

A BEHAVIOR GENETIC STUDY
OF SELF-HARM, SUICIDALITY, AND PERSONALITY
IN AFRICAN AMERICAN AND WHITE WOMEN

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by
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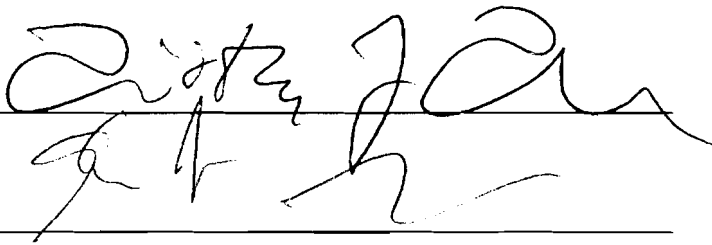
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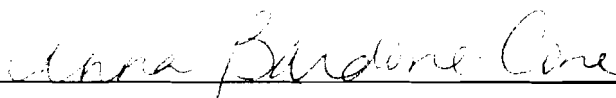
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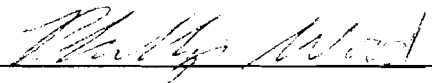
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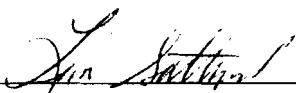
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ABSTRACT

This study is an examination of the genetic and environmental risk factors leading to suicidal and self-harm behaviors in a representative community sample of female adolescents and young adults, and racial differences in the relative strength of those risk factors. Using a behavior genetic twin study design, suicidal and self-harm behaviors proved to be moderately heritable, and were influenced by nonshared environment but not by shared environment. The two phenotypes shared a moderate genetic correlation and a weak environmental correlation. After accounting for genetic influence common to both, self-harm but not suicide attempts had significant unique genetic variance remaining. When examined by race, suicidal behaviors were more common in African American women than White women, while self-harm was equally prevalent. However, the heritability of self-harm and suicide attempts did not vary by race. Further analyses were conducted to determine the extent to which genetic variation in self-harm and suicide attempts could be accounted for by personality traits (Neuroticism and Novelty Seeking) and psychiatric diagnoses. Personality traits and diagnostic status did not differentially predict self-harm or suicidal behavior by race in multivariate regressions. Neuroticism shared significant genetic variance with both self-harm and suicide attempts, and Novelty Seeking shared significant genetic variance with self-harm. Both internalizing and externalizing diagnoses were also genetically correlated with self-harm and suicide attempts. After accounting for genetic influence common to personality, diagnoses, and self-harm or suicide attempts, self-harm but not suicide attempts had significant unique genetic variance remaining.

Self-injurious behaviors are generally conceived as belonging to one of two categories: self-harm or suicide. *Self-harm* is the deliberate and often repetitive infliction of damage or pain to one's own body, without lethal intent (Winchel and Stanley, 1991). Many other terms have been used to describe this behavior including self-injury, self-mutilation, self-inflicted violence, and auto-aggression. *Suicidality* refers to the occurrence of suicidal thoughts (suicidal ideation) or suicidal behavior. Suicidal behavior includes acts of self-harm with lethal intent, usually having the purpose of ending intolerable emotional pain. The outcome may be fatal (suicide) or nonfatal (attempted suicide). These behaviors share many similarities, but may also differ in key aspects.

Demographics of Self-Harm

Statistics concerning the prevalence of self-harm tend to be unreliable due to the private nature of the act, and the fact that many incidents do not reach the attention of mental health professionals. Current estimates for community prevalence rates of self-harm in the United States and in several other countries range from 4% to 7% (Hawton, Rodham, Evans, & Weatherall, 2002; Klonsky, Oltmanns, & Turkheimer, 2003; Patton et al., 1997). It appears that self-harm cuts across the boundaries of education, sexual preference, and socioeconomic status (Favazza and Conterio, 1989), though women are about twice more likely to self-harm than are men.

The most common practice of self-harm is cutting the skin, but other methods include burning, self-hitting, interference with wound healing, severe skin scratching, hair pulling, swallowing small objects or toxic substances, and bone-breaking (Favazza and Conterio, 1989). Self-harming behaviors are typically initiated in early adolescence, around 14 years of age, and have a peak incidence from 16 to 25 years of age (Favazza

and Conterio, 1989). Non-fatal repetition is common after self-harm; about one in six patients repeats over the next year and one in four after 4 years (Owens, Horrocks, & House, 2002). The intent of self-harm has typically been characterized as a maladaptive means of affective regulation via either the endogenous opioid response to bodily injury, dissociation, or distraction by means of physical pain or the sight of blood (although some individuals report feeling no pain during self-harm). Self-harm is typically relied on as a response to overwhelming emotions of anger, shame, or sadness following interpersonal losses or conflicts.¹

¹ To simplify and clarify the relevant issues, this review will not include self-injurious or suicidal behavior in the context of pervasive developmental disorders such as severe mental retardation, autism, and Asperger's syndrome, or genetic conditions such as Prader-Willi, Lesch-Nyhan, and Cri-du-Chat syndromes. In all of these conditions, self-harm behavior is common. However, the self-harm behaviors are different relative to the self-harm behavior of individuals with other psychiatric illnesses such as borderline personality disorder or major depression, tending to be stereotyped, sometimes involuntary, and often much more severe (Bodfish, Symons, Parker, & Lewis, 2000). Furthermore, the contingencies and maintaining factors of self-injurious behavior seem to be qualitatively different in cases of pervasive developmental disorders or genetic conditions, more often (though not exclusively) relating to levels of environmental stimulation rather than to social consequences (Patel, Carr, Kim, Robles, & Eastridge, 2000).

Scant research is available on the differential prevalence of self reports of self-harm by race; the extant literature suggests that self-harm is more common in White individuals². In a sample of adolescents undergoing intensive psychiatric treatment, White individuals were more likely to cut themselves than were members of minority groups (Brown, Houck, Hadley, & Lescano, 2005). Similarly, Sen (2004) reported that among children in 6th to 10th grade, African American individuals were less likely to self-harm than members of other racial groups. These studies suggest a trend in which Whites report relatively higher rates of self-harm, and African Americans relatively lower rates, but the first study has limited generalizability due to the selected clinical sample, and the second is limited by the young age of the participants, who were below the age of peak incidence of self-harm. The differential racial prevalence of self-harm could be better determined by a community sample of older adolescents/young adults.

Demographics of Suicidality

According to the National Comorbidity Study, of a sample of United States residents ages 15-54, 13.5% reported lifetime suicidal ideation, 3.9% a suicide plan, and 4.6% a suicide attempt (Kessler, Borges, & Walters, 1999). In 2000, suicide was the 11th leading cause of death in the U.S. overall (Miniño, Arias, Kochanek, Murphy, & Smith,

² There are a number of terminologies in use for the description of members of racial and ethnic groups living in America. That used by the Centers for Disease Control and Prevention will be adopted here, in which those of European descent are referred to as White, those of African descent are referred to as African-American, those of Asian descent are referred to as Asian-American, etc.

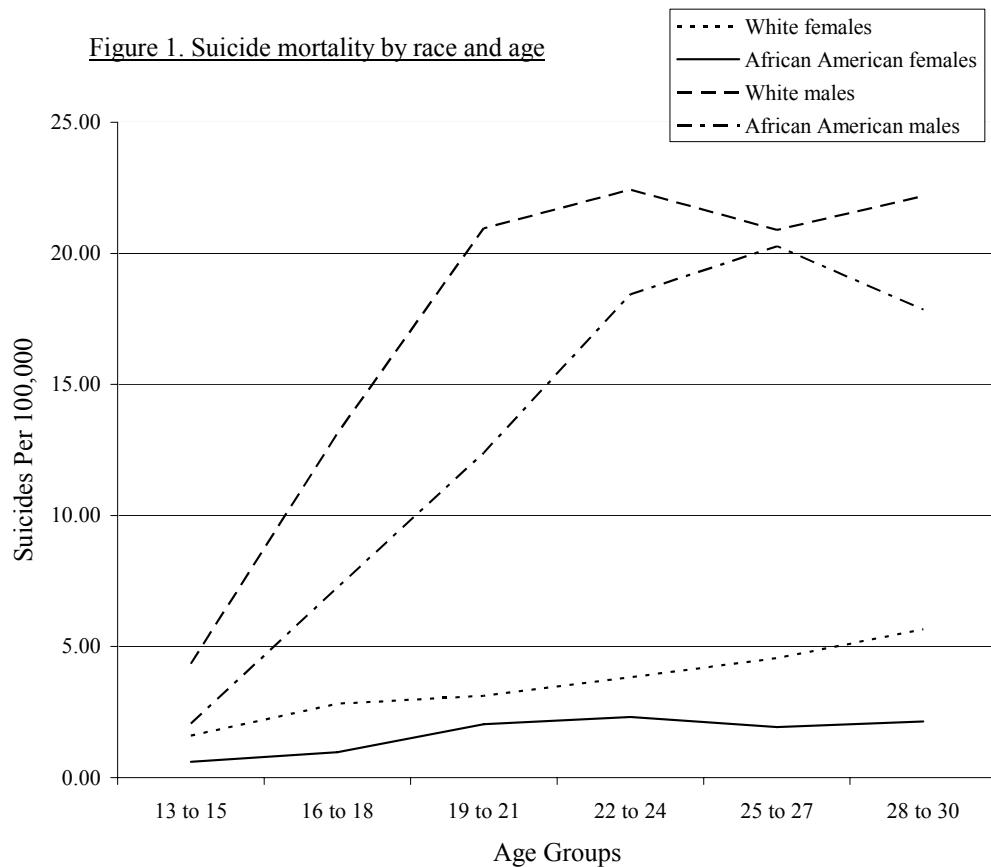
2002). The total number of suicides was 29,350, or 1.2% of all deaths. Suicide deaths actually outnumber homicide deaths by five to three.

Even though most suicides happen among older individuals, suicide is also a significant health problem among adolescents and young adults. In 2000, suicide was the third leading cause of death among 15- to 24-year-olds, following unintentional injuries and homicide (Miniño et al., 2002). Further, there are between 8 and 25 attempted suicides per every suicide death (Moscicki, 2001). Attempted and completed suicide among adolescents in the United States causes economic losses of over 15 billion dollars per year in medical expenses, loss of future earnings, and pain, suffering, and quality of life loss to victims and their families (Miller, Covington, & Jensen, 1999). According to the 1999 national Youth Risk Behavior Survey, 8.3% of U.S. high school students attempted suicide during the year preceding the survey (10.9% of high school females; Centers for Disease Control and Prevention, 2000). Approximately 19-23% of adolescents report lifetime suicidal ideation (Fergusson, Horwood, Ridder, & Beautrais, 2005; Lewinsohn, Rohde, & Seeley, 1996). When assessed from early adolescence through young adulthood, suicidal ideation rates increase during early adolescence, reaching a peak during mid-adolescence at approximately the age of 14–15 years, and decline thereafter (Kessler, Borges, & Walters, 1999; Lewinsohn et al., 1996).

Researchers have also noted some differences in the prevalence of suicidal ideation and attempts among members of various racial and ethnic groups. Among African Americans, suicides are much more common among young adults than older adults, a pattern that differs significantly from Whites (Garlow, Purselle, & Heninger, 2005). The window of vulnerability for African American women is narrow; for them,

the vast majority of completed suicides occur between the ages of 20 and 45. Studies show that African American youth tend to report lower levels of suicidal ideation and fewer suicide attempts than White youth (Blum et al., 2000; Centers for Disease Control and Prevention, 2001; Guitierrez, Osman, Kopper, Barrios, & Bagge, 2000; Kann et al., 2000; Morrison and Downey, 2000; Muehlenkamp, Guitierrez, Osman, & Barrios, 2005). However, these studies fail to further examine the rates of suicidality by sex within ethnic groups, which is a major predictor of suicidal behavior.

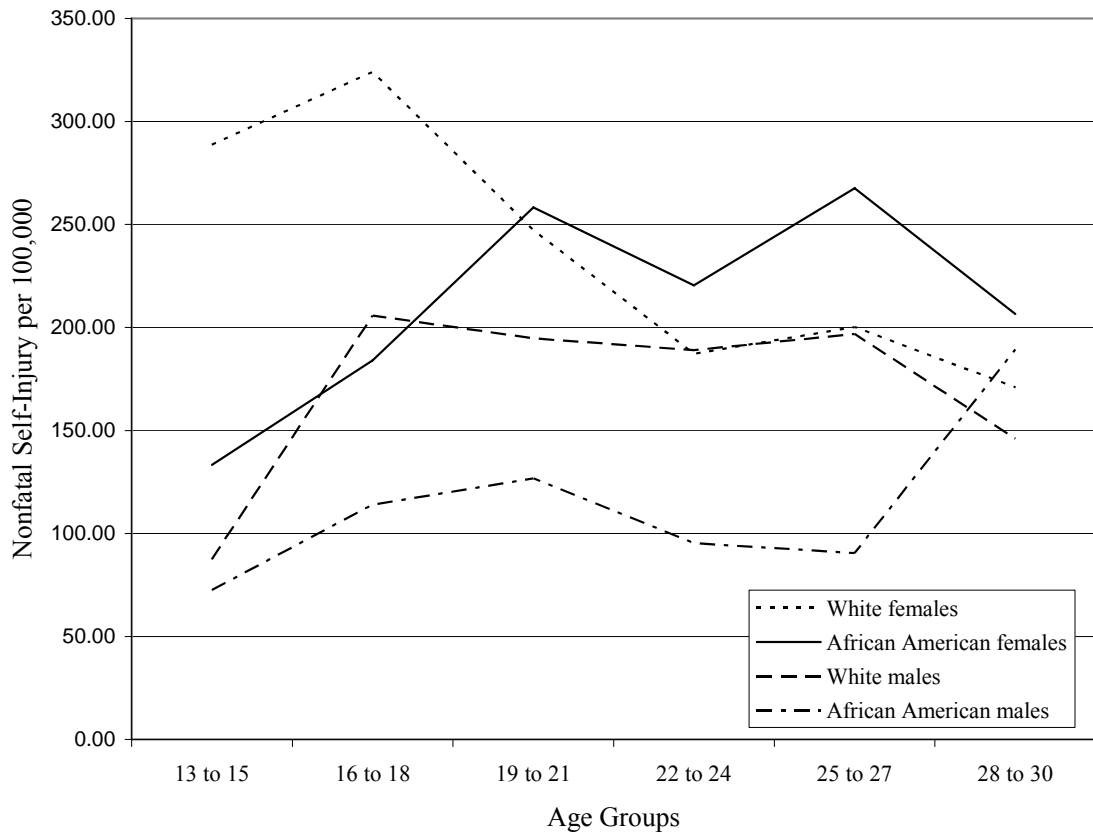
Figure 1 shows data collected by the CDC in 2002 concerning suicide mortality rates by age, sex, and race (National Center for Injury Prevention and Control [NCIPC], 2005). Among adolescents and young adults, deaths by suicide in adolescents and young



adults are more common among males than females, and are more common among Whites than African Americans. However, this pattern may not hold for suicide attempts and/or self-harm.

Figure 2 shows data concerning nonfatal self-injury from cases presenting at US emergency rooms (NCIPC, 2005). Note that this data lacks information on the intent of the self-injurious behavior, whether lethal or nonlethal. Furthermore, many cases of attempted suicide and self-harm do not reach the attention of health care professionals, so this graph shows only a limited sample of what are likely the most medically severe self-injurious behaviors. Despite these limitations, it is significant to note that in this dataset nonfatal self-injuries are more common in women of either race than in men. Also, while

Figure 2. Nonfatal self-injuries presenting at emergency rooms, by race and age



White women are most likely to engage in these behaviors in mid-adolescence, African American women are most likely to engage in these behaviors in early adulthood. Therefore, the results of previous studies on rates of suicide attempts by race, showing that African Americans have lower rates of suicide attempts than do Whites, may obscure the significant findings that there is an important race by sex interaction, and that African American women are actually engaging in nonfatal self-injurious behavior quite frequently compared to other demographic groups.

These findings lead to several further questions, concerning whether the observed racial differences hold for all nonfatal self-injurious behaviors (not just those which are medically treated), whether the observed racial differences also hold for suicide attempts versus self-harm, and whether the apparent developmental trend in which White women have higher rates of nonfatal self-injury in adolescence, while African American women have higher rates of nonfatal self-injury in early adulthood, would hold for suicide attempts and self-harm taken individually. Finally, this data underscores the importance of studying self-harm and suicidal behavior in young women. Despite the fact that males of this age group are more likely to complete suicide, women of this age group are far more likely to attempt suicide and self-harm, particularly in ways that require medical attention.

Distinguishing Self-Harm and Suicide

Despite the fact that self-harm behaviors are sometimes referred to as "parasuicide," most researchers recognize that the self-injurer generally does *not* intend to die. Herpertz (1995) notes that self-injurers themselves distinguish between self-injurious and suicidal acts. According to individuals who self-harm, in comparison to self-harm

behaviors, suicide attempts are repeated less frequently, fail to provide relief, and have less communicative value. Solomon and Farrand (1996) write that "although the [self-injurious and suicidal] acts themselves may blur, their meaning does not. What does emerge, though, is a link between the two acts in that one (self-injury) is an alternative to the other (suicide), and is preferable." In a review of the literature on self-injury, Favazza (1998) notes that recently it has become generally recognized that self-harm is a morbid form of coping, one which is often turned to in an effort to avoid suicide.

Although self-injurious behavior is not in and of itself suicidal in intent, it can easily lead to suicidal ideation or even, when a self-harmer goes too far, suicide itself. The link between self-harm and suicide is a strong one; subsequent suicide occurs in somewhere between 1 in 200 and 1 in 40 self-harm patients in the first year of follow-up and in around 1 in 15 people after 9 or more years (Owens et al., 2002). Although a functional distinction can be drawn between self-harm and suicidal behavior, there is considerable overlap between the two.

The causes of suicidal behavior are not fully understood; the causes of self-harm behavior are even less clear. A better understanding of etiology will lead to the development of more effective prevention strategies to reduce the prevalence of these dangerous behaviors which pose a considerable burden to public health. For example, if the same risk factors lead to both types of behavior, it is likely that prevention programs could be designed to simultaneously reduce the prevalence of both suicidal and self-harm behavior, but if risk factors for these behaviors differ significantly, different prevention strategies may be required. Furthermore, if genetic risk factors are more influential than environmental risk factors, prevention programs might most effectively target individuals

with a family history of the behavior. On the other hand, if shared environmental factors are most influential, prevention programs might most effectively target features of the home environment. Finally, the relative strength of risk factors for self-harm and suicidality may vary by race. If so, this would influence the targets of prevention programs designed for specific racial groups.

Given the enormous costs associated with self-harm and suicidality, some investigators have explored the etiology of these behaviors. Owing to low base rates, they have proven difficult to predict. However, certain factors are reliably associated with self-harm and suicidal behavior. Of these factors, some of the most-studied show that self-harm and suicidal behaviors tend to run in families.

Self-Harm and Suicidal Behaviors are Familial

The self-harm behavior or suicidality of a family member is associated with self-harm behavior. One study has addressed this topic (Hawton, Haw, Houston, & Townsend, 2002). Of a sample of self-harm patients, those with and without a positive family history of suicidality were compared with regard to the nature and repetition of their self-harm episodes, and psychological and psychiatric characteristics. About one-third of the self-harm patients had a family history of suicidal behavior. Self-harm was more frequent in patients' mothers than fathers. The researchers concluded that a family history of suicidal behavior contributes most to the initiation of self-harm behavior rather than to the maintenance of self-harm behavior.

Similarly, a substantial factor in the prediction of suicidal behavior is having family members who have attempted or committed suicide. Evidence that suicide can run in families has been found in both case reports and epidemiological studies. One well-

known case is that of the novelist Ernest Hemingway's family, in which five members over four generations have died from completed suicides. Epidemiological studies, based on clinical patients or community samples, have consistently demonstrated a significantly higher risk for suicidal behavior among family members of suicide victims and attempters (Gould, Fisher, Parides, Flory, & Shaffer, 1996; Roy, 1983). Another study, which included all 21,168 suicides during a 17-year period in Denmark, demonstrated that suicide mortality in the first-degree relatives of suicide victims is 3.5 times that in the first-degree relatives of controls matched for age, sex and date of suicide (Qin, Agerbo, & Mortensen, 2003). Results from this study further indicated that people with a family history of completed suicide relative to those without such a family history are at a 2.1-fold increased risk of committing suicide even after adjusting for differences in individual socioeconomic status and psychiatric history.

Just one study has examined the differential racial familiarity of suicide. Roy (2003) reported that among African American and White substance dependent suicide attempters, the White participants were significantly more likely to have a family history of suicide. This suggests that perhaps genetic influence on suicidal behavior is less strong in African Americans than in Whites. Further research is necessary to replicate and explore the nature of this finding.

Using different methodologies, many other researchers have also come to the conclusion that suicidal behavior runs in families. Farberow and Simon (1969) reported that among 100 suicide victims in Vienna and Los Angeles, 6 had a parent who had committed suicide, a rate over 80 times what would be expected. Similarly, Robins, Schmidt, and O'Neal (1957) found that 11% of 109 suicide attempters had a family

history of suicidal behavior. In another study, one third of 55 callers to a suicide prevention center had a family history of suicidal behavior, and this was significantly more likely to be the case if the caller had previously attempted suicide himself or herself (Murphy, Wetzel, Swallow, & McClure, 1969). Murphy and Wetzel (1982) interviewed individuals hospitalized following a suicide attempt. Of the 127 patients, 14% reported a family history of completed suicide, 27% a family history of attempted suicide, and 6% a family history of suicide threats. One or more of these familial suicidal behaviors was reported by one third of the suicide attempters. Woodruff, Clayton, and Guze (1972) noted a statistically significant increase in the likelihood that a psychiatric outpatient with a history of suicide attempt would also have a family history of suicide attempt, relative to outpatient controls. Interestingly, another study has shown that having a history of suicidality in family members is more strongly linked to suicidality in females than in males (Hjelmeland & Bjerke, 1996).

Similar studies have been conducted to assess family history of suicide among adolescents who have attempted or completed suicide. For example, Tishler, McKenry, and Morgan (1981) found that 22% of 108 adolescent attempters seen at a children's emergency hospital room after attempting suicide had a family history of suicidal behavior. Garfinkel, Froese, and Hood (1982) found that significantly more of 505 adolescent suicide attempters than controls had a family history of suicidal behavior (8% vs. 1%). More recently, Pfeffer, Normandin and Kakuma (1994) found more suicide attempts among the first degree relatives of adolescent suicide attempters compared with first degree relatives of normal control children. In that study, 50% of the mothers of adolescent attempters reported a history of suicide attempt. Several studies have also

reported on family history of suicidal behavior in adolescents who completed suicide. Shaffer (1974) reported that suicidal behavior in a first-degree relative had occurred in 7 of 31 youth suicides. Results from a series of psychological autopsies on 20 adolescent victims of suicide indicated that significantly more of the suicide victims than controls had a family history of suicide (Shafii, Carrigan, Whittinghill, & Derrick, 1985). Finally, in a study of 52 suicide victims under the age of 19, 20 (38%) had a relative who either committed or attempted suicide (Shaffer, Gould, & Trautman, 1985).

One study to date has examined the familial transmission of suicidal ideation. Farmer et al. (2001) examined this issue in a sib-pair study of depressed and healthy participants and their siblings. They concluded that suicidal ideation is not familial, finding no significant sib-pair correlations. This evidence suggests that having thoughts of suicide might be determined by quite different factors than those that determine suicidal behavior. However, this was a moderate size study relying on past-week suicidal ideation (which has a low base rate), and therefore statistical power was low. Further research is necessary to resolve the question of whether suicidal ideation is familial.

