



Multiple Facets of the Heritability of Major Depressive Disorder

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Abstract

The objective of this study was to investigate the existence of associations of a family history of major depressive disorder (MDD) with prevalence rates, severity of condition, age, and symptom type in males and females. Face-to-face interviews were conducted with 43,093 adults through the National Institute on Alcohol Abuse and Alcoholism's 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). Despite having equal vulnerability to heritability, females with a family history were found to have significantly higher prevalence rates of MDD than males; somatic and affective symptoms were equally heritable; and within the sample of those with MDD, individuals with a family history were more likely to have an earlier first onset, more episodes and symptoms, and greater severity of condition compared to those without a family history.

Introduction

Everyone experiences days when they are feeling especially low or down. However, when this feeling persists for weeks at a time, it begins to interfere with everyday life. Feeling melancholy for as long as several weeks is evidence of major depressive disorder (MDD), which is much more than a passing blue mood. Without treatment, symptoms may last for months, even years. MDD is fairly common; at any given time it affects twenty million Americans. There is a lifetime risk of 7%-12% for men and 20%-25% for women (APA, 1994).

The disorder involves a combination of symptoms. Diagnostic criteria in the DSM-IV include one of three given abnormal moods¹ significantly interfering with one's life for at least two weeks and five of nine given physiological

¹(1) Abnormal depressed mood most of the day, nearly every day; (2) abnormal loss of all interest and pleasure most of the day, nearly every day; (3) if 18 years of age or younger, abnormal irritable mood most of the day, nearly every day.

symptoms for the same two weeks. These physiological symptoms include abnormal depressed and irritable moods, loss of pleasure or interest in activities, changes in appetite, insomnia or oversleeping, fatigue, and inappropriate guilt. However, they must not be due to mood-incongruent psychosis, physical illness, medication, street drugs, or normal bereavement. In addition, the respondent must never have had a manic, mixed, or hypomanic episode (APA, 1994).

Studies of MDD have shown that the disorder has a significant heritable basis, which has been reported to be as high as 39% as measured from two large general population-based samples of twins (Kendler & Prescott, 1999). Research on the heritability of MDD is especially important because it has the potential to help those with a family history of the disorder to better understand their level of vulnerability to it. A greater understanding of how the disorder is inherited (e.g., which genes are involved) could potentially lead to highly reliable forms of onset prevention in at risk populations. Recently, researchers have begun to investigate more specific questions regarding the heritability of MDD, studying the differential heritability of MDD based on symptom type, severity, age, and sex.

Symptom Type

The heritability of types of depressive symptoms is a relatively new topic in the MDD literature, so there are few studies concerning it. In a prominent study by Jang, Livesley, Taylor, Stein, and Moon (2004), the heritability of individual depressive symptoms was estimated from a sample of volunteer twin pairs who completed the Beck Depression Inventory (BDI; Beck & Steer, 1993), the Centre for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), and items from the Revised Symptom Checklist (SCL-90-R; Derogatis, 1994) assessing depressive symptoms. Factors were found to be differentially heritable; physiological functions and cognitions that refer to heritable personality traits (e.g., hopelessness and feelings of guilt) showed a strong heritable basis while other symptoms of MDD did not (Jang et al., 2004). The researchers believe this difference suggests that the symptoms that do not appear to be heritable are a response to negative life experiences or a learned association with changes in somatic function rather than being endogenous. This view is consistent with the “kindling” or “prekindling” hypothesis for episodes of depression, in which somatic and personality factors with a genetic base symbolize the genetic liability to the initial depressive response (Kendler, Thornton, & Gardner, 2000). Due to the large environmental influence on all of the MDD symptoms, Jang et al. (2004) hypothesized that subsequent depressive episodes result from a learned association with aversive events which generates other somatic responses, such as continued loss of appetite and sleep problems, as in a classical conditioning model.

