Different outcomes, same etiology? Shared genetic and environmental influences on non-suicidal self injury and suicidal ideation

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Abstract

Importance—Non-suicidal and suicidal self-injury are very harmful behaviours and are associated with several psychiatric disorders. In the recently developed 5th version of the DSM, non-suicidal self-injury and suicidal behaviour disorder are for the first time introduced as conditions in their own right, instead of symptoms of other psychiatric disorders. It is unclear to what extent non-suicidal and suicidal self-injurious behaviours share the same underlying biological mechanisms and are influenced by the same environmental factors.

Objective—To determine the relative importance of genetic and environmental influences on the variation in non-suicidal self-injury and suicidal ideation and their covariation.

Design—Classical twin design, using population-based twin sample in which twins participated in semi-structured telephone interviews between 1996 and 2009 which primarily focused on psychiatric disorders.

Setting—General community.

Participants—10678 male and female Australian adult twins (mean age 32.8 years).
Main Outcome Measures—Lifetime presence of self-reported non-suicidal self-injury (NSSI) and suicidal ideation.

Results—The prevalence of NSSI and suicidal ideation was 4.7% and 26.5% and individuals that engaged in self-harm were much more likely to report suicidal ideation, OR(95% CIs)=8.4 (6.8–10.3). Results from a bivariate genetic model indicated that genetic factors explain a substantial part of the variance in both NSSI (37% for males and 59% for females) and suicidal ideation (41% and 55%, respectively), while residual influences (including nonshared environmental influences and measurement error) explain the remainder of the variance. Shared (family) environment did not seem to play a role. Moreover, both behaviours were strongly correlated (r=0.49 for males and 0.61 for females) and this correlation was largely explained by overlapping genetic influences (62% and 76% for males and females, respectively), whereas residual influences accounted for the remainder of the phenotypic correlation.

Conclusions and Relevance—Results indicated that the substantial correlation between NSSI and suicidal ideation is largely driven by overlapping genetic factors, suggesting that the two behaviours share similar biological underpinnings. Overlapping residual influences also explain part of the covariance between the two traits. Future research should further investigate which genetic and environmental influences underlie the vulnerability in NSSI and suicidal ideation.

Self-injurious behaviours are behaviours that are performed intentionally with the goal to injure oneself and include non-suicidal and suicidal behaviours (i.e., without versus with the intention to die)\(^1\). Lifetime prevalences in adult community samples are estimated 4–6% for non-suicidal self-injury (NSSI; including self-cutting, biting, or burning)\(^2,3\), while they are substantially higher in adolescent (14–47%)\(^4–6\) and clinical samples (21–61%)\(^3,7\). Lifetime prevalences for suicidal behaviours are estimated 9.2% for suicidal ideation, 3.1% for suicidal plans, and 2.7% for suicide attempts\(^8\). Non-suicidal and suicidal self-injurious behaviours are very impairing and associated with an increased risk of psychiatric disorders, such as depression and borderline personality disorder\(^9–12\). In the previous version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)\(^13\), non-suicidal and suicidal self-injury were included only as symptoms of certain mental disorders. For instance, NSSI was included as a symptom of borderline personality disorder, although research indicates it also occurs in individuals without borderline\(^14,15\). In the recently developed 5th edition of the DSM\(^16\), NSSI and suicidal behaviour disorder are introduced as conditions in their own right. The American Psychiatric Association indicates that further research is needed to guide decisions for future editions of the DSM as to whether these conditions should be considered as formal disorders.

Currently, there is a debate about the relationship between NSSI and suicidal self-injury, with some researchers highlighting both behaviours are distinct\(^17\), whereas others are more cautious in clearly differentiating these two behaviours\(^18\). Numerous studies have noted that non-suicidal and suicidal self-injurious behaviours co-occur frequently\(^14,19,20\) and that NSSI longitudinally predicts increased risk of suicidal behaviours\(^21–23\). However, non-suicidal and suicidal self-injurious behaviours can also be distinguished from each other based on the following characteristics, see \(^24\); intention (NSSI are not performed with the intention to die, suicidal behaviours are\(^1,12,25\)), repetition (NSSI has a higher frequency than suicidal behaviours\(^26,14\)), and lethality (NSSI includes methods of low lethality, such as burning,
whereas suicidal behaviours include methods of higher lethality, such as overdose\textsuperscript{26}. Moreover, studies have shown that suicidal self-injurious behaviours are associated with greater levels of psychological and psychosocial impairment compared to NSSI alone\textsuperscript{27,11} It is important to investigate whether non-suicidal and suicidal self-injury have the same underlying biological and environmental mechanisms to determine how distinct or similar both behaviours are. Clarifying this relationship is important for both research and treatment\textsuperscript{24}.