In sum, considerable evidence exists to show that self-harm and suicidal behavior runs in families, and that the familial transmission of suicidal behavior is evident in adolescence as well as adulthood. Only one study to date addresses whether suicidal behavior has stronger familial transmission among different ethnic or racial groups, and no studies address this issue for self-harm. The major limitation of studies reviewed above is that they cannot identify whether the familial resemblance for self-harm and suicidal behaviors is passed on by means of genetic or environmental transmission. Most likely a combination of genetic and environmental transmission is in operation. In

contrast to the previously reviewed studies, behavior genetic studies by design allow for the statistical estimation of separate genetic and environmental influences.

Genetic Influence on Self-Harm and Suicidal Behavior

Behavior Genetic Studies

For quite some time, researchers have speculated that the propensity for suicidal behavior is to some extent genetically inherited. In recent years, there have been several studies conducted on the heritability of suicidal behavior using genetically informative samples (for a review, see Roy, 1992). For example, a few studies using small twin samples have shown that monozygotic (MZ, or identical) twins are more highly concordant for suicidality than are dizygotic (DZ, or fraternal) twins. Because MZ twins share 100% of their genes, while DZ twins share 50% of their genes, yet both types of twins share a family/rearing environment, twin studies allow for the disentanglement of genetic and shared environmental factors. One small study of twins demonstrated that monozygotic twin pairs have significantly greater concordance for both completed and attempted suicide than dizygotic twin pairs (Roy, Segal, Centerwall, & Robinette, 1991). In addition, the living MZ co-twins of suicide completers are more likely to have attempted suicide themselves than living DZ co-twins of suicide completers (Roy, Segal, & Sarchiapone, 1995). However, MZ and DZ twins whose co-twins have died of causes other than suicide do not show differential rates of suicide attempts or suicidal ideation, ruling out the alternative hypothesis that suicidality in co-twins of suicide victims might be due to extreme grief reactions (Segal & Roy, 2001). This evidence clearly suggests that there is in fact a genetic component to suicidal behavior.

Several adoption studies have also examined the issue. Results indicate that suicide is more common among biological relatives of adopted suicide completers than among biological relatives of adopted controls, and suicide attempts more likely among biological relatives of adopted suicide attempters than among biological relatives of adopted controls (Wender et al., 1986). Shulsinger, Kety, Rosenthal, and Wender (1979) examined suicide in the relatives of 71 adoptees with affective disorders who had completed suicide. Substantially more of their biological relatives had completed suicide than their adoptive relatives, although there were no significant differences between the biological and adoptive relatives of a control group of adoptees with affective disorders who had not completed suicide.

In a second study, the same researchers examined another sample of adoptees without known psychiatric disorder who had completed suicide. The biological relatives of these individuals were significantly more likely to complete suicide than the biological relatives of adoptee controls, but there were no suicides in the adoptive families of either group. Shulsinger et al. (1979) therefore suggested that the liability for suicidal behavior is genetically transmitted independently of the liability for psychiatric illness. In sum, these findings suggest that genetic factors are to some degree responsible for suicidality clusters in families.

Despite the plethora of evidence implicating genetic factors in the familial transmission of suicidal behavior, no behavior genetic studies have yet been conducted to assess the heritability of self-harm behavior. Given that one study to date has indicated that self-harm behavior is familial, and given the similarities between suicidal and self-

harm behavior, it is likely that self-harm behavior is to some extent heritable. A behavior genetic study is necessary to support or to disconfirm this hypothesis.

Twin Studies

Three major behavior genetic studies on suicide have been conducted using twin study methods and formal statistical modeling. Statham et al. (1998) studied suicidal behavior among about 6000 adult members of an Australian volunteer twin sample. They focused on four aspects of suicidality: suicidal thoughts, persistent suicidal thoughts lasting more than one day, suicidal plans, and suicide attempts, and also gathered data on treatment and hospitalization for attempted suicides, and any suicides and attempted suicides in first degree relatives. About 23% reported having ever had suicidal thoughts, 8% reported suicidal thoughts lasting more than a day, 6% a suicidal plan, and 2% a suicide attempt. The observed rates of suicidal behavior did not vary by sex or birth cohort (except the very oldest cohort, which had a somewhat lower rate of suicidal behavior). Genetic factors accounted for 44% of reporting any suicidal ideation, any suicidal plan, or suicide attempt on a severity scale (95% CI: 33-51%), and 55% of making a serious suicide attempt. The researchers also ruled out the alternative hypothesis that social contagion, an environmental factor, could account for the greater concordance among MZ twins relative to DZ twins. If social contagion was in operation, co-twins of those who had made suicide attempts would be more likely make an attempt soon afterwards, but the actual time elapsed between attempts in cases where both members of a twin pair attempted suicide was too great to support that hypothesis.

Glowinski et al. (2001) examined the heritability of suicidal behaviors including thoughts, plans, and attempts in an adolescent female twin sample (average age 15).

Over three thousand twins from Missouri participated in the study. The researchers found that 16% reported having ever had suicidal thoughts, 7% reported suicidal ideation lasting more than a day, 5% reported having made a suicide plan, and 4% reported at least one suicide attempt. Notably, African American adolescents were significantly more likely to report a suicide attempt, with a prevalence of 8% in this group. Additive genetic effects accounted for 45% of the variance in suicide attempts (95% CI: 0-73%), shared environmental effects accounted for 8% of the variance in suicide attempts (95% CI: 0-59%), and nonshared environmental effects accounted for 44% of the variance in suicide attempts (95% CI: 27-67%). However, the 95% confidence intervals for both additive genetic and shared environmental effects included zero, likely due to the low base rate of suicide attempts, so the researchers concluded that there is a familial liability to youth suicide attempt, with genetic and shared environmental factors accounting for between 33% and 73% of the variance in risk. The twin/cotwin suicide attempt odds ratio was 5.6 for monozygotic twins and 4.0 for dizygotic twins (after controlling for other psychiatric risk factors). Glowinski et al. were also able to rule out the contagion hypothesis as an alternative explanation for the observed familial transmission of suicidal behavior.

Fu et al. (2002) conducted another behavior genetic study of suicidality in men. In this study, suicidal ideation and attempts were studied in over three thousand twin pairs from the Vietnam Era Twin Registry. The prevalence of suicidal ideation in this sample was 16.1%, and the prevalence of suicide attempts was 2.4%. Results showed that additive genetics accounted for 47% of variance in suicidal ideation and 30% of the variance in suicide attempts (95% CI: 34-53% for suicidal ideation, and 95% CI: 22-34% for suicide attempts). Shared environment accounted for 19% of the variance in suicide

attempts, but none of the variance in suicidal ideation. These researchers then further investigated whether the same genetic and environmental factors influenced both suicidal ideation and attempt (a single liability dimension), versus whether independent genetic and environmental factors influenced risk for suicidal ideation and risk for suicide attempt in those who developed suicidal ideation (independent liability dimensions), versus whether correlated genetic and environmental influences influenced both behaviors. The best fitting model included correlated genetic and correlated environmental factors. In fact, there was complete overlap of genetic influences on suicidal ideation and suicide attempt. The estimated genetic variance specific to attempt after controlling for ideation was zero.

Results from these three twin studies as well as from earlier family and adoption studies clearly show that suicidal behavior is substantially heritable, with heritability estimates ranging from 0.43 to 0.47 for suicidal ideation and 0.30 to 0.55 for suicide attempts. However, to know that suicidality is heritable is only the first step. We must next ask how (or by what mechanism) it is transmitted. Suicidal behavior is far too complex a behavior to be associated with single gene. Likely there are many genes that influence suicidal behavior, and intermediate endophenotypes which are steps in the chain between genotype and observable behavior. Several different theories have been proposed to account for the genetic transmission of suicidal behavior.³

³ The following material concerning mechanisms of genetic transmission applies to suicidal behavior only and not to self-harm behavior, except where noted. Given that there is so little research to date on the heritability of self-harm behavior, there are not yet theories of how it might be genetically transmitted if indeed it proves to be heritable.

Putative Mechanisms of Genetic Transmission

Psychiatric illness. Over 90% of suicide victims have a psychiatric disorder at the time of death (Isometsa et al., 1995; Strakowski, McElroy, Keck, & West, 1996). This proportion is substantially similar for both Whites and African Americans both globally and by type of symptoms (including depressive, psychotic, antisocial, and substance abuse; Castle, Duberstein, Meldrum, Conner, & Conwell, 2004; Oquendo, Lizardi, Greenwald, Weissman, & Mann, 2004). For most psychiatric diagnoses, there is evidence to suggest that each is to some degree heritable (e.g., Kendler, Prescott, Myers, & Neale, 2003). Therefore, it is possible that suicide is familial because psychiatric illness, which sets the stage for suicide, is genetically transmitted. Some evidence exists to lend support to this theory.

Suicide tends to occur in families with psychiatric history. For example, in the Hemingway family a number of the family members, including the novelist himself, suffered mental and/or substance abuse disorders. Previous studies have demonstrated that psychiatric disorders are more prevalent among relatives of suicidal individuals, and individuals with a family history of psychiatric illness are at an increased risk for completed or attempted suicide (Gould, Fisher, Parides, Flory, & Shaffer, 1996; Wagner, 1997). Qin et al. (2003) showed that, in the context of other risk factors, there is an approximately 1.3 relative risk for completed suicide associated with a family history of psychiatric illness leading to hospitalization. Another study demonstrated that an increased risk was associated with a parent's psychiatric history but that the relative risk was not significantly different according to the parent's diagnosis of psychiatric illness (Agerbo, Nordentoft, & Mortensen, 2002). The overall findings from these studies

suggest that there is a genetic susceptibility to suicidal behavior in people with a family history of mental disorders.

There have been several reports of familial aggregation of suicidal behavior in adults (e.g., Egeland and Susse, 1985) and a few family studies of youth suicidal behavior as well (Brent, Bridge, Johnson, & Connolly, 1996; Bridge, Brent, Johnson, & Connolly, 1997), which all found familial factors even when controlling for psychiatric disorders. Tremeau et al. (2005) reported that a family history of suicide predicted more serious suicide attempts, and a greater number of attempts independent of psychiatric diagnosis. Another large study, including 4,262 suicide victims and 80,238 population-based controls, demonstrated that a completed suicide and a hospitalized psychiatric disorder in a parent or sibling act independently as risk factors for suicide in the general population (Qin, Agerbo, & Mortensen, 2002). Their effects could not be explained by socioeconomic, demographic and psychiatric status differences in the population. These findings also demonstrated that a family history of psychiatric illness significantly interacts with an individual's psychiatric status, increasing suicide risk only in people without a psychiatric hospitalization history, whereas a family history of completed suicide significantly increased suicide risk independently of a family history of psychiatric disorders or mental illness in subjects. Those with a family history of suicide were two and a half times more likely to take their own life than were those without such a history. And a family history of psychiatric illness requiring hospital admission increased suicide risk by about 50 percent for those who did not have a history of psychiatric problems themselves. Both types of family history boosted risk, but the effect

was strongest for individuals whose family history included both suicide and psychiatric illness.

In the same study, suicide risk associated was also transformed into population attributable risk. Population attributable risk is the proportion of deaths in the population that could be attributed to a particular cause of mortality (in this case, either family psychiatric history or family history of suicide). A family history of completed suicide accounted for 2.3% of the total suicides while a family history of hospitalized psychiatric illness accounted for 6.8% of the suicides, for a total of 9.1% (Qin et al., 2002). The attributable risk associated with family history is higher for younger people. For instance, Agerbo et al. (2002) estimated that, for people under age 21, about 12.8% of suicides would not occur if exposure to suicidal death and hospitalized psychiatric illness in parents were eliminated. These estimates of population attributable risk would undoubtedly be larger if family history of suicide attempts or family history of psychiatric disorders not resulting in hospitalization were also included.

In sum, current research suggests that the effect of family suicide history on suicide is independent of familial cluster of mental disorders. A family history of psychiatric illness only increases suicide risk through increasing the risk for developing a mental disorder, while a family history of completed suicide significantly increases suicide risk in its own right. Even though the vast majority of suicide victims have a psychiatric illness, most with such a disorder do not commit suicide (e.g., Johns, Stanley, & Stanley, 1986; Roy & Linnoila, 1986). Psychiatric disorder is generally a necessary but insufficient condition for suicide. Therefore, other mechanisms of genetic transmission must be in effect to account for the observed heritability of suicidal behavior.

Serotonin. Molecular studies have implicated biological factors in suicidal behavior. Many scientists now believe that there is an association between suicidal behavior and the molecular genetics of serotonin (for reviews, see Joiner, Brown, and Wingate, 2005; van Heeringen, 2003). Serotonin is a monoamine neurotransmitter which affects many functions including mood, sleep, and appetite. Several indices of serotonergic functioning have been employed in lab studies, including the cerebrospinal fluid (CSF) level of 5-hydroxyindoleacetic acid (5-HIAA, a serotonin metabolite), prolactin response to serotonin agonists such as fenfluramine hydrochloride, and platelet serotonin-related proteins or serotonin content. These three different measures of serotonin function (which are completely unrelated approaches) all show similar results, a serotonin deficiency in patients with a history of suicide attempts, especially highly lethal suicide attempts (Mann et al., 1992).

The D-fenfluramine challenge test gives an indirect index of the functional state of the serotonergic system (Mann et al., 1992). The prolactin response to D-fenfluramine, a specific 5-HT releaser and reuptake inhibitor is a measure of central 5-HT activity. Reduced prolactin levels are suggestive of a deficit in overall central 5-HT function. Compared to a control group of depressed women, women with depression and a history of suicide attempts show a blunted prolactin response to the D-fenfluramine challenge test, and young women with a history of self-harm behavior show an intermediate prolactin response which is significantly blunted relative to controls yet less blunted than women with a history of suicide attempts (Herpertz, Steinmeyer, Marx, Oidtmann, & Saß, 1995).

While serotonin levels can be affected by environmental factors such as diet and medications including the selective serotonin reuptake inhibitors, genetic factors are highly influential. Nielsen et al. (1994) were the first to report an association between suicidal behavior and a molecular genetic variant. Tryptophan hydroxylase (TPH) is the enzyme involved in the biosynthesis of serotonin. It catalyzes the oxygenation of the amino acid tryptophan to 5-hydroxytryptophan, which is decarboxylated to serotonin. Nielsen et al. had earlier identified a polymorphism in the human TPH gene and mapped it to chromosome II. They identified two alleles, U and L. Knowing that low concentrations of 5-HIAA were associated with suicidal behavior, they hypothesized that such individuals may have alterations in genes controlling serotonin synthesis and metabolism. Thus, they studied a group of patients for whom CSF 5-HIAA concentrations had been determined, and found an association between TPH genotype and CSF 5-HIAA concentrations. Further, a history of suicide attempts was significantly associated with either a UL or LL TPH genotype. Nielsen et al. concluded that the L allele is associated with suicidal behavior.

These results have been replicated (Buresi, Courtet, Leboyer, Feingold, & Malafosse, 1997; Mann et al., 1997; Persson, 1999; Pooley, Houston, Hawton, & Harrison, 2003), but at least one study has shown negative findings (Abbar, Courtet, Malafosse, & Castelnaud, 1996). Other allelic association studies have identified possible associations between suicidal behavior and a polymorphism of the serotonin transporter gene (Joiner, Johnson, & Soderstrom, 2002; Mann et al., 2000) as well as several 5-HT receptor loci and 5-HT promoter polymorphisms (Anguelova, Benkelfat, & Turecki, 2003; Campi-Azevedo, Boson, De Marco, Romano-Silva, & Correa, 2003).

One study has also linked attenuated levels of peripheral serotonin with self-harm in adolescent girls (Crowell et al., 2005). This suggests that serotonin may play a role in self-harm as well as suicidal behavior.

Personality traits. Building on evidence from molecular studies, though at a different level of analysis, some researchers have explored the relationship between personality traits and suicidal behavior. Several existing studies have outlined a series of relationships between the traits of impulsivity, aggression, and neuroticism, and suicidal behavior. The observed familial aggregation of suicidality may be due to genetic factors related to these personality traits, which are themselves heritable (for review, see Jang, Vernon, & Livesley, 2001). In particular, impulsivity/aggression is related to the serotonergic system and may be linked to the homozygous LL TPH genotype (New, Goodman, Mitropoulou, & Siever, 2002), as well as to the polymorphism of the serotonin transporter gene which is linked with suicide attempts (Courtet et al., 2004). Impulsivity is linked to lower serotonin levels and may be one of the intervening factors between serotonergic activity and suicidal behavior (Mann, 1987). Several studies have found that lowered CSF 5-HIAA is state-independent: patients who have made suicide attempts and have low CSF 5-HIAA levels continue to have these low levels even after their suicidal symptoms have dissipated, consistent with the characterization of impulsivity as a predisposing, stable personality trait (van Praag, 1986).

Impulsivity has proven to be related to suicide attempts and ideation across a range of studies (Apter, Plutchik, & van Praag, 1993; Beautrais, Joyce, & Mulder, 1999; Brent et al., 1993; Brent, Johnson, et al., 1994; Pfeffer, Newcorn, Kaplan, Mizruchi, & Plutchnik, 1988; Velting, Rauths, & Miller, 2000). Two studies have shown mixed

results. While in a univariate model of suicidality in a longitudinal sample of adolescents, impulsivity significantly predicted suicidal ideation, plans, and attempts, in a multivariate model including prior suicidal behavior and negative life events, impulsivity predicted suicide plans but not ideation or attempts (McKeown et al., 1998). Also, Brezo et al. found that impulsivity contributed to suicidal ideation but not suicide attempts even in the univariate case. In the same study, however, aggression as measured by conduct problems did predict both ideation and attempts. Relative to other psychiatric inpatients, inpatients admitted for a suicide attempt show significantly greater impulsivity scores both at admission and four weeks later, although impulsivity scores fell slightly over time in both groups, suggesting that impulsivity may be both a trait and a state in suicide attempters (Corruble, Damy, & Guelfi, 1999).

Eysenck's Psychoticism dimension is related to impulsivity but also includes tendencies such as tough-mindedness, disregard for common sense, non-acceptance of cultural norms, aggression and inappropriate emotional expression. Psychoticism scores are associated with suicide ideation and attempts, number of suicide attempts, and violent (versus nonviolent) attempts (Edman, Asberg, Levander & Schalling, 1986; Lolas, Gomez, & Suarez, 1991). In another study, recent suicide attempters showed higher scores on both neuroticism and psychoticism from the Eysenck Personality Questionnaire (EPQ) relative to sex- and age-matched surgical controls (Nordstrom, Schalling, & Asberg, 1995). Farmer et al. (2001) found that suicidal ideation is also related to increased EPQ neuroticism and psychoticism. Familial transmission of the liability for suicidal behavior also involves the transmission of aggression, which also overlaps with

impulsivity. In two studies, aggression appears to partially mediate the familial transmission of suicidality (Brent, Bridge, Johnson, & Connolly, 1996; Kim et al., 2005).

Neuroticism is also related to suicidal behavior. Roy (2002) conducted a study to assess whether family history of suicide increases risk of suicidal behavior through the effect of personality. He compared psychiatric outpatients with and without a family history of completed suicide, matched for age, sex, race, and primary diagnosis, and found a nonsignificant trend for higher EPQ neuroticism scores among the family history positive group. Eysenck's Neuroticism dimension was significantly linked to suicidal ideation, suicidal plan, and suicide attempts in the twin study conducted by Statham et al. (1998). Fergusson, Beautrais, and Horwood (2003) found in a longitudinal study that EPQ neuroticism scores at age 14 were significantly predictive of both suicidal ideation and attempts by age 21 in multivariate models including family history of suicide, abuse history, other school and family factors, and other personality traits. Similarly, in another study neuroticism accounted for significant variance in the prediction of serious suicide attempts in a multivariate model including age, gender, hopelessness, and external locus of control (Beautrais, Joyce, & Mulder, 1999).

Some researchers conceptualize suicidality within a diathesis-stress model, in which personality traits constitute the diathesis and an Axis I disorder or major life stress constitutes the stressor. In a test of this model, both trait aggression and impulsivity were significantly greater in patients with a history of suicide attempt relative to non-attempters with the same psychiatric diagnosis (Mann, Wateraux, Haas, & Malone, 1999). Similarly, a combination of impulsivity and aggression predicted suicide attempts in patients with major depressive disorder or bipolar disorder who had recently

experienced a depressive episode (Oquendo, Lizardi, Greenwald, Weissman, & Mann, 2004). Thus, there is some evidence supporting impulsivity as a vulnerability factor which may interact with stresses such as psychiatric illness to result in suicidal behavior.

A few studies have also addressed the relationship between personality traits and self-harm behavior. Results indicate that the same personality traits of neuroticism and impulsivity that are associated with risk for suicidal behavior are also associated with risk for self-harm behavior. One study has shown that neuroticism is related to self-harming behavior (Roy, 1978). Herpertz et al. (1995) also found that a combination of self-directed aggression and symptoms of depression best discriminated women who self-harmed from normal controls. In fact, increased impulsivity and aggression also distinguish suicide attempters who have a history of nonsuicidal self-harm from suicide attempters without such a history (Stanley, Gameroff, Michalsen, & Mann, 2001). While impulsivity also predicts repetition of suicide attempts, results from one study suggest that there is no analogous relationship between impulsivity and repetition of deliberate self-harm behavior (Hawton, Kingsbury, Steinhardt, James, & Fagg, 1999).

Similar to research on mental illness showing that African American and White individuals who attempt suicide have approximately the same rate of different diagnoses, there are comparable findings on the contribution of personality traits to suicidal behavior in different ethnic groups. Roy (2003) noted that in a group of African American and White substance dependent suicide attempters scores on the personality traits of neuroticism, psychoticism, hostility, and extraversion did not significantly differ between groups. However, this study relies on a selected sample of individuals with a particular psychiatric diagnosis, and the findings may not generalize to an unselected population. In

addition, no research has yet addressed whether the strength of the relationship between personality traits and self-harm varies among ethnic groups. Further research is needed to determine whether the finding that impulsivity and neuroticism are associated with self-harm behavior generalizes across members of different ethnicities.

Shared Environmental Influence on Self-Harm

There are a number of putative shared environmental factors that may contribute to the familial transmission of self-harm behavior. These are a range of experiences that siblings share by virtue of growing up together in the same family, and which serve to make siblings more similar. Results from a wide array of studies give evidence that shared environmental experiences in the family of origin are significantly associated with later self-harm behavior.⁴ A wide range of family stressors predict self-harm behavior in children, adolescents, and adults. These variables often relate to family instability and chaos.

⁴ None of the studies reviewed under the headings of shared and nonshared environmental influences employ behavior genetic methodologies unless specifically noted. Therefore, the nature of familial transmission cannot be definitely determined. The distinction made here between genetic, shared environmental, and nonshared environmental factors is conceptual and not absolute; there is sure to be overlap between these categories. Results from these studies are reviewed to support the hypothesis that shared and nonshared environmental influences are important predictors of self-harm and suicidal behavior; and therefore shared and nonshared environmental influences will likely be significant sources of variance in the present study.

Functional and structural characteristics of the family are related to self-harm behavior. One class of family variables that is significantly associated with self-harm is early separation from parents (Gratz, Conrad, & Roemer, 2002). In one study, over half of a sample of self-harming adolescents experienced a significant disruption in attachment, having been abandoned by one or both parents early in childhood (Simpson and Porter, 1981). Early separation from parents certainly interferes with the process of forming secure attachments, and is likely a marker for other family variables such as parental incarceration or substance abuse. Robinson and Platt (1993) found that in a group of self-harming alcoholics, separation from father at an early age predicted self-harm behavior above and beyond the genetic influence of having an alcoholic father. Vivona et al. (1995) studied children and adolescents on a psychiatric inpatient unit. They found that patients who self-harmed reported more frequent disruptions in caretaking, increased likelihood of foster home placement, and a greater number of primary caretakers throughout childhood. Self-harm seems to be associated with significant disruption in the parent-child relationship, often including separation of parent and child. Children and adolescents who self-harm often lack a long-lasting, secure attachment to any adult caretaker.

