Discrepancies Between Adolescent, Mother, and Father Reports of Adolescent Internalizing Symptom Levels and Their Association With Parent Symptoms

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Discrepancies among informants on measures of internalizing symptoms in children and adolescents are common in the literature. One reason proposed for such discrepancies is that psychopathology may distort or bias third-party reports. In the present study, measures of adolescent internalizing symptom levels were completed by adolescents aged 13 to 18 years and their mothers and fathers. Parents also completed measures of their own depression, anxiety, and stress symptoms. Parent symptoms explained a small amount of variance in discrepancies between informants. Specifically, mothers' depression and stress symptoms were associated with discrepancies regarding sons' symptomatology and fathers' anxiety and stress symptoms were associated with discrepancies regarding daughters' symptomatology. Implications of informant discrepancies for both clinical practice and research are discussed. J Clin Psychol: 66:978–995, 2010.

Keywords: psychological assessment; depression; anxiety; stress; adolescents; parents

The inclusion of multiple informants in the psychological assessment of children and adolescents has become the norm in clinical and research practice. Most professionals now agree that the assessment process should ideally include the child and his or her parents, as well as other informants as appropriate (Kendall &

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JOURNAL OF CLINICAL PSYCHOLOGY, Vol. 66(9), 978–995 (2010)
Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/jclp.20695

Flannery-Schroeder, 1998; Silverman & Saavedra, 2004). This multiinformant approach is purported to be more thorough as it provides information about the child from different perspectives and across different situations, which cannot be gleaned from one informant alone. It also provides a means of verifying informant reports, which may be consciously or unconsciously influenced by personal motives and characteristics. However, clinicians and researchers utilizing a multi-informant approach are often confronted with discrepancies among informant reports and must determine the meaning of such discrepancies and how discrepant reports should be integrated. Given the potential implications of informant discrepancies for both psychological treatment decisions and the interpretation of research findings, understanding the nature and source of such discrepancies has become an important and prolific field of psychological assessment research. However, despite the progress that has been made in this field, gaps and inconsistencies remain.

In a comprehensive meta-analysis of cross-informant correlations of child and adolescent behavioral and emotional problems, Achenbach, McConaughy, and Howell (1987) reported that although agreement between parents was generally high ($r = .59$), agreement between parents and children was low ($r = .25$). Subsequent studies have confirmed these patterns of informant agreement regarding child and adolescent internalizing symptoms (e.g., Berg-Nielsen, Vika, & Dahl, 2003; Briggs-Gowan, Carter, & Schwab-Stone, 1996; Duhig, Renk, Epstein, & Phares, 2000; Seiffge-Krenke & Kollmar, 1998; Sourander, Helstela, & Helenius, 1999) and disorders (e.g., Edelbrock, Costello, Dulcan, Conover, & Kala, 1986; Foley et al., 2004; Grills & Ollendick, 2003; Jensen et al., 1999).

Various reasons have been proposed to account for discrepancies between parent and child reports including limitations in children's cognitive and expressive language abilities and parents' awareness of, and exposure to, their child's symptoms (particularly more inconspicuous internalizing symptoms; Cantwell, Lewinsohn, Rohde, & Seeley, 1997; Grills & Ollendick, 2002). In addition, the effect of the parents' own emotional state on their reports of their child's symptomatology is receiving increasing interest. One of the most consistent findings in this area is that maternal depression is associated with mothers reporting higher levels of child psychological difficulties, including internalizing symptoms and disorders, relative to other informants (Berg-Nielsen et al., 2003; Chilcoat & Breslau, 1997; Najman et al., 2000; Youngstrom, Loeber, & Stouthamer-Loeber, 2000); although there are exceptions (Luoma, Koivisto, & Tamminen, 2004; Tarullo, Richardson, Radke-Yarrow, & Martinez, 1995). Research on other types of parent psychopathology is sparser, particularly regarding fathers. Nonetheless, similar findings have been reported for mothers' anxiety (Chilcoat & Breslau, 1997; Najman et al., 2000) and stress (Martin, Ford, Dyer-Friedman, Tang, & Huffman, 2004; Youngstrom et al., 2000), and fathers' depression (Ivens & Rehm, 1988) and anxiety (Jensen, Traylor, Xenakis, & Davis, 1988).

Sex differences in the expression and perception of internalizing symptoms between males and females, and differences in relationship characteristics among parent–child dyads may lead to variations in informant agreement and in observed relationships between parent psychopathology and their reports of child symptoms. Indeed, studies have noted sex differences in informant agreement; however, findings have been mixed. Specifically, some studies have reported better agreement between parents and daughters than between parents and sons (Seiffge-Krenke & Kollmar, 1998), whereas others have reported the opposite pattern (Krain & Kendall, 2000; Sourander et al., 1999), mixed patterns (Tarullo et al., 1995), or no sex difference

(Duhig et al., 2000; Edelbrock et al., 1986). Few studies could be found that have examined sex differences in the relationship between parent psychopathology and reports of child internalizing symptoms, and once again findings are mixed. In one study (Briggs-Gowan et al., 1996), mothers' depressive symptoms were related to greater discrepancies between mothers' and sons' reports regarding child internalizing symptoms, but unrelated to discrepancies between mothers' and daughters' reports. Mothers' anxiety symptoms were unrelated to both mother–son and mother–daughter discrepancies in this study. In contrast, Jensen et al. (1988) found that mothers' depressive and anxiety symptoms were related to greater discrepancies between mothers' and fathers' reports of their sons' but not their daughters' internalizing symptoms. This latter study also reported that fathers' anxiety (but not depressive) symptoms were significantly related to discrepancies between mothers' and fathers' reports regarding their daughters', but not their sons', internalizing symptoms. One further study reported that, after statistically controlling for child and parent self-reported anxiety symptoms, both mothers' and fathers' depressive symptoms were positively related to their reports of daughters', but not sons', anxiety symptoms (Krain & Kendall, 2000).