Severity

Research suggests that recurrent depression is more heritable than nonrecurrent depression (e.g., Bertelsen, Harvald, & Hauge, 1977). This finding is supported by a study involving hospital-ascertained samples of depressed individuals; such samples are typically predominated by severe recurring cases and produce higher heritability estimates than community-based samples (McGuffin, Katz, Watkins, & Rutherford, 1996; McGue & Christensen, 2003). This finding also implies an association between heritability and severe recurring forms of MDD. An explanation for this association may be the diathesis-stress hypothesis, which states that genetic factors predispose an individual to a certain disorder, but that environmental stress factors must occur in order for the potential risk to manifest itself (Gerrig & Zimbardo, 2004). When faced with aversive events, those without a family history of MDD must cope solely with the events whereas

those with a family history must cope with the events in addition to dealing with the effects of genes that predispose them to an onset of MDD. Negative life events may then trigger the expression of these genes in the form of a depressive episode. Therefore, a combination of genetics and an atypical home environment may lead to more severe recurring forms of MDD in individuals with a family history.

Age

There is contradictory evidence regarding the theory that the heritability of depression varies as a function of age. It has been reported that while environmental factors have a greater influence on depressive symptoms between ages 8 and 11, genetic factors exert a more substantial influence by ages 11 to 16 (Thapar & McGuffin, 1994). Gatz, Pedersen, Plomin, Nesselrode, and McClearn (1992) reported that the heritability of a depression symptom score was 18% for those older than 60 as opposed to 3% for those younger than 60. Similarly, in a longitudinal study of male twins aged 59 to 70 at intake, it was found that the heritability of a depression symptom score increased from 25% to 55% over ten years as participants aged (Carmelli, Swan, Kelly-Hayes, Wolf, Reed, & Miller, 2000). These studies suggest that genetic influence on depression symptomatology increases with age.

Contrary to this research, in a large cross-sectional study of pairs of Swedish twins ranging in age from 45 to 95, no differences were found in heritability across the range of age groups (Johnson, McGue, Gaist, Vaupel, & Christensen, 2002). In a study of elderly Danish twins, McGue & Christensen (2003) also found no age-based differences. Due to these contradictions in the literature, more research is required to determine the role of age in the heritability of MDD.

Sex

Like the studies concerning age, studies attempting to determine whether the heritability of MDD varies between the sexes have produced conflicting results. The heritability of DSM-III-R MDD was found to be 44% in females and 24% in males from the large Australian volunteer twin registry (Bierut et al., 1999). The application of DSM-IV criteria did not significantly change this study's heritability estimates (36% for females and 18% for males). Kendler, Gardner, Neale, and Prescott (2001) reported consistent results using DSM-III-R criteria.

The difference between the heritability of MDD across the sexes has been explained by biological and psychosocial factors including hormones (Golombok & Fivush, 1994; Nolen-Hoeksema & Girgus, 1994), experiences specific to adolescent girls (Brooks-Gunn & Warren, 1989; Petersen, Sarigiani, & Kennedy, 1991), X-linked genes (McGuffin & Katz, 1989), and stereotyped expectations that females are more emotional than males (Golombok & Fivush, 1994).

However, the application of more stringent criteria for MDD diagnosis has led to results contrary to those discussed above. In a study of twins, Kendler et al. (2001) used the DSM-III-R plus significant episode-related impairment (DSM-III-R+I) and Washington University (WU) criteria instead of using the DSM-IV criteria.¹ Using these criteria, Kendler et al. found

¹ The DSM-III-R+I criteria were used because the DSM-IV's assessment of female-female twin pairs lacks an item assessing episode-related distress which is required to rate criterion; requiring impairment rather than impairment or distress should make DSM-III-R+I criteria more narrow than DSM-IV criteria. The WU criteria were used because they require one month of dysphoric mood plus at least five of eight criteria:

heritability to be equal across the sexes. This finding has been consistent with other studies using the DSM-IV (McGuffin et al., 1996) and the Cambridge Mental Disorders of the Elderly Examination (CAMDEX; Johnson et al., 2002; McGue & Christensen, 2003). As can be seen from the contradictory body of past research, whether heritability is a function of sex remains unresolved.

