Published in final edited form as:

J Am Psychoanal Assoc. 2008 June ; 56(2): 515–550. doi:10.1177/0003065108319727.

DEPRESSION AND INTERNALLY DIRECTED AGGRESSION: GENETIC AND ENVIRONMENTAL CONTRIBUTIONS

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Abstract

This study uses behavior genetic (BG) methodology to investigate Freud's theory of depression as aggression directed toward the self (1930) and the extent to which genetically and environmentally influenced aggressive tendencies contribute to depressive symptoms. Data from the Twin and Offspring Study in Sweden (TOSS) is used to demonstrate how, in estimating shared and unique environmental influences, BG methods can inform psychoanalytic theory and practice, particularly because of their shared emphasis on the importance of individual experience in development. The TOSS sample consists of 909 pairs of adult twins, their partners, and one adolescent child per couple. The Center for Epidemiologic Studies Depression Scale (Radloff 1977) was used to measure depressive symptoms and the Karolinska Scales of Personality (Schalling and Edman 1993) to measure internally directed aggression. Genetic analyses indicated that for both men and women, their unique experiences as well as genetic factors contributed equally to the association between internally directed aggression and depressive symptoms. These findings support Freud's theory that constitutionally based differences in aggression, along with individual experiences, contribute to a person's depressive symptoms. Establishing that an individual's unique, not shared, experiences and perceptions contribute to depressive symptoms and internally directed aggression reinforces the use of patient-specific treatment approaches implemented in psychoanalytic psychotherapy or psychoanalysis.

Freud's theory of depression as aggression directed inward (1930), while considered by many to be a basic tenet of psychoanalytic theory, lacks strong empirical validation. The absence of such validation is due, in part, to the psychoanalytic tradition of assessing

individuals based on a model that emphasizes individual differences and patient-specific treatment approaches. It also reflects a belief that nuances of affect and object relationships require information gleaned from detailed clinical material, obtained under conditions of trust, as well as verification in the analyst's observations of transference and countertransference. However, Freud's theory of depression, as well as many other important psychoanalytic contributions to psychology run the risk of being overlooked or discarded if large-scale investigations that can be generalized to many individuals are not conducted (Gunderson and Gabbard 1999).

Though some psychoanalysts (e.g., André Green [see Sandler, Sandler, and Davies 2000]) maintain that research is concerned with what can be observed, whereas psychoanalysis is concerned specifically with what cannot be (the unconscious), many studies have demonstrated that large-scale research methods are not restricted to the conscious arena (Shedler 2004) and can be effective in addressing theoretical and clinical debates. By examining components of Freud's theory of depression, this study aims to illustrate that behavior genetic (BG) research methods can be particularly valuable in studying psychoanalytic theories and treatment approaches because of their shared emphasis on the importance of individual experience in development. BG methods will test Freud's theory that the association between depressive symptoms and internally directed aggression is influenced by both genetically influenced characteristics and individual experiences and is different in men and women. To allow readers a better grasp of the psychoanalytic relevance of these methods, a brief description of BG theory and principles follows.

BEHAVIOR GENETICS

Behavior genetics is the study of the pattern of genetic and environmental influence on a phenotype (observable behavior or trait). Phenotypes result from the sum of genetic and environmental influences. In this study the focus is on internally directed aggression and depressive symptoms. There are three main BG designs: family studies, adoption studies, and twin studies. A twin study is the basis of this report.

Twin studies draw inferences about the relative genetic and environmental influences on a phenotype, not from direct measurement of genes or environment, but by using pairs of twins who vary in genetic relatedness in known ways. Twin studies examine the patterns of phenotypes between identical, or monozygotic (MZ = 100% genetically similar), and fraternal, or dizygotic (DZ = ~ 50% genetically similar), twin pairs to see if they vary in similarity according to their degree of genetic relatedness. If a phenotype is substantially influenced by genetics, as is the case with height, MZ twins within a pair will be similar to each other in height to a much greater degree than DZ twins within a pair. As with genetic influences, environmental influences are inferred through variations in similarity between MZ and DZ twin pairs.

As shown in Table 1, environmental influences are broken down into nongenetic contributions that make twins from the same family similar to each other (shared environment) and those that make twins from the same family different from each other (unique environment). If a phenotype is substantially influenced by shared environment, as is the case with IQ, MZ twins within a pair will be similar to each other to the same degree as DZ twins within a pair. Similar patterns among MZ and DZ types suggest that something beyond genetics is shared and can include socioeconomic status, similar parenting, or sharing the same classroom. If a phenotype is substantially influenced by unique environment, as is the case with marital quality, neither MZ nor DZ twins within a pair will be similar to each other, despite genetic relatedness. In fact, MZ twins who grow up in the same home are an ideal test of the effects of the unique environment because that is the only

influence that can make the twins within the pair dissimilar to each other. Thus, the unique environment construct offers a chance to capture and measure a fundamentally psychoanalytic notion that two individuals can experience the same event and come away with two completely different perceptions and outcomes. Unique environment may include actual differences in rearing or perceptual differences (i.e., trauma, birth order, parental treatment, and accidents).

BG can also examine the processes connecting two phenotypes or traits. In the study reported here, the focus is on the connection between depression and a measure of personality. The connection could be due to genetic influences common to the two, shared family experiences, or environmental experiences unique to each twin. By tapping into the pattern of genetic and environmental influences on internally directed aggression and depressive symptoms, this study allows for the examination of some of the key components of Freud's theory of the relationship between aggression and depression, to be discussed below.

Difficulties in understanding BG arise from two sources. First, its methodology is unfamiliar to most behavioral scientists, as well as to many statisticians, and can therefore obscure the value of the research. For this reason, the methods section in this paper provides an overview of BG methodology, while a more detailed description of the statistics is reserved for Appendices A and B. Secondly, these analyses deal with classes of variables, not specific variables. Here genes are not measured directly, nor can the genes common to an observed association be identified. The same is true for the classes of environmental variables. While it can be determined that an association is due to specific variables that distinguish one family from another in the childhood of the subject, these variables cannot be identified.

Why then bother with these complex analyses if only latent variables, or classes of variables, can be measured? As it will hopefully be made clear, it is precisely this form of analysis that has proven to be of enormous help by weighing in on more specific theories. Perhaps the best example in the literature is the often observed association between parental negativity and the development of antisocial behavior in the child. Repeatedly, behavioral geneticists have reported that a major portion of this association is due to genetic influences that evoke or shape parental hostility *toward* the child and antisocial behavior *by* the child (Neiderhiser et al. 1999; Reiss et al. 2000). These BG findings forced all researchers to rethink a large number of theories, including psychoanalytic ones, that interpreted these associations as reflecting purely psychosocial phenomena. It is precisely this use of BG that is deployed here. Thus, this study serves not only to test a psychoanalytic theory of depression but to illustrate the relevance of BG to psychoanalysis, particularly because of their shared emphasis on the importance of individual experience in development.

