Brief Report

Family History of Psychological Problems in Generalized Anxiety Disorder

Katie A. McLaughlin*, Evelyn Behar, and T.D. Borkovec
Penn State University

The current investigation examined self-reported family history of psychological problems in a large sample of individuals diagnosed with generalized anxiety disorder (GAD) and nonanxious controls. The GAD participants were all individuals receiving cognitive–behavioral therapy as part of two large randomized clinical trials. Family history information was obtained from the Anxiety Disorders Interview Schedule-Revised (ADIS-R; DiNardo & Barlow, 1988). The results indicate that, compared to control participants, individuals with GAD were more likely to have family members with anxiety problems, but not other psychological problems. Possible mechanisms for the familial transmission of GAD are discussed. © 2008 Wiley Periodicals, Inc. J Clin Psychol 64: 905–918, 2008.

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The development of virtually every type of psychopathology involves factors operating at the biological, psychological, and social levels. Behavior genetics research has become an invaluable tool in identifying the relative contributions of genetic and environmental factors to the etiology of mental disorders. As research in behavior genetics has progressed in the last several decades, it has become increasingly clear that genetic factors play a considerable role in the development of psychopathology (see Rutter, 2002; Turkheimer, 2000). However, generalized anxiety disorder (GAD) represents a mental disorder for which genetics have not been found to play a substantial role. As such, identification of environmental factors associated with the development of GAD represents an important area for

*Katie McLaughlin is now at the Department of Psychology, Yale University, New Haven, CT.

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Correspondence concerning this article should be addressed to: Katie McLaughlin, Department of Psychology, Yale University, P.O. Box 208205, New Haven, CT 06520; e-mail: katie.mclaughlin@yale.edu

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research. The current study utilizes the family history method to identify specific environmental risk factors within families that are associated with GAD.

Among the anxiety disorders, behavior genetics research has revealed a strong genetic component to the etiology of panic disorder (Kendler et al., 1995), and prospective studies have identified specific psychological risk factors (e.g., anxiety sensitivity; McNally, 2002; Schmidt, Lerew, & Jackson, 1999). In contrast, evidence suggests that genetic factors do not play as prominent a role in the etiology of GAD. A number of twin studies have been conducted examining the relative contribution of genetic and environmental factors in the etiology of GAD. The first of these studies found that genetic factors were largely not involved in the transmission of GAD (Torgersen, 1983). A twin study conducted by Kendler, Neale, Kessler, Heath, and Eaves (1992) that included a large number of female twin pairs also reported low heritability of GAD (19%–30%). Similarly, a twin study using both male and female twin pairs estimated the role of genetics to be modest, in the 15%–20% range (Hettema, Prescott, & Kendler, 2001). A somewhat higher, but still moderate, estimate of the heritability of GAD (40%) was found in a study of middle-aged male twin pairs taken from the Vietnam Era Twin Registry (Scherrer et al., 2000). Taken together, these studies indicate that genetic factors may play some role in the etiology of GAD, but that environmental factors play a predominant role.

Evidence consistently suggests that GAD aggregates in families, despite the modest contribution of genetic factors. A meta-analysis on the genetic epidemiology of anxiety disorders, which took into account relevant family history and twin studies, confirmed that GAD aggregates in families and that genetics play only a minor role in this aggregation (Hettema, Neale, & Kendler, 2001). The aggregation of GAD in families despite a lack of strong genetic influences suggests that environmental influences within families, such as parental psychopathology or family dynamics, may be involved in its etiology. Recent prospective data from a representative birth cohort revealed a number of environmental risk factors measured in childhood that were uniquely associated with future onset of GAD, but not depression, in adulthood (Moffitt et al., 2007). The majority of the risk factors identified were related to quality of the home environment. Specifically, childhood adversity (including low socioeconomic status and maltreatment), and maternal internalizing symptoms were associated with GAD onset (Moffitt et al., 2007). Importantly, these factors were found to be specifically associated with the development of GAD and did not represent global risk factors for any type of psychopathology. These findings are consistent with evidence suggesting a role for familial environmental factors in the etiology of GAD.