Homelessness, defined as having ever lived on the streets and having deviant subsistence strategies, is also very strongly associated with self-harm behavior in adolescents (Ayerst, 1999; Tyler, Whitbeck, Hoyt, & Johnson, 2003). Researcher have found that runaway youth used self-harm as a maladaptive coping strategy for depression or stress, whereas adolescents living with their families were more likely to use productive problem solving or to discuss their problems with a trusted family member or

other adult. This theme of low family support in times of stress being related to self-harm behavior is supported by several further lines of evidence.

Among children who live with their families, low levels of family support predict self-harm behavior (Ellis, Gormley, Ellis, & Sowers, 2003). Low family support is exemplified by poor parent-child communication and low paternal and maternal warmth. An earlier study similarly demonstrated that the absence of a family confidant, less family cohesion, and poor adolescent-parent communication were among the strongest predictors of adolescent self-harm, even after controlling for other putative predictors (Tulloch, Blizzard, & Pinkus, 1997). Where Ayerst (1999) found that homeless youth used self-harm as a coping strategy for stress and depression, McLaughlin, Miller, and Warwick (1996) similarly found that adolescents who self-harmed reported using it as a coping strategy in face of significant problems with family relationships. In a small study, individuals engaging in self-harm described having a good relationship with one parent and a sharply negative relationship with the other more often than control participants (Carroll, Schaffer, Spensley, & Abramowitz, 1980). Another finding from this study was that those who self-harmed often described having a prohibition on talking about or expressing anger within the family. Favazza and Conterio (1989) reported that common family experiences in self-harm patients included excessive anger yet suppressed expression of emotions, lack of affection, and double messages. Self-harm may serve as a veiled expression of anger when feelings of anger are common but verbal communication concerning that emotion is not permitted. Martin, Rozanes, Pearce, and Allison (1995) noted that family dysfunction seemed to influence self-harm behavior indirectly, through other mediating variables such as adolescent depression.

In another study of self-cutters, neglect was significantly related to self-harm (van der Kolk, Perry, & Herman, 1991). The authors found that exposure to physical or sexual abuse, physical or emotional neglect, or chaotic family conditions during childhood and adolescence were all reliable predictors of the frequency and severity of cutting. Furthermore, the earlier the abuse began, the more likely the subjects were to cut and the more severe their cutting. Significantly, the authors noted that while sexual abuse was most strongly related to the incidence of self-harm behavior, neglect was most strongly related to the prevalence of self-harm behavior. They wrote “although childhood trauma contributes heavily to the initiation of self-destructive behavior, lack of secure attachments maintains it. Those ... who could not remember feeling special or loved by anyone as children were least able to ...control their self-destructive behavior.” They further suggested that the immaturity of the central nervous system of children may make them vulnerable to flawed biological self-regulation as a consequence of trauma and neglect. At least one study has failed to replicate this association between neglect and self-harm (Wiederman, Sansone, & Sansone, 1999). However, another study has shown that insecure attachment style is associated with self-harm behavior, partially confirming the theory proposed by van der Kolk and colleagues (Gratz et al., 2002).

Lastly, family discord or domestic violence in family of origin are also linked to later self-harm behavior (Carroll et al., 1980). Children who witness violence between their parents or primary caregivers have a similar level of risk for future self-harm as do children who are themselves the targets of physical abuse.

Not only do variables specific to the family predict suicidality, but variables at the cultural or societal level also have influence. Such predictors are also a part of the shared

environment for siblings growing up in the same family. For example, characteristics of a given geographical region including poverty, education, income level, and occupation are all predictive of suicide, in that areas with lower socioeconomic status tend to have higher rates of suicide (Rehkopf & Buka, 2006).

The prediction of self-harm behavior from shared environmental experiences is complex and multidimensional. Practically every severe stressor and risk factor studied has been shown to predict self-harm behavior. This evidence supports the existence of a strong set of shared environmental influences on self-harm behavior.

Shared Environmental Influence on Suicidal Behavior

As with self-harm behavior, certain aspects of family functioning and structure seem to increase the likelihood that children growing up in such a family will later attempt or complete suicide. These family variables are quite similar to those that are associated with self-harm behavior. Further, there are a set of family variables that appear to confer protection against the initiation of suicidal behavior.

One multivariate study provided results on a number of important family variables and their relationships with suicidality (Dube et al., 2001). In this study, self-reported suicide attempts were correlated with the total number of adverse childhood experiences: including emotional, physical, and sexual abuse; household substance abuse, mental illness, and incarceration; and parental domestic violence, separation, or divorce. The lifetime prevalence of having at least 1 suicide attempt was 3.8%. Adverse childhood experiences (ACE) in any category increased the risk of attempted suicide 2- to 5-fold. The ACE score had a strong, graded relationship to attempted suicide during childhood, adolescence, and adulthood. Compared with persons who reported no such experiences

(with a 1% prevalence of attempted suicide), the adjusted odds ratio of ever attempting suicide among persons with 7 or more experiences was 31 (with a 35% prevalence of attempted suicide).

Another multivariate study examined adolescent suicide attempts in the context of an array of family variables (Fergusson & Lynskey, 1995). These researchers found that children who belonged to low SES families or families high in conflict, who had mothers from the lowest decile of maternal emotional responsiveness, and who had moved and changed schools frequently were more likely to attempt suicide than children from a control group. While adolescent psychopathology was itself a major predictor of suicide attempts, Fergusson and Lynskey concluded that suicide attempts were the result of a causal chain in which childhood problems were related to later psychopathology which in turn predicted suicidality.

A few specific family risk factors have also been linked to suicidality. Low support from parents predicted suicide attempts in both previously hospitalized and non-hospitalized adolescents (Groholt, Ekeberg, Wichstrom, and Haldorsen, 2000). Similarly, low family support predicts suicidal ideation in African American youth (Harris and Molock, 2000). In the extreme form of low parental support, childhood neglect is also associated with suicidal behavior (Silove, George, and Bhanvani-Sankarum, 1987). Somewhat conflicting findings are noted from another study in which parental overprotection during childhood was associated with suicide attempts in later life (Curran, Fitzgerald, and Greene, 1999). Finally, a global measure of family dysfunction predicted suicidality across the continuum of behaviors, including suicidal ideation, suicide planning, and suicide attempts (Martin et al., 1995). After controlling for

psychiatric illness, having less frequent and less satisfying communication with either parent distinguished adolescent suicide completers from controls (Gould et al., 1996). Another study showed that family discord was associated with completed suicide in adolescents (Brent, Perper, et al., 1994). One study has indicated that the strength of the relationship between low family support and suicidal ideation and attempts is similar among both African American and White youth (Perkins and Hartless, 2002).

Several variables related to family constellation are significantly associated with suicidality. Children of single parents have increased risk of both suicide and suicide attempts. Even after adjusting for a number of factors, including the parent's mental health, addiction, and socioeconomic status, children of single parents still face tougher odds than children raised in two parent homes (Weitoft, Hjern, Haglund, & Rosen, 2003). Also, compared to peers in intact families, adolescents whose parents had divorced were significantly more likely to attempt suicide (Rodgers and Rose, 2002). Girls are particularly sensitive to the effects of remarriage (Ponnet et al., 2005). Rubenstein, Halton, Kasten, Rubin, and Stechler (1998) also noted that parental separation, divorce, and remarriage were associated with adolescent suicidality, in that order of strength. However, family cohesiveness also helped to alleviate risk in non intact families. The increased risk of suicidality dependent on family constellation continues throughout adulthood. Even grown children of parents who separated before the child's 16th birthday are at increased risk for suicide (Mullen, Martin, Anderson, Romans, & Herbison, 1996).

Many of the family variables mentioned up to this point are relevant to the long-term prediction of suicidal behavior. However, family variables are also relevant to the short-term prediction of suicidal behavior. For example, most members of a sample of

adolescents who were admitted to the hospital after a medication overdose reported that the suicide attempt was an impulsive act precipitated by an argument with parents, in the context of other known long-range predictors of suicidality such as low family support and insecure attachment (Schmidt, 2001). In addition, a similar study of adolescent suicide attempters found that family conflict was the most frequently reported stressor preceding a suicide attempt, and that family illness was the second most frequently reported stressor preceding a suicide attempt (Davies and Cunningham, 1999). A combination of early exposure to neglectful and overprotective parenting, followed by recent familial or interpersonal conflict or separation immediately preceding a suicide attempt best predicted suicidality in a clinical sample of adults (Silove et al., 1987). This relationship between recent family conflict and suicide attempts may be stronger for women, especially younger women, than for men (Hjelmeland and Bjerke, 1996).

Qin et al. (2002) assessed whether family history of completed suicide and mental illness are risk factors for suicide. They found that a family history of completed suicide and psychiatric illness significantly and independently increased suicide risk above and beyond the effects of socioeconomic status and the psychiatric history of suicide completers. Interestingly, a history of family psychiatric illness significantly raised suicide risk only in those without a history of psychiatric illness, whereas a family history of suicide increased suicide risk irrespective of psychiatric illness. The authors concluded that completed suicide and psychiatric illness in relatives are both risk factors for suicide, and that the effect of family suicide history is independent of the familial cluster of mental disorders. This evidence suggests that the effect of having a mentally ill parent

may go beyond the simple genetic transmission of the illness to include environmental effects.

Finally, some characteristics of the shared environment are actually associated with lower rates of suicidality. Research shows that family cohesion, communalism, and religious values are salient protective factors against suicidal behavior, particularly among the African American population (Harris & Molock, 2000; O'Donnell, O'Donnell, Wardlaw, & Stueve, 2004; Range et al., 1999). Family adaptability and cohesion and social embeddedness and support are associated with lower rates of suicide attempts in African American adults, even among those who are depressed and from a lower SES (Compton, Thompson, & Kaslow, 2005). Religiosity, which is often adopted from the family of origin, is particularly significant in that it predicts a strongly negative attitude towards suicide (Anglin, Gabriel, & Kaslow, 2005; Neeleman, Halpern, Leon, & Lewis, 1997). African American individuals generally are more religious and are less accepting of suicide than are White individuals (Neeleman, Wessley, & Lewis, 1998).

In summary, many family characteristics predict suicidal behavior. Several family characteristics predict long term risk of suicidal behavior, including low support and warmth and family constellation. In the short term, family conflicts predict suicide attempts in individuals already at risk for suicidal behavior. It is particularly striking that early childhood experiences and qualities of family life can have lifelong effects on the risk for suicidal behaviors. Models positing the potentiating effect of recent interpersonal stressors in addition to pre-existing vulnerability factors in the form of adverse family childhood experiences have garnered strong empirical support.

Most shared environmental risk factors for self-harm and suicidal behaviors are highly similar, including childhood abuse or neglect, low parental warmth and support, high family conflict, early separations from parents, parental marital status, and family history of psychiatric illness or suicidal behavior. Even though these two classes of behavior may be functionally distinct, they do predict one another and are etiologically similar.

Many of the shared environmental experiences that are associated with self-harm and suicidal behavior, including neglect, domestic violence, and homelessness, are more common among African American youth than White youth (Cousineau, 2001; Department of Health and Human Services, 2005). However, while African Americans tend to experience more of the risk factors leading to self-harm and suicidal behavior, they also tend to experience more of the protective factors such as religiosity. It is difficult to judge whether the net shared environment is more or less predictive of suicidal behavior in African Americans relative to Whites. Most importantly, mean level differences between ethnic groups in either predictors or outcomes do not necessarily imply differential correlation or a differential developmental course (Rowe, Vazsonyi, and Flannery, 1994). No research has yet addressed the question of whether the strength of the relationship between these shared family experiences and subsequent self-harm or suicidal behavior varies between members of different ethnicities.

Nonshared Environmental Influence on Self-Harm

Finally, nonshared environmental factors are those that differ for siblings from the same family and serve to make children growing up in the same family more different

from one another. In particular, one nonshared environmental factor has been related to risk of self-harm behavior: childhood physical and sexual abuse.

Childhood trauma in the form of physical and sexual abuse is strongly linked with later self-harm. In one study, 62% of repetitive self-cutters reported a history of at least one form of childhood abuse, whether physical, sexual, or both (Favazza and Conterio, 1989). In another study, 96% of self-harming women reported a history of childhood sexual abuse (Romans, Martin, Anderson, Herbison, & Mullen, 1995). Other studies have confirmed the findings that self-harm behavior in adolescents and adults is strongly and significantly associated with both childhood sexual and physical abuse (Low, Jones, MacLeod, Power, & Duggan, 2000; Wiederman et al., 1999; for a review, see Santa Mina & Gallop, 1998).

Unfortunately not all studies distinguish between abuse at the hands of a family member and abuse perpetrated by a non relative. Presumably most cases of physical abuse were perpetrated by the parents or primary caregivers. Some results show that sexual abuse by a relative, particularly by a father or stepfather, is more strongly related to later self-harm behavior than is sexual abuse by a non relative (Romans et al., 1995). Also, Turell and Armsworth (2000) reported that among incest survivors, those with greater duration or frequency of abuse or who endured multiple abuses were more likely to self-harm. Therefore cases of incest do lead to self-harm, and the more severe the abuse, the greater the likelihood of later self-harm behavior.

In summary, all forms of childhood abuse are strongly associated with later self-harm behavior. Given these findings, the nonshared environment likely plays a significant role in predicting self-harm behavior.

Nonshared Environmental Influence on Suicidal Behavior

A number of factors that would not necessarily be shared between siblings in a family are also linked to suicidality. These include demographic and social role characteristics, and childhood physical or sexual abuse.

Results from the Epidemiologic Catchment Area Program show that being separated or divorced, having low educational attainment, being unemployed, or being in active alcoholism or cocaine abuse are all risk factors for suicide attempts (Petronis, Samuels, Moscicki, & Anthony, 1990). Even cohabiting with a partner is associated with significantly greater risk of suicide relative to being married. Also, the death of a spouse by any cause is linked with suicide (Agerbo, 2003). This relationship is twice as strong when the spouse committed suicide. Furthermore, the risk of suicide increases when a spouse is admitted to a hospital for a psychiatric disorder (Agerbo, 2003). These findings could not be accounted for by assortative mating.

Similar to self-harm behavior, both childhood physical and sexual abuse are significantly associated with later suicide attempts (for a review, see Santa Mina & Gallop, 1998). The impact of emotional, physical, and sexual abuse is related in a linear fashion to suicide risk, such that women who experienced one, two, or three forms of abuse are proportionately more likely to attempt suicide (Dube et al., 2001) or to have suicidal ideation (Gutierrez, Thakkar, & Kuczen, 2000). This linear relationship between the types of abuse experienced and suicide attempts has also been documented in a sample of African American women (Anderson, Tiro, Price, Bender, and Kaslow, 2002). The severity of abuse is associated with more harmful and long-lasting effects (Beitchman et al., 1992), with increased distress (Pillay and Schoubben, 2001), and with

severity of suicidal behavior (Bensley, Van Eenwyk, Spieker, & Schoder, 1999). Finally, in a sample of nearly 15,000 adolescents, physical and sexual abuse were significantly associated with suicide attempts in both White and African American students, and the strength of that association did not significantly differ between the racial groups (Perkins & Hartless, 2002). Roy (2003) replicated this finding of racial invariance in a sample of African American and White substance dependent suicide attempters.

Bergen, Martin, Richardson, Allison, and Roeger (2003) further examined mediators of the relationship between childhood sexual abuse and later suicidality. Findings from this study of a community sample of young adolescents indicate that childhood sexual abuse is indirectly related to suicide attempts through pathways encompassing hopelessness and depressive symptoms, indicators of suicide risk such as ideation, plans, threats, and self-harm behavior, as well as directly related to suicidality. Childhood sexual abuse was independently and directly associated with suicide attempts in girls but not boys, although the latter result may be due to lack of statistical power in the study due to a small percentage of boys reporting abuse.

In summary, childhood emotional, physical, and sexual abuse are all associated with future suicidal behavior. Increased severity of abuse, and increases in the number of types of abuse suffered combine to increase future risk of suicidality. The effects of childhood sexual abuse are likely mediated by hopelessness and depressive symptoms. The nonshared environment likely exerts influence on later suicidal behavior. Current evidence suggests that while African Americans do experience approximately twice the rate of abuse than do Whites (Department of Health and Human Services, 2005), the strength of the relationship between abuse and suicidal behavior is similar between the

ethnic groups. There is no available research to indicate whether the strength of the relationship between abuse and self-harm behavior is also similar between members of different ethnicities.

The Present Study

Although some researchers draw a functional distinction between self-harm and suicidal behavior, the etiology of these two behaviors is remarkably similar. Both are familial, possibly implicating genetic influence. While suicidal behavior is clearly heritable based on results of previous behavior genetic research, there is as of yet no research on the heritability of self-harm behavior. Similar shared and nonshared environmental factors also appear to be associated with increased prevalence of both types of behavior, and these factors are common to both Whites and African Americans. No formal tests have yet been conducted on the relative influence of genetic and environmental risk among members of different ethnic groups. Finally, certain personality traits are also clearly associated with these behaviors, and preliminary evidence suggests that these personality traits may represent an intermediate step between genotype and phenotype.

Given the above evidence, the following hypotheses are addressed in the current study:

(1): An investigation of racial differences in the prevalence of suicidal thoughts, plans, attempts, and self-harm behaviors. This will replicate and extend earlier results on the differential racial prevalence of suicidal behavior. Prior research suggests that suicide attempts are about equally prevalent in adolescent African American and White females, yet African American female teens are much less likely to report suicidal ideation or

suicide plans (Centers for Disease Control and Prevention, 2000). Some evidence suggests that self-harm is less common among African Americans than Whites, but the studies showing this finding have limited generalizability.

(2): An examination of the heritability of lifetime history of suicidal behavior, including ideation, plans, and attempts, and of self-harm behavior both generally and by race. A substantial genetic contribution to variance in self-harm behavior is likely, partly because of its relationship with personality traits which are known to be inherited and partly because of its overlap with suicidal behavior which is similarly known to be inherited. Given the available research evidence on the influence of factors such as family functioning and childhood abuse, nonzero effects of the shared environment and nonshared environment are anticipated. Finally, given available evidence showing that low family support, abuse, and neglect predict suicidal behaviors equally well in both African Americans and Whites, the shared and nonshared environment will most likely have similar contributions to these behaviors in both ethnic groups.

(3): An exploration of the expected correlation between self-harm behavior and suicide attempts and determination of the extent to which genes, shared, and nonshared environment contribute to the association between the two behavioral phenotypes. Given that the similar factors are associated with risk for both behaviors, common genetic and environmental influences should predict a significant amount of variance in both self-harm and suicidal behavior. If this proves to be the case, it would simplify the design of prevention and intervention programs which could target both types of behavior simultaneously. Currently, self-harm and suicide are often treated as two separate and functionally distinct classes of behavior.

(4): An examination of the joint heritability of self-harm/suicide attempts and the personality traits of neuroticism and impulsivity in multivariate behavior genetic models. It is unlikely that genes code directly for anything as complex as these behaviors, but more likely that genes code for intermediate phenotypes that increase risk for suicidal and self-harm behavior. This study could clarify the impact of individual-level risk factors including the personality traits of impulsivity and neuroticism on suicidal and self-harm behavior. Prior studies have shown that certain personality traits are reliable predictors of both self-harm and suicidal behavior. These personality traits are associated with these behaviors in both Whites and African Americans, and are heritable. Therefore, common genetic factors will likely account for a significant amount of variance in self-harm behavior and suicidal behavior, and the personality traits of neuroticism and impulsivity. These results could further guide the understanding of the pathways from genes to self-harm and suicidal behaviors.

Additionally, this study will examine whether the expected common genetic influence on personality and self-harm behavior and the expected common genetic influence on personality and suicide attempts are each independent of genetic influence on psychiatric illness. Results from previous studies suggest that while psychiatric illness does account for some of the heritability of suicidal behavior, there is also genetic influence on suicidal behavior that cannot be explained by diagnosis. Genetic influence on personality traits could account for genetic influence on suicidal behavior that is independent of psychiatric diagnosis.

Data from this study will be unique and significant in several ways. This will be the first study of the differential prevalence of self-harm by race. Little is known about

the frequency of deliberate self-harm within varying racial groups; a concern given that self-harm is rapidly increasing in prevalence (Briere & Gil, 1998). Furthermore, this will be one of only a handful of studies to systematically investigate the differential prevalence of suicidality by race. Next, this will be the first behavior genetic study of self-harm behavior. No previous research has established whether self-harm behavior is heritable or is substantially influenced by genetic factors. Also, this study will be the first to explore etiological similarities between self-harm and suicidal behavior. Self-harm and suicide attempts are correlated behaviors, yet clinicians often treat them as distinct. These behaviors may actually be quite similar etiologically.

Another unique aspect of this study is the analysis of the differential impact of risk factors on self-harm and suicidality by race. I will determine whether risk factors operate similarly or differently between African American and White females. Finally, this study extends prior research on the genetic influence on suicidality by testing whether personality traits represent an endophenotype between genes and behavior. No prior behavior genetic studies of suicidality have gone beyond estimating heritability to exploring the mechanism of genetic transmission. This has significant implications for prevention, because while gene therapy would be an unlikely option for those with a propensity for self-harm or suicidal behavior, personality styles might be a viable target. In this way genetic risk for self-harm and suicidality might be ameliorated without changing the genes.

Implications for Prevention

The simple prevalence estimates from this study will guide prevention efforts by highlighting in which demographic groups self-harm behavior and suicidal ideation,

plans, and attempts are most common. Also, as summarized above, behavior genetic studies are quite important for determining genetic contributions to a phenotype, as well as any potential tie to endophenotypes (e.g., impulsivity) which could be targeted by means of prevention and/or treatment efforts (Heath, Slutske, Bucholz, Madden, & Martin, 1997). As previously stated, in the absence of a suitable endophenotype, genetic influence on risk would not likely be a first-line target for prevention programs.

However, behavior genetic studies are equally important for identifying environmental influences on the phenotype. For example, a behavior genetic study that implicated nonshared, but not shared, environmental influences would guide future studies as to what major variables to incorporate into etiological models (e.g., extrafamilial sexual abuse but not parental neglect). Such findings would ultimately inform and focus further prevention efforts.

In addition, results from this study will show whether the same risk factors, in the same proportions, affect self-harm and suicidal behavior in both White and African American female adolescents. These findings will suggest whether prevention programs targeting specific variables could be utilized for both populations, or whether different prevention programs targeting different risk factors may be required. Finally, behavior genetic studies will inform etiological models of self-harm and suicidal behavior by documenting the importance of certain factors that may interact with genetic predisposition to confer higher risk, and set the stage for studies of gene by environment interactions that would be quite informative for prevention efforts (Moffitt, 2005; Moffitt, Caspi, & Rutter, 2006).

Method

Participants

Participants in this study were drawn from the Missouri Adolescent Female Twin Study (MOAFTS), a prospective population-based sample of same-sex female twin pairs born in Missouri to Missouri residents. Ascertainment of families began in 1995 and is ongoing. Electronic records of multiple births were obtained from the Missouri Division of Vital Statistics under a law which allows access for research purposes. Twins adopted at birth were excluded to prevent unintentional disclosure of adoption secrets. MOAFTS twins were then located using computerized and manual techniques based on other records from the Department of Revenue and Division of Vital Statistics (drivers licenses, marriage records, etc.) and from commercial databases. These techniques resulted in the successful location of 95% of female twin pairs identified from birth records.

After a family was located, initial contact with parents/guardians was attempted by phone. If the family consented, further information regarding the study was mailed and the initial intake interview was scheduled. Of identified families, 89% were successfully contacted. For the first wave of the study, 3,416 adolescent twins were interviewed between 1995 and 2000, with a response rate of 86%. Parental interviews were also completed with at least one parent or guardian (usually the biological mother) in 86% of contacted families. The MOAFTS twin pairs were divided into birth cohorts. A cohort-sequential/accelerated longitudinal design was employed, with the first cohorts of families targeted for assessment when their twins were 13, 15, 17, or 19, and subsequent families scheduled for assessment as the twins reached these ages. More detailed

descriptions of sample ascertainment and study design (Heath, Madden, & Bucholz, 1999) as well as the representativeness of the MOAFTS sample (Heath et al., 2002) are also available.