The current study aimed to examine the levels of agreement and discrepancies between adolescent, mother, and father reports of adolescent internalizing symptom levels, and to determine whether discrepancies in these informants' reports are related to maternal and paternal depression, anxiety, and stress symptoms. The focus of the study was not on reports of the presence or absence of specific symptoms or disorders, but agreement and discrepancies in the overall *level* of internalizing symptomatology reported. The distinction between *agreement* and *discrepancies* is also crucial as each index provides important, yet distinct, information regarding multi-informant data (Kazdin, 1994). Agreement is typically reported as the correlation between informant reports and indicates similarities in rank orders of scores provided by informants. Discrepancies reflect the degree to which informant reports differ and can reveal patterns in informant reports (e.g., who reports higher or lower levels of symptoms).

The study design expanded on previous research in a number of ways. First, past research has tended to focus on school-aged children, or a mixture of children and adolescents, whereas the current study included only adolescents. Given that adolescence is a time when self reports are likely to be increasingly sought, more heavily weighted, and often relied upon as the sole source of information, understanding informant discrepancies during this period is of particular importance to clinical and research procedures. Second, the study included both mothers and fathers, and explored parent and adolescent sex differences. As noted, few studies have examined sex differences in this field and results have been mixed. Further, although family-based research has become more inclusive of fathers, they remain under-represented. Third, the study examined three types of parent symptoms: depressive, anxiety, and stress. By comparison, the vast majority of past research has focused on maternal depression, and studies that have examined parent stress have tended to assess role-related stress rather than general stress (Martin et al., 2004; Youngstrom et al., 2000).

The study also improved upon previous methods of analysis in this field. First, when reporting on agreement and discrepancies between informants, previous studies have typically reported only correlations and mean scores or mean discrepancies. By also describing the distribution of informant discrepancies, this

study allowed for the examination of the frequency and magnitude of informant discrepancies and the identification of patterns in informants' discrepancies.

Second, when analyzing associations between reports of adolescent internalizing symptom levels and parent symptoms, the dependent variable in the current study was discrepancies between parent reports and either adolescent or spouse reports. As parent and adolescent internalizing symptoms are likely to co-occur due to genetic and environmental factors (Goodman & Gotlib, 1999; Hughes & Gullone, 2008), it is necessary to examine parent reports relative to criterion informant reports. Although there is no gold standard for criterion informants, and each has its limitations (see Richters, 1992 for a discussion), the adolescent and the spouse are arguably appropriate choices given that the former has firsthand knowledge of the behavior in question and the latter occupies a very similar role in the adolescent's life to the target parent. Although many studies have statistically controlled for criterion reports to examine the effect of parent symptoms on parent-reported child psychological adjustment, Richters (1992) pointed out that this may produce misleading results. In a review of the literature on maternal depression distortion, he argued that the dependent variable should be the discrepancy between reports from the mother and the criterion informant, rather than the mother-reported child symptoms after controlling for the criterion report, as each may yield different conclusions under different circumstances.

Third, the current study controlled for discrepancies in the other dyad when analyzing the contribution of parent symptoms to informant discrepancies. That is, parent–adolescent discrepancy was controlled when predicting mother–father discrepancy from parent symptoms and mother–father discrepancy was controlled when predicting parent–adolescent discrepancy from parent symptoms. It was reasoned that there may be sources of discrepancies that are common across informant dyads. For example, mother and father reports may both differ from adolescent reports due to characteristics of the parenting role (e.g., being an observer of their adolescent's behavior in the home, and having an investment in their adolescent's well-being). Similarly, adolescent reports may differ from both parent reports due to the adolescent's maturation level, social desirability, tendency to agree or disagree with others, or conspicuousness of their symptoms. Furthermore, as the parent and adolescent measures were not identical in the current study, this methodology allowed for some control over potential confounding related to differences in the measures that were common between mother–adolescent dyads and father–adolescent dyads. Discrepancies within the other dyad therefore provided a proxy measure of other sources of discrepancies allowing for a more powerful test of the effect of parent symptoms on parent reports of adolescent symptom levels.

On the basis of previous research, in the current study it was hypothesized that (1) parent–adolescent agreement would be in the low to moderate range and mother–father agreement would be higher, (2) adolescents would report higher levels of internalizing symptoms than parents (Edelbrock et al., 1986; Foley et al., 2004; Jensen et al., 1999; Seiffge-Krenke & Kollmar, 1998; Sourander et al., 1999), and (3) mothers would report higher levels of internalizing symptoms than fathers (Luoma et al., 2004; Seiffge-Krenke & Kollmar, 1998). It was further hypothesized that (4) higher levels of parent depressive, anxiety, and stress symptoms would be associated with greater informant discrepancies regarding adolescent symptoms. Given the limited and somewhat equivocal findings of past research, analyses of sex differences remained exploratory.

Method

Participants and Procedure

The study was approved by the institutional ethics committee, the Victorian Department of Education and Training, and the Melbourne Catholic Education Office. Participants were recruited through two private and four public secondary schools and advertisements in university e-mail bulletins. Voluntary informed consent was given in writing by parents and adolescents. No compensation was provided for participation.