BACKGROUND

Psychoanalytic Theory and Depression

Freud, depression and internally directed aggression—Freud began his well-known exploration of depression and its link to aggression in “Mourning and Melancholia” (1917). He conceptualized that in response to the experience of a loss, the individual internalizes the lost object as a way of holding on to it. The outcome of maintaining the loss is twofold: it helps to ease the grieving, and it provides an alternative target for hostility toward the lost object.

Several years later, in *Civilization and Its Discontents*, Freud (1930) expanded on these views in the context of family and society. According to Freud, as the result of an internal set of standards and values by which one judges him- or herself (i.e., a superego),

individuals may unconsciously inhibit their hostility or aggression to avoid the negative consequences of expressing it externally; thus they preserve their place in the family or, on a larger scale, society. This process results in internalized hostility that predisposes a person to depressive symptoms. Freud proposed that the link between depression and internally directed aggression is mediated by the superego and is due, in part, both to one's expectation of aggression (derived from unique perceptions of one's parents) and to one's own innate level of aggressiveness. "What it amounts to is that, in the formation of the superego, and the emergence of a conscience, innate constitutional factors and influences from the real environment act in combination" (Freud 1930, p. 92).

Further psychoanalytic views of depression—Freud's view of depression and its link to aggression has been elaborated and modified by numerous theorists. Abraham's critical contributions to the theory of depression (1911), though provisional, actually preceded "Morning and Melancholia" (Freud 1917). Abraham viewed depressed individuals as having suffered an enormous frustration at the hands of a loved one and therefore unconsciously longing to destroy the object of affection. This perspective served as a foundation for later theorists who proposed that the intense love and hate for the same object resulted in guilt, leading to self-reproach, the need for punishment, an obligation to protect the loved one from the destructive impulses, and, finally, tormenting depression (Klein 1940; Winnicott 1950; Bibring 1953; Bowlby 1960; Blatt 1974).

In Bibring's model of depression (1953), depressive symptoms result from frustrated aspirations and the experience of helplessness. An individual's incapacity to fulfill his or her wishes to be loved, loving, secure, and good, rather than unworthy of love, insecure, aggressive, and destructive, leads, according to Bibring, to depression. In Bowlby's view (1960), separation from the mother induces powerful and intolerable hate in the child. This unmanageable and unacceptable hate is pushed down and displaced onto others and the self.

Building on Freud's early sensitivity to the developmental origins of depression, Blatt (1974) differentiated between "anacletic" and "introjective" depression. Anacletic depression is characterized by an intense longing for closeness and a complementary fear of abandonment. Introjective, or self-critical, depression is markedly different in that the pain is related to powerful guilt, inferiority, a sense of worthlessness, and internally directed aggression.

More recently, Brenner (1991) and Milrod (1988) have offered somewhat contrasting contemporary views of depression. Brenner's theory rests on the notion that depression is an affect or personality trait that is present in all individuals. He theorizes that depressive symptoms speak to the structure of defenses and affect regulation, rather than to the etiology or origins of the conflict. In fact, he argues that internally directed aggression is not the cause of depression but rather a possible defensive response, as are depressive symptoms, to depressive affect. In stark contrast, Milrod argues that depression is not an affect but a mood, or temporary ego state. According to Milrod, depressive symptoms are not defenses; rather, he proposes that all depressive symptoms result from anger directed toward the self in consequence of loss.

Depression, internally directed aggression, and psychoanalytic research—The process of turning aggression inward is unconscious and therefore cannot be measured directly; however, it may be manifested in observable behaviors or perceptions. For instance, consider a person who is treated rudely by a waiter at a restaurant, but does not complain, argue, or do anything to defend himself. This person may feel uncomfortable or even angry, but have little insight into his inaction or inhibition. However, according to Freud, this person may be punishing himself, or turning his aggression inward.

While the content of the unconscious is not easily tapped, one's observable behaviors can be used to infer some of the struggles and conflicts that infuse the unconscious. Thus, studying the conscious and measurable manifestations of aggression can be a rich source of data leading to an empirical understanding of the association between internally directed aggression and depressive symptoms, and can provide an informative test of Freud's theory of depression.

Some studies have already considered the link proposed by Freud between internally directed aggression and depressive symptoms. One such study found that as their symptoms improved, depressed patients became less self-critical, displaying less self-directed aggression (Mayo 1967). Similarly, Blackburn (1974) showed that patients whose depressive symptoms improved directed less hostility inward than those who experienced no relief, though externally directed aggression remained the same for both groups. Another study found that depression-prone subjects showed a higher level of internally directed aggression and a lower level of externally directed aggression than did healthy controls (Perris et al. 1984).

By and large, psychoanalytic theories and research support a link between aggression and depressive symptoms. Few studies, however, have examined or been able to infer the extent to which the association between aggressive tendencies and depressive symptoms can be explained by genetic and experiential factors. As noted above, Freud proposed that there are both biological and experiential origins of aggression and its association with depression. Therefore, if Freud's theory is correct, the link between internally directed aggression and depressive symptoms should in part be explained by genetic and experiential factors. In other words, the same genetic and experiential factors that explain variation in aggressiveness should also in part explain variation in depressive symptoms within a population. Revisiting and testing, within a BG framework, Freud's view that this association is influenced both by unique experiences of the environment and by genetics is a useful starting point from which to attempt to reconcile various theoretical differences now current.

Behavior Genetics, Depression, and Aggression

Behavior genetic studies have already found that depression and aggression are influenced by a combination of genetic makeup and unique experiences. Several such studies have examined the genetic and environmental influences on different forms of aggression (Brendgen et al. 2006; DiLalla 2002; Miles and Carey 1997; Rhee and Waldman 2002). Brendgen et al. (2006) cite results suggesting that genetic influences account for an estimated 50 percent of the variance in aggression (estimates range from 40 to 80 percent); unique environment accounts for the remainder. Shared environment, all nongenetic influences that make siblings similar to one another, was calculated to have little to no influence on these tendencies. Notably, these investigations have not found differences between genders in the magnitude of genetic and environmental influence on aggression.