The primary methods of data collection were telephone interviews with parents and twins and mailed self-report questionnaires for the twins. All interviews were given by lay interviewers with two weeks of training, including supervised telephone diagnostic interviews with community volunteers. All interviews were monitored until the interviewer attained a consistent standard of performance. Further, all interviews were audio taped unless the interviewee refused. The project coordinator then reviewed a 5% sample of the interviews to verify adherence to the project protocol. Different interviewers were always assigned to each twin from the same pair, and interviewers were unaware of the diagnostic status of family members. To maintain confidentiality and improve the comfort level for interviewees, respondent booklets enabled them to respond to sensitive questions with letters or numbers. A consent form in the respondent booklet was reviewed with the parents, and verbal consent was obtained before proceeding with the interview. Data for the current analysis come from three waves of assessment: the initial interview (Wave 1), a self-report assessment conducted roughly 1 year later (Wave 2), and another interview following about 4 years after that (Wave 4). See Table 1 for demographic characteristics of the sample at Waves 1 and 4.

Measures

Zygoty

A brief zygosity interview was conducted with the parents and/or guardians of twins over the telephone. Respondents were asked whether the twins have the same eye,

Table 1. Participant demographics

	Wave 1 (N=3436)	Wave 4 (N=3736)
Zygoty		
Monozygoty	1925 (56.0%)	2071 (55.4%)
Dizygoty	1511 (44.0%)	1665 (44.6%)
Age		
	<i>M</i> =15.5 (<i>SD</i> =2.42)	<i>M</i> =21.7 (<i>SD</i> =2.75)
12-13	1186 (34.5%)	
14-15	735 (21.4%)	
16-17	684 (19.9%)	
18-19	682 (19.8%)	1027 (27.5%)
20-21	136 (4.0%)	835 (22.4%)
22-23	13 (0.4%)	825 (22.1%)
24-25		618 (16.5%)
26+		430 (11.5%)
Race		
Caucasian	2928 (85.2%)	2945 (78.8%)
African American	453 (13.2%)	477 (12.8%)
Other	19 (0.6%)	21 (0.6%)
Missing	36 (1.0%)	293 (7.8%)
Educational attainment		
(Household education at Wave 1; twin education at Wave 4)		
Without high school diploma	258 (7.5%)	292 (8.8%)
High school diploma/GED	1388 (40.4%)	1059 (31.8%)
Some college	1103 (32.1%)	1239 (37.2%)
4-year degree or more	684 (19.9%)	740 (22.2%)

Table 1 (cont'd). Participant demographics

	Wave 1 (N=3436)	Wave 4 (N=3736)
Marital status		
Single	3288 (95.7%)	2512 (68.2%)
Cohabiting	69 (2.0%)	453 (12.1%)
Married	79 (2.3%)	735 (19.7%)
Annual income		
(Household income at Wave 1; twin income at Wave 4)		
\$0-5,999	153 (3.8%)	840 (25.4%)
\$6,000-14,999	320 (7.9%)	842 (25.5%)
\$15,000-23,999	412 (10.2%)	943 (28.5%)
\$24,000-34,999	591 (14.6%)	439 (13.3%)
\$35,000-74,999	1596 (39.5%)	233 (7.0%)
\$75,000+	968 (23.4%)	13 (0.4%)

Note: due to missing data, some totals by category do not equal the total for the wave of assessment

hair, and skin coloring and were asked about the height and weight of each twin. Further questions regarding zygosity, included in a follow-up interview, were based on established questionnaires designed to assess zygosity in twin samples (see Loehlin and Nichols, 1976). These questions concerned how often parents, teachers, and strangers had difficulty telling the twins apart and whether the twins looked as alike as “two peas in a pod” or like regular sisters. These questions have been determined to yield 95% or greater accuracy when compared with zygosity determinations made by genotyping

(Eaves, Eysenck, & Martin, 1989). Responses to these questions were submitted to a computer algorithm to determine zygosity.

For twins added to the sample after the first phase of recruitment, this initial parent interview is missing, and thus zygosity cannot be determined by the same algorithm. Of 2261 families in which at least one twin participated in the study, 436 were missing the parent interview. In those cases, zygosity was estimated by an alternative method based on items concerning twin physical similarity from the Wave 1 and 4 interviews. The two estimation methods produce convergent results. Of 1569 families with both estimates of zygosity available, the zygosity estimate was the same in 90.1% of families ($\kappa=0.81$, $p < 0.001$).

Self-Harm and Suicidal Behavior

Self-harm and suicidal behaviors were assessed by a semi-structured interview entitled Suicidal Thoughts and Behaviors (see Appendix A). At Wave 1, this section includes 13 question categories about lifetime suicidal ideation, plans, and attempts, as well as a question about deliberate self-harm. Family history of suicide attempts and completed suicide was also assessed by this interview. At Wave 4, detailed questions concerning the method and intent of suicide attempts were also included. The Suicidal Thoughts and Behaviors interview has shown good interrater reliability (Buchholz et al., 1994). Any responses that suggested an imminent risk of self-harm were noted and followed up by a call from the principal investigator.

The reliability and validity of the item concerning deliberate self-harm was assessed as part of the current study in a sample of 1113 undergraduates at a large Midwestern university. See Appendix B for complete analysis and results. Participants

responded to a series of self-report items in a web-based assessment, including the item on self-harm from the Suicidal Thoughts and Behaviors Interview, as well as a series of items from the Deliberate Self-Harm Inventory (Gratz, 2001). Results indicate that the self-harm item from the Suicidal Thoughts and Behaviors Interview has good validity (though validity is only fair in African Americans) but the item tends to underestimate the prevalence of self-harm in young women.

Personality

Personality traits were measured by self-report questionnaires mailed to the twins at Wave 2. These questionnaires covered a wide range of variables including alcohol and drug use, religiosity, family characteristics, and personality. 2754 individuals completed the two personality scales relevant to this analysis. A number of twins had some missing data on items from the personality inventories. To include as many individuals as possible in the analyses, the total score for a personality scale was prorated for any individual that provided data on at least 80% of the items for that scale (essentially, for any individual that had missed no more than 2 or 3 items on a scale).

Neuroticism was assessed by the Junior Eysenck Personality Questionnaire (JEPQ; Eysenck and Eysenck, 1975; see Appendix C for items). Eysenck's model of personality includes three domains: Neuroticism, Extraversion, and Psychoticism. The JEPQ also includes a validity scale entitled "Lie." High Neuroticism scorers tend to be moody, nervous, easily distressed, and sensitive, while low scorers tend to be stable, confident, poised, and emotionally self-controlled. Previous research has found that the JEPQ has good reliability and validity (Maltby and Talley, 1998). Further, the dimensions of the EPQ including Neuroticism are heritable, with substantial additive

genetic influence (Gillespie, Johnstone, Boyce, Heath, & Martin, 1991). In the current sample, the average Neuroticism score was 5.44 out of 12 ($SD=3.31$), and coefficient alpha for Neuroticism was .81. The means differed significantly between Whites and African Americans, with African Americans scoring significantly higher on this scale (5.3 vs. 6.4, $t = 5.0, p < .001$).

Impulsivity was assessed by the Novelty Seeking scale of the Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987; see Appendix C for items). The TPQ measures three dimensions of personality: Novelty Seeking, Harm Avoidance, and Reward Dependence, which are conceptualized as being biologically based. High scorers on the Novelty Seeking trait tend to be impulsive, exploratory, fickle, excitable, quick-tempered, and extravagant, while low scorers tend to be reflective, rigid, loyal, stoic, slow-tempered, and frugal. The trait dimensions of the TPQ are heritable and are substantially influenced by additive genetics (Gillespie, Johnstone, Boyce, Heath, & Martin, 1991; Stallings, Hewitt, Cloninger, Heath, & Eaves, 1996), and there is evidence supporting the reliability and validity of TPQ scales (Cloninger, Pryzbeck, & Svrakic, 1991; Svrakic, Pryzbeck, & Cloninger, 1991). In the current sample, the average Novelty Seeking score was 8.95 out of 18 items ($SD=3.27$), and the coefficient alpha of the scale was .67. The mean Novelty Seeking score did not differ significantly between African Americans and Whites.

Psychiatric Diagnosis

Lifetime DSM-IV diagnoses were also assessed as part of the semi-structured interviews administered at Waves 1 and 4. The diagnostic portion of the interview was adapted from two other measures: the *Diagnostic Interview for Children and Adolescents*

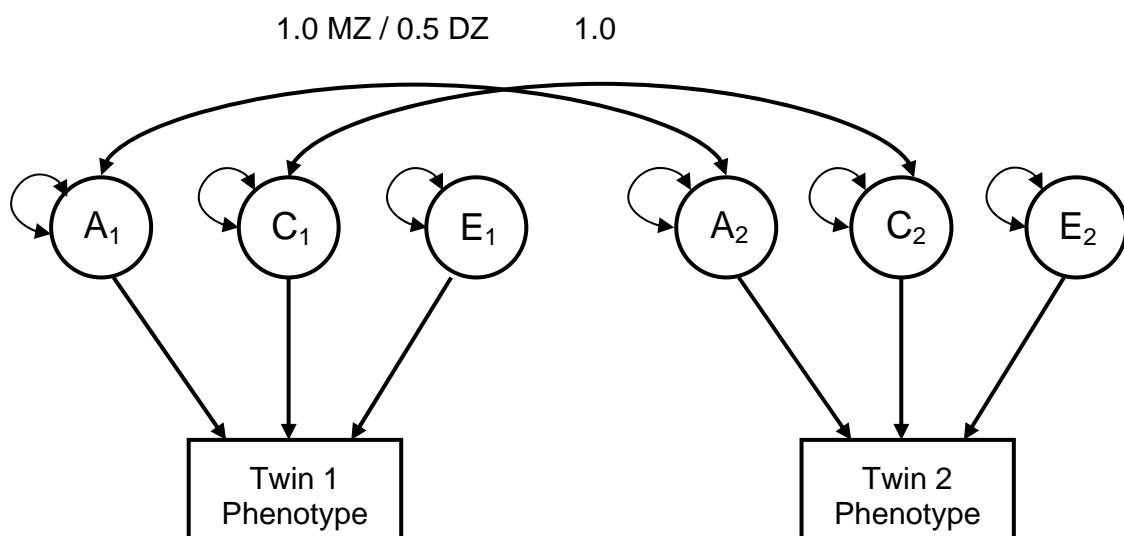
(DICA; Reich, 1996), and the *Semi-Structured Interview for the Assessment of the Genetics of Alcoholism* (Bucholz et al., 1994; COGA, 1996). There is evidence to show that the DICA has good reliability and validity (Reich, 2000). At Wave 1, the following diagnoses were assessed: depression, social phobia, specific phobia, generalized anxiety disorder, alcohol abuse and dependence, oppositional defiant disorder, and conduct disorder. Additionally, all twins age 16 and older were assessed for panic disorder, agoraphobia, and drug abuse. At Wave 4, all twins were assessed for depression, social phobia, panic disorder, agoraphobia, anorexia, bulimia, binge eating disorder, alcohol abuse and dependence, drug abuse and dependence, conduct disorder, and antisocial personality disorder.

Statistical Analyses

Univariate Analyses

The univariate model is shown in path diagram form in Figure 3. Biometric analyses break down observable individual differences in phenotype into three sources of variance. These three sources of information accounting are additive genetic effects (A),

Figure 3. Univariate Biometric Model



shared environmental effects (C), and nonshared environmental effects (E). These univariate models fall under the paradigm of basic genetic epidemiology, with the goal of quantifying the degree to which individual differences in liability to illness results from genetic or environmental factors (Kendler, 2005). The structure of twin data allows the relative effects of genetic and environmental influences to be estimated. MZ twins shared all of their genes identical by descent, while DZ twins share on average half of their genes. Therefore, additive genetic influences are perfectly correlated between members of a monozygotic (MZ) twin pair and 50% correlated between members of a dizygotic (DZ) twin pair. Additive genetic influences account only for simple genetic effects in which multiple loci interact additively to contribute to the phenotype and exclude any influence by dominance (interactions between more than one allele at a given locus) or epistatic (interactions between alleles at multiple loci) effects.

Shared environmental influence includes any experiences that make twins growing up together more similar. This may include factors relating to the family, school, or neighborhood, or larger societal and cultural influences. Shared environmental influences are assumed to promote resemblance equally in MZ and DZ twins (this concept is known as the equal environments assumption or EEA). The equal environments assumption holds that any shared environmental factors that contribute to risk for a given trait are equally correlated between both types of twins. While MZ twins are more likely to be treated similarly in certain aspects (to be dressed alike as children, for instance), the EEA specifies that these differences in the shared environment between MZ and DZ twins are not influential for the trait of interest. This assumption has been

tested for various forms of psychopathology and results indicate that this assumption is reasonable for such traits (Cronk et al., 2002; Xian et al., 2000).

Nonshared environmental influences are any idiosyncratic nonshared experiences that do not influence similarity for twins growing up together. This can include parental behavior that varies towards each twin; peers, friends, or teachers not common to both twins; and life experiences outside the family. Nonshared environmental influences are uncorrelated between members of a twin pair and also include measurement error.

By comparing MZ and DZ correlations on the variables of interest, the type and magnitude of these three influences on individual differences can be estimated. Ignoring zygosity, a greater-than-chance resemblance within both types of twin pairs for some characteristic suggests that the characteristic is familial. Because the shared environment promotes resemblance equally in MZ and DZ pairs, significantly greater similarity among MZ pairs than DZ pairs implies that there is genetic influence on the phenotype under investigation. If the degree of similarity is the same for MZ and DZ twins, the characteristic is most likely influenced by shared environment and not by genetics. Both genetic and shared environmental influences are indicated when DZ correlations are greater than one half the MZ correlations. A fourth source of variance, non-additive or dominance genetic effects (D) are implied when DZ correlations are less than one-half the MZ correlations.

First, a series of univariate models was tested, to estimate the genetic and environmental contribution to lifetime presence or absence of suicidal ideation, plans,

and attempts, and self-harm behavior, as assessed at Waves 1 and 4.⁵ The first series of models replicate results published by Glowinski et al. (2001), though the second series are based on data collected five years later than that used for the previously published results. Submodels of the full univariate model were tested by dropping latent etiological factors. If dropping a genetic or environmental factor results in a significant worsening of model fit, the latent factor was retained. At each step, the most parsimonious model with adequate fit was retained.

Next, the factorial invariance of the univariate models by race was examined. This was accomplished by comparing the fit of models with the A, C, and E parameters constrained to be equal between African American and White women against models in which those parameters were free to vary between groups. If the constrained model fit the data best, then additive genetics, shared environment, and nonshared environment do not vary in their effect on suicidal behavior between groups; if the free model fit the data best, then the relative impact of these factors varies by ethnicity. This test was done for each phenotype of interest: suicidal ideation, plans, attempts, and self-harm behavior.

Multivariate Analyses

A series of multivariate models were tested to investigate the predicted phenotypic covariation among lifetime presence or absence of self-harm behavior and suicide attempts and personality traits. These multivariate models fit with the advanced

⁵ Suicide completions were too rare in this sample to be used as an outcome variable.

Suicide attempts are used instead as a proxy (suicide attempts are one of the best predictors of suicide completions) and also are of interest in their own right. As cited earlier, suicide attempts represent a significant public health burden among adolescents.

genetic epidemiology paradigm (Kendler, 2005) by going beyond simple investigations of heritability to investigate common genetic and environmental influences on different phenotypes and the mediation of genetic risk on self-harm and suicidality by personality traits.

To best equate the two variables as actions, suicide attempts were used as the index of suicidality rather than plans or ideation. The first multivariate model tested the existence of common genetic and environmental influences on self-harm behavior and suicide attempts. This is an independent pathway model, in which the common genetic and environmental influences have direct and independent influences on each of the phenotypes. Covariance between the behaviors is attributable to some combination of shared environmental, nonshared environmental, and/or additive genetic influences. The remainder of the variance for each variable is attributable to some combination of environmental and genetic influences which are unique to each disorder. The test of the hypothesis is whether the common genetic and environmental factors, with paths to both self-harm behavior and suicide attempts, are important for good model fit.

The remaining multivariate models were based on Cholesky models, which are similar to multiple regression analysis. The (expected) observed covariation between neuroticism, Novelty Seeking, and self-harm behavior or suicide attempts was broken down into genetic and environmental components. To test this hypothesis, separate models were used for the phenotypes of self-harm behavior and suicide attempts. In each, the test of whether common genetic factors account for observed covariation between personality traits and self-harm behavior or suicidality is in the test of the paths from the common genetic factor to self-harm/suicidal behavior.

Similarly, separate models were tested to examine the influence of psychiatric diagnoses and personality traits on self-harm behavior and suicide attempts. These test whether genetic influence on personality traits exerts an influence on self-harm and suicidal behavior above and beyond the genetic influence of psychiatric illness. In the current sample at Wave 1, diagnoses of alcohol abuse or dependence, specific phobia, conduct disorder, generalized anxiety disorder, major depression, and social phobia were all significantly associated with risk for suicide attempts (Glowinski et al., 2001).

As in the univariate case, submodels of the best fitting multivariate model for each hypothesis addressed were tested by dropping latent etiological factors or some of their component paths. If dropping a common genetic or environmental factor resulted in a significant worsening of model fit, the latent factor was retained, and subsequent submodels dropping each of its component paths were tested. At each step, the most parsimonious model with adequate fit was retained and used as the basis of comparison for subsequent submodels.

Results

Epidemiology

See Table 2 for the frequencies of suicidal and self-harm behaviors at Waves 1 and 4, as well as the frequencies of suicidal and self-harm behavior reported at any timepoint. The latter variables were added in light of the fact that many individuals who reported lifetime suicidal or self-harm behavior at Wave 1 failed to report lifetime suicidal behavior at Wave 4. See Appendix D for further discussion and analysis of the test-retest reliability of these variables. Finally, a new variable was constructed which represented a continuum of severity of suicidal behavior. Any individual having no reported suicidal behavior had a score of 0, having brief suicidal ideation was scored a 1, having suicidal ideation lasting longer than a day was scored a 2, making a suicide plan was scored a 3, and making a suicide attempt was scored a 4. This variable will be referred to as “suicidality” as it is an index of all suicidal behaviors assessed in this study.

All suicidal behaviors, including brief ideation (lasting less than one day), sustained ideation (lasting one day or longer), and making a suicide plan or attempt were significantly more common in African American women than White women at both waves of assessment. The suicidality variable was also significantly greater in African American women than in White women at both waves of assessment. The prevalence of self-harm, however, did not differ by race (the latter finding was also noted in a sample of college students; see Appendix B, Table B2).

Owing to the fact that the data gathered concerns lifetime behaviors and is not a detailed history, a full investigation of the longitudinal course of self-harm and suicidality could not be carried out. However, it is interesting to note that the reported age of onset

Table 2. Frequencies of suicidality and self-harm

	<u>Total sample</u>	<u>Whites</u>	<u>African- Americans</u>
<u>Wave 1</u>	N=3436	N=2928	N=453
Brief suicidal ideation	550 (16.0%)	449 (15.3%)	94 (20.8%) ^a
Sustained suicidal ideation	233 (6.8%)	181 (6.2%)	50 (11.0%) ^a
Suicide plan	170 (4.9%)	127 (4.3%)	42 (9.3%) ^a
Suicide attempt	143 (4.2%)	107 (3.7%)	35 (7.8%) ^a
Self-harm	162 (4.7%)	134 (4.6%)	24 (5.3%)
<u>Wave 4</u>	N=3736	N=2945	N=477
Brief suicidal ideation	678 (18.1%)	496 (16.8%)	108 (22.7%) ^a
Sustained suicidal ideation	262 (7.0%)	183 (6.2%)	47 (9.9%) ^a
Suicide plan	221 (5.9%)	146 (5.0%)	51 (10.7%) ^a
Suicide attempt	192 (5.2%)	127 (4.3%)	41 (8.6%) ^a
Self-harm	190 (5.1%)	142 (4.8%)	24 (5.0%)
<u>Any reported</u>	N=4352	N=3425	N=594
Brief suicidal ideation	1010 (23.2%)	756 (22.0%)	174 (29.3%) ^a
Sustained suicidal ideation	441 (10.1%)	319 (9.3%)	88 (14.8%) ^a
Suicide plan	352 (8.1%)	245 (7.1%)	82 (13.8%) ^a
Suicide attempt	283 (6.5%)	190 (5.5%)	68 (11.4%) ^a
Self-harm	312 (7.2%)	239 (7.0%)	45 (7.6%)

^a significantly differs from prevalence in Whites based on chi-square test, $p < .05$

for suicidal ideation, plans, and attempts as assessed at Wave 4 did not differ significantly between African American and White women, nor did the total number of suicide attempts or self-harm episodes. What did differ was the recency of the last self-harm episode reported at the Wave 4 interview, with African Americans reporting the most recent incident occurring at age 18.7, and Whites reporting the most recent incident occurring at age 16.5 ($t = -2.61$, 166 df, $p = .01$). This finding supports CDC data indicating that in late adolescence/early adulthood, African American women are continuing to self-injure while the prevalence of self-injury in White women decreases.

See Table 3 for frequencies of all lifetime psychiatric diagnoses at Waves 1 and 4.

Table 3. Frequencies of lifetime psychiatric diagnoses

	Total sample	Whites	African-Americans
<u>Wave 1</u>	N=3444	N=2937	N=453
Depression	342 (10.0%)	285 (9.7%)	54 (11.9%)
Social Phobia	456 (13.2%)	360 (12.2%)	87 (19.2%) ^d
Specific Phobia	378 (11.0%)	289 (9.8%)	83 (18.3%) ^d
Generalized Anxiety Disorder	39 (1.1%)	35 (1.2%)	4 (0.9%)
Panic Disorder	21 (1.6%) ^a	16 (1.5%) ^b	5 (2.5%) ^c
Agoraphobia	33 (2.6%) ^a	26 (2.4%) ^b	7 (3.5%) ^c
Alcohol Abuse	130 (3.8%)	123 (4.2%)	6 (1.3%) ^d
Alcohol Dependence	165 (4.8%)	155 (5.3%)	8 (1.8%) ^d
Drug Abuse	54 (1.2%) ^a	49 (4.6%) ^b	4 (2.0%) ^c
Oppositional Defiant Disorder	203 (5.9%)	152 (5.1%)	48 (10.6%) ^d
Conduct Disorder	51 (1.5%)	30 (1.0%)	21 (4.6%) ^d

Table 3 (cont'd). Frequencies of psychiatric diagnoses

<u>Wave 4</u>	<u>Total sample</u> N=3755	<u>Whites</u> N=2946	<u>African- Americans</u> N=480
Depression	773 (20.6%)	572 (19.3%)	128 (26.7%) ^d
Social Phobia	503 (13.4%)	356 (12.0%)	86 (17.9%) ^d
Panic Disorder	108 (2.9%)	81 (2.7%)	16 (3.3%)
Agoraphobia	53 (1.4%)	40 (1.4%)	6 (1.3%)
Anorexia	28 (0.7%)	25 (0.8%)	1 (0.2%) ^d
Bulimia	18 (0.5%)	14 (0.5%)	0
Binge Eating Disorder	4 (0.1%)	3 (0.1%)	1 (0.2%)
Alcohol Abuse	162 (4.3%)	138 (4.7%)	12 (2.5%) ^d
Alcohol Dependence	263 (7.0%)	212 (7.2%)	25 (5.2%)
Drug Abuse	89 (2.4%)	73 (2.5%)	8 (1.7%)
Drug Dependence	86 (2.3%)	65 (2.2%)	13 (2.7%)
Conduct Disorder	19 (0.5%)	10 (0.3%)	4 (0.8%)
Antisocial Personality Disorder	18 (0.5%)	10 (0.3%)	4 (0.8%)

^a diagnosis assessed only in older twins; N=1515

^b diagnosis assessed only in older twins; N=1074

^c diagnosis assessed only in older twins; N=200

^d significantly differs from prevalence in Whites based on chi-square test, $p < .05$

The same problem with test-retest reliability noted in the assessment of suicidal and self-harm behavior was again noted in the assessment of diagnoses. Many individuals who reported significant lifetime symptoms of mental illness at Wave 1 did not report those symptoms at Wave 4. See Appendix D for further discussion and analysis of this issue.