Limitations of previous studies

Many of the previous studies on the heritability of MDD have similar limitations. The research of Jang et al. (2004) on symptom type was restricted by a relatively small sample size and scarcity of males, resulting in low power and generalizability. In addition, all twin research studies using non-separated twins are limited by the assumption that any greater similarity in the twins must be the result of their greater genetic similarity rather than any greater similarity in environment; this can underestimate environmental influence on the heritability of MDD. The work of Gatz et al. (1992) and Carmelli et al. (2000) regarding age was limited by moderately sized samples and restricted age ranges, also affecting power and generalizability. Finally, the studies of Bierut et al. (1999) and Kendler et al. (2001) on variation between the sexes were criticized for their use of the DSM-III-R, which gives broad diagnostic criteria for MDD. Using the DSM-III-R may reduce the significance of the results by including participants who would not be diagnosed with MDD under the more restrictive diagnostic criteria of the DSM-IV.

The present study attempts to address the limitations of the previous research. It uses the data set from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a cross-sectional survey of a representative sample of the United States population.² The NESARC has a large number of male and female participants; it is not a twin study, so there are no problematic environmental assumptions; the age range is 18 years and older; and it uses the DSM-IV criteria to define MDD.

This study serves to add to the research literature on the heritability of MDD and provide answers to the debated questions about the heritability of MDD as a function of symptom type, severity, age, and sex. Based on the results of Jang et al. (2004) on differential heritability of depressive symptoms, it is hypothesized that the present study will find that physiological functions have a significant heritable basis while other symptoms do not. It is expected that recurrent depression is more heritable than nonrecurrent depression (Bertelsen et al., 1977). The literature on age is contradictory, so it is unknown whether heritability is a function of age. Finally, based on the conflicting results of studies using DSM-IV diagnostic criteria, it is also unknown whether women are more vulnerable to heritability than are men.

Methods

Participants

Data was obtained from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a nationally representative sample of the adult population of the United States conducted from 2001–2002. The target population was the civilian noninstitutionalized population, 18 years and older. The sample included the military living off-base and persons

poor appetite/weight loss, sleep difficulty, energy loss, agitation/retardation, guilt, difficulty concentrating, and suicidal ideation.

² The NESARC serves as the primary source for information and data on the U.S. population for alcohol and drug use, abuse and dependence, and associated psychiatric and other medical comorbidities.

living in households, boarding houses, rooming houses, nontransient hotels and motels, shelters, facilities for housing workers, college quarters, and group homes.

All potential NESARC respondents were informed about the nature of the survey, the statistical uses of the survey data, the voluntary aspect of their participation, and the federal laws that provided for the confidentiality of the survey information. The respondents consenting to take part in the interview after having received this information, were interviewed, and were not compensated for their participation. The research procedure was given full ethical review and approval from the U.S. Census Bureau and U.S. Office of Management and Budget.

Face-to-face personal interviews were conducted with 43,093 respondents of which 18,518 (43%) were male and 24,575 (57%) were female. The overall survey response rate was 81.2%. The sample is not an exact representation of the population at large. The number of Blacks, Hispanics and young adults (ages 18–24) relative to other respondents in the sample is greater than their actual percentage in the overall population.

Interviewers and Training

Approximately 1800 lay interviewers from the U.S. Census Bureau collected data via computer assisted personal interviewing. On average, the interviewers had five years of experience working on health-related national surveys. All completed a 5-day self-study course followed by a 5-day in-person training session at one of the U.S. Census Bureau's regional offices. Interview quality was ensured by regional supervisors who re-contacted a random 10% of all respondents by telephone and re-asked a set of 30 questions from different parts of the interview in order to verify answers.

Assessment

All diagnoses in the NESARC were made according to the criteria of the DSM-IV using the National Institute on Alcohol Abuse and Alcoholism's Alcohol Use Disorder and Associated Disabilities Interview Schedule - DSM-IV Version (AUDADIS-IV), a fully structured diagnostic interview designed for use by professional, non-clinical interviewers. The AUDADIS-IV is used to screen respondents for the central feature of each mood and anxiety disorder (e.g., MDD is signified by a period of at least two weeks during which there is depressed mood or loss of interest or pleasure in nearly all activities) and those not passing the screen disregard the corresponding interview section. Diagnostic criteria for MDD in the DSM-IV include one of three given abnormal moods interfering with one's life for at least two weeks, and five of nine given physiological symptoms for the same two weeks. These symptoms must not be due to mood-incongruent psychosis, physical illness, medication, street drugs, or normal bereavement. In addition, the respondent must have never had a manic, mixed, or hypomanic episode (APA, 1994).