Sullivan, Neale, and Kendler (2000) conducted a metaanalysis of the genetic epidemiology of major depression, using data from five family studies. This metaanalysis concluded that *genetic* factors in combination with unique experiences account for the variance in depressive symptoms across family members. Experiences that are shared by family members do not account for this variance.

Taken together, these studies indicate that aggression and depression are influenced by both genetic makeup and unique experiences. These findings are consistent with Freud's view that constitutional factors may predispose individuals to develop certain personality traits (e.g., aggression) and depression. However--and this is a central question in this paper--what

remains to be known is whether the *association* between depression and aggression is explained by a common set of genetic factors and experiences, as proposed by Freud. Many investigations have linked certain broad personality attributes (e.g., neuroticism, child anxiety, sensitivity to environmental stress) to the emergence of depression and found these links to be genetically influenced (Kendler et al. 1993, 2006; Eaves, Silberg, and Erkanli 2003; McGue and Bouchard 1998). These studies are relevant and important to the study reported here because internally directed aggression as a temperamental (constitutional) correlate of depression has not yet been studied.

Gender and Depression

The psychoanalytic literature on gender is extensive and can be traced back to Freud's controversial views of child development. To state these in brief and admittedly simplified fashion, Freud supposed that for various developmental and anatomical reasons both sexes assign a higher value to masculinity than to femininity. As one consequence of this social structure, he viewed girls as more influenced by hostility and as responding more impulsively to their emotions (Freud 1925). Over the past few decades, however, critiques and revisions of Freud's theory of gender development have appeared, and a number of alternative theories have been proposed (Chasseguet-Smirgel 1988; Gilligan 1982; Benjamin 1992; Chodorow 1980). Despite these alterations of Freud's perspective, psychoanalytic and nonpsychoanalytic research and theory justifiably continue to support a model of development that recognizes major differences between the sexes, differences thought to be influenced by biology as well as by men's and women's experiences.

One striking difference is that depression and depressive symptoms are nearly twice as common in women than in men (Kessler et al. 1994). Behavior genetic research has also pointed to differences between men and women in the pattern of genetic and environmental influence on depression. The literature suggests that depression may be more heritable in boys than in girls (Eley and Stevenson 1999; Eaves et al. 1997; Murray and Sines 1996); equally heritable in male and female adults (Sullivan, Neale, and Kendler 2000; Kendler and Prescott 1999; Kendler et al. 1995; Lyons et al. 1998); and, among the elderly, more heritable in men than in women (Jansson et al. 2004). While the debate over the nuances of gender differences is ongoing, there is no doubt that differences exist. For this reason this study will conduct all analyses separately by gender. While studies suggest differences in prevalence and pattern of depression among the sexes, this literature does not offer insight into the impact of gender on the association between internally directed aggression and depression. Thus, there is no expectation regarding how gender will impact the particular points of interest in this study.

OVERVIEW

Psychoanalytic and BG studies of depression and aggression have been successful in enhancing our understanding of complex psychological processes and have demonstrated the value of both large-scale empirical research and individual case studies. BG studies have introduced methods applicable to the testing of psychoanalytic theories and have shown that gaps in psychoanalytic knowledge can be studied using BG methods. Studies suggesting that aggression and depressive symptoms are associated have not yet explored (a) whether the link between aggression and depressive symptoms differs for men and women and (b) whether the association between internally directed aggression and depressive symptoms is influenced by both genetics and unique individual experiences. BG offers a uniquely compatible method for addressing both of these unexplored psychoanalytic questions.

Results suggesting that unique environmental influences (i.e., individual experiences) in combination with innate factors account for the majority of the association between

internally directed aggression and depression would support Freud's view (1930). However, if genetic factors alone influence the association between internally directed aggression and depression, this could mean that a heritable temperament or mode of affect regulation renders a person susceptible to depression, and that the impact of individual experiences is negligible. Alternatively, if shared environmental influences account for the association between internally directed aggression and depression, this would contradict Freud's theory, as such findings would suggest that objective experiences shared by siblings explain the origins of illness and aggression, minimizing the contributions both of unique perceptions/representations and of constitutional differences in temperament.

While a longitudinal examination of *causality* (posing the question, Does internally directed aggression lead to depressive symptoms or vice versa?) is still needed in order to fully examine Freud's theory, a causal hypothesis is imbedded in this study. In other words, if Freud's theory is correct, then specific genes would be found to account for the association between internally directed aggression and depression. Since aggression precedes depression developmentally, it can be inferred that common genetic influences first contribute to internally directed aggression, which in turn contributes to depressive symptom formation.

Results of a study of this kind can also provide insight into the appropriate level and timing of interventions, thus providing an opportunity to assess the merit of psychoanalytic techniques in treating depressive symptoms. For instance, finding that depressive symptoms are predominantly influenced by events and experiences shared by family members suggests that treatment would probably be most effective on a family or group therapy level, rather than on an individual level. Individual treatment approaches (e.g., psychoanalysis or psychoanalytic psychotherapy) would be therapeutically indicated when differences between perceptions and actual events are thought to influence symptoms. Questions of intervention are especially relevant today, given the trend in the mental health field of treating depression psychopharmacologically and thus as an illness with predominantly biological roots. Favoring less costly, manual-guided short-term treatments to long-term, highly individualized intensive therapies with no evidence to validate their efficacy, managed health care companies have little incentive to support psychoanalytic treatments. The techniques used in twin studies such as this one can therefore be important, not only in testing specific psychoanalytic approaches, but in providing the larger clinical community some insight into these interventions.

RESEARCH METHODS

The Twin and Offspring Study in Sweden (TOSS) was designed to (1) examine the associations between parent-child and marital relationships and the mental health of women and men; (2) examine the associations between parent-child relationships and child mental health and explore possible mediators of these associations; and (3) disentangle, using a twin-family approach, the genetic, shared environmental, and unique environmental influences for both sets of associations. The study reported here capitalizes on research methods selected for other purposes. In exchange for valuable access to a twin sample, it was accepted that the data are not precisely tailored to psychoanalytic theory.