Two hundred seventy-seven adolescents completed written questionnaires in one sitting either in groups at school (84%) or at home. Parents were posted written questionnaires to complete at home and return by mail. Two-hundred nineteen adolescents had at least one parent who also completed questionnaires (212 mothers, 156 fathers) and were therefore included in the current study. Of these, 149 adolescents had two parents who completed questionnaires. Two-tailed *t*-tests indicated there were no statistical differences on any of the adolescent measures between adolescents whose parents participated and those who did not ($p > .05$).

The demographic characteristics of the sample are shown in Table 1. Occupational prestige was based on parents' most recent paid job and scored using the Australian Bureau of Statistics' Australian Standard Classification of Occupations (Australian Bureau of Statistics, 1997) together with the Australian National University Fourth Edition (ANU4) scale (Jones & McMillan, 2001). The ANU4 ratings range from 0 to 100, with higher scores reflecting greater occupational prestige. As can be seen in Table 1, the adolescent sample was mostly Australian born and of middle to high socioeconomic status as indicated by parental education, occupation, and income.

Measures

Adolescent internalizing symptoms. Adolescents reported their levels of depressive and anxiety symptoms using the Reynolds Adolescent Depression Scale-Second

Table 1
Characteristics of the Sample

	Adolescents	Mothers	Fathers
N	219	212	156
Male adolescent, *n* (%)[a,b]	96 (44)	92 (43)	64 (41)
Female adolescent, *n* (%)[b]	123 (56)	120 (57)	92 (59)
Age in years, *M* (*SD*)	15.7 (1.1)	45.7 (5.2)	48.0 (5.9)
Age, range	13.1–18.7	29.4–66.8	33.8–78.0
Australian born, *n* (%)	200 (93)	172 (81)	115 (74)
Tertiary degree or higher, *n* (%)	–	70 (33)	65 (42)
Occupational prestige, *M* (*SD*)	–	53.6 (23.8)	59.6 (21.9)
Occupational prestige range	–	12.4–100	7.2–100
Income ≥ AUD$75,000, *n* (%)	–	57 (37)	57 (37)
Relationship to adolescent, *n* (%)			
Biological parent	–	203 (96)	145 (93)
Step-parent	–	5 (2)	8 (5)
Other[c]	–	4 (2)	3 (2)

[a]Valid percent used throughout table.
[b]Indicates sex of target adolescent, for example, 43% were mothers of male adolescents.
[c]Included adoptive parents, foster parents, and grandparents.

Edition (RADS-2; Reynolds, 2002) and the Revised Children's Manifest Anxiety Scale (RCMAS; C.R. Reynolds & Richmond, 1985), respectively. The RADS-2 consists of 30 statements rated on a 4-point scale indicating the frequency with which depressive symptoms are experienced (1 = *almost never*, 4 = *most of the time*). It has been reported to have high levels of internal consistency ($\alpha = .92$) and high levels of test-retest reliability over 2 weeks ($r = .77$ to .84; Reynolds, 2002). Validity of the RADS-2 has been supported by its high correlations with other measures of depression, moderate to high correlations with measures of adolescent well-being (e.g. suicidal ideation, anxiety, self-esteem), and weak correlations with measures of social desirability and academic attainment (Reynolds, 2002). In the current study, the internal consistency coefficient was .94.

The RCMAS comprises 28 items assessing anxiety symptoms. Adolescents indicate whether or not the item is true of them (0 = no, 1 = yes). It has been reported to have high levels of internal consistency ($\alpha = .80$–.83) and high levels of test-retest reliability over 3 weeks ($r = .98$; C.R. Reynolds & Richmond, 1985). The validity of the RCMAS as a measure of chronic anxiety has been supported by its high correlations with measures of child trait anxiety and nonsignificant correlations with measures of child state anxiety (C.R. Reynolds & Richmond, 1985). In the current study, the internal consistency coefficient was .86.

Parents reported on levels of adolescent internalizing symptoms using the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). The CBCL internalizing scale comprises 26 items canvassing three types of internalizing behaviors: anxious/depressed, withdrawn/depressed, and somatic complaints. Parents rate each behavior on a 3-point scale indicating how true it is of their child (0 = *not true*, 1 = *somewhat or sometimes true*, 2 = *very true or often true*). The scale has been reported to have high levels of internal consistency ($\alpha = .90$) and good test-retest reliability over 8 days ($\alpha = .91$) to 2 years ($\alpha = .70$; Achenbach & Rescorla, 2001). Extensive validation studies of the CBCL have found it to discriminate well between clinically referred and nonreferred children, and to correlate highly with other measures of child and adolescent well-being (Achenbach & Rescorla, 2001). In the current study, the internal consistency coefficients were .91 (mother) and .89 (father).

The CBCL, RADS-2, and RCMAS raw scores were converted into sex-specific *T*-scores using information from the publisher's manual for each measure. RADS-2 and RCMAS T-scores were highly correlated ($r = .74$, $p < .001$) and thus were averaged to produce one score for adolescent-reported internalizing symptoms, which correlated highly with the original scores (RADS-2: $r = .93$, $p < .001$; RCMAS: $r = .94$, $p < .001$). The use of *T*-scores allowed for the description of informant agreement and discrepancies in reported levels of adolescent internalizing symptoms relative to population norms on a metric frequently used in clinical and research practice.

