risk for child psychopathology including both internalizing and externalizing symptoms, as also noted above. This is consistent with findings of Marmorstein and Iacono (2004), who reported that the presence of either MDD or CD was related to maternal depression and paternal antisocial behavior in an adolescent community-based sample. However, the exact mechanisms of risk between paternal antisocial behavior and child depression remains unclear.

In contrast to results obtained from discrete vs. dimensional analyses of paternal ASPD, implications for specific theories of comorbidity changed when maternal depression was analyzed as a dichotomy vs. a continuum. As might be expected, the categorical approach yielded less specificity than a severity-based approach. This may indicate that the diagnostic criteria for MDD, requiring only a single episode of depression, may be too inclusive to adequately identify those at risk for transmitting depression from one generation to another. Brennan and colleagues (2000) found that factors such as depression chronicity and severity were more informative than the presence or absence of lifetime episodes, suggesting heterogeneous outcomes with respect to heritability of the disorder. As an important aside, continuous measures of psychopathology are also more informative than dichotomous measures for psychometric reasons that have nothing to do with the phenomenology of any particular disorder (Beauchaine, 2003; Beauchaine & Marsh, 2006). In this regard, loss of variability that occurs when a continuous measure is dichotomized almost always results in attenuated statistical power, and can produce misleading outcomes (MacCallum, Zhang, Preacher, & Rucker, 2002). Thus, psychometric precision may be the source of the increased specificity provided by the continuous approach.

One limitation of the current study involves the nature of the sample. Although every effort was made to access psychopathological individuals from a broad range of community sources by targeting schools, clinics, city buses, and community based locations such as libraries and grocery stores, recruitment was necessarily reliant on self-enrollment. Thus, the willingness of participant parents to identify their child's problems and enroll in a study where they were asked to report personal information of a sensitive nature may have introduced a bias into the sample. On the other hand, such recruiting methods are likely to reach a more representative pool of families than would be found only at clinics, as the vast majority of pediatric CP and depression cases go untreated (U.S. Public Health Service, 2000).

A second limitation is the possibility of invalid data for paternal antisocial characteristics. Because the measure of paternal antisocial behavior was derived from maternal reports, there may be inaccuracies resulting from lack of maternal knowledge in cases of limited contact with the father (most likely resulting in underestimates of antisocial tendencies), biased reporting on the part of the mother (which could result in inaccurately failing to report antisocial activities or in inaccurately inflating antisocial tendencies), or inaccurate reporting of paternal lineages, either because the mother has mislead the child about paternal identity or because she is not sure about paternal identity.

Sole reliance on maternal reports may have also introduced bias into the severity of symptoms reported for the child. Because over half of the mothers in the sample had a lifetime diagnosis of depression, their depression status may have distorted their view of their children's symptoms. This has been shown among depressed mothers who are asked to report about their children's behavioral problems (Chi & Hinshaw, 2002). However, in the current sample, children in the conduct problem group were no more likely than controls to have mother reporting lifetime depression. Moreover, only 7% of the mothers in this study reported a current depressive episode when enrolled in the study. Nevertheless, future research should evaluate how patterns of family history relate to comorbidity defined by a multitude of assessment measures.

In addition, the design of the current study did not enable us to dissociate mechanisms of intergenerational transmission of risk such as heritability, environmental programming of biological functioning, or environmental shaping of symptom expression. It is likely that parental psychopathology contributes to offspring psychopathology in each of these domains. Paternal antisocial behavior may confer genetic risk to offspring for disruptive behavior disorders, but may also contribute to negative environmental conditions (Thornberry, Smith, & Howard, 1997) that could lead to depressive symptoms such as verbal or physical abuse, emotional neglect, and environmental instability and stress. Similarly, maternal depression may increase environmental stress and emotional neglect linearly as severity increases, thus increasing susceptibility of offspring to the development of depression. The design of this study did not allow for an analysis of familial dynamics over the course of children's development, and more sophisticated behavioral genetics designs are necessary to separate genetic influences from environmental effects, particularly environmental processes that are influenced by genetic factors (i.e., gene $\times$ environment interactions; see Moffitt, 2005; Moffitt, Caspi, & Rutter, 2006). This is especially so given that paternal depression and maternal antisocial behavior were not assessed and may have contributed in meaningful ways to the intergenerational transmission of risk. Furthermore, a multigenerational assessment of family history may be valuable as recent research has demonstrated moderating effects of grandparent psychopathology on grandchild symptom presentation (Weissman et al., 2005). However, missing data on family members beyond the parental generation presented an insurmountable challenge in the present study. Further clarification of these issues will require researchers to locate and interview several members of the extended family.

Additionally, given the cross sectional data that were available, we could not address important questions regarding developmental processes affecting comorbidity. Parental psychopathology both contributes to and is affected by child behavior over time. For instance, interactions between parental psychopathology and child temperament may affect specific symptom expression (Mun, Fiztgerald, Von Eye, Puttler, & Zuker, 2001). Research has shown that factors such as anxiety and social isolation alter trajectories for antisocial children, with anxiety attenuating and social isolation potentiating delinquent outcomes (Rutter, Giller, & Hagell, 1998). How such moderating variables alter the effects of parental psychopathology on children may have important implications for treatment. Longitudinal research has also indicated that comorbid symptoms both within and across the externalizing and internalizing spectra tend to covary over time. That is, increases in depression tend to occur with increases in CD, and not necessarily before or after such increases (Lahey et al., 2002). Whether these covarying changes derive from common biological mechanisms or environmental influences remains to be determined.

This study was also unable to assess potential moderating effects of sex on the expression of psychopathology, which are likely to change over the course of development. The age range of children in the current study probably influenced the unequal sex distribution across the groups. Males were overrepresented in diagnostic groups while the control group had a larger proportion of females. This pattern is not surprising given that males are more likely to exhibit externalizing symptoms (Simonoff et al., 1997) and equally likely to exhibit internalizing symptoms compared with females within this age range (Angold & Rutter, 1992; Hankin & Abramson, 1999; Nolen-Hoeksema & Girgus, 1994). However, rates of depression increase sharply among females after puberty (Angold, Costello, Erkanli, & Worthman, 1999), suggesting that patterns of comorbidity and familial transmission may appear different at different developmental time points. Because a greater proportion of males were recruited in all three diagnostic groups, concerns over sex effects were not serious. However, the relatively lower number of females, particularly in the CP group, reduced power too much to explore sex effects. It is possible that child sex may affect how parental psychopathology is expressed among children and further exploration of this possibility is warranted. Finally, the relatively low participation of racial minority subjects precluded an analysis of specific effects of race on familial patterns of psychopathology. Collapsing across racial categories, child diagnostic groups did not differ in racial minority status. However, the sample size was not sufficient for analyses assessing a possible differential relationship between child and parent diagnosis based on racial minority status.

In summary, the results of this study suggest that comorbidity between internalizing and externalizing symptoms in children is likely to be a product of high rates of comorbidity across parents as well. From a clinical perspective,

increased rates of parental comorbidity may be a significant impediment to child treatment unless addressed. Furthermore, approaches to treatment may need to be varied to reflect the range of underlying pathology. Results from this study do not provide support for primary treatment of a single disorder, but rather treatment that addresses both psychological disturbances.

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