The Sample

The TOSS sample was drawn from the Swedish Twin Registry (Lichtenstein et al. 2002; Pedersen, Lichtenstein, and Svedberg 2002) at two different times. The first cohort comprises 326 pairs of adult twin women, their male partners, and one adolescent child per couple. The second sample was collected seven years later and comprises 350 male and 233 female adult twin pairs, their spouses, and one adolescent child per couple. The two cohorts are comparable on all of the demographic factors examined and on the constructs relevant

for this study. The two cohorts are therefore combined in all analyses, resulting in a total sample of 909 pairs of twins, 861 with reported zygosity (128 female and 258 male MZ twins; 286 female and 189 male DZ twins). Zygosity was assessed through genotyping for most of the twins in the first cohort. For twins who have not yet been genotyped or who did not provide usable DNA samples, zygosity was determined through the self-report question, “As children, were you and your sibling as similar as two berries?” This question has been shown to be over 98 percent reliable when compared with genotyping (Lichtenstein et al. 2002). The twins’ average age was 45.4 years (± 5 years); the range was 33 to 60.

Measures

Assessment included a variety of relationship, personality, and mental health measures. Only those used in the current study will be described here.

Depression—The Center for Epidemiologic Studies Depression Scale (CES-D; Radloff 1977) was used to measure depressive symptoms in the twins and their spouses.

The CES-D is a 20-item self-report scale based on diagnostic interviews assessing DSM-IV criteria for major depression. Each item represents a symptom of depressive disorder. Respondents rate how frequently they experienced each symptom during the week prior to completing the questionnaire, on a four-point Likert scale ranging from “0: not at all” to “3: almost all the time.” The total depression score used in this report is the sum of these ratings, with a possible range of 0 to 60; a cutoff score of 16 is the most commonly used criterion for prevalence of depression (Radloff 1977). In the TOSS sample, 342 subjects exceeded the cutoff score of 16 (i.e., were in the clinical range of depression). Internal consistency as measured by Cronbach’s alpha is high across a variety of populations (Radloff and Locke 2000); Cronbach’s alpha for the CES-D total depression scale in this sample was .92.

The content, concurrent, and discriminant validity of the CES-D have been demonstrated (Weissman et al. 1977). This is important to note because while the CES-D assesses depressive symptoms in the week prior to completing the questionnaire, it has also been shown to be a sensitive tool for assessing lifetime risk for depressive symptoms and detecting change in symptoms over time, which it does as well as, or better than, more lengthy self-report scales and clinical interviews (Weissman et al. 1977).

Aggression—The Karolinska Scales of Personality (KSP; Schalling and Edman 1993) is designed to measure dimensions of temperament and personality with a focus on markers for vulnerability to psychopathology. Respondents rate the frequency with which they experienced each item, from “not true at all” to “very much true.” The alphas of the KSP subscales in this sample were acceptable, ranging from .69 to .74. The two subscales indexing aggressive expression--internally directed aggression (inhibited aggression) and externally directed aggression (verbal aggression)--were used in this report. Table 2 provides sample questions from the KSP aggression subtests. A number of studies, both psychoanalytic (Perris et al. 1984; Wilczek et al. 1998, 2004; Gustavsson et al. 1997; Weinryb et al. 1992) and nonpsychoanalytic (Ojehagen, Johnsson, and Traskman-Bendz 2003; Ortet et al. 2002), have found the KSP aggression scales to be stable measures of personality over time.

As Freud’s theory proposes, the process of internalizing aggression is unconscious and therefore cannot be measured directly; however, the KSP includes items that capture the manifestations of the unconscious. For example, item 20 states, “I find it embarrassing to say something if I have been shortchanged.” This set of emotions and behaviors is consistent with Freud’s conceptualization of internalized aggression. Therefore, while the content of

the unconscious is not easily tapped, the KSP can measure observable behaviors to infer some of the struggles and conflicts that may infuse the unconscious.

Analyses

Regression analysis—In the first stage of analysis, nongenetic multiple regression analyses were conducted to establish associations among the constructs. The contributions of internally and externally directed aggression to depressive symptoms were measured. The shared and unique variance accounted for by internally and externally directed aggression to depressive symptoms was also computed.

Correlational analysis—Following the regression analyses, *intraclass* twin correlations and *cross-trait* correlations were conducted to help generate estimates of the genetic and environmental influences on each construct separately, as well as on the correlation between the constructs. Unlike standard Pearson correlations, which are used when two distinct variables are being compared, say X and Y, intraclass and cross-trait correlations were used because there was no clear way to categorize one twin in a pair as twin 1 or twin 2. Double-entered data was also used to guard against the possibility that the initial designation of a twin, as either twin 1 or twin 2, was not random. In other words, each member of a twin pair was entered twice, once as twin 1 and a second time as twin 2.

The intraclass correlations were used to estimate the separate genetic and environmental contributions to one of the phenotypes or constructs of interest, in this case either internally directed aggression or depressive symptoms. Estimates of genetic and environmental influence on each individual construct were generated by comparing the MZ within-pair correlations for a construct to the DZ within-pair correlations for a construct. For example, to compute the intraclass twin correlation for depressive symptoms, twin 1's depressive symptom score was correlated with twin 2's. Likewise, to compute the intraclass twin correlation for internally directed aggression, twin 1's aggression score was correlated with twin 2's. Separate correlations are generated for MZ and DZ twins, allowing for a comparison of correlations between zygosity types.

Interpretations of the intraclass correlations were made based on the assumption that MZ twins share 100 percent of their segregating genes and DZ twins share, on average, 50 percent. A correlation of 1.0 indicates perfect resemblance within a pair, whereas a correlation of 0.0 suggests no resemblance within a pair. Figure 1 illustrates predicted patterns of correlations that can be interpreted to estimate genetic (first column of bars), shared environmental (second column), or unique environmental (third column) influences. As laid out in the bar graph, genetic influences are indicated when MZ twins resemble each other to a greater extent than DZ twins: MZ twin correlation is greater than the DZ twin correlation. If the correlation for the MZ twins is twice that of the DZ twins, then additive genetic effects are inferred. This indicates that multiple genes are contributing to a phenotype in an additive fashion. Shared environmental influences, sources of twin *similarity* due to nongenetic factors, are indicated when MZ and DZ twin correlations are similar and substantial in magnitude. Finally, if the MZ and DZ correlations are small and similar in magnitude, then unique environmental influences are inferred. Unique influences are all nongenetic factors that make family members *different* from one another, so the expectation is for all correlations to be low. Estimates of unique influences may also include error of measurement.

The cross-trait correlations were used to generate estimates of genetic and environmental influence on the *covariance* or correlations between internally directed aggression and depressive symptoms by comparing MZ within-pair and DZ within-pair correlations. These correlations were computed by correlating the aggression ratings for twin 1 with the

depression ratings for twin 2, separately by zygosity type. In other words, the cross-trait twin correlations examined the degree to which twin 1's internally directed aggression score predicted twin 2's depressive symptoms score. Cross-trait twin correlations are interpreted the same way as the intraclass twin correlations: if genetic influences are significant, the pattern of correlations will adhere to the pattern of genetic relatedness; if shared environmental influences are present, the correlations should be consistently large across sibling types; and if unique environmental influences are important, the correlations will be low for both MZ and DZ twins.