Parent symptoms. Parent symptoms were measured using the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995b). This measure consists of 42 statements reflecting symptoms of depression, anxiety, and stress in adults. Each statement is rated on a 4-point scale indicating how much the statement applied to the parent during the past week (0 = *did not apply to me at all*, 3 = *applied to me very much or most of the time*). The DASS has been reported to have high internal consistency ($\alpha = .84$–.91; Lovibond & Lovibond, 1995b), and good test-retest reliability over 2 weeks in clinical samples ($r = .71$–.81; Brown, Chorpita, Korotitsch, & Barlow, 1997). The validity of the depression and anxiety scales has

been supported by their high correlations with the Beck Depression Inventory ($r = .74$) and Beck Anxiety Inventory ($r = .81$), respectively (Lovibond & Lovibond, 1995a), and the tendency for higher mean scores to be reported for clinical samples compared to nonclinical samples (Antony, Bieling, Cox, Enns, & Swinson, 1998). The stress scale represents a persistent state of overarousal and low frustration tolerance (Lovibond & Lovibond, 1995b). Although there is no direct diagnostic equivalent, the stress scale correlates highly with measures of depression, anxiety, and negative affect ($r = .54$ to .72; Crawford & Henry, 2003). In the current study, the internal consistency coefficients were .95 and .93 (depression), 90 and .84 (anxiety), and .92 and .92 (stress) for mothers and fathers, respectively.

Results

Informant Agreement

Table 2 presents the mean scores for parent and adolescent reports of adolescent symptom levels. A 2×3 (Adolescent Sex $\times$ Informant) mixed design analysis of variance (ANOVA) indicated no significant differences in mean scores between reports of sons and daughters, $F(1,146) = 0.29$, $p > .05$, or between reports by adolescents, mothers, and fathers, $F(2,292) = 1.77$, $p > .05$. There was also no significant interaction effect between adolescent sex and informant, $F(2,292) = 1.02$, $p > .05$.

Pearson's product moment correlations between parent and adolescent reports are presented in Table 3.[1] Correlations between parent and adolescent reports were all significant ($p < .01$) and mostly in the moderate range as expected. Parent–son agreement ($r = .44$ and .50) appeared stronger than parent–daughter agreement ($r = .28$ and .39). Mother–father agreement appeared stronger than parent–adolescent agreement, being in the high range for sons ($r = .66$) and the moderate range for daughters ($r = .46$). Differences in correlations within son and daughter subgroups were tested using William's t test for dependent correlations (Steiger, 1980). As some adolescents did not have data from both parents, there were different sample sizes for mother–adolescent and father–adolescent dyads. William's t test does not allow for different sample sizes, thus only cases with data from all three informants were analyzed (i.e., using listwise deletion of missing cases). With regard to the hypothesis that mother–father agreement would be greater than parent–adolescent agreement, only one comparison was significant: mother–father agreement was significantly stronger than mother–son agreement, $t(57) = 3.65$, $p < .001$, one-tailed, Cohen's $d = .67$. In addition, father–son agreement was significantly stronger than mother–son agreement, $t(57) = 2.35$, $p < .05$, two-tailed, Cohen's $d = .43$. Exploratory analysis of informant agreement for sons compared to daughters using Fisher's z test for independent correlations with all possible cases included indicated no significant differences ($p > .05$, two-tailed).

Informant Discrepancies

Discrepancy scores were calculated for each pair of informants using symptom T-scores. Adolescent self-reported internalizing symptoms were subtracted from mother-reported adolescent internalizing symptoms. A negative mother–adolescent discrepancy score therefore indicated that mothers reported lower levels of

[1]Interclass correlations were also computed to check for additive and multiplicative bias as suggested by Richters (1992), however, the coefficients were near identical so are not reported here.

Table 2
Descriptives of Parent and Adolescent Reports of Adolescent Symptoms and Parent Psychopathology

	Adolescent report	Mother report	Father report
Sons			
n	93	92	64
M (*SD*)	48.5 (9.5)	47.8 (11.2)	48.6 (11.9)
Range (min, max)	29.0, 70.0	32.0, 74.0	32.0, 76.0
Daughters			
n	124	120	92
M (*SD*)	49.2 (10.4)	49.2 (11.5)	46.5 (10.2)
Range (min, max)	26.0, 76.0	31.0, 82.0	31.0, 79.0

symptoms than adolescents, whereas a positive score indicated that mothers reported higher levels of symptoms than adolescents. Adolescent self-reported internalizing symptoms were subtracted from father-reported adolescent internalizing symptoms to produce the same direction of scores for father–adolescent discrepancy. Finally, father-reported adolescent internalizing symptoms were subtracted from mother-reported adolescent internalizing symptoms. Hence, a negative mother–father discrepancy score indicated that mothers reported lower levels of symptoms than fathers; a positive score indicated that mothers reported higher levels of symptoms than fathers.

Descriptive statistics for discrepancy scores are shown in Table 4. As can be seen, there were wide variations in scores, with discrepancies ranging from −39 to +30. A 2 × 3 (Adolescent Sex × Informant Dyad) mixed design ANOVA indicated no significant differences in mean discrepancy scores between sons and daughters, $F(1,146) = 2.24$, $p > .01$, or between dyads, $F(2,292) = 1.30$, $p > .01$. There was also no significant interaction effect between adolescent sex and informant dyad, $F(2,292) = 1.15$, $p > .01$. Although mean discrepancy scores approximated zero (range = −1.8–1.4), indicating close agreement overall, inspection of the distribution of scores indicated that approximately one third of discrepancy scores exceeded one *T*-score standard deviation (i.e., ∓ 10); 37% and 38% for mother–adolescent discrepancy, 32% and 43% for father–adolescent discrepancy, and 23% and 30% for mother–father discrepancy, for sons and daughters, respectively.