Some racial differences in diagnostic prevalence were noted; these are generally consistent with the existing literature on psychiatric diagnoses concerning female African American and White adolescents and young adults (Ezpeleta, Keeler, Alaatin, Costello, & Angold, 2001; Kilgus, Pumariaga, Cuffe, & Kim, 1995; Kessler et al., 1994; White & Grilo, 2005).

Univariate Analyses

Twin Similarity

Two measures of similarity were estimated for MZ and DZ twins. See Tables 4 and 5 for probandwise concordances (the proportion of affected twins who have an affected twin) and tetrachoric correlations. These statistics indicate that there is a moderate degree of similarity between twins on the phenotypes of interest, which is for the most part statistically significant, judging by the confidence intervals around the tetrachoric correlations. The similarity between identical twin pairs appears to be greater than the similarity between fraternal twin pairs, implicating the role of genetic factors influencing suicidal and self-harm behaviors. Lastly, similarity is greater for the “any reported” variables than for either Wave 1 or Wave 4 reports of suicidality or self-harm.

Univariate Biometric Models

Next, univariate biometric models for suicidal ideation, plans, attempts, suicidality, and self-harm were estimated for behaviors reported at Wave 1 and Wave 4, as well as any reported behavior across either wave. Note that the Wave 1 tables replicate findings by Glowinski et al. (2001), with the addition of the suicidality and self-harm variables. Tables 6 through 8 show the relative fit of models including all three sources of variance on suicidal and self-harm behavior (A, which is additive genetics, C, which is

Table 4. Probandwise concordances

	<u>MZ twins</u>	<u>DZ twins</u>
Wave 1 brief suicidal ideation	.432	.392
Wave 1 sustained suicidal ideation	.235	.170
Wave 1 suicide plan	.177	.187
Wave 1 suicide attempt	.246	.148
Wave 1 self-harm	.289	.131
<hr/>		
Wave 4 brief suicidal ideation	.411	.295
Wave 4 sustained suicidal ideation	.109	.072
Wave 4 suicide plan	.160	.121
Wave 4 suicide attempt	.174	.076
Wave 4 self-harm	.371	.160
<hr/>		
Any reported brief suicidal ideation	.490	.372
Any reported sustained suicidal ideation	.242	.151
Any reported suicide plan	.209	.160
Any reported suicide attempt	.250	.123
Any reported self-harm	.426	.188

Table 5. Tetrachoric correlations and 95% confidence intervals

	<u>MZ twins</u>	<u>DZ twins</u>
Wave 1 brief suicidal ideation	.589 [.477, .686]	.365 [.219, .499]
Wave 1 sustained suicidal ideation	.487 [.287, .652]	.261 [.047, .458]
Wave 1 suicide plan	.415 [.172, .616]	.372 [.130, .580]
Wave 1 suicide attempt	.558 [.331, .732]	.354 [.057, .602]
Wave 1 suicidality	.536 [.426, .633]	.360 [.225, .485]
Wave 1 self-harm	.582 [.391, .732]	.296 [.001, .551]
<hr/>		
Wave 4 brief suicidal ideation	.502 [.393, .600]	.269 [.127, .404]
Wave 4 sustained suicidal ideation	.149 [-.072, .356]	-.005 [-.249, .235]
Wave 4 suicide plan	.349 [.122, .546]	.180 [-.054, .400]
Wave 4 suicide attempt	.387 [.161, .580]	.093 [-.193, .362]
Wave 4 suicidality	.467 [.364, .561]	.231 [.097, .360]
Wave 4 self-harm	.678 [.525, .795]	.339 [.084, .560]
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Any reported brief suicidal ideation	.566 [.481, .642]	.323 [.210, .428]
Any reported sustained suicidal ideation	.401 [.241, .544]	.203 [.022, .374]
Any reported suicide plan	.514 [.370, .638]	.357 [.191, .508]
Any reported suicide attempt	.527 [.367, .661]	.227 [.030, .411]
Any reported suicidality	.511 [.431, .585]	.272 [.167, .372]
Any reported self-harm	.614 [.486, .721]	.373 [.187, .538]

Table 6. Wave 1 Univariate Biometric Models

	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Brief suicidal ideation	.447 [.098, .684]	.142 [.000, .432]	.411 [.315, .523]	-6.47	.014	5.35	.148	3 (saturated)
	.605 [.503, .694]	*	.395 [.306, .497]	-1.82	.015	.828	.363	1 (χ^2 difference)
	*	.492 [.402, .575]	.508 [.425, .598]	3.72	.041	6.37	.012	1 (χ^2 difference)
	*	*	1.0	97.9	.137	103	.000	2 (χ^2 difference)
Sustained suicidal ideation	.452 [.000, .646]	.035 [.000, .456]	.513 [.355, .713]	-7.37	.020	5.26	.153	3 (saturated)
	.493 [.314, .646]	*	.507 [.354, .686]	-1.98	.008	.023	.881	1 (χ^2 difference)
	*	.375 [.228, .508]	.626 [.492, .773]	-2.31	.027	2.51	.113	1 (χ^2 difference)
	*	*	1.0	21.4	.069	26.1	.000	2 (χ^2 difference)
Suicide plan	.086 [.000, .608]	.329 [.000, .542]	.586 [.384, .774]	-3.67	.000	2.33	.507	3 (saturated)
	.470 [.263, .644]	*	.530 [.356, .737]	-4.14	.004	1.53	.216	1 (χ^2 difference)
	*	.393 [.224, .543]	.607 [.457, .776]	-5.60	.000	.069	.794	1 (χ^2 difference)
	*	*	1.0	12.1	.058	19.7	.000	2 (χ^2 difference)

Table 6 (cont'd). Wave 1 Univariate Biometric Models

	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Suicide attempt	.407 [.000, .732]	.151 [.000, .598]	.443 [.268, .668]	-4.97	.000	1.03	.794	3 (saturated)
	.574 [.368, .736]	*	.427 [.264, .632]	-6.73	.000	.242	.623	1 (χ^2 difference)
	*	.474 [.295, .626]	.526 [.374, .705]	-5.58	.000	1.39	.239	1 (χ^2 difference)
	*	*	1.0	16.9	.060	25.9	.000	2 (χ^2 difference)
	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Suicidality	.347 [.023, .621]	.187 [.000, .454]	.465 [.372, .573]	-40.7	.007	43.3	.414	42 (saturated)
	.556 [.459, .641]	*	.444 [.359, .541]	-41.0	.007	1.68	.195	1 (χ^2 difference)
	*	.458 [.372, .538]	.502 [.463, .628]	-38.2	.008	4.43	.035	1 (χ^2 difference)
	*	*	1.0	54.2	.049	98.9	.000	2 (χ^2 difference)
	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Self-harm	.571 [.000, .729]	.011 [.000, .546]	.418 [.271, .609]	-2.16	.011	3.84	.280	3 (saturated)
	.583 [.402, .729]	*	.417 [.271, .598]	-4.16	.007	.001	.971	1 (χ^2 difference)
	*	.480 [.320, .619]	.520 [.381, .681]	-1.08	.020	3.08	.079	1 (χ^2 difference)
	*	*	1.0	27.6	.067	33.8	.000	2 (χ^2 difference)

Note: best fitting model is in bold print; bracketed ranges are 95% confidence intervals for parameter estimates

Table 7. Wave 4 Univariate Biometric Models

	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Brief suicidal ideation	.465 [.119, .598]	.037 [.000, .326]	.498 [.402, .608]	-5.20	.000	.799	.850	3 (saturated)
	.506 [.405, .599]	*	.494 [.401, .595]	-7.14	.000	.059	.808	1 (χ^2 difference)
	*	.405 [.317, .487]	.595 [.513, .683]	-1.96	.026	7.01	.008	1 (χ^2 difference)
	*	*	1.0	71.8	.109	81.9	.000	2 (χ^2 difference)
Sustained suicidal ideation	.122 [.000, .316]	.000 [.000, .229]	.878 [.684, 1.00]	-4.43	.000	1.57	.666	3 (saturated)
	.122 [.000, .316]	*	.878 [.684, 1.00]	-6.43	.000	.000	.999	1 (χ^2 difference)
	*	.080 [.000, .240]	.920 [.760, 1.00]	-5.92	.000	.507	.477	1 (χ^2 difference)
	*	*	1.0	-7.00	.000	1.43	.490	2 (χ^2 difference)
Suicide plan	.338 [.000, .533]	.011 [.000, .395]	.651 [.467, .872]	.042	.019	6.04	.110	3 (saturated)
	.351 [.148, .533]	*	.649 [.467, .852]	-1.96	.015	.002	.965	1 (χ^2 difference)
	*	.265 [.102, .417]	.735 [.583, .898]	-8.62	.018	1.10	.295	1 (χ^2 difference)
	*	*	1.0	7.17	.045	11.1	.004	2 (χ^2 difference)

Table 7 (cont'd). Wave 4 Univariate Biometric Models

	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Suicide attempt	.361 [.000, .549]	.000 [.000, .375]	.640 [.451, .852]	-3.10	.000	2.90	.407	3 (saturated)
	.361 [.149, .549]	*	.640 [.451, .852]	-5.10	.000	.000	.999	1 (χ^2 difference)
	*	.267 [.090, .430]	.733 [.570, .910]	-2.85	.008	2.25	.134	1 (χ^2 difference)
	*	*	1.0	3.79	.028	10.9	.004	2 (χ^2 difference)
	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Suicidality	.471 [.169, .559]	.000 [.000, .251]	.529 [.441, .631]	-39.0	.009	45.1	.346	42 (saturated)
	.471 [.374, .559]	*	.529 [.441, .626]	-41.0	.008	.000	.999	1 (χ^2 difference)
	*	.367 [.285, .446]	.633 [.554, .715]	-31.9	.012	9.02	.003	1 (χ^2 difference)
	*	*	1.0	36.0	.038	79.0	.000	2 (χ^2 difference)
	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Self-harm	.677 [.145, .792]	.000 [.000, .465]	.322 [.208, .475]	-2.49	.011	3.51	.319	3 (saturated)
	.677 [.532, .792]	*	.322 [.208, .468]	-4.49	.005	.000	.999	1 (χ^2 difference)
	*	.556 [.421, .671]	.444 [.329, .579]	1.84	.033	6.33	.012	1 (χ^2 difference)
	*	*	1.0	53.7	.093	60.2	.000	2 (χ^2 difference)

Note: best fitting model is in bold print; bracketed ranges are 95% confidence intervals for parameter estimates

Table 8. Univariate Biometric Models for Any Reported Suicidality or Self-Harm

	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Brief suicidal ideation	.487 [.212, .634]	.069 [.000, .299]	.444 [.366, .531]	-7.22	.017	5.28	.153	3 (saturated)
	.564 [.484, .637]	*	.436 [.363, .516]	-2.40	.011	.322	.571	1 (χ^2 difference)
	*	.451 [.382, .517]	.549 [.483, .618]	9.47	.049	12.2	.000	1 (χ^2 difference)
	*	*	1.0	144	.143	149	.000	2 (χ^2 difference)
Sustained suicidal ideation	.350 [.027, .475]	.000 [.000, .257]	.650 [.525, .786]	-1.49	.015	4.52	.211	3 (saturated)
	.350 [.215, .475]	*	.650 [.525, .785]	-3.49	.011	.000	.999	1 (χ^2 difference)
	*	.259 [.148, .365]	.741 [.635, .852]	.893	.025	4.38	.036	1 (χ^2 difference)
	*	*	1.0	19.2	.053	24.7	.000	2 (χ^2 difference)
Suicide plan	.332 [.000, .512]	.037 [.000, .374]	.631 [.488, .798]	1.19	.023	7.19	.066	3 (saturated)
	.375 [.225, .513]	*	.625 [.487, .775]	-7.71	.019	.035	.851	1 (χ^2 difference)
	*	.290 [.167, .406]	.710 [.594, .833]	1.05	.022	1.86	.173	1 (χ^2 difference)
	*	*	1.0	20.0	.061	22.8	.000	2 (χ^2 difference)

Table 8 (cont'd). Univariate Biometric Models for Any Reported Suicidality or Self-Harm

	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Suicide attempt	.473 [.103, .612]	.000 [.000, .435]	.527 [.388, .685]	1.18	.023	7.18	.066	3 (saturated)
	.473 [.315, .612]	*	.527 [.388, .685]	-8.22	.019	.000	.999	1 (χ^2 difference)
	*	.350 [.218, .472]	.650 [.528, .702]	4.85	.038	5.67	.017	1 (χ^2 difference)
	*	*	1.0	28.5	.066	31.4	.000	2 (χ^2 difference)
	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Suicidality	.512 [.261, .591]	.009 [.000, .219]	.479 [.410, .560]	-39.8	.004	44.2	.377	42 (saturated)
	.522 [.447, .591]	*	.478 [.409, .553]	-41.7	.003	.007	.933	1 (χ^2 difference)
	*	.406 [.341, .468]	.594 [.532, .660]	-25.6	.019	16.3	.000	1 (χ^2 difference)
	*	*	1.0	103	.051	147	.000	2 (χ^2 difference)
	A	C	E	AIC	RMSEA	χ^2	<i>p</i>	df
Self-harm	.540 [.104, .712]	.062 [.000, .435]	.397 [.288, .528]	-5.78	.000	.218	.975	3 (saturated)
	.608 [.486, .713]	*	.392 [.287, .514]	-7.69	.000	.095	.758	1 (χ^2 difference)
	*	.499 [.389, .589]	.501 [.402, .611]	-1.81	.012	5.97	.015	1 (χ^2 difference)
	*	*	1.0	63.5	.092	73.3	.000	2 (χ^2 difference)

Note: best fitting model is in bold print; bracketed ranges are 95% confidence intervals for parameter estimates

shared environment, and E, which is nonshared environment) as well as AE, CE, and E only models. For example, for brief suicidal ideation at Wave 1 as shown on Table 6, the initial confidence interval for C in an ACE model includes zero. When this parameter is dropped in the AE model, the AIC improves relative to that of the ACE model (lower values of the AIC indicate better fit). The RMSEA, another index of model fit, does not change substantially. The chi-square difference test is nonsignificant, showing that dropping the C parameter does not result in significant model misfit above the ACE model. Therefore, the AE model is shown in bold as the best fitting model, because dropping C or dropping both A and C results in increased AIC and RMSEA, and significant chi-square difference tests, indicating that the CE and E models have worse fit than the original ACE model.

Results across outcomes show that the influence of shared environment on most variables at most timepoints was not significant, and could be dropped from models without incurring important model misfit to the data. The AE model, including only the influence of additive genetics and nonshared environment, proved to be the best fit in all cases with the exception of suicide plan at Wave 1 and sustained suicidal ideation at Wave 4.

Note that results from several models from Wave 1 and 4 were somewhat equivocal in that the initial confidence intervals for both A and C included zero. Chi-square difference tests suggested that dropping either parameter did not result in significant model misfit yet dropping both did result in model misfit (Wave 1: sustained suicidal ideation, suicide plan, suicide attempt, self-harm; Wave 4: suicide plan, suicide attempt). In these instances the best fitting model as suggested by model fit indices is

highlighted, whether AE or CE, but with the caveat there is not enough power to distinguish between models with statistical significance. No models for the “any reported” variables were similarly equivocal.

No obvious developmental trends were noted between the models at Waves 1 and 4; the relative influence of genetics and environment seems fairly constant at the two timepoints (at which individuals had an average age of 16 and 22, respectively). Importantly, the AE model also fit best for all “any reported” variables (across both timepoints), and these variables are likely to be the most valid. From this point on, only the “any reported” variables will be used in further analyses.

Considering the “any reported” variables, additive genetics accounted for a substantial proportion of the variance in suicidal and self-harm behaviors, ranging from 35% to 61%. Notably, self-harm was the most strongly influenced by additive genetics. Nonshared environment also accounted for significant variance in these behaviors.

Effect of Age on Heritability

Of possible concern with these models was the effect of age. Age at interview is almost perfectly correlated among twin pairs, the overwhelming majority of whom were interviewed at the same age. Age will also predict suicidal and self-harm behaviors, because twins interviewed at younger ages will not yet have gone through the age of most risk for onset of these behaviors. Older twins will of course be more likely to report self-harm or suicide attempts.

In this sample, significant point-biserial correlations confirm that age is associated with self-reports of self-harm and suicidal behavior. At Wave 1, age at interview was correlated with brief and sustained suicidal ideation, suicide plans, suicide attempts, and

self-harm at .16, .09, .08, .06, and .04, respectively ($p < .05$ for all correlations). At Wave 4, age at interview was again correlated with brief and sustained suicidal ideation, suicide plans, and suicide attempts at .07, .04, .05, and .05, respectively ($p < .05$ for all correlations). However, there was no association between age at interview and reported history of self-harm at Wave 4. The correlations between age and suicidality showed a similar pattern, with significant correlations of .12 and .07 at Waves 1 and 4. Therefore, age at interview weakly but significantly predicts suicidal behaviors at Waves 1 and 4, and predicts self-harm at Wave 1 but not at Wave 4.

The probandwise concordances by age were calculated for reports at Waves 1 and 4. Table 9 shows the results of age on twin similarity for reported suicidal and self-harm behaviors. No clear developmental trends were noted; twin reports of suicidal and self-harm behaviors do not consistently become more or less concordant over time.

Age contributes equally to MZ and DZ twin similarity, thereby possibly inflating the estimate of influence by the shared environment. One method developed to address this problem is to include age in the biometric model, parsing out the effect of age from the shared environment (Neale and Maes, 2004; Neale and Martin, 1989). These age-corrected models were tested for all suicidal and self-harm behaviors at Waves 1 and 4 (see Figure 4 for the path model). Results did show a very slight decrease in estimated value of shared environment for Wave 1 models with age included (average of .04 change in the parameter estimate); estimates of the effect of shared environment were virtually unchanged for Wave 4 models with age included. For the suicidality variable, the effect of age within a biometric model was zero at Wave 4. At Wave 1, the effect of age on suicidality approached but did not reach significance.

Table 9. Probandwise concordances by age

	MZ twins	DZ twins
Wave 1 brief suicidal ideation		
12-13	.407	.367
14-15	.467	.346
16-17	.321	.393
18+	.489	.232
Wave 1 sustained suicidal ideation		
12-13	.173	.381
14-15	.182	.174
16-17	.240	.067
18+	.313	.125
Wave 1 suicide plan		
12-13	0	.400
14-15	.190	.200
16-17	.211	.190
18+	.222	0
Wave 1 suicide attempt		
12-13	.267	.267
14-15	.429	.154
16-17	.286	.154
18+	.091	0
Wave 1 self-harm		
12-13	.118	0
14-15	.231	.154
16-17	.125	.316
18+	.583	0

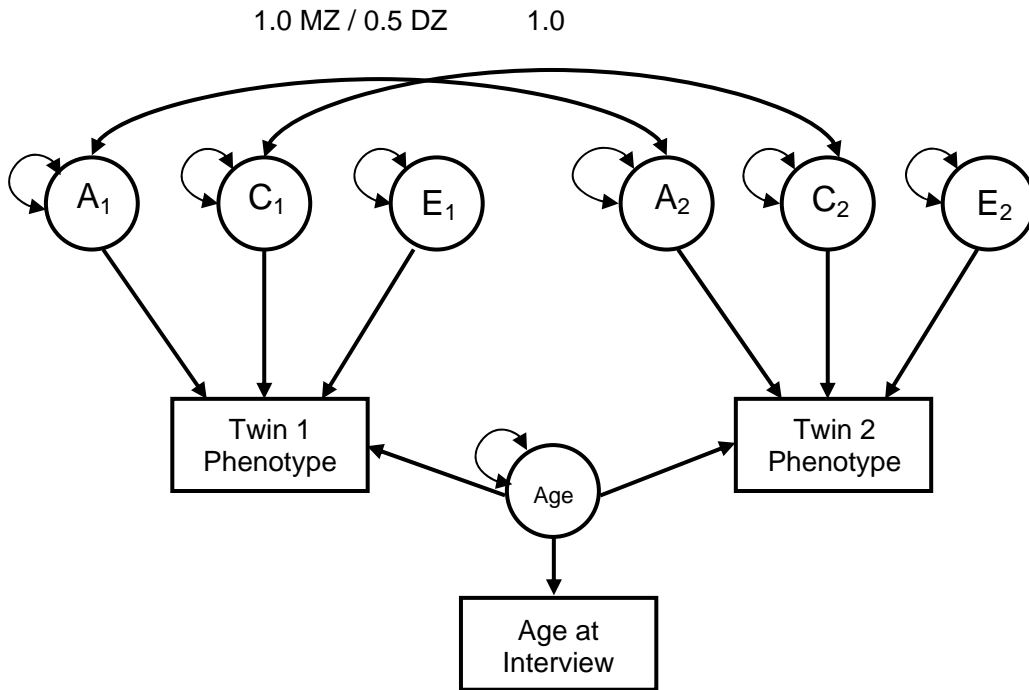
Note: For 12-13, N=613 pairs; for 14-15, N=380 pairs; for 16-17, N=361 pairs; for 18+, N=485 pairs

Table 9 (cont'd). Probandwise concordances by age

	MZ twins	DZ twins
Wave 4 brief suicidal ideation		
18-19	.415	.393
20-21	.417	.415
22-23	.366	.250
24+	.431	.198
Wave 4 sustained suicidal ideation		
18-19	.138	0
20-21	.071	.167
22-23	.154	0
24+	.087	.111
Wave 4 suicide plan		
18-19	0	.261
20-21	.222	0
22-23	.105	0
24+	.216	.162
Wave 4 suicide attempt		
18-19	.167	.118
20-21	.087	0
22-23	.235	0
24+	.235	.133
Wave 4 self-harm		
18-19	.333	.182
20-21	.261	.190
22-23	.364	0
24+	.500	.235

Note: For 18-19, N=514 pairs; for 20-21, N=447 pairs; for 22-23, N=431 pairs; for 24+, N=610 pairs

Figure 4. Age-corrected univariate biometric model



Overall, the effect of age on reports of suicidal and self-harm behaviors within these age corrected biometric models was very small and was never significant. Including age in the path model never changed the model selected as the best fit to the data and had very little effect on parameter estimates. Therefore, results of the univariate biometric models are presented without age correction.

Effect of Race on Heritability

To determine whether the relative contribution of genetics and environment to suicidal and self-harm behaviors varies by race, the invariance of the univariate models by race was tested (Millsap & Yun-Tein, 2004). When first tested separately by race, the AE model was appropriate for every variable among both White and African American women, and influence of the shared environment could be dropped without significantly

worsening model fit. Therefore the joint models were tested including only additive genetics and nonshared environment.

In each case, the fit of a model specifying unique loadings by race for A and E was not significantly better than the fit of a model which specified a single set of loadings for A and E, and the more parsimonious model is retained (see Table 10 for results). For example, for brief suicidal ideation, the parameters estimated by race (indicated in the first line of the table and separated by slashes) are somewhat different. However, the model with shared parameters by race (indicated in the second line of the table) has better measures of fit, including a lower RMSEA and higher CFI and TLI, and the chi-square difference test is nonsignificant.