A number of familial factors may be associated with liability for GAD. Of these, familial psychopathology may be a particularly potent risk factor for GAD onset. Specifically, internalizing symptoms in family members may predispose individuals to developing GAD through a variety of mechanisms, including vicarious learning. The role of vicarious learning in the acquisition of fear is well documented in primates (e.g., Cook & Mineka, 1989), and stress reactivity in rats has been demonstrated to be transmitted to offspring through behavioral modeling (Francis, Diorio, Liu, & Meany, 1999). The importance of observational learning in the acquisition of fears in children has also been experimentally established (Gerull & Rapee, 2002). Moreover, a large community study reported that over 50% of children reported vicarious learning experiences to be responsible for the development of their fears (Ollendick & King, 1991). As such, vicarious learning may play a role in the transmission of anxiety, including worry and GAD, from

parents to children. Children of parents with an anxiety disorder are seven times more likely to develop an anxious condition than those with parents lacking an anxiety diagnosis (Turner, Beidel, & Costello, 1987), and parental expression of fears mediates the relationship between anxiety in children and anxiety in their mothers (Muris, Steerneman, Merckelbach, & Meesters, 1996). It is likely that a child who witnesses chronically anxious or worrisome behavior in a parent or important relative may begin to model that same behavior, perceiving many future threats and worrying about those threats. Anxious parents have also been found to reciprocate and reinforce threatening interpretations of ambiguous stimuli as well as avoidant responses in their children, as compared to nonanxious parents who reinforce prosocial plans (Barrett, Rapee, Dadds, and Ryan, 1996; Dadds, Barrett, Rapee, & Ryan, 1996). It is noteworthy that chronic worry and GAD are characterized by intolerance of uncertain or ambiguous situations (e.g., Dugas, Gagnon, Ladouceur, & Freeston, 1998). Parental modeling or reinforcement of avoidant responses in such situations may strengthen children's fears of, and avoidance responses to, uncertainty, which may eventually contribute to the development of GAD.

The aggregation of GAD within families has been found to be largely disorder-specific, suggesting that having a relative with GAD predisposes an individual specifically to developing GAD. However, in one study familial transmission of GAD was partially accounted for by parental major depression (Kendler, Davis, & Kessler, 1997). Generalized anxiety disorder in children was seen frequently in instances where parents had either GAD or major depression, or both. Moreover, individuals with GAD are more likely to have a family history of depression than individuals without the disorder (Reich, 1995). As such, increased vulnerability for developing GAD may be associated with presence of either parental anxiety or depression. A number of studies have found that GAD and major depression share a common genetic vulnerability (Kendler, 1996; Kendler, Neale, Kessler, & Heath, 1992; Kendler, Prescott, Myers, & Neale, 2003; Kendler, Gardner, Gatz, & Pedersen, 2007). Moreover, worry, the central defining feature of GAD, has been demonstrated to occur in all of the anxiety disorders (Barlow, 1988), as well as in major depression (Chelminski & Zimmerman, 2003). As such, a child may be exposed to a family environment in which negative affect and worry are prominent without having a family member who actually has GAD. Exposure to worry and chronic negative affectivity in one's parents may increase a child's likelihood of developing GAD through observational learning. It seems likely, therefore, that the presence of any anxiety or mood problems in a parent or important relative may contribute to the development of GAD.

On the other hand, the presence of externalizing types of psychopathology, such as substance abuse, among family members may also increase risk for GAD. Parents with alcohol and substance abuse problems have been consistently found to provide less predictable parenting and to create less predictable home environments for children than parents with no substance use problems (Johnson, 2002; Ross & Hill, 2004). Moreover, marital discord, divorce, and child abuse and neglect are more common in families with an alcohol-abusing parent (Johnson, 2001; Ker & Hill, 1992; Sher, Gershuny, Peterson, & Raskin, 1997), and children of alcoholic parents exhibit less secure attachments to caregivers than children of nonalcoholic parents (Eiden, Edwards, & Leonard, 2002). Lack of controllability over one's environment is a prominent contributor to anxiety (Barlow, 1988). Early experiences with lack of control over one's environment may lead to anxiety via an influence on locus of control, attributional style, and other cognitive vulnerabilities to anxiety

(see Chorpita & Barlow, 1998). As such, it is likely that individuals who are exposed to unpredictable and uncontrollable home environments as a results of parental substance abuse may also be predisposed to developing GAD.

The purpose of the present investigation was to determine whether individuals with GAD were more likely to have family members with psychological problems than individuals with no current or past history of psychopathology. To address this question, individuals with diagnosed GAD and individuals with no current or past psychopathology provided reports of history of psychopathology among their relatives. Self-reported family history of psychopathology has been found to provide useful and valid diagnostic information in prior research on familial aggregation of psychopathology. For example, self-reported family history of major depression was found to predict future episodes of depression among family members after controlling for the presence of a major depression diagnosis in family members based on personal interview (Kendler & Roy, 1995). We hypothesized that individuals with GAD, as compared to controls, would report having a higher number of family members with psychological problems overall. Specifically, we predicted that higher rates of anxiety and mood problems and substance abuse among family members would be present in individuals with GAD as compared to controls.