The skewness and kurtosis indices indicated that discrepancies were normally distributed. Thus, there was no clear pattern to suggest that one type of informant consistently reported higher or lower levels of symptoms than another. There were moderate to large significant intercorrelations between discrepancy scores, suggesting that greater discrepancies between one dyad were associated with greater discrepancies between other dyads.[2] This reinforced the need to control for discrepancies between other dyads in subsequent analyses.

Parent Symptoms and Reports of Adolescent Symptoms

Mean scores on the DASS were 3.3 ($SD = 5.8$; depressive), 1.9 ($SD = 4.1$; anxiety), 6.3 ($SD = 6.0$; stress) for mothers, and 3.1 ($SD = 4.8$; depressive), 1.6 ($SD = 2.8$;

[2]The negative correlation between father-adolescent and mother-father discrepancies is a function of the way discrepancies were calculated and does not represent any variation in the relationships between discrepancies.

Table 3
Pearson's Correlations Between Parent and Adolescent Reports of Adolescent Symptoms and Parent Psychopathology

		Sons' symptoms			Daughters' symptoms		
		Adolescent report	Mother report	Father report	Adolescent report	Mother report	Father report
Adolescent symptoms	Mother report[a]	.44** (.30**)	–		.39*** (.32**)	–	
	Father report[a]	.50*** (.52***)	.66*** (.66***)	–	.28** (.28**)	.46*** (.46***)	–
Mother symptoms	Depressive	.15	.45***	.27*	.28**	.23**	.26**
	Anxiety	.08	.25**	.22*	.15*	.18*	.24*
	Stress	.13	.40***	.16	.23**	.26**	.29**
Father symptoms	Depressive	.16	.21	.25*	.10	.04	.18*
	Anxiety	.12	.29*	.38**	.10	−.02	.23*
	Stress	.24*	.25*	.43***	.16	.11	.33**

[a]See Table 4 for *n* per dyad. Correlations in parentheses are with listwise deletion of missing data (sons, $n = 60$; daughters, $n = 88$).
*$p < .05$; **$p < .01$; ***$p < .001$, one-tailed.

Table 4
Descriptives and Pearson's Correlations of Discrepancies Between Informants of Adolescent Symptoms

	Sons' discrepancies			Daughters' discrepancies		
	Mother–adolescent	Father–adolescent	Mother–father	Mother–adolescent	Father–adolescent	Mother–father
n	90	63	61	120	92	88
M (*SD*)	−0.7 (11.0)	−0.9 (10.9)	−1.3 (9.3)	0.2 (12.0)	−1.8 (12.3)	1.4 (11.0)
Range (min, max)	−29.5, 27.0	−22.0, 29.0	−26.0, 19.0	−39.0, 30.0	−27.5, 27.5	−29.0, 30.0
Skewness (SE)	−0.20 (.25)	0.53 (.30)	−0.40 (.31)	−0.23 (.22)	0.43 (.25)	0.16 (.26)
Kurtosis (SE)	0.11 (.50)	0.83 (.60)	0.74 (.60)	0.35 (.44)	−0.28 (.50)	0.40 (.51)
Mother–adolescent	–			–		
Father–adolescent	.65***	–		.60***	–	
Mother–father	.51***	−.33**	–	.45***	−.45***	–

**$p < .01$; ***$p < .001$, one-tailed Pearson's correlations.

anxiety), 6.7 ($SD = 5.9$; stress) for fathers. These equated to mean percentile scores of 44, 46, 39, 43, 45, and 42, respectively (Crawford & Henry, 2003). As can be seen in Table 3, there were significant positive correlations between parent and adolescent symptoms, particularly within informant. That is, mother self-reported symptoms correlated with mother-reported adolescent symptoms, and father self-reported symptoms correlated with father-reported adolescent symptoms. Mother self-reported symptoms also correlated significantly with daughter, but not son self-reported symptoms, and father self-reported stress symptoms correlated significantly with son self-reported symptoms.

Parent Symptoms and Informant Discrepancies

To examine the contribution of parent symptoms to discrepancies between informant reports of adolescent symptom levels, a series of hierarchical regression analyses was undertaken with mother–adolescent, father–adolescent, and mother–father discrepancies, in turn, as the dependent variable. Prior to analysis, discrepancies were recalculated following the conversion of adolescent and parent reports of adolescent symptoms into *z* scores as recommended by De Los Reyes and Kazdin (2004). Without conversion, De Los Reyes and Kazdin have demonstrated that results can be unduly influenced by one informant.

In each regression analysis, discrepancy within the other dyad was entered in the first step to account for other shared sources of discrepancies. Parent depressive, anxiety, or stress symptoms were then entered in the second step. For example, in the first analysis mother–son discrepancy was entered as the dependent variable, father–son discrepancy was entered as the first predictor followed by mothers' depressive symptoms. The regressions predicting father–adolescent discrepancy from fathers' symptoms similarly controlled for mother–adolescent discrepancy, whereas those predicting mother–father discrepancy from mothers' symptoms and fathers' symptoms controlled for father–adolescent discrepancy and mother–adolescent discrepancy, respectively. Separate analyses were conducted for parent depressive, anxiety, and stress symptoms due to strong intercorrelations between these variables ($r = .60$ to $.81$, $p < .001$). This also allowed for the effect of each type of symptom to

be evaluated independently rather than utilizing a combined score. All analyses were conducted separately for sons and daughters.