The separate estimates by race do not appear to differ much for any of the variables related to suicidal behavior. However, the separate estimates by race for self-harm do appear quite different despite the nonsignificant chi-square difference test indicating that a model with shared loadings is most appropriate. This is most likely due to the fact that only 3 African American twin pairs were concordant and positive for lifetime self-harm, so estimates for this group were somewhat unstable. Further, power for these analyses was limited given the relatively low base rate of the behaviors assessed, and the size of the smaller racial group (594 African Americans). The most powerful analyses given base rates were those for suicidal ideation and the suicidality continuum; the least powerful analyses were those for suicide attempts and self-harm. These results should be considered exploratory and not definitive.

Table 10. Influence of Genetics and Environment on Suicidal and Self-Harm Behavior By Race

	<u>A</u>	<u>E</u>	<u>RMSEA</u>	<u>CFI</u>	<u>TLI</u>	χ^2	<u>df</u>	<u>p</u>	χ^2 <u>difference</u>
<u>Brief suicidal ideation</u>									
White/African American	.55/.67	.45/.33	.038	.974	.985	12.3	7	.09	
Combined	.57	.43	.030	.979	.991	13.2	9	.15	$\chi^2 = 1.1, 2 \text{ df}, p = .58$
<u>Sustained suicidal ideation</u>									
White/African American	.38/.28	.62/.72	.012	.974	.987	8.64	8	.37	
Combined	.36	.64	.001	.989	.998	8.91	10	.54	$\chi^2 = .38, 2 \text{ df}, p = .54$
<u>Suicide plan</u>									
White/African American	.39/.24	.61/.76	.044	.590	.795	16.2	8	.04	
Combined	.36	.64	.036	.669	.867	16.6	10	.08	$\chi^2 = .58, 2 \text{ df}, p = .75$
<u>Suicide attempt</u>									
White/African American	.43/.56	.57/.42	.032	.872	.936	12.3	8	.14	
Combined	.47	.53	.022	.922	.969	12.6	10	.26	$\chi^2 = .59, 2 \text{ df}, p = .74$

Table 10 (cont'd). Influence of Genetics and Environment on Suicidal and Self-Harm Behavior By Race

Suicidality

White/African American	.51/.57	.49/.43	.047	.931	.977	25.6	12	.01	
Combined	.52	.48	.040	.946	.983	23.7	13	.03	$\chi^2 = .08, 1 \text{ df}, p = .78$

Self-harm

White/African American	.61/.28	.39/.72	.000	1.00	1.00	5.42	8	.71	
Combined	.59	.41	.000	1.00	1.00	7.47	10	.68	$\chi^2 = 2.0, 2 \text{ df}, p = .36$

Note: best fitting model is in bold print. Chi-square difference tests do not equal the raw difference in chi-squares owing to the use of the WLSMV estimator

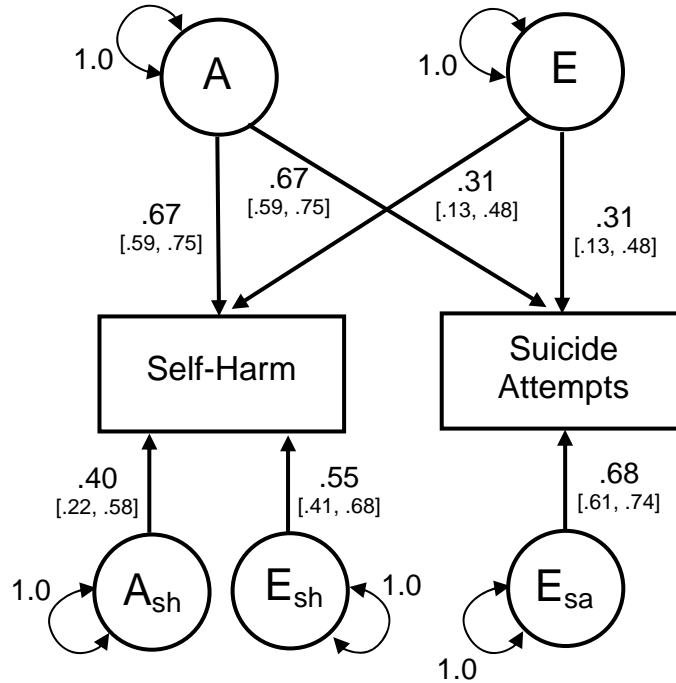
Multivariate Analyses

Self-Harm and Suicidal Behavior

These models tested the genetic correlation between suicidal and self-harm behavior, as well as the extent to which each are affected by common and unique environmental factors. Lifetime suicide attempts and self-harm behavior were significantly associated (OR= 8.11, 95% CI: 6.10 – 10.78). This relationship was invariant by race; in a logistic regression, the interaction of race by self-harm did not predict suicide attempts above and beyond self-harm. The association was then broken down by means of an independent pathway model. Given that neither behavior was significantly affected by shared environment, only common and unique latent variables for both additive genetics and nonshared environment were modeled. The unique genetic factor predicting suicide attempts proved to be nonsignificant, and could therefore be dropped without significantly worsening model fit.

See Figure 5 for the final model. This model did not show significant misfit to the data ($\chi^2_{13\text{ df}} = 8.0, p = 0.84; \text{RMSEA} = 0.000$). The resulting genetic correlation between suicide attempts and self-harm was .45 ($p < .001$), and the nonshared environmental correlation was .09 ($p = .014$). A genetic correlation is a measure of association between genes that determine two traits, and is estimated by multiplying the paths from each phenotype to the latent genetic factor. Similarly, an environmental correlation is a measure of association between environmental sources of variance that determine two traits, and is estimated by multiplying the paths from each phenotype to the latent environmental factor.

Figure 5. Shared genetic and environmental influence in self-harm and suicide attempts



Note: bracketed ranges are 95% confidence intervals for parameter estimates

Personality, Self-Harm, and Suicide Attempts

Before testing the multivariate models including personality and suicidal and self-harm behavior, I first established that the personality traits of interest are heritable and are significantly related to the dependent variables. In a univariate biometric model, the familial transmission of Neuroticism was best described by the AE model, with $A = .46$ (95% CI: .43 - .50) and $E = .54$ (95% CI: .51 - .56). The familial transmission of Novelty Seeking was also best described by the AE model, with $A = .23$ (95% CI: .21 - .27), and $E = .77$ (95% CI: .73 - .80). Both traits are significantly heritable, though Neuroticism is much more strongly influenced by additive genetics than is Novelty Seeking.

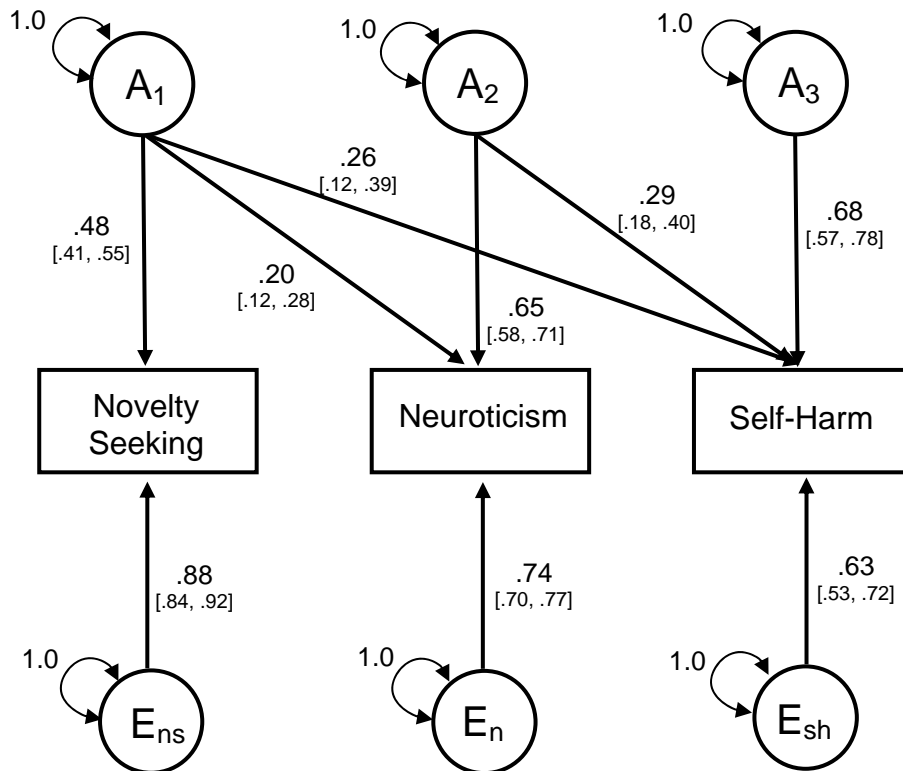
Next, I determined the relationship between the personality traits and suicidal and self-harm behavior. Neuroticism, but not Novelty Seeking, was significantly related to

lifetime suicide attempts ($r = .16, p < 0.001$). Both personality traits proved to be significantly related to lifetime self-harm. In a logistic regression with both traits entered, $B = 0.16$ for Neuroticism ($p < .001$), and $B = 0.06$ for Novelty Seeking ($p = 0.01$).

Next, multivariate logistic regressions were tested to investigate the differential relationship between personality and self-harm and suicidal behavior by race (White versus African American). The interaction terms of race by each personality trait did not predict either behavior over and above the main effects of personality and race. Neuroticism and Novelty Seeking have similar relationships with suicide attempts and self-harm in both White and African American young women.

See Figure 6 for the final Cholesky model of self-harm and personality. Both Neuroticism and Novelty Seeking shared significant genetic variance with self-harm, but

Figure 6. Self-harm, Neuroticism, and Novelty Seeking

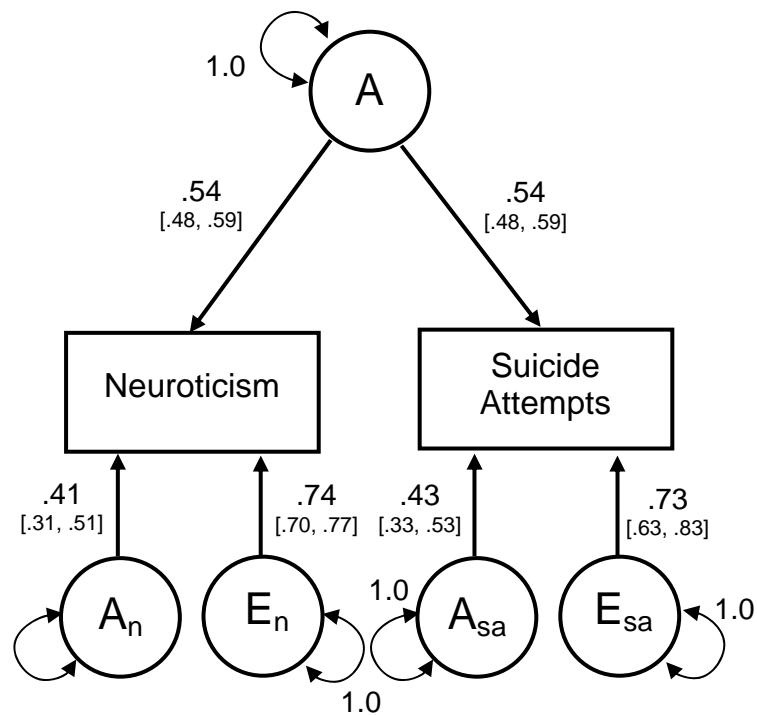


Note: bracketed ranges are 95% confidence intervals for parameter estimates

no environmental variance was shared among the two personality traits and self-harm, so cross loadings from the shared environment factors were all dropped. The fit of the final model was good ($\chi^2_{33\text{ df}} = 28.1, p = 0.71$; RMSEA = 0.011). The genetic correlation between Neuroticism and self-harm was .24 ($p < .001$); the genetic correlation between Novelty Seeking and self-harm was .12 ($p < .001$). Neuroticism shares a moderate amount of genetic variance with self-harm, while Novelty Seeking shares a small amount of genetic variance with self-harm.

The final model for suicide attempts and personality is displayed in Figure 7. In the initial model, the common nonshared environmental factor proved nonsignificant and was dropped from the model. The fit of the final model was good ($\chi^2_{16\text{ df}} = 19.3, p = 0.25$; RMSEA = 0.013). The resulting genetic correlation between suicide attempts and

Figure 7. Suicide attempts and Neuroticism



Note: bracketed ranges are 95% confidence intervals for parameter estimates

Neuroticism was .29 ($p < .001$), indicating that the phenotypes share a moderate degree of genetic variance. Each also had significant unique genetic variance as well as unique nonshared environmental variance.

Diagnostic Status, Self-Harm, and Suicide Attempts

Similarly, I examined the heritability of diagnoses of mental illness, and their relationship with suicidal and self-harm behavior. See Table 11 for odds ratios between the selected diagnoses and outcome variables. All of the listed diagnoses are significantly associated with both self-harm and suicide attempts. Major depression is the only diagnosis that has a significantly different relationship with the two outcomes; it is a stronger predictor of suicide attempts than of self-harm although it is significantly associated with both. This set of diagnoses was selected based on the strength of

Table 11. Odds Ratios and 95% Confidence Intervals between Diagnoses, Self-Harm and Suicide Attempts

	<u>Self-Harm</u>	<u>Suicide Attempt</u>
Major Depression	4.56 [3.60 – 5.77]	8.62 [6.64 – 11.2]
Social Phobia	2.61 [2.05 – 3.33]	3.07 [2.39 – 3.94]
Panic Disorder	3.59 [2.29 – 5.64]	7.16 [4.77 – 10.8]
Any Internalizing	4.08 [3.20 – 5.21]	7.58 [5.69 – 10.1]
Conduct Disorder	10.1 [6.15 – 16.7]	6.50 [3.80 – 11.1]
Alcohol Dependence	3.14 [2.35 – 4.21]	2.97 [2.19 – 4.03]
Drug Abuse	4.51 [3.00 – 6.79]	2.81 [1.75 – 4.51]
Drug Dependence	3.31 [1.92 – 5.72]	5.70 [3.46 – 9.36]
Any Externalizing	4.33 [3.37 – 5.58]	3.54 [2.70 – 4.63]

association with self-harm and suicide attempts, to make the test of whether personality predicts those outcomes over and above psychiatric diagnosis maximally conservative.

Mental illnesses can be conceived as belonging to two broad spectrums of psychopathology: internalizing and externalizing (Krueger, 1999). These broad spectrums of psychopathology are strongly heritable and disorders belonging to the same spectrum share common genetic underpinnings (Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Middeldorp, Cath, Van Dyck, & Boomsma, 2005). Further, there is evidence to suggest that these two dimensions of psychopathology have somewhat independent relationships with suicide attempts (Hills, Cox, McWilliams, & Sareen, 2005). Therefore, to simplify the models, the diagnoses were collapsed into two dichotomous variables: presence of any internalizing disorder (depression, social phobia, or panic disorder), and presence of any externalizing disorder (conduct disorder, alcohol dependence, drug abuse, or drug dependence). Table 11 also includes odds ratios for the internalizing and externalizing variables in relation to self-harm and suicide attempts.

Univariate biometric models were tested for internalizing and externalizing disorders. For internalizing disorders, the best fitting model was an AE model. A was .39 (95% CI: .31 - .46) and E was .61 (95% CI: .54 - .69). For externalizing disorders, the best fitting model was an ACE model. A was .23 (95% CI: .09 - .37), C was .26 (95% CI: .14 - .40), and E was .51 (95% CI: .46 - .56). Both types of psychopathology are significantly affected by genetics and nonshared environment, and externalizing disorders are also affected by shared environment.

In multiple logistic regressions, internalizing and externalizing diagnoses accounted for significant variance in both suicide attempts and self-harm above and

beyond the personality traits of Neuroticism and Novelty Seeking. However, once the diagnoses were entered, Novelty Seeking was no longer a significant predictor of self-harm. Novelty Seeking did not predict suicide attempts even in the bivariate case.

Therefore it will be dropped from further models, as Novelty Seeking does not contribute to either phenotype above and beyond the effects of diagnostic status. See Table 12 for results from logistic regressions including internalizing and externalizing diagnoses as well as Neuroticism.

Table 12. Logistic Regressions Predicting Self-Harm and Suicide Attempts from Neuroticism and Psychiatric Diagnoses

Self-harm

	B	S.E.	Wald χ^2	df	<i>p</i>
Neuroticism	.116	.025	22.109	1	.000
Internalizing Diagnosis	.938	.166	32.021	1	.000
Externalizing Diagnosis	1.212	.170	50.786	1	.000
Constant	-4.043	.197	421.144	1	.000

Suicide Attempt

	B	S.E.	Wald χ^2	df	<i>p</i>
Neuroticism	.131	.027	24.277	1	.000
Internalizing Diagnosis	1.658	.196	71.950	1	.000
Externalizing Diagnosis	.869	.185	22.104	1	.000
Constant	-4.670	.235	394.195	1	.000

Next, the relative predictive power of diagnostic status on self-harm and suicide attempts in different races was tested. Logistic regressions of diagnostic status on self-harm and suicide attempts were tested, adding the effect of race as well as the interactions of race by internalizing diagnosis and race by externalizing diagnosis. Neither interaction term was significant in predicting self-harm, so diagnostic status is not differentially associated with self-harm by race. However, the interaction of race by internalizing disorder was significant in predicting lifetime history of suicide attempt ($B = -.91, p = .008$). The interaction of race by externalizing disorder was not significant. African American women who have an internalizing disorder are less likely to attempt suicide than are White women who have an internalizing disorder.

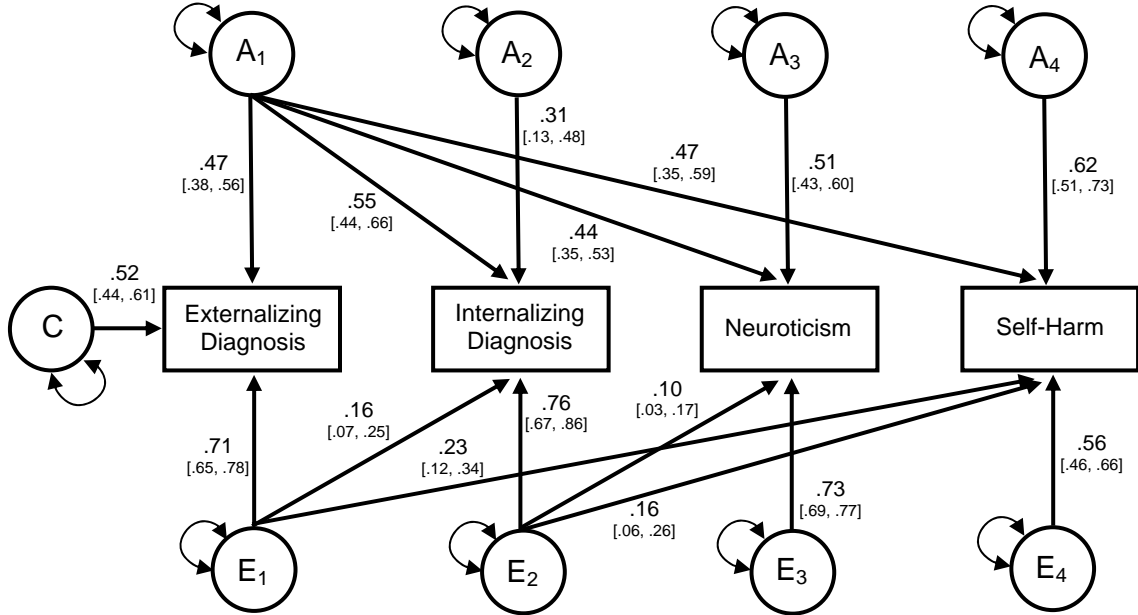
The phenotypic associations between self-harm, suicide attempts, and psychiatric diagnoses were further probed by a series of bivariate behavior genetic analyses. Self-harm and internalizing diagnoses had a genetic correlation of .28 ($p < .001$) and an environmental correlation of .13 ($p < .001$). Conversely, self-harm and externalizing diagnoses had a genetic correlation of .14 ($p < .001$), and an environmental correlation of .25 ($p < .001$). Suicide attempts and internalizing diagnoses had a genetic correlation of .31 ($p < .001$), and an environmental correlation of .24 ($p < .001$). Suicide attempts and externalizing diagnoses had a genetic correlation of .34 ($p < .001$), but did not have a significant environmental correlation. Both self-harm and suicide attempts shared genetic variance with internalizing and externalizing diagnoses. Nonshared environment plays a role in the association between internalizing diagnoses and both self-harm and suicide attempts. Nonshared environment also affects the association between externalizing

diagnoses and self-harm, but has no effect on the association between externalizing diagnoses and suicide attempts.

In multivariate models including both externalizing and internalizing diagnoses, unique genetic variance remained for both self-harm and suicide attempts after controlling for shared variance with both sets of diagnoses. For self-harm, 66% of the genetic variance remained unexplained, and for suicide attempts, 60% of the genetic variance remained unexplained. While psychiatric diagnoses do account for a significant amount of genetic variance in these behaviors, they cannot fully account for the heritability of self-harm and suicide attempts.

Finally, the relationships between psychiatric diagnoses, Neuroticism and self-harm and suicide attempts were examined in two multivariate behavior genetic models (see Figures 8 and 9). Nonsignificant paths were dropped from the full Cholesky model to determine the most parsimonious model with adequate fit. For self-harm, the final model displayed in Figure 8 did not show significant misfit to the data ($\chi^2_{44 \text{ df}} = 47.0, p = 0.35; \text{RMSEA} = 0.008$). Additionally, the fit of the model shown did not differ significantly from the fit of the full Cholesky model ($\chi^2 \text{ difference}_{5 \text{ df}} = 7.86, p = 0.16$). In the case of self-harm, one common genetic factor accounted for significant variance in externalizing disorders, internalizing disorders, Neuroticism, and self-harm. The latter three variables also had significant unique genetic variance. Self-harm did not share significant genetic variance with Neuroticism above and beyond genetic variance shared with psychiatric diagnoses. Also, self-harm shared environmental variance with externalizing diagnoses, internalizing diagnoses, and Neuroticism, as well as having significant unique environmental variance.

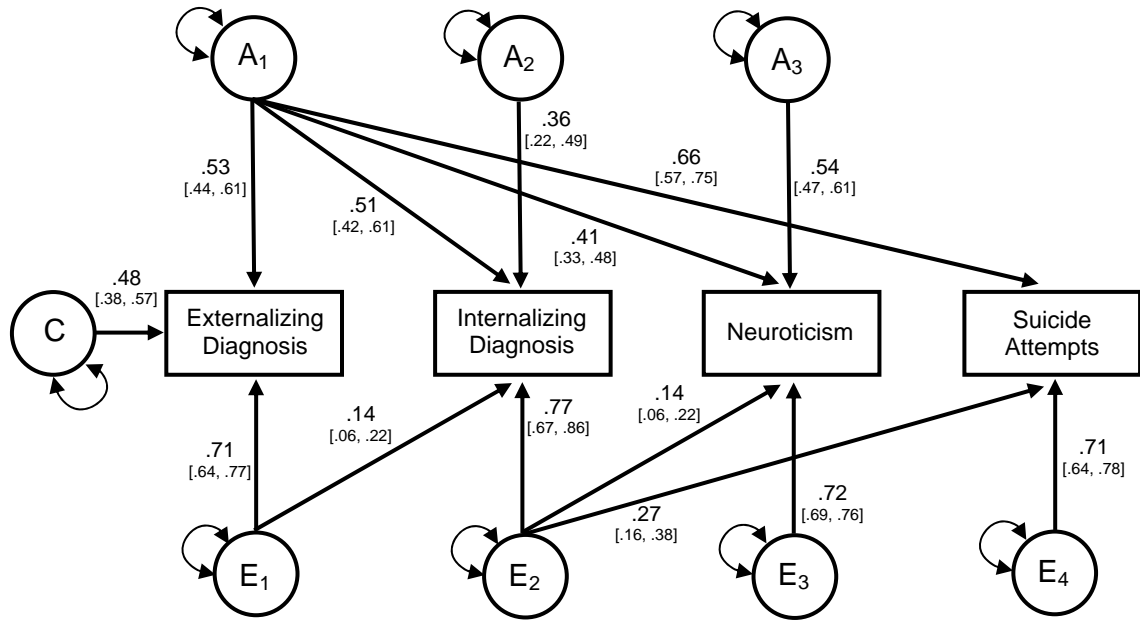
Figure 8. Self-harm, Neuroticism, and psychiatric diagnosis



Note: bracketed ranges are 95% confidence intervals for parameter estimates

Similar results were noted in the model for suicide attempts. The final model, displayed in Figure 9, did not show significant misfit to the data ($\chi^2_{47 df} = 47.1, p = 0.47$; RMSEA = 0.001). Additionally, the fit of the model shown did not differ significantly from the fit of the full Cholesky model (χ^2 difference $7 df = 1.71, p = 0.97$). Again, one common genetic factor accounted for significant variance in externalizing disorders, internalizing disorders, Neuroticism, and suicide attempts. Notably, there was no unique genetic variance for suicide attempts not shared with the diagnoses or Neuroticism. Suicide attempts shared environmental variance with internalizing diagnoses and Neuroticism, but not externalizing diagnoses. Suicide attempts also had significant unique environmental variance. Similar to the findings for self-harm, suicide attempts did not share significant genetic variance with Neuroticism above and beyond genetic variance shared with internalizing and externalizing diagnoses.