Genetic model-fitting analysis—As a third step, so-called biometric model-fitting techniques were used to generate more rigorous estimates of genetic and environmental influence than those offered by the intraclass and cross-trait correlational analyses. As with the correlational analyses, model-fitting analyses were conducted in two stages to help generate estimates of the genetic and environmental influences on each construct separately (univariate analyses), as well as on the correlation between the constructs (bivariate analyses). In the first stage, univariate genetic analyses, like the intraclass correlations, estimated the genetic and environmental contributions for each construct separately. In the second stage, bivariate genetic analyses, like the cross-trait correlations, estimated the genetic and environmental contributions to the *covariance* between internally directed aggression and depressive symptoms. Bivariate genetic analyses also provide estimates for the variance in depressive symptoms that does not covary with internalized aggression.

Using Mx software specifically designed for BG studies, model-fitting techniques test the fit between the observed phenotypic data and the assumptions that are known about patterns of genetic relatedness among twins (Neale, 1997; Neale et al. 2002; Eaves et al., 1989; Jinks and Fulker 1970; Loehlin 1992; Neale and Cardon 1992). The basic assumptions of the model are consistent with the correlational patterns depicted in Figure 1. To the degree that the observed data deviate from or mimics the assumptions of the model, genetic, shared environmental, and unique environmental estimates are generated. Model-fitting techniques also provide confidence intervals for the estimates of genetic and environmental influence and provide statistics of goodness of fit, thus indicating whether the hypothesized models are reasonable. A more detailed description of the model-fitting analyses is provided in Appendices A and B. [AU: NO C AND D.]

RESULTS

Regression Analysis

The findings from regression analysis indicated that there was a substantial association between depressive symptoms and internally directed aggression, explaining 9% ($r = .28$) of variance in depressive symptoms. Conversely, there was a weak association between externally directed aggression and depressive symptoms, explaining only 1% ($r = .12$) of variance in depressive symptoms. Though these finding may not seem robust at first glance, finding that a single variable explains 9% of the variance in a complicated construct such as depression is indeed strong and worth investigating further. It is also important to note that significance levels are not reported because the percentage and pattern of variance accounted for in depressive symptoms are of much greater relevance.

Correlations

Intraclass correlations—The intraclass correlational results for MZ and DZ twins can be found in Table 3. The intraclass correlations for MZ and DZ male and female twins for depressive symptoms are listed in the top two rows of results. The bottom rows list the correlations for MZ and DZ female and male twins' reports of internally directed

aggression. The significance levels are not reported, because the pattern of variance accounted for is of much greater relevance. For both depressive symptoms and internally directed aggression in women, the MZ correlations were slightly more than twice that of the DZ twins, consistent with additive genetic influences. In addition, the MZ and DZ correlations were not substantial or similar in magnitude, indicating that shared environmental effects were negligible, whereas unique environmental effects were present. As was the case for female twins' reports of internally directed aggression and depressive symptoms, for men the MZ correlations were greater than twice that of the DZ twins, indicating genetic influences. In addition, the MZ and DZ correlations were not substantial or similar, indicating that unique influences were also present.

Cross trait correlations—The cross-trait correlations are presented in Table 4. For both men and women, the MZ correlation was greater than the DZ correlation, which indicates some genetic influences on the association. The correlations were not very large for either MZ or DZ twins, also suggesting that unique environment contributes to the covariance between depressive symptoms and internally directed aggression. Finally, these correlations do not suggest a shared environmental influence, as the DZ correlation was less than half the MZ twin correlation, both of which were small. The absence of shared environmental influences is an important finding, as it reinforces a psychoanalytic model that emphasizes individual differences.

Model Fitting

Univariate genetic model fitting—In order to translate the model-fitting estimates into a somewhat more meaningful interpretation of the levels of genetic and environmental influences on depressive symptoms and internalized aggression separately, the estimated percentage of influences on depressive symptoms and internally directed aggression is presented in Table 5. Overall, the results of the univariate genetic model fitting confirm the results of the intraclass correlations by indicating that genetic and unique environments were the dominant influences for men and women for both internally directed aggression and depressive symptoms. For both sexes, unique environment had a greater influence on depressive symptoms than did genetic factors. Internally directed aggression in women and men seems almost equally influenced by genetic factors and unique environment. As predicted by the intraclass correlational results, shared environment had no influence on internally directed aggression or depressive symptoms in either men or women. Since the univariate analyses indicated that shared environment did not contribute to depressive symptoms or internally directed aggression, it was not explored further in the bivariate analyses.

Bivariate genetic model fitting—The estimated percentage of the correlation between internalized aggression and depressive symptoms that can be explained by each factor is presented in Table 6. For women, genetic factors accounted for 45% of the correlation, and unique environment accounted for 55%. For men, genetic factors accounted for 40% of the correlation, and unique environment accounted for 60%. As anticipated by the cross-trait correlations, genetic and unique environmental influences account for the covariance of internally directed aggression and depressive symptoms for *both* men and women.

DISCUSSION

This study was an effort to examine Freud's theory of depression – its characterization as aggression directed inward-- to illustrate that behavior genetic (BG) research methods can be particularly valuable in studying psychoanalytic theories and treatment approaches because of their shared emphasis on the importance of individual experience in

development. Though previous studies have demonstrated an association between depressive symptoms and aggression (Mayo 1967; Blackburn 1974; Perris et al. 1984), none have explored this association within a behavior genetics framework, and in failing to do so have neglected many key components of Freud's theory, including the notion that the link between aggression and depressive symptoms differs for men and women and is influenced by both genetics and unique individual experiences. In addition to enhancing theory, it is possible, by studying the pattern of genetic and environmental influences on the association between aggression and depressive symptoms, to gain a greater understanding of the etiology, and to some extent the directionality, of the mechanisms at work, as well as to inform treatment and prevention. Each of the main findings of this study is discussed below.

Internally directed aggression and depressive symptoms

In order to move forward with the genetic analyses, it was first necessary to confirm the findings of previous investigations by establishing an association between internally directed aggression and depressive symptoms. Had no association been found, subsequent analyses would have been thrown into question by the possibility that (a) the Swedish sample was aberrant, (b) the measurements of aggression and depressive symptoms were unrelated to previous psychoanalytic investigations, or (c) some other, unknown confounding factor was at work. The results indicated that internally directed aggression indeed accounted for a significant amount of variance in depressive symptoms, and justified conducting genetic analyses to further investigate Freud's theory.