Mothers' symptoms. Overall, the significant models explained between 11 and 54% of the variance in informant discrepancies (see Table 5). Only the model predicting mother–father discrepancies for sons from mother anxiety symptoms was not significant. Discrepancy within the other dyad contributed a significant amount of unique variance in each analysis (24 to 51%) except for the analyses predicting mother–father discrepancy for sons. Mothers' depressive and stress symptoms each explained additional variance in the discrepancy between mother and son reports (4% each) and in the discrepancy between mother and father reports of sons' symptoms (7% each). Thus, mothers who reported experiencing higher levels of depressive or stress symptoms tended to report higher levels of internalizing symptoms in their sons compared to levels reported by sons and fathers. Mothers' symptoms did not explain any additional variance in the discrepancy between mother and daughter reports, nor between mother and father reports of daughters' symptoms. Mothers' anxiety symptoms were not significant in any analysis.

Fathers' symptoms. Overall, the models explained between 15 and 53% of the variance in informant discrepancies (see Table 6). Discrepancy within the other dyad contributed a significant amount of unique variance in each analysis (15 to 51%). Fathers' depressive, anxiety, and stress symptoms did not explain any additional variance in informant discrepancies regarding sons' symptoms. However, fathers' anxiety and stress symptoms explained additional variance in the discrepancy between father and daughter reports (3% and 4%, respectively) and in the discrepancy between father and mother reports of daughters' symptoms (5% each). Thus, fathers who reported experiencing higher levels of anxiety and stress symptoms tended to report higher levels of internalizing symptoms in their daughters compared to levels reported by daughters and mothers. Fathers' depressive symptoms were not significantly related to discrepancies regarding daughters' symptoms.[3]

Discussion

Consistent with past research (e.g., Achenbach et al., 1987; Briggs-Gowan et al., 1996; Seiffge-Krenke & Kollmar, 1998), the current study found notable differences between parent and adolescent reports of adolescent internalizing symptom levels. First, as expected, there were typically only moderate levels of agreement between parent and adolescent reports of adolescent internalizing symptoms, and somewhat higher agreement between mother and father reports. Dyad comparisons indicated that mother–father agreement and father–son agreement were both significantly stronger than mother–son agreement. Further, although informant agreement tended toward being stronger for sons compared to daughters, differences were not significant. Second, although the overall mean discrepancies between informant reports of adolescent internalizing levels were small, the distribution of discrepancies indicated that there were large differences between some informant reports. These results suggest that although some parents and adolescents agree in their reports of

[3]When the three types of parent symptoms were combined into a single composite score, father symptoms significantly predicted father–daughter discrepancy and mother–father discrepancy regarding daughters. Mother symptoms did not significantly predict discrepancies in any analyses. Details available from the authors.

Table 5
Hierarchical Regression Analyses Testing the Associations Between Mothers' Symptoms and Discrepancies Between Informant Reports of Adolescent Internalizing Symptoms

Step	Predictor variables	*B* (*SE*)	*β*	*t*	ΔR^2	ΔF	R^2	*F*
				Mother–son discrepancy				
1	Father–son discrepancy	.83 (.11)	.70	7.48***	.51	59.15***	.51	59.15***
2[a]	Mother depressive symptoms	.07 (.03)	.20	2.09*	.04	4.30*	.54	33.41***
	Mother anxiety symptoms	.00 (.05)	−.01	−0.07	.00	0.00	.51	29.07***
	Mother stress symptoms	.04 (.02)	.20	2.13*	.04	4.52*	.54	33.63***
				Mother–daughter discrepancy				
1	Father–daughter discrepancy	.58 (.08)	.61	7.13***	.38	52.08***	.38	52.08***
2[a]	Mother depressive symptoms	−.01 (.02)	−.06	−0.75	.00	0.56	.38	26.19***
	Mother anxiety symptoms	−.04 (.03)	−.12	−0.14	.01	1.89	.39	26.00***
	Mother stress symptoms	−.01 (.02)	−.05	−0.58	.00	0.33	.38	17.45***
				Mother–father discrepancy (sons)				
1	Father–son discrepancy	−.17 (.11)	−.21	−1.60	.04	2.79	.04	2.56
2[a]	Mother depressive symptoms	.07 (.03)	.27	2.07*	.07	4.36*	.11	3.50*
	Mother anxiety symptoms	.00 (.05)	−.01	−0.07	.00	0.01	.04	1.26
	Mother stress symptoms	.04 (.02)	.27	2.13*	.07	4.52*	.11	3.62*
				Mother–father discrepancy (daughters)				
1	Father–daughter discrepancy	−.42 (.08)	−.49	−5.23***	.24	26.28***	.24	27.35***
2[a]	Mother depressive symptoms	−.01 (.02)	−.07	−0.75	.01	0.56	.25	13.88***
	Mother anxiety symptoms	−.04 (.03)	−.13	−1.38	.02	1.89	.26	14.76***
	Mother stress symptoms	−.01 (.02)	−.05	−0.57	.00	0.33	.24	13.73***

[a]Separate regressions were conducted for each symptom measure in Step 2. Step 1 is identical for each analysis so is not repeated here.
*$p < .05$; **$p < .01$; ***$p < .001$.