Additionally, the AUDADIS-IV includes questions regarding family history of mood disorders, measures of onset recency of each disorder, sufficient measures of duration criteria (e.g., the repetitiveness of symptoms required to assess clinical significance), and measures of self-medication related to anxiety and mood disorders.

For the present analyses, affective and somatic depression symptoms were summed up separately to show their prevalence rates. Age categories were created to include younger adults age 18-49 and older adults 50 and above. Groups of males and females with and without a family history of MDD were constructed.

Results

Heritability of Depression

Categorical variables and continuous variables were analyzed using chi-squares and analysis of variance, respectively. Within the sample, 7839 (18.2%) individuals were classified as having MDD in their lifetime. A total of 8803 (22.6%) had one or more parents with MDD; 3144 (35.7%) were male and 5659 (64.3%) were female. Prevalence of MDD was significantly greater in those with a family history of MDD than those without a family history ($\chi^2 = 4699.6, p < .0001$). For those with a family history, females developed MDD significantly more often than males, showing rates of 46.7% and 33.7%, respectively ($\chi^2 = 4699.6, p < .0001$; see Figure 1). A logistic regression controlling for secondary family history (grandparents with MDD) showed that primary family history alone (only mothers and fathers with MDD) made males 4.1 times and females 4.0 times more likely to inherit MDD ($p < .0001$).

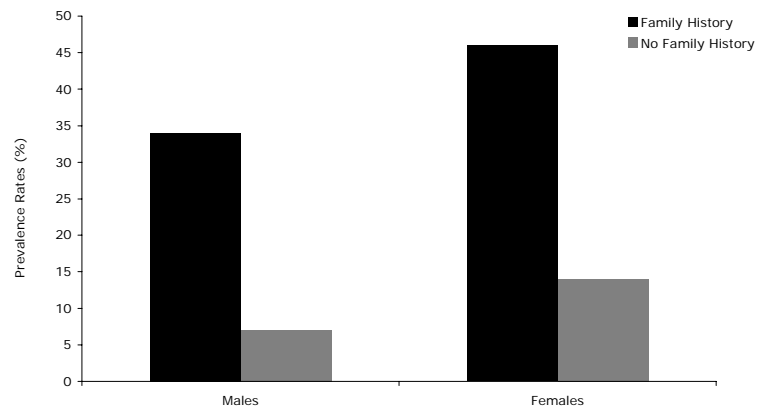


Figure 1. Prevalence rates of MDD in males and females

Symptom Type

Among those who had been diagnosed with MDD, the average numbers of both affective and somatic symptoms present were higher in individuals with family history than in those without a family history ($F = 37182.7$ for affective, $F = 44560.2$ for somatic, p 's $< .0001$; see Table 1). Average rates were significantly higher in women than in men within both groups ($F = 2350.4$ for affective, $F = 2139.1$ for somatic, p 's $< .0001$).

TABLE 1
MDD Characteristics for Males and Females with or without Family History of MDD

	Age of First Onset		Duration of Longest Episode		Number of Episodes		Number of Affective Symptoms		Number of Somatic Symptoms	
	M	F	M	F	M	F	M	F	M	F
Family Hx	27.3	27.7	291	367	10.9	11.7	2.7	3.5	3.4	4.6
No Family Hx	33.3	34.7	363	341	9.3	8.1	0.7	1.1	0.8	1.4
MDD with Family Hx							6.4		8.6	
MDD with no Family Hx							5.6		8.1	
ANOVA <i>p</i> -value	****		n.s.		****		****		****	

**** *p* < .0001.

Note: "Family Hx" stands for Family History. Episodes are measured in days. Sex-wise comparisons for MDD with and without Family Hx were not run and only aggregated data are reported for number of affective symptoms and number of somatic symptoms.