Method

Participants

The 151 individuals diagnosed with GAD (102 women, 49 men) and 76 controls (51 women, 24 men) were part of two larger treatment-outcome investigations of cognitive–behavioral therapy for GAD (Borkovec & Costello, 1993; Borkovec, Newman, Pincus, & Lytle, 2002). Of the GAD participants, 24 (19 women, 5 men) did not complete treatment. The mean age of the participants was 37.3 years ($SD = 11.9$). 88.5% of participants were Caucasian ($N = 201$), 4.0% were Black ($N = 9$), 4.4% were Hispanic ($N = 10$), 1.3% were of Middle Eastern descent ($N = 4$), and 1.8% were Indian ($N = 3$). Participants were recruited primarily through flyers and community advertisements in newspapers. Eighteen of the GAD participants were referred to the study by mental health professionals who had clinical practices in the study area. Local clinicians were given information about the study and asked to refer any patients that qualified for GAD and were interested in participating in a trial of CBT treatment. 69 (45.7%) of the participants with GAD met criteria for another anxiety disorder, and 17 (11.3%) met criteria for a mood disorder (major depression or dysthymia).[1] The entirety of the sample was recruited from the central Pennsylvania area, which is a predominantly lower middle-class to middle class area. The average educational attainment of the sample was 16.5 years ($SD = 2.2$). Data on income were not collected.

Diagnosis of GAD was determined using the Anxiety Disorders Interview Schedule-Revised (ADIS-R; DiNardo & Barlow, 1998); GAD status was confirmed by a second ADIS-R interview conducted by an independent assessor within 2 weeks of the initial diagnostic assessment. The mean duration of GAD was 14.83 years. Control participants were frequency-matched on age, gender, ethnicity, and education level to GAD participants such that one control participant was recruited

[1]The comorbidity rates for this sample were derived from data from Borkovec, Abel, and Newman (1995) and Newman, Przeworski, and Borkovec (2001).

for every 2 participants with GAD. A single ADIS interview was conducted with controls to ensure the absence of any past or current diagnosable conditions as well as the absence of any history of psychotherapy or pharmacological treatment.

Measures

The ADIS-R (DiNardo & Barlow, 1998) is a semistructured interview designed to assess the mood, anxiety, and somatoform disorders and to rule out the presence of other Axis I diagnoses (e.g., substance use and psychotic disorders). The ADIS-R was designed to assess the symptoms of anxiety and mood disorders in a more detailed fashion than other structured interviews, such as the Structured Clinical Interview for DSM-IV (SCID-I; First, Spitzer, Gibbon, & Williams, 1997). The ADIS-R is comprised of questions that assess each of the diagnostic criteria for all of the anxiety disorders (panic disorder, GAD, social phobia, specific phobia, obsessive–compulsive disorder, and posttraumatic stress disorder), the mood disorders (major depressive disorder, bipolar disorder, and dysthymia), and the somatoform disorders (somatization disorder, hypochondriasis, and body dysmorphic disorder). The ADIS-R also screens for current and past psychotic disorders and substance use disorders. Interviewers provide a severity rating for each diagnosis for which a participant has met diagnostic criteria on a 0 to 8 scale. The severity ratings reflect the amount of distress and functional impairment associated with each diagnosis and use the following anchors: 0 (*absent/none*), 2 (*mild; slightly disturbing/disabling*), 4 (*moderate; definitely disturbing/disabling*), 6 (*severe/markedly disturbing/disabling*), and 8 (*extreme; very disturbing/disabling*). In addition to assessing the *DSM* criteria, the ADIS-R also includes dimensional ratings (also on a 0–8 scale) of disorder features that aid in differential diagnosis and severity assessment. Interviewers rate fear and avoidance of numerous social situations in the social phobia section, excessiveness and difficulty controlling worry across a number of domains in the GAD section, persistence and distress of different types of obsessions and frequency of compulsions in the OCD section, and fear and avoidance of phobic objects and situations in the specific phobia section. The ADIS-R has demonstrated good to excellent interrater reliability for each of the anxiety and mood disorders (Brown & Barlow, 1992; Di Nardo, Moras, Barlow, Rapee, & Brown, 1993; Sanderson, DiNardo, Rapee, & Barlow, 1990). The interrater reliability and validity of anxiety and mood disorder diagnoses are comparable to those for other diagnostic interviews such as the SCID and the Schedule for Affective Disorders and Schizophrenia-Lifetime Anxiety Version (see Mannuzza et al., 1989; Williams et al., 1992). Moreover, this interview represents the gold standard for assessing the anxiety and mood disorders, is more comprehensive in assessment of anxiety and mood symptoms than other diagnostic interviews, such as the SCID, and has been used as the primary diagnostic measure in innumerable treatment outcome studies (e.g., Barlow, Craske, Cerny, & Klosko, 1989; Barlow, Rapee, & Brown, 1992), including the field trials for anxiety and mood disorders (e.g., Zinbarg et al., 1994) for the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*; American Psychiatric Association, 1994).