Genetic, unique environmental, and shared environmental influences

The results of the genetic analyses lent support for Freud's theory by indicating that unique environment and genetic factors contributed, in equal measure, to the association between internally directed aggression and depressive symptoms in both men and women. Shared environment had no influence on that association for either gender. Finding a large unique influence is in line with behavior genetic studies that have consistently pointed to unique environment as the most influential source of environmental variance for personality and psychopathology (Plomin 1990).

It is important to note that while unique environment captures nongenetic differences among siblings, these results do not by themselves support the notion that the differences are influenced by perceptions or representations of experiences and relationships, as suggested by Freudian theory. They could reflect actual differences in environmental circumstances for developing individuals, as suggested by Kohut and Seitz (1963). Kohut views internally directed aggression as the residual of real experiences with parental authority. He sees the degree to which an individual avoids external punishment as directly related to the original parent's capacity to be loving and approving (Kohut and Seitz 1963). Notably, there is data suggesting that in adolescence it is unlikely that unique environmental influences arise solely from actual differences between siblings (Reiss et al. 2000). However, that is not uniformly the case in adulthood. For instance, the objectively verifiable quality of marriage and of the spouse contributes an important share of unique environment in midlife women, and quite specifically for depressive symptoms (Spotts et al. 2004). Therefore, while a unique influence is consistent with Freudian theory, it does not categorically support it and the question of perception versus actual reality remains.

With regard to this question, the absence of a shared environmental influence in this study's findings is relevant. While the degree to which unique environmental influences reflect reality or perception is unknown, it is known that shared environment largely reflects actual external experiences, most likely from childhood (Pederson et al. 1999). Therefore, finding no shared environmental influence reduces the impact actual events may have on the

association between depressive symptoms and internally directed aggression. While the absence of shared environmental influence may seem unremarkable, these results should not be overlooked. To demonstrate the weight of this construct, shared environment has been found to play an important role in autonomy, social responsibility, similarity in parental treatment, sociability, sibling relationships, and adolescent adjustment, just to name a few (Reiss et al. 2000). In sum, finding that shared environment does not influence the association between depressive symptoms and internally directed aggression weighs in favor of the trends in psychoanalytic theory, including Freud's 1930 reformulation, that consider constitutional factors and individual representations of events as contributing to depressive symptoms.

Gender differences

There was no expectation regarding how gender would affect the results and separating the analyses by sex provided interesting information. Overall, the genetic and environmental pattern of influence on the association did not differ by sex and therefore undermined Freud's theory that women are more influenced by hostility. However, there were some moderate gender differences found. Examining the genetic and environmental contributions to internally directed aggression, independently of depressive symptoms, revealed that genetic factors have a slightly greater influence on women than on men. One explanation for this finding could be that aggression is socially less acceptable in women, and thus is less stimulated by the environment, and that when innately determined is harder to contain. Notably, men and women had common patterns of environmental and genetic influence for depressive symptoms when analyzed independent of internally directed aggression. Finally, this study examined not the prevalence of depressive symptoms or internally directed aggression in men and women, but rather the pattern of genetic and environmental influence.

Clinical implications

This study set out to examine a psychoanalytic theory of depressive symptoms using a BG model; the idea was to build on innovative avenues of research developed by other researchers, with the ultimate aim of informing psychoanalytic theory and practice. The large unique environmental influence on the association indicates that intervention would be more successful on an individual level, not a family level. Additionally, it is a common misconception that genetically influenced constructs cannot be changed. Over time, however, it not uncommon to find that genetic influences and environmental contributions shift.

Inferences about causality (i.e., whether internally directed aggression leads to depressive symptoms or vice versa), which should be made with caution, can aid in determining the timing of interventions and prevention strategies. If it is the case that internally directed aggression leads to depressive symptoms, clinically it may be that discouraging individuals from directing aggression inward may reduce or prevent the onset of depressive symptoms. Notably, in a paper written in 1975, Sternbach suggests that the type of depressive symptoms might determine if external expression of aggression will help diminish or increase symptomatology.

Limitations and future directions

Four main points will be addressed in this section. First, it is necessary to discuss the issues that may surface when using self-report questionnaires to measure depressive symptoms and internally directed aggression. It is important to note that the CES-D and the KSP, and similar instruments, have been widely used in psychoanalytic studies to measure depressive symptoms and internally directed aggression (Blatt and Homann 1992; Wilczek et al. 1998, 2004; Ekselius, Hetta, and von Knorring 1994). Admittedly, the use of self-report and

correlational data leave the findings open to a number of interpretations and cannot directly access the unconscious mechanisms proposed by psychoanalytic theory to be at work. Studying the expected manifestations of these mechanisms in behavior and thought using self-report measures provides an opportunity to capture the psychoanalytic concepts. Based on the continued efforts to test the validity of measures used (Boyd et al. 1982; Breslau 1985; Gatz et al. 1993), it is reasonable to expect that the CES-D will come very close to measuring what clinicians would consider depressive symptoms and lifetime risk for depressive symptoms, thereby presenting an opportunity to study and revise our understanding of the illness. While the KSP cannot directly measure the unconscious processes involved in turning aggression inward, its questions can appraise some of the potential manifestations (e.g., a person's inability to speak out when someone cuts into the line). It is important to emphasize that the methodology used in this study is not presented with the intention of replacing traditional case studies that focus on underlying dynamics. Both methods have their value and can complement each other by offering an opportunity for optimal understanding.

Second, the question of causality still needs to be addressed. While a longitudinal study would be needed to fully examine this issue, the data presented here nonetheless offer clues into the mechanism behind the link between internally directed aggression and depressive symptoms. A finding that specific genes account for this association, and the knowledge that aggression is an earlier developmental achievement than depression, may suggest that internal aggression leads causally to depressive symptoms.

Third, there is the possibility of genotype-environment (GE) interaction. GE interaction is conceptualized as the genetic control of sensitivity to varying environments (Kendler and Eaves 1986). In a linear GE interaction, the genetic effects increase or decrease as a function of the environmental variable. A nonlinear interaction occurs when the genetic effects are expressed at extremely high or extremely low levels of the environmental variable. The most common type of GE interaction occurs where the environment is unique. The less common type, where the environment is shared, would be expressed in the genetic estimate. While estimating GE interaction is beyond the scope of this paper, future research will test for its influence on the association between internally directed aggression and depression. With Freudian theory in mind, it would be interesting to examine whether the expression of the genetic effects of depressive symptoms and internally directed aggression increase as a function of extremely harsh parenting.