Severity

Those with a family history of MDD exhibited a greater number of MDD symptoms ($\chi^2(9) = 6118.9, p < .0001$) and episodes lasting at least two weeks than did those without a family history of MDD ($F = 10.79, p < .0001$; see Table 1). The difference in the average duration of longest episodes between groups of individuals with or without family history was not found to be significant ($F = 0.71, p > .05$; see Table 1). Compared to those without a family history of MDD, those with a family history were 4.6 times more likely to have gone to a counselor, doctor, or therapist for help to improve their moods ($\chi^2 = 3824.3, p < .0001$); 4.7 times more likely to have stayed overnight in a hospital because of depression ($\chi^2 = 790.7, p < .0001$); 5.1 times more likely to have gone to the emergency room because of depression ($\chi^2 = 752.5, p < .0001$); and 4.6 times more likely to have been prescribed a medicine or drug to improve their moods ($\chi^2 = 3247.6, p < .0001$; see Table 2).

TABLE 2
Severity of MDD

	Family Hx	No Family Hx	χ^2 (df=1)	<i>p</i>
Went to counselor/therapist/ doctor for help to improve mood	2389 (27.1%)	1771 (5.9%)	3824.3	****
Stayed overnight in hospital because of depression	583 (6.6%)	420 (1.4%)	790.7	****
Went to emergency room for help because of depression	495 (5.6%)	372 (1.1%)	752.5	****
Doctor prescribed medicine/ drug to improve mood/	2014(22.9%)	1513 (5.0%)	3247.6	****

make you feel better

**** $p < .0001$.

Age

The onset of MDD in those with a family history tended to occur earlier ($F = 135.8$, $p < .0001$; see Tables 1 and 3). There were no significant differences across the sexes in either group (p 's $> .05$). After controlling for a secondary family history, those with a primary family history were 1.6 times more likely to have onset from ages 8-11 ($p < .05$) and 11-16 ($p < .0001$).

TABLE 3
First Onset Relative to Age Fifty

	Family Hx		No Family Hx	
	<i>M</i>	<i>F</i>	<i>M</i>	<i>F</i>
First onset < 50 yrs old	1074 (93.2%)	2657 (93.0%)	1036 (84.6%)	2182 (81.9%)
First onset > 50 yrs old	78 (6.8%)	201 (7.0%)	189 (15.4%)	482 (18.1%)

Discussion

This study was designed to investigate the existence of associations of a family history of MDD with prevalence rates, symptom type, severity of condition, and age in males and females. The results provide evidence that having a family history of MDD has strong associations with all variables considered for both sexes.

Prevalence

Prevalence rates of MDD were significantly higher in those with a family history of MDD (33.7% for males, 46.7% for females) than in those without a history (8.2% for males, 13.9% for females; $\chi^2 = 4699.6$, $p < .0001$). These rates were higher than the previous heritability estimates using DSM-IV criteria, which were 18% for males and 36% for females (Kendler et al., 2001), though they are similar to Beirut et al.'s (1999) finding of 44% in women and 24% in men using DSM-III-R criteria. This discrepancy is not unusual as heritability rates often differ between studies. While some of this inconsistency is due to varying diagnostic criteria, another reason may be disparities in the assessment process, such as the use of interviewers with unequal levels of training or with varying degrees of familiarity with the patient. Small differences in the assessment process can explain contrasting results across studies.

Despite the higher prevalence rate in females, a logistic regression showed that after controlling for the association with a secondary family history, males and females with a primary family history were equally vulnerable to the onset of MDD. The finding of equal vulnerability is consistent with the findings of several previous studies (e.g., Johnson et al., 2002; Kendler et al., 2001; McGue & Christensen, 2003; McGuffin et al., 1996), and implies that the presence of a secondary family history may be a less accurate predictor of MDD in females than in males. This finding could be due to additional factors influencing the onset of MDD in females, which are

reflected in the higher prevalence rate of MDD in females than in males for people with and without family histories. As explained in previous studies, these other factors could be hormones, X-linked genes, experiences specific to adolescent girls, or stereotyped expectations that girls are more emotional than boys (see Brooks-Gunn & Warren, 1989; Golombok & Fivush, 1994; McGuffin & Katz, 1989; Nolen-Hoeksema & Girgus, 1994; Petersen et al., 1991).