Procedure

Participants were enrolled over a 5-year period. Trained clinical assessors (advanced graduate students) administered the ADIS-R interviews, including the initial

diagnostic interview and the second interview to confirm diagnostic status. In the family history section of the ADIS-R, participants are asked to report the psychological problems of any immediate family members. Specifically, they are asked, "Has anybody in your family ever been treated or hospitalized for anxiety, depression, alcohol or drug abuse, or other such problems, or had any such problems but did not seek treatment?" The clients' verbatim reports for each mentioned family member were transcribed.

Two independent raters coded the psychological problems as reflecting one of five categories: anxiety problems, mood problems, substance use problems, anger management problems, or other types of mental problems. The "other" category included eating disturbances, schizophrenia, other psychotic problems, and any other type of psychological difficulties that did not fit into one of the previous categories. The anxiety and mood categories were created based on predictions about positive family history of these types of psychological problems in individuals with GAD. The substance use and anger management categories were created after examination of the data revealed numerous verbatim reports of those types of psychological problems. The other category includes a number of disorders that were not reported with a high enough frequency to warrant creation of another category of psychological problems. Low frequency for each of these types of psychopathology (e.g., eating disorders) would have resulted in unreliable estimates in significance testing, and thus these low frequency categories were collapsed into a single category labeled *other*. Raters were bachelors degree students in psychology and underwent 10 hours of training in using the coding system. Raters were unaware of GAD or control group status.

Results

Interrater Reliability

The reliability of problem categories determined by percentage agreement calculation was high (94.69%). To account for base rate of agreement, an intraclass correlation coefficient was calculated for the primary diagnosis for each reported relative and revealed excellent agreement on category classifications, $r(353) = .90$, $p < .001$.

Family History of Psychological Problems

Of the 151 GAD participants in this study, 34 (22.5%) reported no family members with any history of psychological problems. The remaining 117 (77.5%) participants reported 323 family members with a history of at least one psychological problem. Of the 76 control participants, 23 (30.3%) reported no family members with a history of psychological problems. The remaining 53 (69.7%) participants reported a total of 92 relatives with psychological problems. Table 1 presents the frequencies of each category of psychological problem for each diagnostic group. Table 1 also presents the frequency of having at least one family member with a problem in each category for GAD participants and controls.

To examine the number of GAD participants who had at least one family member with a family history of each psychological problem relative to controls, a series of chi-square tests of independence were conducted. The association between group status (GAD, control) and psychological problem status (yes, no) was examined for each psychological problem category as well as for all categories combined

Table 1
Number of Relatives With Each Type of Psychological Problem (and Number With at Least One Relative With Each Problem).

	GAD	%	Control	%
Problem category	($n = 151$)		($n = 76$)	
Anxiety problems	137(80)	53.0	26(23)	30.3
Mood problems	79(48)	31.8	31(23)	30.3
Substance abuse problems	67(44)	29.1	24(17)	22.4
Other psychological problems	34(26)	17.2	10(9)	11.8
Anger management problems	6(5)	3.3	1(1)	1.3
Any psychological problem	323(117)	77.5	92(52)	68.4

Note. GAD = Generalized anxiety disorder. Percentage column represents the percentage of the GAD and control group who reported at least one family member with each type of psychological problem.

Table 2
Contingency Tables of GAD Status and Family History Status for Each Psychological Problem Category

Anxiety		GAD	Control
	Family history present	80	23
	Family history absent	71	53
Depression		GAD	Control
	Family history present	48	23
	Family history absent	103	53
Substance use		GAD	Control
	Family history present	44	17
	Family history absent	107	59
Anger management		GAD	Control
	Family history present	5	75
	Family history absent	146	1
Other problems		GAD	Control
	Family history present	26	9
	Family history absent	125	67
Total problems		GAD	Control
	Family history present	117	52
	Family history absent	34	24

Note. GAD = Generalized anxiety disorder.