Figure 9. Suicide attempts, Neuroticism, and psychiatric diagnoses



Note: bracketed ranges are 95% confidence intervals for parameter estimates

Discussion

Suicidal and self-harm behaviors are strongly heritable in a population of adolescent and young adult women. The nonshared environment also plays a considerable role in shaping these behaviors, but not the shared environment. Both types of behaviors are predicted by Neuroticism, as well as by internalizing and externalizing diagnoses. Novelty Seeking is not an important predictor. Suicide attempts and self-harm share genetic variance with Neuroticism and psychiatric diagnoses. While genetic influence on suicide attempts can be fully accounted for by the combination of those predictors, self-harm has unexplained genetic variance after allowing for shared genetic variance with Neuroticism and diagnostic status.

Suicidal behaviors are more common among African American women than White women in adolescence and early adulthood. However, no difference in the prevalence of self-harm was noted. The predictors of suicidal and self-harm behavior, including personality traits and psychiatric diagnoses, generally did not vary by race. There are many commonalities in the etiology of suicidal and self-harm behaviors in African American and White Midwestern women.

Prevalence

Self-harm had a lifetime prevalence of 7.2% in this sample. This is within the range of reported community lifetime prevalences, though on the high end, probably because participants in this study reported on their behavior close in time to the major period of risk for self-harm. The prevalence may also be high because the sample consisted entirely of women, and previous research has suggested that women are more likely to self-harm than men. However, in the sample of college students described in

Appendix B, women and men were equally likely to report self-harm, casting some doubt on that explanation.

Suicide attempts had a lifetime prevalence of 6.5% in this sample. Also, 23.2% reported suicidal ideation, 10.1% reported suicidal ideation lasting longer than a day, and 8.1% reported a suicide plan. These estimates of the rate of suicidal behaviors are similar to lifetime prevalences estimated from other adolescent or young adult samples. Also consistent with previous research, these rates are higher than those reported by adult community samples, suggesting that perhaps recall bias plays a role in the reporting of suicidal behaviors.

Prevalence By Race

One particularly striking finding was that the entire spectrum of suicidal behaviors including ideation, plans, and attempts were more common among African American women than White women. In fact, the rates of suicide plans and attempts among African American women were double the rates of White women. This result is in the opposite direction of what had been anticipated based on previous literature, which fairly consistently indicated that Whites had higher rates of suicidality than African Americans at all ages. However, these studies have largely failed to fully explore the sex by race by age interaction. Perhaps suicidality is more common in young African American women than White women, while it is less common in young African American men than White men. Also, there may be a cohort effect in operation. Some studies have shown that the rate of suicide attempts among African Americans, including African American women, has been steadily increasing (Gary, Yarandi, & Scruggs, 2003). Results of this study may reflect a historical trend whereby the rate of suicidality

among African American young women has recently superseded that of White young women. Further studies would be necessary to definitely establish this.

By contrast, self-harm was equally prevalent among women of both races. Though previous studies suggested that Whites were more likely to engage in self-harm than members of ethnic minority groups, those studies included younger participants than those in the current study. Among the twins in the current sample, as well as the college students described in Appendix B, there was no significant difference in the prevalence of self-harm between White and African American women.

Heritability of Self-Harm and Suicidal Behavior

As predicted, both genetics and nonshared environment played significant roles in predicting self-harm and suicidal behaviors. This confirms earlier research on the heritability of suicidal ideation and attempts, and extends these findings to self-harm for the first time. More than half of the variance in suicidal ideation can be attributed to additive genetics, less so for suicidal ideation lasting longer than a day or suicide plan. For those reports (“any reported” variables), additive genetics accounted for about a third of the variance, still a substantial proportion but notably less in magnitude than genetic variance predicting suicidal ideation.

Further, there were some anomalies in the biometric models of these two variables. Reports of having made a suicide plan at Wave 1 were best described by a CE model, while reports of suicidal thoughts lasting longer than a day at Wave 4 were best described by an E model. At Wave 1, both MZ and DZ twins showed a limited but similar degree of resemblance for having made a suicide plan. At Wave 4, neither MZ nor DZ twins were significantly correlated on having suicidal thoughts lasting longer than

a day. There is likely some noise in reports of the these two variables. It is probably easier and thus more reliable to report the qualitative distinction between having thought about suicide versus never having thought about suicide, rather than the subtler quantitative decision as to how long those thoughts lasted, or even as to whether a plan was definitively formed. Poor reliability would inflate the estimate of the influence of nonshared environment, and limit the ability of biometric models to detect effects of genetics or shared environment.

For suicide attempts, nearly half the variance could be attributed to additive genetics, with the remaining variance attributable to nonshared environment. This is consistent with previous estimates from twin studies, and extends these findings to a population of young adult women. Suicidality, representing the continuum of suicidal behaviors, showed a similar pattern. This finding supports the concept of a range of related suicidal behaviors, sharing a common etiology.

The shared environment did not emerge as a significant predictor for any of these outcomes. Estimates for the influence of the shared environment were fairly consistent for all suicidal behaviors and self-harm at both waves of assessment as well as the “any reported” variables, and were very nearly zero. Shared environment was therefore dropped from most models, although the confidence intervals were wide enough that a limited effect of the shared environment cannot be conclusively ruled out. This finding apparently conflicts with substantial evidence suggesting that many predictors which seemingly fall into the category of shared environment do predict self-harm and suicide attempts (such as low levels of family support or neglect). Apparently these predictors do not function in the most obvious or simple fashion.

One possible explanation for this result is gene-environment interaction. Studies showing that low family support and poor attachment are related to suicidal and self-harm behavior generally rely on retrospective self-report measures. Individuals who are predisposed to engage in self-harm or suicidal behavior may also be more likely to perceive low family support, or even to evoke it.

One study has already shown that differences in adolescent sibling reports of parenting style are associated with differences in concurrent level of suicidal ideation between siblings (Wagner & Cohen, 1994). This suggests that adolescents who have suicidal ideation may be perceiving parenting style differently than those who do not, or perhaps are evoking a different parenting style than are their siblings (of course, this correlational study cannot rule out the possibility that the causal factor is that the siblings receive different treatment from their parents). If reports of low family support are to some extent determined by the same genetic factors that predict suicidal or self-harm behavior, then the effect of low family support will emerge as a genetic predictor, and not as part of the shared environment. Future studies of the relationship between family variables and self-harm or suicidal behavior should rely on prospective measures with multiple informants.

An investigation of the effect of age at interview on biometric modeling revealed that age correction was not necessary for accurate parameter estimates. The weak correlation between age and reports of suicidal and self-harm behaviors did not significantly or importantly affect estimates of the influence of shared environment. This is possibly because the peak age of incidence for lifetime self-harm is around 16, and the peak incidence of suicidal ideation among adolescents is 15 (with a decline thereafter

throughout the early twenties). The lifetime prevalence of these behaviors in women may not increase enough from mid-adolescence through early adulthood to influence estimates of their heritability.

In analyses exploring the differential heritability of suicidal and self-harm behaviors by race, combined models with shared parameters for the influence of genes and nonshared environment fit better for all outcomes than did models with separate heritability estimates. This was a somewhat surprising outcome given that many putative environmental predictors such as neglect and homelessness are more common among African Americans than Whites. If the effects of genetic and environmental predictors on suicidal and self-harm behavior were simply additive, one would expect that a group of people who experience greater levels of environmental influence on these outcomes would also show a greater proportion of environmental influence relative to genetic influence. However, these findings suggest that there may be a threshold effect with respect to the genetic effect on suicidal and self-harm behavior. If that is the case, some minimal level of genetic predisposition may be a necessary prerequisite for the development of these behaviors, regardless of the level of environmental influence present.

Alternatively, it is also possible that buffering effects more common in the cultural environment of African Americans (such as religiosity) to some extent counter the increased prevalence of negative environmental influence. The various factors may more or less even out. Also, as mentioned previously, what appear on the surface to be environmental factors may in fact have a genetic basis as in the case of a gene-environment interaction.

Joint Heritability of Self-Harm and Suicide Attempts

Self-harm behavior and suicide attempts were significantly associated in this sample. The two behaviors shared a moderate degree of genetic risk, and a limited but significant degree of environmental risk. Neither was significantly influenced by shared environment. This evidence supports the hypothesis that self-harm and suicidality are etiologically similar. The two behaviors share genetic and environmental risk factors in common.

Notably, there was no unique genetic risk for suicide attempts over and above that shared with self-harm, yet there was unique genetic risk for self-harm over and above that shared with suicide attempts. The substantial remaining genetic variance associated with self-harm implies that it is a more complex phenomenon genetically than are suicide attempts. Both had unique environmental influence, suggesting that there is a combination of environmental influence and measurement error specific to each phenotype.

Personality, Self-Harm, and Suicide Attempts

Neuroticism proved to be an important predictor of both suicide attempts and self-harm. It significantly predicted both phenotypes in the bivariate case. Additionally, there was no difference in the predictive power of Neuroticism for suicide attempts of self-harm by race. That is, Neuroticism functions similarly in its relationship to both behaviors in White and African American young women.

In contrast, Novelty Seeking as a measure of impulsivity proved to be relatively unimportant in the prediction of self-harm and suicidal behavior. It was not related to suicide attempts even in the simplest bivariate case, and predicted self-harm only weakly.

The most likely explanation for this negative finding is that Novelty Seeking did not tap the aspects of impulsivity most likely to predict self-harm and suicidal behavior. Novelty Seeking encompasses a number of tendencies, some of which are not likely to predict self-harm or suicidal behaviors, such as being extravagant or exploratory. Certainly there are many people who like to try new things but stop far short of harming themselves. Also, Novelty Seeking was not strongly heritable in this sample, limiting the extent to which it would be possible to find shared genetic variance between it and suicidal or self-harm behaviors.

Several researchers have constructed taxonomies of impulsivity. Eysenck and Eysenck (1977) divided impulsivity into the elements of risk taking, lack of planning, and making up one's mind quickly. Patton, Stanford, and Barratt (1995) separated impulsivity into three components: acting on the spur of the moment (motor activation), not focusing on the task at hand (attention), and not planning and thinking carefully (lack of planning). Using those classifications, the type of impulsivity that might be expected to predict suicidal and self-harm behavior is risk taking or acting on the spur of the moment rather than lack of planning. Also, impulsive aggression has proven to be a particularly strong predictor of both self-harm and suicidal behaviors, as previously discussed, and of psychiatric diagnoses in general (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). A measure of impulsivity that tapped impulsive aggression, or that was simply a cleaner measure of acting without thinking might well have shown a stronger relationship with self-harm and suicidal behavior.

In a multivariate behavior genetic model, self-harm shared significant genetic variance with both Neuroticism and Novelty Seeking. There was no shared

environmental variance between either of the personality traits and self-harm. These personality traits could be characterized as endophenotypes of self-harm, as personality traits are putatively more elementary psychological characteristics. One definition of endophenotype is a measurable component on the pathway between disease and distal genotype (Gottesman & Gould, 2003). Four criteria must be present: (1) The endophenotype is associated with illness in the population, (2) the endophenotype is heritable, (3) the endophenotype is primarily state-independent (manifests in an individual whether or not illness is active), and (4) within families, endophenotype and illness co-segregate. Results from this study have shown that both personality traits are associated with self-harm, and are heritable. Prior research has demonstrated that personality traits are generally stable and should therefore be relatively independent of whether an individual is currently self-harming. Finally, results from the multivariate model show that self-harm is associated with Neuroticism and Novelty Seeking, and that this association has a genetic basis.

The genetic correlation between self-harm and Neuroticism was twice the genetic correlation between self-harm and Novelty Seeking. This is likely further evidence to show that Novelty Seeking was an imperfect measure of the type of impulsivity associated with self-harm, as evidence from previous research would have suggested a greater association between impulsivity and self-harm than was observed here. Despite the shared genetic variance with personality, self-harm still had significant and substantial unique genetic variance. These two personality traits can account for part of the effect of genetics on self-harm, but a moderate proportion of genetic variance is left unexplained.

Neuroticism also shared significant genetic variance with suicide attempts in a behavior genetic model. The two phenotypes did not share environmental variance. Suicide attempts also had a moderate proportion of unique genetic variance that could not be explained by Neuroticism. Neuroticism could also be characterized as an endophenotype of suicide attempts.

Diagnostic Status, Self-Harm, and Suicide Attempts

Psychiatric diagnoses were associated with both self-harm and suicide attempts. Internalizing disorders were significantly more predictive of suicide attempts than self-harm, but externalizing disorders had a similar strength of relationship with both outcomes.

No racial differences were found in the strength of association between diagnoses and self-harm or suicide attempts, with the exception of the predictive value of an internalizing diagnosis relative to suicide attempts. While having an internalizing diagnosis was associated with suicide attempts in both African American and White women, the relationship was significantly stronger among White women. Some research supports the idea that externalizing behaviors such as aggression and drug use are better predictors of suicidal behavior in African Americans than internalizing symptomatology (Willis, Coombs, Drentea, & Cockerham, 2003). Perhaps protective factors including religiosity and family support buffer the effects of internalizing disorders in African American women, who may have a supportive social network to turn to when experiencing depression or anxiety. On the other hand, both internalizing and externalizing disorders were predictive of self-harm in African American and White women, with no significant differences in the strength of the relationships by race.

Psychiatric diagnoses of internalizing or externalizing disorders also share significant genetic and environmental variance with self-harm and suicide attempts. In a multivariate behavior genetic model, self-harm shared significant genetic variance with Neuroticism and both internalizing and externalizing diagnoses. However, shared genetic variance with Neuroticism did not predict self-harm above and beyond psychiatric diagnoses. Neuroticism does not make a unique genetic contribution to self-harm. Also, self-harm did have significant unique genetic variance above and beyond that shared with Neuroticism and internalizing and externalizing diagnoses. The combination of psychiatric diagnoses and Neuroticism cannot fully account for the genetic influence on self-harm; other genetic factors must be in operation. Finally, self-harm shared environmental variance with both types of disorders. Nonshared environmental experiences can shape both internalizing and externalizing disorders as well as self-harm.

By contrast, the combination of Neuroticism and internalizing and externalizing diagnoses fully accounted for genetic variance in suicide attempts. There was no genetic variance unique to suicide attempts remaining after controlling for Neuroticism and psychiatric diagnoses. Suicide attempts also had nonshared environmental influence in common with internalizing diagnoses, but did not have nonshared environmental influence in common with externalizing diagnoses. As with self-harm, Neuroticism did not share genetic variance with suicide attempts above and beyond psychiatric diagnoses.

In sum, Neuroticism does predict self-harm and suicide attempts above and beyond internalizing and externalizing diagnoses, but not through unique genetic or environmental pathways. Rather, a high level of Neuroticism signifies increased genetic loading on a broad factor of genetic predisposition to psychopathology. This factor

confers risk for externalizing and internalizing disorders, Neuroticism, and either suicide attempts or self-harm. The effects of this broad genetic factor can fully account for the heritability of suicide attempts, but can only partially account for the heritability of self-harm.

Limitations

This study had a few limitations which should be kept in mind. Firstly, suicide attempts and self-harm were each assessed with a single item from a measure with unknown psychometric properties. This is fairly common practice for research on suicidality, but there are associated problems with reliability and validity with such an approach. The study in Appendix B demonstrated that the self-harm item showed a reasonable degree of convergence with a psychometrically sound measure of self-harm, but that it likely underestimates the prevalence of self-harm, and misses individuals who self-harm by methods other than cutting or burning.

The demographics of the population under study also limit the conclusions that can be made. Participants come from an epidemiological sample that well represents individuals belonging to the community sampled, that is, young women living in the Midwest. Study results are of great relevance to this and similar populations. Of course, findings have limited generalizability to individuals of other ages, those living in different geographic regions, or to men.

Future Directions

Results from this study suggest several directions for future research in suicidality and self-harm. First, future studies on self-harm and personality should further examine the relationship between self-harm and impulsivity with a more appropriate measure of

impulsivity, such as the Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995) or even behavioral or laboratory measures of impulsivity. Gorlyn (2005) suggests that the best approach for determining the relationship between impulsivity and suicidal behavior in adolescents would be to use a combination of self-report and performance measures, which often have low intercorrelations and may tap different yet important aspects of impulsivity. Further efforts involving measures impulsivity would not be as important for studies of suicide attempts, at least in terms of looking for endophenotypes, as genetic variance in suicide attempt could be fully accounted for without allowing for impulsivity. However, impulsivity may well explain further genetic variance in self-harm that went unaccounted for by Neuroticism and psychiatric diagnoses in the current study.

Also, results from this study raise the issue of why suicidal behaviors are more common in African American women than White women, yet self-harm is equally prevalent. Given that similar risk factors predict both types of behavior, and the behavior share significant genetic and environmental variance, it would be expected that the prevalence of the behaviors would be linked (i.e., if suicide attempts were relatively more common in a given population, self-harm would likely be more common as well). Some risk and/or protective factors must be in operation to differentially affect the prevalence of suicidality versus self-harm by race. Results from such work could not only clarify the epidemiology of self-harm and suicidality by race, but could explicate the differences between self-harm and suicidal behavior in general by identifying risk factors that operate differently on the two phenotypes.

It is also possible that cultural and social factors play a role in the observed differences. Perhaps self-harm is simply less accepted culturally among African

Americans than suicidal behavior, although research indicates that suicidal behavior itself is frowned upon in African American communities. There may also be differences in the vocabulary used by members of different ethnicities (the behavior labeled as a suicide attempt by a member of one group might be labeled self-harm by another).

Implications for Prevention

These findings also carry a number of implications for prevention of suicidal and self-harm behavior. Suicidal and self-harm behaviors are relatively rare, and universal prevention efforts requiring even moderate investment per person may not make sense from the perspective of a cost-benefit analysis. Results from this study could guide future selected prevention programming, by identifying high risk groups. For example, given the substantial heritability of self-harm and suicidal efforts, prevention efforts might well target individuals with a known family history of such behaviors. Given that self-harm and suicide attempts share genetic variance, a family history of either behavior suggests risk for both types of behavior. Using family history as a means to identify high risk groups for selected prevention should be as about effective in reducing suicidal and self-harm behaviors for African American women as for White women.

Also, prevention programs could target individuals with high levels of Neuroticism, internalizing disorders including depression, panic disorder, or social phobia, or externalizing disorders including conduct disorder, alcohol dependence, drug abuse, or drug dependence. All of these diagnoses and Neuroticism are related to both self-harm and suicidal behaviors. In particular, individuals with comorbid diagnoses from the previous list and high Neuroticism would be at high risk, given that they would likely have a very high loading on a broad genetic factor predicting psychopathology as well as

suicide attempts or self-harm. Again, this strategy should be equally effective at identifying high risk African American women as White women, as the relationship between personality traits and diagnoses, and self-harm or suicide attempts differed little by race.

Additionally, the nonshared environment played a significant role in both self-harm and suicidal behaviors. Future research should make efforts to discover what specific aspects of the nonshared environment best predict suicide attempts and self-harm. Prevention efforts directed at that risk factor could reduce the prevalence of both behaviors. For example, previous research has indicated that childhood abuse is predictive of both outcomes. However, behavior genetic studies would be necessary to verify that childhood abuse is largely a nonshared environmental predictor. If abuse within the home is most often associated with self-harm or suicide attempts, and siblings within the same home experience that abuse and become similarly likely to self-harm or make a suicide attempt, the abuse may contribute to shared environmental influence rather than nonshared environmental influence.

Summary

Taken together, there were more similarities than differences in the etiology of self-harm and suicidal behaviors between African American and White young women. Although the prevalence of suicidal behaviors was greater in African American women than White women, the same risk factors including Neuroticism, internalizing, and externalizing diagnoses influenced both self-harm and suicidal behavior in women of either race.

Another focal question of this study was to determine whether self-harm and suicide attempts are points along a continuum of self-injurious behavior, or whether there are any qualitative differences between the two. In support of the former, self-harm and suicide attempts share genetic and environmental risk. Also, both share genetic risk with Neuroticism, suggesting that it is an endophenotype for both behaviors. Self-harm and suicide attempts share genetic risk with internalizing and externalizing diagnoses. In sum, the genetic influence on both behaviors is quite similar.

On the other hand, some important differences between self-harm and suicide attempts emerged. Self-harm has a substantial amount of genetic variance that is not shared with suicide attempts. Also, the genetic variance in self-harm could not be fully explained by the combination of Neuroticism, internalizing diagnoses, and externalizing diagnoses while suicide attempts had no unique genetic variance above and beyond the combination of those variables. The two phenotypes also had varying relationships with psychiatric diagnoses. Internalizing disorders, especially depression, were more predictive of suicide attempts than self-harm. Also, self-harm had shared environmental variance with externalizing diagnoses, but suicide attempts did not.

Given the above evidence, there is not a clear answer to the question originally posed. Suicidal and self-harm are etiologically very similar, yet could not fairly be characterized as points on a continuum given their differences. There is still empirical value in conceptualizing them as separate phenomena, which should be tempered with an understanding of their fundamental interrelatedness.

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Appendix A

Suicidal Thoughts and Behaviors Interview, Wave 1

Now I am going to ask you some (further) questions about suicide.

1. Have you ever thought about taking your own life?
 - A. Did those thoughts last for more than a day?
 - B. Did you ever have a plan? (How did you plan to take your life?)
 - C. How old were you when you first/last had thoughts about taking your own life?

2. Have you ever tried to take your own life?
 - A. How many times?
 - B. How old were you the first/last time you tried to take your own life?

3. How did you try to take your life? (Tell me about your most serious attempt.)

4. Did anyone know at the time that you were trying to take your life?

5. Did you try to take your life when you thought no one would be around?

6. Did you require medical treatment after you tried to take your life?

7. Were you admitted to hospital after the attempt? (What ward were you admitted to?)

8. Did you really want to die?

9. Did you think that (METHOD RECORDED IN 3) would kill you?

10. (When you made your most serious attempt), did you try to take your life while you were:

- A. Feeling depressed?
 - B. Drinking heavily?
 - C. Feeling extremely good or high?
 - D. Using drugs?
 - E. Having strange thoughts or experiences or seeing visions?
11. (Other than when you tried to take your own life), did you ever hurt yourself on purpose, for example, by cutting or burning yourself?
12. Have any of your relatives ever committed suicide? (IF YES: who was that? Any other relatives?)
13. (Apart from anyone you've already told me about) Have any of your (other) relatives ever attempted suicide? (IF YES: who was that? Any other relatives?)