Table 6
Hierarchical Regression Analyses Testing the Associations Between Fathers' Symptoms and Discrepancies Between Informant Reports of Adolescent Internalizing Symptoms

Step	Predictor variables	*B* (*SE*)	*β*	*t*	ΔR^2	ΔF	R^2	*F*
				Father–son discrepancy				
1	Mother–son discrepancy	.61 (.08)	.71	7.69***	.51	59.15***	.51	59.15***
2[a]	Father depressive symptoms	.00 (.03)	.01	0.09	.00	0.01	.51	29.08***
	Father anxiety symptoms	.07 (.04)	.15	1.64	.02	2.67	.53	31.77***
	Father stress symptoms	.03 (.02)	.16	1.81	.03	3.12	.53	32.37***
				Father–daughter discrepancy				
1	Mother–daughter discrepancy	.65 (.09)	.61	7.22***	.38	52.08***	.38	52.08***
2[a]	Father depressive symptoms	.02 (.02)	.11	1.25	.01	1.57	.39	27.00***
	Father anxiety symptoms	.07 (.03)	.19	2.23*	.03	4.98*	.41	29.73***
	Father stress symptoms	.04 (.02)	.19	2.27*	.04	6.81*	.41	29.86***
				Mother–father discrepancy (sons)				
1	Mother–son discrepancy	.39 (.08)	.54	4.82***	.29	24.20***	.29	24.20***
2[a]	Father depressive symptoms	.00 (.03)	−.01	−0.09	.00	0.01	.30	11.90***
	Father anxiety symptoms	−.07 (.04)	−.18	−1.64	.03	2.67	.33	13.79***
	Father stress symptoms	−.03 (.02)	−.20	−1.81	.04	3.27	.33	14.21***
				Mother–father discrepancy (daughters)				
1	Mother–daughter discrepancy	.35 (.09)	.39	3.88***	.15	15.04***	.15	15.04***
2[a]	Father depressive symptoms	−.02 (.02)	−.13	−1.25	.02	1.57	.16	8.35***
	Father anxiety symptoms	−.07 (.03)	−.22	−2.23*	.05	4.98*	.20	10.35***
	Father stress symptoms	−.04 (.02)	−.22	−2.27*	.05	5.14*	.20	10.45***

[a]Separate regressions were conducted for each symptom measure in Step 2. Step 1 is identical for each analysis so is not repeated here.
*$p < .05$; **$p < .01$; ***$p < .001$.

adolescent internalizing symptom levels, there are many others who do not. However, in contrast to previous studies (Luoma et al., 2004; Seiffge-Krenke & Kollmar, 1998; Sourander et al., 1999), the discrepancy distributions revealed no clear patterns to suggest that one type of informant reported higher or lower adolescent symptom levels relative to other types of informants.

As expected, parent symptoms were positively correlated with their reports of adolescent symptoms. Moreover, parent symptoms were associated with discrepancies in reports of adolescent internalizing symptom levels. However, the amount of variance accounted for was small, and findings varied by sex and parent symptom type. Overall, mothers' depressive and stress symptoms were associated with greater discrepancies in reports regarding sons but not daughters, whereas fathers' anxiety and stress symptoms were associated with greater discrepancies in reports regarding daughters but not sons. The findings regarding parent stress symptoms are particularly noteworthy given that past studies of parent stress are scarce and have tended to focus on maternal role-related stress rather than general stress (Martin et al., 2004; Youngstrom et al., 2000).

The findings regarding parent depressive and anxiety symptoms are consistent with some previous studies (Briggs-Gowan et al., 1996; Jensen et al., 1988), but contrast with others (Chilcoat & Breslau, 1997; Ivens & Rehm, 1988; Krain & Kendall, 2000; Najman et al., 2000). However, the comparability of findings across studies is limited. For instance, unlike the current study and others (Briggs-Gowan et al., 1996; Jensen et al., 1988), studies that reported significant findings where this study did not, have tended to utilize clinical samples (Ivens & Rehm, 1988; Krain & Kendall, 2000) or did not examine findings by child sex (Chilcoat & Breslau, 1997; Ivens & Rehm, 1988; Najman et al., 2000). This suggests parent symptomatology may be more strongly related to reports of child symptoms in clinical populations. It also highlights the need to further examine parent and child sex differences, including potential underlying causes of such differences, for example, the ways that sons and daughters express symptoms to their parents, the sequelae of psychopathology for mothers compared to fathers, and the relationship characteristics of same- and opposite-sex parent-child dyads.

In general, significant associations between parent psychopathology and discrepancies in reports of child and adolescent symptom levels have been put forth as support for the assertion that psychopathology distorts parents' perceptions of their children's symptomatology. Such distortions may be due to mood-congruent perception, appraisal, and recall (Chilcoat & Breslau, 1997) or other psychological processes such as projection (Briggs-Gowan et al., 1996). Alternatively, parent psychopathology might reduce tolerance for problem behaviors, such that parents, who experience high levels of stress, worry, and hopelessness, label age-appropriate or normative behaviors as symptomatic (Briggs-Gowan et al., 1996; Chilcoat & Breslau, 1997). Notably, the significant findings related to parent stress in the current study provide some support for this last explanation. Nonetheless, firm conclusions regarding causality cannot be inferred from the relationships observed herein. For example, it may be that parents perceived higher levels of adolescent symptoms for other reasons, and this perception in turn impacted upon parent symptomatology. Furthermore, it has been argued that parents affected by psychopathology may actually be more accurate informants, such that their tendency to report higher levels of child symptoms is a result of a "real" relationship between parent and child psychopathology combined with affected parents' heightened sensitivity to, and knowledge about, such issues relative to nonaffected informants (Briggs-Gowan

et al., 1996; Richters, 1992; Tarullo et al., 1995). Although this is a feasible explanation for the relationships observed in this and other studies, given the lack of a gold standard for assessing adolescent psychopathology, it is a difficult hypothesis to test.