A fourth issue to take into consideration is whether the genetic and environmental influences on extreme levels of depressive symptoms differ from those for the full distribution of symptoms in adults. Such a finding would suggest that the etiology of depressive symptoms differs according to the magnitude of symptomatology and that studies conducted on the full distribution of symptoms may obscure the true mechanisms of depressive symptom formation. Currently most behavior genetics research on depression in adults can be divided into two domains: research conducted on subjects diagnosed with Major Depression (MD) (Kendler et al. 2006, 2007; Sullivan, Neale, and Kendler 2000); and research conducted on the full distribution of depressive symptoms collected from the general population (Reiss et al. 2001; Gatz et al. 1992; Silberg et al. 1990). Despite being fairly consistent (genetic influences = ~ 30% and unique environmental = ~70%), especially with respect to finding negligible shared environmental influences, these studies may be disguising important differences in patterns of etiology by either assuming that depression is a categorical illness (rather than a continuum of severity) or by assuming that the full continuum of symptoms is normally distributed. Several studies have examined this issue in adolescent populations (Rende et al. 1993, 2006) and found that genetic influences were significant at low levels of symptoms and nonsignificant at high levels. The reverse was found to be true for shared

environmental influences. Overall, they concluded that there are significantly different etiological patterns found in low and high levels of depressive symptoms. Currently, there do not appear to be any studies that have investigated if and how the patterns of influence found to operate on the adolescent samples change in adulthood; such a study would seem warranted.

CONCLUSION

It is no surprise that genetic and environmental influences work in combination to influence mental health and relationships. However, establishing that unique environment (not shared environment) in combination with genetic factors contributes to the association between internally directed aggression and depressive symptoms offers additional clarification and reinforcement of Freud's view that it is likely to be one's subjective perception of an event, in combination with inborn traits, that contributes to one's outcome and experience.

This study is the first of its kind to use BG methods and twin data to test psychoanalytic theories and demonstrate that BG analysis can serve a major function in expanding psychoanalytic research. Deciphering the genetic and environmental interplay can not only assist in refining theory but can go further by informing etiology, prevention, intervention, and technique. In addition, the unique environmental construct that behavior genetic methods capture and measure is central to psychoanalytic thinking and treatment. Demonstrating the extent to which individuals experience the same event and come away with a different perception of it has direct implications for the psychoanalytic tradition of treating patients based on a model that emphasizes individual differences. An elucidation of the genetic and environmental manifestations of psychopathology can potentially provide a great deal of empirical validation and support for psychoanalytic contributions to psychology.

Acknowledgments

This project was supported by National Institute of Mental Health grant R01MH54610 and a grant from the Swedish Medical Research Council. The first author was supported in part by the Samuels Foundation Fellowship for Psychoanalytic Research from the Washington Psychoanalytic Society and the George Washington University Department of Psychiatry and Behavioral Sciences. The authors thank Debbie Feldheim, Jenn Ulbricht, Dorothy Holmes, and Richard Fritsch for their help in preparing this paper. Submitted for publication

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APPENDIX A: UNIVARIATE MODEL FITTING

The univariate path diagram is presented in Figure A1. The univariate path model is programmed with the assumptions about the genetic relatedness among MZ and DZ twins. In the hypothesized models 'A' is the latent (not directly observed) variable representing the additive genetic variance shared by twin pairs on each construct, and is set to correlate at 0.5 for DZ twins and 1.0 for MZ twins. This reflects the degree of genetic relatedness of the twins: MZ twins share 100% of their genes and DZ twins share, on average, 50% of their genes. 'C' is a latent variable representing shared environmental influences on the two twins and is set to correlate 1.0 for both MZ and DZ twins because by definition shared environment is equal for all family members in the same home. Finally, E represents the residual variance (unique environmental influence and measurement error) that does not correlate for twin 1 and twin 2. This path is set to zero because by definition unique environmental influences are different for each twin, serving to make twins different from each other. Based on the degree that the observed data deviate from or mimic the assumptions of the model, the genetic and environmental influences on either construct (internally directed aggression or depressive symptoms) are estimated.

Figure A1.

The univariate estimates of the genetic and environmental influences and 95% confidence intervals for female and male depressive symptoms are presented in Figure A2. For females, the parameter estimate was .33 for the path leading from the latent genetic variable 'A' to depressive symptoms, indicating that 33% of the variance in depressive symptoms in women is genetically influenced. For females, the parameter estimate was .67 for the path leading from the latent unique environmental variable 'E' to depressive symptoms, indicating that 67% of the variance in depressive symptoms in women is influenced by unique environment. The paths for the male estimates can be traced in the same way.

Female

Male

Figure A2.

Figure A3 presents the univariate estimates for internally directed aggression in men and women. Estimates of the genetic and environmental influences and 95% confidence intervals for female and male internally directed aggression are mapped on to the univariate path model presented in Figure A1. For females, the parameter estimate was .48 for the path leading from the latent genetic variable 'A', indicating that 48% of the variance in internally directed aggression in women is influenced by genetics. The paths for the other paths can be traced in the same way.

Female

Male

Figure A3.

APPENDIX B: BIVARIATE MODEL FITTING

The path diagram for the bivariate estimates is presented in Figure B1. The bivariate path diagram presented is known as a Cholesky decomposition and is approximately analogous to stepwise regressions. As in the univariate model, this model includes latent genetic, shared environmental, and unique environmental factors. The latent variables 'A,' 'C,' and 'E' represent the genetic, shared environmental, and unique environmental influences on depression that covary with internally directed aggression for twin 1 and twin 2. Each path from the latent construct can be traced to the observed variable. The path values for the bivariate model are set to the same values as the univariate model and are based on the same assumptions. 'A' is set to 1.00 for MZ twins and 0.5 for DZ twins; 'C' is set to 1.00 for both MZ and DZ twins; and 'E' is set to 0.0 for both MZ and DZ twins. In the figure the paths leading from latent variable 'A' represent the genetic influences on internally directed aggression (a_{11}) and the genetic influences on the covariation between internally directed aggression and depressive symptoms (a_{12}). Similarly, the paths leading from latent variable 'C' represent the shared environmental influences on internally directed aggression (c_{11}) and on the covariation between internally directed aggression and depressive symptoms (c_{12}). Since the univariate genetic analyses indicated that shared environment did not contribute to depressive symptoms or internally directed aggression, it was not estimated in the bivariate analyses. Finally, the paths leading from latent variable 'E' represent the estimated unique environmental influences on internally directed aggression (e_{11}), and the unique environmental influences on the covariation between internally directed aggression and depressive symptoms (e_{12}). The paths marked a_{22} , c_{22} , and e_{22} , contain the parameter estimates for genetic and environmental influences on depressive symptoms that are not correlated with internally directed aggression. The parameters a_{22} , c_{22} , and e_{22} , respectively can be squared to determine how much variance in depression remains after the contributions of internally directed aggression are considered.