Appendix A (cont'd)

Suicidal Thoughts and Behaviors Interview, Wave 4

Now I am going to ask you some (further) questions about suicide.

1. (You told me earlier that you had thoughts about taking your life.) Have you ever thought about taking your own life?
 2. How old were you when you (first/last) thought about taking your own life?
 - A. Did those thoughts about taking your own life ever last for more than a day?
 - A1. What was the longest period of time that you had these thoughts, that is, how many days in a row?
 - A2. How old were you when you first/last had thoughts about taking your life that lasted more than one day?
 - B. (You told me earlier that you had planned a way of taking your own life.) Did you ever plan a way of taking your own life?
 - B1. Tell me about your most serious plan.
 - C. How old were you the (first/last) time you had a plan for taking your own life?
 - C1. What plan have you had within the past week for taking your life?
 - C2. Are you still having thoughts about taking your own life?
3. How did you try to take your life? (Tell me about your most serious attempt.)
 - A. Did you sustain any injury?
 - B. What did you take?
 - B1. How many pills did you take altogether?
 - a. How much did you take?
 - B2. Did you lose consciousness?
 - B3. Was your stomach pumped?
 - C. Did you get sutures?
 - D. How long had you been planning this particular attempt to take your life?

4. How old were you when you tried to take your life (for this most serious attempt)?

5. Did anyone know at that time that you were trying to take your life?

6. (For your most serious attempt) Did you try to take your life when you thought no one would be around?

7. At the time you made your (most serious) attempt did you really want to die?

8. Did you think that (METHOD RECORDED IN N3) would kill you?

9. Did you make this (most serious) attempt while you were:
 1. Feeling depressed or down?
 2. Drinking?
 3. Using illegal drugs?
 4. Having strange thoughts or experiences or seeing visions?
 5. Feeling extremely good or manic?
 6. Experiencing anything else? IF YES: What else were you experiencing?

10. Had anything upsetting to you happened before you made this (most serious) attempt?
 - A. What happened? Anything else?
 - B. How long before you made this attempt did (this/these things) happen?

11. Did you see a psychiatrist, psychologist, social worker or counselor after you tried to take your life?

12. Did you go to an emergency room or were you taken to an emergency room after you tried to take your own life?
 - A. After you tried to take your life (during this most serious attempt), were you admitted to a hospital because of emotional or stress problems?

13. Did you receive treatment for medical problems resulting from this suicide attempt?

14. Were you admitted to a hospital for the treatment of medical problems resulting from this suicide attempt?

15. Have you ever come close to taking your life but then stopped before making an actual attempt?

A. What were you going to do then? (Tell me about the time you came closest to taking your life but did not make an actual attempt?)

B. How many times have you come close to taking your life but then stopped before making an actual attempt?

C. How old were you (when/the first time/the last time) you came close to taking your life but stopped before making an actual attempt?

16. (Apart from when you were trying to take your life) Did you ever hurt yourself on purpose, for example by cutting or burning yourself?

A. In your entire life, how many times have you done this?

B. How old were you the first/last time that happened?

17. Have any of the following people ever attempted or committed suicide? (IF YES: How old were you when that happened (the first time)?)

18. Did (any) <REL> die as a result of a suicide attempt?

Appendix B

Reliability of the measurement of self-harm

I conducted an additional study to determine the reliability of the interview question used to assess the prevalence of self-harm. As an untested single-item instrument, it may or may not have sufficient reliability to be valid. One method of determining convergent validity would be to compare the item to a measure of self-harm with proven reliability and validity. The Deliberate Self-Harm Inventory is one such measure (DSHI; Gratz, 2001). This instrument is a 17-item behaviorally based questionnaire which assesses various aspects of self-harm including frequency, severity, duration, and type of self-harming behavior. The DSHI has an internal consistency of $\alpha = .82$. The 3-week test-retest reliability is moderate, with $r = .92$ for number of self-harming behaviors endorsed, and $\phi = .68$ for the classification of individuals as self-harming or not self-harming (Gratz, 2001). It shows good convergent and discriminant validity, correlating strongly with other measures of self-harm and with measures of borderline personality and correlating weakly (but significantly) with measures of social desirability and suicidal behavior.

Undergraduate students from a large Midwestern university took part in the reliability study. The students were enrolled in an introductory psychology course and were awarded course credit for their participation in research. Written consent was obtained from all participants, and alternatives such as writing papers were available to those students who did not wish to participate in research studies. The self-harm item from the Suicidal Thoughts and Behaviors Interview (STB; in questionnaire form; see Table B1, item 1) was administered along with items from the DSHI (see Table B1, items

2-8). These items included one general question about self-harm and 6 questions about specific methods of self-harm. The survey, which included several other questionnaires for other studies, was available on the Internet over the course of one week.

A total of 1108 students took part in the study. 46.8% of the participants were female, and the average age was 19.1 (SD=1.5, range=17 to 43). 86.6% of the participants were White, 6.4% African American, 3.5% Asian/Asian Pacific, 1.4% Latino/Hispanic, and 2.2% from other racial groups. See Table B2 for the obtained frequencies of self-harm variables in the sample.

Table B1. Self-harm items administered

Respond to the following questions with NO = 1 or YES = 2.

1. Other than trying to take your own life, did you ever hurt yourself on purpose, for example, by cutting or burning yourself?
2. Have you ever intentionally harmed or injured yourself without intending to kill yourself?
3. Have you ever intentionally cut yourself without intending to kill yourself?
4. Have you ever intentionally burned yourself without intending to kill yourself?
5. Have you ever intentionally severely scratched yourself (to the extent that scarring or bleeding occurred) without intending to kill yourself?
6. Have you ever intentionally stuck sharp objects such as needles, pins, or staples into your skin (not including tattoos or body piercings) without intending to kill yourself?
7. Have you ever intentionally banged your head against something or punched yourself (to the extent that you caused a bruise to appear) without intending to kill yourself?
8. Have you ever intentionally prevented wounds from healing without intending to kill yourself?

Table B2. Frequencies of self-harm behaviors

	<u>Total</u>	<u>Men</u>	<u>Women</u>	<u>White</u>	<u>African-American</u>
STB:					
Self-harm	117 (10.1%)	68 (11.4%)	49 (9.7%)	105 (11.0%)	5 (7.1%)
DSHI:					
Self-harm	124 (11.1%)	70 (11.7%)	54 (10.7%)	114 (12.0%)	5 (7.1%)
Cut	79 (7.1%)	44 (7.4%)	35 (6.9%)	66 (6.9%)	5 (7.1%)
Burn	59 (5.3%)	51 (8.5%) ^b	8 (1.6%)	56 (5.9%)	2 (2.9%)
Scratch	75 (6.8%)	43 (7.2%)	32 (6.3%)	69 (7.2%)	5 (7.1%)
Puncture	113 (10.2%)	76 (12.8%) ^b	37 (7.3%)	99 (10.4%)	8 (11.4%)
Bang/punch	151 (13.7%)	112 (18.7%) ^b	39 (7.7%)	139 (14.6%) ^c	4 (5.7%)
Prev. healing	65 (5.9%)	38 (6.4%)	27 (5.3%)	58 (6.1%)	2 (2.9%)
Any method	306 (27.7%) ^a	199 (33.4%) ^{a,b}	107 (21.1%) ^a	273 (28.7%) ^a	15 (21.4%) ^a

^a significantly greater than prevalence estimate from either self-harm item for this group, $p < .05$

^b significantly greater than in women, $p < .05$

^c significantly greater than in African Americans, $p < .05$

The prevalence of self-harm as estimated by the STB item and the DSHI general self-harm item did not differ significantly, either in the total sample or in any subgroup by gender or race. Furthermore, for the most part the same individuals endorsed both items (see Table B3). Yule's Y assessing agreement was good in all groups except among African Americans, where agreement was only fair. Therefore, the STB item used in the MOAFTS study functions similarly to the item from the DSHI in a population of young adults and shows good convergent validity.

Table B3. Convergent validity of two self-harm items

<u>Self-harm (STB)</u>	<u>Self-harm (DSHI)</u>		
	Absent	Present	
<u>Total sample</u>			
Absent	957	28	
Present	22	95	
			Yule's Y=.85
<u>Men</u>			
Absent	512	16	
Present	15	53	
			Yule's Y=.83
<u>Women</u>			
Absent	445	12	
Present	7	42	
			Yule's Y=.87
<u>White</u>			
Absent	822	25	
Present	16	89	
			Yule's Y=.86
<u>African American</u>			
Absent	61	2	
Present	3	2	
			Yule's Y=.64

However, one significant problem with the STB assessment of self-harm can be noted. The prevalence of self-harm from either of the single, general items is 2 to 3 times less than the prevalence of self-harm from several questions about specific methods of self-harm (denoted "any method" in Table B2). Some of the methods specified may tap

behaviors that do not fit strict conceptions of self-harm (for example, punching a wall in anger). Nevertheless, this finding underscores the importance of using a series of specific questions to best estimate the prevalence of self-harm. Neither age nor race were associated with denying the general items yet endorsing a specific method of self-harm, but men were significantly more likely than women to endorse a specific method of self-harm without endorsing either general self-harm item (20.0% of men vs. 11.3% of women; $\chi^2_{1\text{ df}} = 15.0, p < .001$). Perhaps men are less likely to label their self-injurious behaviors as such due to the belief that self-harm is typically found in women, although the current study shows that men are just as likely to engage in self-harm as women. It is also possible that men are engaging in behaviors that are somewhat outside the stereotypical realm of self-harm, yet are tapped by the questions concerning specific methods of self-harm, such as the previously mentioned “punching a wall.”

Next, I further tested whether the general items consistently underestimated any subgroup of self-injurers by type. Among individuals who answered the STB item in the negative but endorsed at least one self-harm method, scratching, puncturing, banging/punching, and preventing wound healing were all significantly more common than in those who answered the STB item in the affirmative and endorsed at least one self-harm method. However, among individuals who answered the DSHI item in the negative but endorsed at least one self-harm method, all specific methods of self-harm were significantly more common than in those who answered the DSHI item in the affirmative and endorsed at least one self-harm method.

In other words, the STB item accurately samples individuals who cut or burn themselves, but fails to consistently pick up individuals who self-harm in other ways. The

DSHI item consistently undersamples self-harming individuals who use any method. This can be accounted for quite simply by fact that the STB question includes the examples of cutting and burning, while the DSHI item offers no examples. It may be that the examples are necessary to make explicit the definition of intentional self-injury; the participant's internal definition may not be the same as the definition the researcher intends. It may also be the case that having examples jogs the memory of the participant.

Given this nonequality of the wording of the questions, I compared agreement between endorsement of the STB item and endorsement of either the DSHI general item or the specific items on cutting and burning. By this I adjusted for the fact that the STB item offers those two specific examples but the DSHI item does not. This adjustment changed agreement as follows: Yule's $Y=.88$ for the full sample, Yule's $Y=.83$ in males, Yule's $Y=.91$ in females, Yule's $Y=.87$ in Whites, and Yule's $Y=.70$ in African Americans. Agreement was slightly improved by this change. This finding suggests the importance of offering specific examples of self-harm methods when assessing this population, given that the agreement improves when equating the two means of assessment by the specific examples offered.

In summary, the STB self-harm item demonstrates moderate validity in young adults. The STB item likely undersamples the true prevalence of self-harm by missing many individuals who self-harm by methods other than cutting and burning. Future studies should rely on measures of self-harm that include multiple items explicitly referring to various methods of self-harm. For purposes of the current study, these findings show that the validity of the STB item in young adult women is good though it includes many false negatives. Generalizability to the MOAFTS sample is somewhat

limited as the reliability study was conducted in college students instead of a population sample. However, the age range, racial distribution, and geographic region are quite similar.

Appendix C

Neuroticism items from the Junior Eysenck Personality Questionnaire

Instructions: Please answer each question by putting a circle around the “YES” or “NO” following the question. There are no right or wrong answers. Work quickly and do not think too long about the exact meaning of the questions.

1. Do you often feel guilty about things you've said or done?
2. Do lots of things annoy you?
3. Do you ever feel “just miserable” for no good reason?
4. Do you often feel life is very dull?
5. Do you worry about awful things that might happen?
6. Are your feelings rather easily hurt?
7. Do you often feel “fed-up”?
8. Do you worry for a long while if you feel you have made a fool of yourself?
9. Do you sometimes feel life is just not worth living?
10. Do you often feel lonely?
11. Do you sometimes feel specially cheerful and at other times sad without any good reason?
12. Do you often need kind friends to cheer you up?

Appendix C (cont'd)

Novelty Seeking items from the Tridimensional Personality Questionnaire

Instructions: Read each statement carefully, but don't spend too much time deciding on the answer. Please circle "T" for TRUE or "F" for FALSE for every statement, even if you are not completely sure of the answer. Remember, there are no right or wrong answers – just describe your own personal opinions and feelings.

1. I often try new things just for fun or thrills, even if most people think it is a waste of time.
2. When nothing new is happening, I usually start looking for something that is thrilling or exciting.
3. I often do things based on how I feel at the moment without thinking about how they were done in the past.
4. I often break rules and regulations when I think I can get away with it.
5. I am much more reserved and controlled than most people.
6. I am slower than most people to get excited about new ideas and activities.
7. I like to think about things for a long time before I make a decision.
8. I often follow my instincts, hunches, or intuition without thinking through all the details.
9. I usually think about all the facts in detail before I make a decision.
10. I nearly always think about all the facts in detail before I make a decision, even when other people demand a quick decision.
11. I can usually do a good job at stretching the truth to tell a funnier story or to play a joke on someone.
12. I have trouble telling a lie, even when it is meant to spare someone else's feelings.
13. I am better at saving money than most people.
14. I often spend money until I run out of cash or get into debt from using too much credit.
15. Because I so often spend too much money on impulse, it is hard for me to save money – even for special plans like a holiday.

16. I hate to make decisions based only on my first impressions.
17. I enjoy saving money more than spending it on entertainment or thrills.
18. I like to stay at home better than to travel or explore new places.

Appendix D

Consistency and inconsistency in longitudinal assessment

In many previous longitudinal studies of self-report measures, researchers have discovered significant problems with test-retest reliability. For example, Scott and Aneshensel (1997) found that only 55% of individuals who initially reported a history of sexual abuse also reported the history one year later. Klein and Rubovits (1987) also noted that agreement on a measure of stressful life events was only 52% at an interval of 20 weeks. The same phenomenon has been reported concerning measures of mental illness, where symptoms initially reported are subsequently denied at a follow-up assessment (e.g., Zanarini et al., 2000).

Similarly, in the current study, test-retest reliability from Wave 1 to Wave 4 was not strong. In fact, a significant proportion of the sample reported lifetime suicidal or self-harm behaviors at Wave 1 but reported no such lifetime behaviors at Wave 4, a logical inconsistency. See Table D1 for the reported behaviors of the 2820 individuals who were interviewed at both time points. Of positive reports at Wave 1, 49.4% of those reporting brief suicidal ideation, 69.1% of those reporting sustained suicidal ideation, 68.8% of those reporting a suicide plan, 50.9% of those reporting a suicide attempt, and 67.5% of those reporting self-harm denied these behaviors at the time of the Wave 4 interview. This low reliability poses a serious threat to the validity of the reports of suicidal and self-harm behaviors.

Why is it that reports of suicidal and self-harm behaviors are so inconsistent? Several explanations seem probable. Firstly, a form of mental reclassification may alter the individual's interpretation of past events. For example, a young woman who cuts her

Table D1. Longitudinal reports of lifetime suicidal and self-harm behavior

<u>Wave 1</u>	<u>Wave 4</u>	
	Absent	Present
<u>Brief suicidal ideation</u>		
Absent	2130	259
Present	213	218
<u>Sustained suicidal ideation</u>		
Absent	2524	121
Present	121	54
<u>Suicide plan</u>		
Absent	2583	108
Present	86	39
<u>Suicide attempt</u>		
Absent	2618	82
Present	54	52
<u>Self-harm</u>		
Absent	2603	86
Present	83	40

wrists superficially may initially deem it a suicide attempt, but with time she may decide that the injury was not serious enough to qualify as an attempt and relabel the act as self-harm. Suicide attempts are often accompanied by ambivalent intent to die (Hamdi, Amin, & Mattar, 1991); self-harm behavior is highly correlated with subsequent suicide

attempts (Owens et al., 2002). Thus the behaviors may exist on a continuum with fuzzy conceptual boundaries between the two. Secondly, as the individual matures she may become more reticent to share such personal information, especially if she feels that self-harm was a stage she has now outgrown. Finally, simple forgetting is possible but quite unlikely to be a major explanatory mechanism given the psychological significance of these behaviors.

Further analysis was conducted on the subgroup who reported suicidal or self-harm behavior at Wave 1, but either reported it or failed to report it at Wave 4 (N=106 for suicide attempts and N=123 for self-harm). Considering that more medically serious attempts, attempts made with a clear intention to die, or more recent attempts might be more consistently reported, a series of chi square statistics was tested. Hospitalization for medical problems subsequent to the attempt or medical treatment did not significantly predict consistent reporting of suicide attempts. However, treatment by a psychiatrist or hospital admission subsequent to the attempt for treatment of emotional problems did predict consistent reporting. Therefore, treatment that addressed the emotional or psychological aspects of the behavior was associated with more consistent reporting, but not treatment addressing only the medical consequences.

No variables associated with suicidal intent significantly predicted consistent reporting (including whether anyone knew about the planned attempt at the time, whether anyone was around, whether the individual “really wanted to die” or whether she believed the method would kill her). The number of attempts was significantly associated with consistent reporting, such that those reporting higher numbers of attempts at Wave 1 were more likely to report that they had ever attempted suicide at Wave 4. Recency of

attempts did not predict consistency. For self-harm, recency of the behavior did not predict consistent reporting (no further descriptive variables concerning self-harm were available for testing). It was not possible to conduct a multivariate analysis to test the relative strength of all the predictors identified due to the fact that the interview for suicidal behaviors was constructed with many skip-outs and thus few individuals had data on all the variables of interest.

Cotwin reports of suicide attempts were also examined as a potential source of collaborating information. These were examined for cotwins of individuals who reported suicide attempts at Wave 1 and either reported or failed to report a suicide attempt at Wave 4 (see Table D2). The odds ratio for consistent or inconsistent reporting of suicide attempts by cotwin report of suicide was not significant at Wave 1 (OR = 2.17, 95% CI: .91 – 5.16). However, the odds ratio for cotwin reports at Wave 4 was significant (OR = 10.35, 95% CI: 3.70 – 28.95). This indicates that a correlate of consistent reporting of a suicide attempt in Wave 1 and 4 is a cotwin report of attempted suicide at Wave 4.

Further analysis was conducted among 34 twin pairs where both twins reported a suicide attempt by one twin of the pair at Wave 1, and both twins participated in both waves. Of these, in 13 instances both twins reported the attempt at both waves, and in 11 instances both twins reported the attempt at the Wave 1 but not Wave 4. In 5 pairs, the twin identified as having made the attempt did not report it at Wave 4 although the cotwin did, and in 5 further pairs the reverse was true. This is a significant level of agreement between the twins over time (OR = 5.72, 95% CI: 1.31 – 25.05). Perhaps as the twin who initially reported a suicide attempt changes how she thinks about that event,

Table D2. Cotwin reports and consistent and inconsistent self-reporting of suicide attempt

<u>Cotwin</u>	<u>Respondent</u>		
	<u>Inconsistent Reports of Suicide Attempt</u>	<u>Consistent Reports of Suicide Attempt</u>	
<u>Wave 1</u>			
No attempt	33	19	52
Attempt	16	20	36
	49	39	88
<u>Wave 4</u>			
No attempt	46	20	66
Attempt	6	27	33
	52	47	99

she shares her thoughts and opinions with her cotwin. These findings are evidence of a shared reinterpretation of the event between assessment waves.

Next, the hypothesis of reclassification of behaviors between the categories of suicide attempt and self-harm was examined. Of individuals who reported a suicide attempt or self-harm at least once during either interview, only 8 (2%) gave responses that suggested a re-evaluation of the behavior (reporting a suicide attempt but not self-harm at Wave 1, then reporting self-harm and not a suicide attempt at Wave 4, or the reverse). This accounted for only 3% of inconsistent reports. Further investigation revealed that due to much different ages at the time of the behavior in question between waves, 5 of these cases appeared to be referring to different events. For example, one

individual reported at Wave 1 that she had self-harmed at age 16, and then reported at Wave 4 that she had made 3 suicide attempts between the ages of 23 and 27. Another participant described a suicide attempt made at 13 at the Wave 1 interview, then reported 3 incidents of self-harm from ages 16 to 18 at the Wave 4 interview. In none of these cases did the age of the reported behavior match between waves, but in 3 cases, the age differed by just one year. This leaves open the possibility that the participant may have been referring to the same event. However, the small number of cases where reclassification of the event from suicide attempt to self-harm (or the reverse) between waves is possible rules it out as a major explanatory mechanism for the observed inconsistency in the reporting of self-harm and suicidal behaviors.

In summary, individuals who consistently report a lifetime history of suicide attempts are more likely to have had inpatient or outpatient psychological treatment for suicidal behavior, or to have made a greater number of attempts. Twins of individuals who reported a suicide attempt at Wave 1 but denied suicide attempts at Wave 4 were significantly less likely to report that their twin had made a suicide attempt. Additionally, there is evidence that inconsistent reporting of suicide attempts over time may be deliberate as evidenced by the greater than chance agreement between twins. It may be that not only the twin who engaged in the behavior but her cotwin as well change their opinion on whether a behavior constitutes a suicide attempt or not. Reclassification between suicide attempt and self-harm is not a major factor in inconsistent reporting.

For the purposes of the current study, so that conclusions are not limited to individuals who make more serious or frequent attempts, suicidal behavior reported at either time point will be used in all analyses. Further analyses will be conducted on a new

set of variables reflecting any endorsement of the suicidal or self-harm behavior (at either wave), on the assumption that these positive reports are valid in the majority of cases. It is unlikely that there would be many false positives given the stigma and shame that are potentially associated with reporting such behaviors.

Similarly, for psychiatric diagnoses inconsistency rather than consistency was most characteristic of individuals assessed at two waves. Of positive reports at Wave 1, 48.3% of those reporting depression, 71.9% of those reporting social phobia, 62.5% of those reporting panic disorder, 91.3% of those reporting agoraphobia, 91.8% of those reporting alcohol abuse, 77.2% of those reporting alcohol dependence, 84.1% of those reporting drug abuse, and 95.0% of those reporting conduct disorder denied having had those symptoms at the time of the Wave 4 interview. See Table D3 for the diagnoses of 2840 individuals who completed the diagnostic interview at both waves of assessment.

Severity of the disorder at Wave 1, as assessed by symptoms counts for each disorder, did not predict consistency of reporting in any case. Further, the method of assessment is unlikely to have affected the reliability of reporting relative to in-person interviews. In a comparison of telephone versus face-to-face diagnostic interviews of substance abuse and dependence, Cohen and Vison (1995) found that test-retest reliability did not vary by method. Therefore, to ensure reliability and match the technique used to address the same problem with suicidal and self-harm behaviors, further analyses in the main study will be based on lifetime diagnoses reported at either wave of assessment.

Table D3. Longitudinal reports of psychiatric diagnoses

<u>Wave 1</u>	<u>Wave 4</u>	
	Absent	Present
<u>Depression</u>		
Absent	2163	392
Present	128	137
<u>Social Phobia</u>		
Absent	2248	226
Present	263	103
<u>Panic Disorder</u>		
Absent	998	34
Present	10	6
<u>Agoraphobia</u>		
Absent	1017	18
Present	21	2
<u>Alcohol Abuse</u>		
Absent	2621	109
Present	101	9
<u>Alcohol Dependence</u>		
Absent	2566	151
Present	95	28

Drug Abuse

Absent	995	20
Present	37	7

Conduct Disorder

Absent	2794	6
Present	38	2

VITA

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