Although the findings point to a potentially important source of informant discrepancies in reports of adolescent internalizing symptoms, much of the variance in informant discrepancies remained unexplained, and parent symptoms accounted for only a small amount of variance in informant discrepancies (3 to 7%). Thus, it remains possible that parent symptoms may not be a critical influencing factor in informant reports, particularly with nonclinical samples. It is noteworthy that in all but one model, the largest proportion of explained variance was accounted for by discrepancies in the other dyad. This suggests a need to examine factors that are common across informant dyads. Additionally, the distribution of discrepancies suggests that it is necessary to examine factors likely to result in reporting of lower levels of adolescent symptoms relative to other informants as well as those that may result in reporting of higher levels. It is also likely that there are some factors common to both parents and adolescents including social desirability, cognitive attribution style, tolerance levels, and response style.

Further to the issues already raised, there are a number of limitations relevant to the current study that must be acknowledged. First, given the use of nonparallel measures, the observed discrepancies between parent and adolescent reports may in part be due to differences in measure wording and content. However, the study did not aim to make symptom by symptom comparisons, but to examine reported levels of symptomatology. All measures have been demonstrated to be reliable and valid measures of the target construct (i.e., adolescent internalizing symptoms levels) and the use of standardized T-scores allowed for valid comparisons of this construct between parents and adolescents. Nonetheless, future research would benefit from the inclusion of parallel measures, particularly structured clinical interviews, which are designed for both parent and child report and allow for more in-depth analysis of specific internalizing symptoms (e.g., Anxiety Disorders Interview Schedule, C.R. Reynolds & Kamphaus, 2004; or the Behavior Assessment System for Children, Silverman & Albano, 1996).

Second, although there was a wide range of scores on the symptom measures, the sample was recruited from the community, was generally of middle to high socioeconomic status, and was not asked about prior psychological diagnoses or treatment. It may be that participants with elevated levels of symptoms were underrepresented or were of insufficient number to reveal significant effects. Notably, mean parent symptoms scores fell below the 50th percentile. Further, in clinical samples, child and adolescent symptomatology may be more overt, thus leading to greater informant agreement. However, even in clinically referred samples parent–child agreement tends to be low (Edelbrock et al., 1986; Grills & Ollendick, 2003). Nonetheless, differences between clinical and nonclinical populations could also alter interactions between parent and adolescent symptomatology. Clearly, generalization of the current findings to clinical populations should be made with caution, and future research should consider how these populations may differ both quantitatively and qualitatively.

Finally, although the total sample was large, analyzing the data by parent and adolescent sex reduced sample sizes, particularly for father–son dyads. In addition, despite the large number of analyses, Bonferroni corrections were not made so as to maximize the likelihood of detecting relationships given the relatively novel aspects of the study (e.g., stress, sex) and the reduced power in some analyses. However, this

may have increased the chance of Type I error. Replication of the study in larger and more diverse research samples is therefore recommended.

These issues aside, the study and others in this area have important implications for the use of multiple informants. Given the inherent issues of the multi-informant approach, the value of including other informants in the assessment of adolescents must be carefully considered by clinicians and researchers. In clinical settings there are obvious advantages in collecting information from multiple informants regarding adolescent, and possibly even adult, functioning (cf. Achenbach, Krukowski, Dumenci, & Ivanova, 2005). However, clinicians adopting such an approach must remain cognizant of informant discrepancies and methods for combining information from multiple informants (e.g., Piacentini, Cohen, & Cohen, 1992). Studies have found that, more often than not, treatment begins without clinicians, parents, and children agreeing on a single target problem (Hawley & Weisz, 2003). Such lack of agreement may impact upon the success of treatment and its long-term outcomes.

The use of multiple informants in adolescent research is more complex. In many cases there is often clear theoretical justification for multi-informant data. For example, prevalence studies typically include multiple informants when there is no clear consensus favoring diagnoses based on one informant over another. However, the inclusion of multiple informants in other studies may be questionable. For example, including multiple informants for the purpose of providing additional support for hypotheses via the replication of findings across informants can be misleading. Specifically, given demonstrated discrepancies across informants, hypotheses are not necessarily weakened when replication of findings in such studies is not yielded. Critical to the decision to include multiple informants in research is the nature of the research question and the construct under investigation. For example, if the focus is on the impact of an individual's symptoms on their family, the family members' perceptions of the individual's symptoms may be as, if not more, important than the individual's perception of their symptoms. Thus, a multi-informant approach would be justified. In contrast, a validation study of an adolescent-report measure may not be improved by the inclusion of a parent-report version, as one would only expect low to moderate correlations between adolescent and parent reports, particularly if the construct being measured is not directly observable.

Researchers who decide to include multiple informants need to carefully consider how they will analyze resulting data. Although methods for combining informant reports are available (Achenbach & Rescorla, 2001; Kraemer et al., 2003; Piacentini et al., 1992), the richness of information may be lost and some research questions may be more effectively answered by examining informant reports separately. Researchers should also consider the extent to which their selected sample may be affected by factors related to informant discrepancies. Future research is needed to shed more light on the specific factors that should be considered but may include, for example, age, sex, parent psychopathology, relationship quality, and communication.

In conclusion, the study findings provide further support for the contribution of parent psychological symptoms in observed discrepancies between adolescent, mother, and father reports of adolescent internalizing symptom levels. The study has extended previous research by investigating three types of parent symptoms and examining relationships across mothers, fathers, sons, and daughters. Future research should focus on identifying the types of parent psychopathology that are of most importance and for whom, and further understanding the processes through

which parent psychopathology may affect parent reports. Research also needs to examine other factors which may be of equal, if not greater, importance to understanding informant reports. Theoretical models of informant discrepancies are beginning to be developed (e.g., De Los Reyes & Kazdin, 2005), which will hopefully generate further research and advance our knowledge of the factors which influence both parent and adolescent reports.

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