Symptom Type

The numbers of affective and somatic symptoms in those with a family history (2.7 and 3.5, respectively, for males; 3.4 and 4.6, respectively, for females) were significantly higher than in those without a history (0.7 and 1.1, respectively, for males; 0.8 and 1.4, respectively, for females; $F = 2350.4$ for affective, $F = 2139.1$ for somatic, $p < .0001$). This finding indicates that regardless of whether or not a diagnosis of MDD is warranted, people with a family history are likely to have more MDD symptoms than those without a history. After limiting the two groups to participants who had been diagnosed with MDD, those with a family history still had higher symptom levels ($F = 37182.7$ for affective, $F = 44560.2$ for somatic, $p < .0001$). This result suggests that individuals with a family history are likely to have more symptoms of MDD during a depressive episode; a greater number of symptoms could lead to significant interference with healthy functioning in additional facets of one's life, contributing to a more widespread and, therefore, severe form of MDD. The levels of affective symptoms were significantly lower than levels of somatic symptoms regardless of a family history ($F = 37182.7$ for affective, $F = 44560.2$ for somatic, $p < .0001$). This result is most likely due to the interview having fewer questions regarding the presence of various affective symptoms than somatic symptoms.

Though individuals with a family history had more of both symptom types, the difference in the numbers of symptoms between groups was marginally greater for affective symptoms (6.4-5.6 = 0.8, $F = 37182.7$, $p < .0001$) than somatic symptoms (8.6-8.1 = 0.5, $F = 44560.2$, $p < .0001$), implying that, in general, somatic symptoms are no more heritable than affective symptoms. This finding does not drastically conflict with the findings of Jang et al. (2004) because they did not explicitly categorize all somatic and affective symptoms into two groups. However, they did find that physiological functions had a stronger heritable basis than most other symptoms, which is not consistent with the present study's findings. The disparity may be explained by the use of different diagnostic criteria. Jang et al. (2004) used the BDI, CES-D and SCL-90-R; while certain factors of these resemble DSM-IV criteria for MDD, there are a number of differences resulting from the previous study's added inclusion of co-morbid symptoms/disorders often observed in patients with MDD. The presence of these extra factors could explain the inconsistent results. More credence may be given to the present study's finding of equal heritability across symptom types considering Jang et al.'s (2004) diagnostic criteria included additional symptoms that are not part of the DSM-IV criteria for MDD.

Severity

In every category where severity of MDD was measured, excluding duration of the longest episode, those with a family history had more severe characteristics on average than those without a history. No significant differences were found between those with and without a family history for the duration of the longest episode, suggesting that length of episode has no association with heritability. However, the fact that people with a family history were found to be much more likely than those without a history to have stayed overnight in a hospital, gone to the emergency room, and gone to a doctor for a prescription because of depression, suggests that individuals with a family history are much more likely to require medical assistance for

depression. The finding that those with a family history of MDD experienced more depressive episodes on average ($F = 10.79, p < .0001$) might be accounted for by heritability, a different environment due to a family history of MDD, or a combination of the two. In other words, the higher number of episodes could be a direct result of genes that predispose the individual to an onset of MDD, or the home environment could be negatively affected by the condition of a depressed parent, leading to a more severe form of MDD in the child. It seems likely that a combination of genetics and the environment, as explained by the diathesis-stress hypothesis (Gerrig & Zimbardo, 2004), is the most accurate explanation for a higher number of depressive episodes. According to the hypothesis, individuals with a family history who are living with the depressed family member should be especially vulnerable to recurrent depression because they are genetically predisposed to onset and are likely to have increased exposure to environmental stress factors which would activate the genes.