(see Table 2 for contingency tables for each problem category). Generalized anxiety disorder status was associated with a higher frequency of positive family history of anxiety disorders, $\chi^2(1) = 10.53$, $p < .001$. On the other hand, GAD status was not associated with a higher frequency of positive family history of mood disorders, $\chi^2\ (1) = .06$, $p > .05$, substance abuse, $\chi^2\ (1) = 1.18$, $p > .05$, anger management problems, $\chi^2\ (1) = 0.78$, $p > .05$, or for psychological problems falling into the other category (e.g., eating disturbances, psychosis), $\chi^2(1) = 1.12$, $p > .05$. When all types of problems were collapsed into one category, there was a trend for GAD status to be associated with a positive family history of psychological problems overall, but this trend did not reach statistical significance, $\chi^2(1) = 2.18$, $p < .15$.

Gender Differences

To determine whether family history of psychological problems differed by gender, a series of chi-square tests of independence were performed. The association between gender and family history of each type of psychological problem was examined. These analyses revealed that gender was not associated with a family history of anxiety disorders, $\chi^2(1) = 0.06$, $p > .05$, mood disorders, $\chi^2(1) = .92$, $p > 0.05$, substance abuse, $\chi^2(1) = 3.70$, $p > 0.05$, anger management problems, $\chi^2(1) = 0.32$, $p > 0.05$, or for psychological problems falling into the other category, $\chi^2(1) = 0.17$, $p > 0.05$. When all types of problems were collapsed into one category, gender was not associated with a positive family history of psychological problems, $\chi^2(1) = 1.76$, $p > .05$. To examine whether gender differences existed in the extent to which GAD status was associated with a positive family history of psychological problems, a series of univariate ANOVAs were conducted with GAD status and gender as between-groups factors and number of family members with a history of each psychological problem as the dependent variable. These analyses revealed no gender by GAD status interactions for any type of psychological problem or for all psychological problems collapsed. As such, gender did not moderate the association between GAD status and family history of psychological problems.

Discussion

The purpose of this study was to examine self-reported family history of psychological problems among individuals with diagnosed GAD as compared to individuals who had no current or past history of psychopathology. We hypothesized that individuals with GAD would report having more relatives with anxiety and mood problems than individuals with no psychopathology. The results of this investigation are consistent with our prediction that individuals diagnosed with GAD would have a significant positive family history of anxiety problems compared to nonanxious controls. Unexpectedly, GAD was not associated with a family history of mood problems. Individuals with GAD did not have a greater family history of depression, despite more than half of those with GAD reporting a family member with mood problems. Likewise, no differences between the two groups existed for family history of substance abuse problems, anger problems, or other problems. Due to the significant prevalence of anxiety problems in the families of GAD participants, the family members of individuals diagnosed with GAD were marginally more likely to have psychological problems overall than family members of individuals without the disorder. No gender differences were found in the frequency of having a family member with a history of psychological problems.

The results of this study indicate that anxiety problems are more common in family members of individuals with GAD than in family members of nonanxious controls. These findings suggest that having an immediate family member with an anxiety disorder may be associated with an increased likelihood of developing GAD. Future research should aim to identify the mechanisms by which having a family member with an anxiety disorder increases the risk for the development of GAD. Although this study cannot shed light on what those mechanisms might be, several hypothesized pathways will be briefly mentioned as fruitful avenues for future research into familial transmission of GAD. It is important to note that these hypothesized mechanisms are speculative at this point and warrant further investigation. As mentioned in the Introduction section, vicarious learning

mechanisms may underlie familial transmission of anxiety (e.g., Barrett et al., 1996; Gerull & Rapee, 2002; Muris et al., 1996). Another possibility is that parents with anxiety disorders are more likely to have children with insecure attachment. Retrospective evidence suggests that individuals diagnosed with GAD do report attachment difficulties in childhood (Cassidy, 1995; Schut et al., 1997). Role-reversed/enmeshed relationships with caregivers were specifically prominent. If children are forced to care for a parent as well as themselves, they may come to view the world as a dangerous and threatening place and have doubts that they can cope with future events. Finally, lack of control may also lead to the development of GAD in families with anxiety problems. Whaley, Pinto, and Sigman (1999) found that mothers with an anxiety disorder are more controlling/less autonomy-granting in interactions with their children. With fewer opportunities to develop a sense of, and skill in, having control, a child could well develop a fear of the future and little confidence in being able to cope with it. Each of these potential mechanisms of familial transmission of anxiety warrants further investigation.