Figure B1.

The bivariate parameter estimates and 95% confidence intervals for the genetic, shared environmental and unique environmental contributions to the association between twins' report of depressive symptoms and internally directed aggression and for the unique variance of depressive symptoms are presented in Figure B2. Figure B2 maps the standardized parameter estimates and confidence intervals for the bivariate genetic model onto the bivariate path model presented in Figure B1. For women, the genetic parameter estimates were .70 for the path leading from 'A' to internally directed aggression (a_{11}), .18 for the path from 'A' to depressive symptoms (a_{12}), .55 for the path leading from 'a' to depressive symptoms (a_{22}). The paths for each of the other latent factors can be traced in the same way. The total covariance (Genetic + Shared E. + Unique E.) in depressive symptoms accounted for by internally directed aggression can be estimated by multiplying the paths from each factor to each observed variable, and then summing their products: e.g., total association = $(a_{11} \times a_{12}) + (c_{11} \times c_{12}) + (e_{11} \times e_{12})$ (Neale and Cardon 1992; Loehlin 2004). For instance, the equation for the total association for females is $[\cdot70 \times \cdot18] + [-- \times --] + [\cdot72 \times \cdot22] = \cdot29$. The total association can then be divided into genetic ($a_{11} \times a_{12}$), shared environmental ($c_{11} \times c_{12}$), and unique environmental ($e_{11} \times e_{12}$) contributions, enabling one to determine the relative importance of each latent factor to the association. For instance, in

women the genetic factors accounted for 45% of r_{est} : e.g., $a_{11} \times a_{12}/r_{\text{est}} = .13/.29 = .45$) and the unique factors accounted for 55% of the association: e.g., $e_{11} \times e_{12}/r_{\text{est}} = .16/.29 = .55$).

Female

Male

Figure B2.

Please note that overall fits of the univariate and bivariate models were tested using both chi-square (χ^2) and Akaike's information criteria (AIC; Tanaka 1993). In contrast to most indicators of significance, a model is considered a good fit to the data when χ^2 is nonsignificant. Although this is a commonly used statistic, it has been found that χ^2 values are likely to reject a model that fits the data well but imperfectly because they are highly sensitive to sample size and actually improve when more parameters are added to the model (Mulaik et al. 1989; Neale and Cardon 1992), so alternative forms of testing the fit of the model are often used. A commonly used alternative is the Aikake's Information Criteria, an

index that takes into account both the χ^2 value and the degrees of freedom associated with the model. The best fitting and most parsimonious model is indicated by the smallest AIC. For a more technical discussion of model fitting techniques see Loehlin (1992, 2004).

Figure 1.

Sample Correlational Patterns. MZ and DZ within pair correlations are compared to see if they vary in similarity according to their degree of genetic relatedness. Genetic influences are indicated when $MZ = 1.0$ and $DZ = .5$. The pattern of correlations corresponds to the genetic similarity (e.g., height, voice, foot size). Shared environmental influences are indicated when $MZ = 1.0$ and $DZ = 1.0$. The Pattern of correlations suggests that something is shared for both twin types that is nongenetic (e.g., IQ or sib relationships). Unique environmental influences are indicated when $MZ = 0$ and $DZ = 0$. A pattern of low correlations suggests that none of the twin types are similar to each other (e.g., marital relationships, schizophrenia).

Table 1

Description of Genetic and Environmental Influence

Genetic	Additive	Ⓐ	Genes influence phenotype equal to the sum of each gene individually
Environmental	Shared	Ⓒ	Nongenetic influence on phenotype making family members similar .
	Unique	Ⓔ	Nongenetic influence on phenotype making family members different .

Table 2**KSP Sample Questions**

Scale	Item
Internally Directed Aggression (Inhibited Aggression)	If I am badly treated in a restaurant I have difficulty complaining.
	When someone pushes in front of me in a queue I usually say something. *
	I find it embarrassing to say something if I have been short-changed.
	If someone tells me off I get sad rather than angry
Externally Directed Aggression (Verbal Aggression)	I easily start arguing with those who are of different opinion.
	If someone annoys me I don't mind telling them what I think.
	When I get angry I sometimes say nasty things.
	If someone shouts at me, I shout back.

Note. Scales' original names are given in parentheses.

* Item reversed when scored.

Table 3

Intraclass Twin Correlations

Measure	MZ	DZ	Interpretation
Depressive Symptoms			
Female	.36	.14	A and E
Male	.44	.15	A and E
Internally Directed Aggression			
Female	.50	.21	A and E
Male	.28	.05	A and E

Note. Interpretations are based on a comparison of the correlation between MZ twin 1 and MZ twin 2 with the correlation between DZ twin 1 and DZ twin 2 within MZ or DZ twin pairs.

'A' represents genetic influences. 'E' represents unique environmental influences.

Table 4

Cross Twin Correlation

Internally Directed Aggression and Depressive Symptoms	MZ	DZ	Interpretation
Female	.13	.02	A and E
Male	.16	.05	A and E

Note. Correlations between internally directed aggression ratings for twin 1 with depressive symptoms ratings for twin 2. 'A' represents genetic influences. 'E' represents unique environmental influences.

Table 5

Univariate Model Fitting Results

	% of Contribution		
	Genetic	Shared Environment	Unique Environment
Depressive Symptoms			
Female	33	0	67
Male	26	0	74
Internally Directed Aggression			
Female	48	0	52
Male	43	0	57

Note. Univariate model fitting estimates for the genetic and environmental influences on depressive symptoms and internally directed aggression.

Table 6

Bivariate Model Fitting Results

	% of Contribution to the Covariance		
	Genetic	Shared Environment	Unique Environment
Female	45	0	55
Male	40	0	60

Note. Bivariate model-fitting estimates for the genetic and environmental influences on the *associations* among depressive symptoms and internally directed aggression.