Age

This study's results imply that heritability varies as a function of age. A logistic regression showed that people with a family history were 1.6 times more likely to have onset from ages 8-11 ($p < .05$) and 11-16 ($p < .0001$) than those without a history. However, the effect was more significant from ages 11 to 16, suggesting that genetic factors may begin to have a greater influence at age 11. This finding is consistent with previous research (e.g., Thapar & McGuffin, 1994). For those with a family history, the first onset of MDD was significantly earlier and there were no considerable differences between the sexes in either group. Consistent with this result, out of the participants with MDD, only 7% of those with a family history had the first onset at age 50 or older compared to 17.3% of those without a history ($\chi^2 = 203.5, p < .0001$). This finding further suggests that family history influences the first onset occurring earlier in life. It also may help to explain why the results of the present study and past research indicate that recurrent depression is more heritable than nonrecurrent depression: people with a family history have more time in their lives to experience additional episodes of MDD because they have an earlier first onset. This explanation could account for a higher average number of episodes in individuals with a family history of MDD compared to those without a family history. However, as explained above, the higher number could also be the result of a combination of genetics and a home environment negatively affected by a depressed parent. A regular interaction between genes predisposing a person to MDD and aversive events within the home could cause multiple depressive episodes as well as an earlier onset.

In summation, this study found that despite having equal vulnerability to heritability, females with a family history had significantly higher prevalence rates than males; somatic and affective symptoms were found to be equally heritable; and within the sample of those with MDD, individuals with a family history were more likely to have an earlier first onset, more episodes and symptoms, and greater severity of condition compared to those without a family history.

Limitations

The present study has several limitations. First, the exclusion of institutionalized people may have eliminated relevant cases considering the results, which suggest that those with a family history of MDD are more likely to have severe conditions. Institutionalized people are likely to be those with the most severe conditions, so it is possible that many of them would have been added to the family history group, leading to even more significant results. Second, the data set used in this study is limited by its self-report measure of assessment for depression symptomatology. While this measure is psychometrically sound, its accuracy is compromised by

issues of compliance, the avoidance or denial of depressed affect (Cytryn & McKnew, 1974), and anxiety about revealing secrets or making mistakes (Birleson, Hudson, Buchanan & Wolff, 1987). Third, the NESARC data is limited to people living in the United States whose life experiences may differ in important ways from those in other cultures. This limitation affects the extent to which these results may be generalized beyond the United States. Fourth, the study was limited in how it could assess whether heritability is a function of age because of the cross-sectional design of the data. There were no variables regarding the age at which each episode occurred, when specific symptoms were present or not, or whether episodes at later points in life were typically more severe than others. Finally, the questions asked in the interview showed little attention to the respondents' environment. Onset of MDD in children could be a result of environmental influence and not heritability. For example, children with a primary family history of MDD may learn to adopt the depressed mindset of their parent(s). It is also possible that parents with MDD could be affected by their condition to the extent that they are unable to provide a happy, healthy home environment, influencing the onset of MDD in their children. Heritability and environment should be compared through studies of twins who have been adopted into different homes.

Implications for Future Research

Future research should attempt to quantitatively measure the impact on MDD of hormones, X-linked genes, experiences specific to adolescent girls, and stereotyped expectations that girls are more emotional than boys. This information may help to explain the higher prevalence rate in females despite this study's finding of vulnerability equal to males. In addition, future studies should control for the behavior of the depressed parent in order to determine the household environment's association with the heritability of MDD. The information garnered by such work may assist families in reducing the likelihood of a depressive episode by showing them how to limit environmental stress factors in the home.

Conclusion

This study sheds light on the differential heritability of MDD based on symptom type, severity, age, and sex. Somatic and affective symptoms were found to be equally heritable. However, within the sample of those with MDD, individuals with a family history were more likely to have an earlier first onset, more episodes and symptoms, and greater severity of condition compared to those without a family history. Despite having equal vulnerability to heritability, females with a family history had significantly higher prevalence rates. These results and their implications for future research may help those with a family history of the disorder to better understand their vulnerability to MDD. With a greater knowledge of the associations between heritability, the environment, and specific genes, advancements can be made in the prevention and treatment of the disorder. People with MDD and their families will then have the opportunity to obtain a higher degree of control over their lives that they never had before, and their overall quality of life may improve as a result.

Michael Sandler is a senior and graduate psychology student at Wesleyan University (BA '07 / MA '08) with thoughts of pursuing a career in school psychology. He wishes to thank Professor Lisa Dierker for her guidance during the research process and Kirsten Haller for her editorial assistance on previous versions of this manuscript. Correspondence may be addressed to msandler@wesleyan.edu.

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