Our prediction that individuals with GAD would also have higher rates of mood problems among their family members was not supported. Approximately one third of the GAD participants reported having a family member with a history of a mood problem, indicating a greater prevalence of mood problems among family members with GAD than the general population given that prevalence rates for depression are approximately 16%–18% (Kessler et al., 1994; Kessler et al., 2003). Importantly, the high rate of family history of mood problems among GAD participants in this study is consistent with evidence indicating high comorbidity between GAD and major depression (Brown & Barlow, 1992; Brown, Campbell, Lehman, Grisham, & Mancill, 2001) and similar genetic liability factors that predispose to both disorders Kendler et al., 1992; Kender et al., 2007). However, given the relatively high rate of mood problems among family members of control participants, GAD was not significantly associated with a family history of mood problems. This likely resulted from the high rate of depression among relatives in the control group; nearly one third of control participants reported a family member with depression. It is unclear why the control group evidenced higher rates of depression among their family members than would be expected based on population prevalence data. One potential explanation for the higher rate of mood problems among control participants is the relatively low-income community from which all participants were drawn. Low socioeconomic status (SES) is consistently associated with higher rates of depression (Blazer, Kessler, McGonangle, & Swartz, 1994; Bruce, Takeuchi, & Leaf, 1991). Control participants were matched to GAD participants on educational attainment, meaning that any differences between the groups cannot be accounted for by differences in this marker of SES. However, low SES among control participants may be an explanation for higher rates of mood problems among their family members than would be expected. Another explanation for our finding of elevated depression among family members of control participants may be imprecision in the family history method. However, it is unlikely that control participants consistently overreported depression among their family members making this interpretation less plausible.

Limitations

The largest limitation of the current study involves the use of self-reported information on family history. This method of collecting information is susceptible

to error (Roy, Walsh, & Kendler, 1996). It is possible that individuals with a psychiatric disorder may be more likely to report psychiatric symptoms in their family members for a number of reasons. Those with a disorder may be more able to recognize symptomatology in their relatives or may be more inclined to label certain behaviors as pathological than individuals without a history of psychiatric problems. Some evidence indicates that this may, in fact, be the case. Individuals with major depression and generalized anxiety disorder have been found to report that their parents have the same disorder that they have been diagnosed with (i.e., either GAD or depression) at higher rates than their unaffected twin (Kendler et al., 1991). However, some studies have found the opposite; family history reports have high specificity, but low sensitivity at identifying family members with psychopathology (see Kendler & Roy, 1995). Given this limitation, future studies should aim to identify family history of psychiatric disorders in individuals with GAD using structured interviews of family members, which represents a method less susceptible to bias.

Despite the limitations inherent in self-reported family history of psychological problems, Kendler and Roy (1995) argue that this method does provide useful information about family transmission of mental disorders. This conclusion was based on their finding that reported family history of major depression independently predicted family aggregation of major depression in relatives other than the proband with major depression. Positive family history also independently predicted subsequent major depressive episodes in the proband (Kendler & Roy, 1995). As such, we feel that despite the limitations inherent in this method, self-reported family history of psychological problems provides useful information relevant to the study of familial transmission of psychopathology. This method provides an important first step in identifying patterns of aggregation of disorders in families and highlights potential avenues for future research that can elucidate mechanisms by which family history of psychopathology increases risk for the development of mental disorders.

A final limitation involves generalizability of our findings. Our sample was predominantly Caucasian and predominantly women. Given that GAD is more common among women relative to men (see Barlow, 2002), the gender distribution of our sample was representative of the gender distribution of GAD in the general population. However, our sample did not include a representative proportion of racial/ethnic minority participants. As such, future studies examining family history of psychopathology among individuals with GAD should aim to include representative proportions of racial/ethnic minorities in their samples.

Conclusions

The results of this study suggest that individuals with generalized anxiety disorder have a higher occurrence of anxiety problems among their relatives than do individuals without the disorder. These results build upon previous family history findings by examining reports of self-reported family history in individuals with GAD compared to nonanxious controls. Positive family history of anxiety disorders may represent an important environmental etiologic factor in the development of GAD. The present findings suggest that future research should be conducted from a family systems perspective to identify causal family factors involved in the etiology of GAD.

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