Twin studies represent an adequate method to answer this question, as they determine the genetic and environmental influences on traits, but also the extent to which genetic and environmental factors are shared between traits. Studies on suicidal self-injury have found that 30–55\% of the variance in suicide attempts and 43–56\% in suicidal ideation could be attributed to genetic factors\textsuperscript{28–31}. Studies on NSSI are very rare and findings are inconsistent. For instance, one study showed that thoughts of NSSI were moderately heritable (36\%), whereas acts of NSSI were not heritable and solely explained by environmental influences\textsuperscript{32}, although it needs to be noted that this study is limited by a small sample size of 483 twin pairs. Contrarily, results from an unpublished study among female twins showed that more than half the variance in self-injury was explained by genes. Moreover, this study is –to our knowledge– the only study that has examined the overlapping genetic and environmental influences on NSSI and suicide attempts. Results showed that both behaviours shared a moderate amount of genetic and a very small amount of unique environmental risk\textsuperscript{31}. However, because this study is unpublished, results should be interpreted with caution.

Using a large sample of male and female twins, the present study determined the relative importance of genetic and environmental influences on NSSI and suicidal ideation as well as on the covariance between both behaviours.

**Methods**

**Participants**

The study sample consisted of identical (monozygotic; MZ) and non-identical (dizygotic; DZ) twins from the Australian Twin Registry, a population-based twin registry. Between 1996 and 2009 the twins participated in various semi-structured telephone interviews focused on psychiatric disorders (see\textsuperscript{33–35}). In each of these studies, twins completed the same items about NSSI and suicidal ideation. Verbal informed consent was obtained from all participants. Procedures were approved by the Human Studies Committee at Washington University and the Ethics Committee at Queensland Institute of Medical Research.

The combined sample comprised 10678 twins (4429 males and 6249 females), including 1154 female MZ, 693 male MZ, 932 female DZ, 594 male DZ, 1038 opposite sex DZ pairs, and 1856 single twins (single twins were retained as they increase precision of the threshold estimates). The participants’ age ranged from 19 to 75 years ($M=32.76$, $SD=6.99$). Zygosity was determined based on standard items about physical similarity, a procedure with high (at least 95\%) concurrence with DNA typing\textsuperscript{36}.  

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Measures

The interview was an adaption of the SSAGA (Semi-Structured Assessment for the Genetics of Alcoholism), which assesses psychiatric disorders in adults and has been shown to be reliable. NSSI was assessed using the question “Other than when you tried to take your own life, did you ever hurt yourself on purpose, for example, by cutting or burning yourself?” and suicidal ideation was assessed using the question “Have you ever thought about taking your own life?”. Answers were coded with “yes” or “no”.

Data analysis

Descriptive statistics were calculated using SPSS 20.0. In accordance with standard twin analysis, genetic analyses employed maximum-likelihood modelling procedures using the statistical package Mx. Measures were analyzed as raw dichotomous data, assuming that a normally distributed continuum of liability is cut in two at a certain threshold, yielding two observed categories. In maximum-likelihood modelling, the goodness-of-fit of a model is distributed as chi-square ($\chi^2$). By testing the change in chi-square ($\Delta \chi^2$) against the change in degrees of freedom ($\Delta df$), we tested whether dropping or equating specific model parameters significantly worsens the model fit. We used the classical twin design, in which the variance in NSSI and suicidal ideation as well as the covariance between them is portioned into genetic (additive, A, and non-additive, D) and environmental (shared, C, and residual, E) influences. Additive genetic variance includes the influence of summed allelic effects on the liability of a trait, whereas non-additive variance includes dominance (allelic interactions within genes) and epistasis (interaction between multiple genes). Shared environmental variance results from environmental influences shared within twin pairs, making them more similar to each other (e.g., family environment), whereas residual environmental variance represents the variance due to unique experiences as well as measurement error.

Portioning of variance into genetic and environmental components can be achieved because MZ twins share 100% of their genes, whereas DZ twins share on average 50% of their segregating genes. Individual differences in phenotypes are the result of a combination of genetic and environmental influences. Structural equation modelling was used to determine which combination fits the observed data best. Moreover, by examining cross-twin cross-trait correlations, we partitioned the covariance between NSSI and suicidal ideation into genetic and environmental parts. Additional information on the classical twin design can be found elsewhere.

A limitation of the classical twin design is that C and D are confounded and therefore cannot be tested simultaneously in a model with only twins reared together. The choice of an ACE or ADE model depends on the pattern of MZ and DZ correlations. If the DZ correlation is greater than half the MZ correlation, C is estimated, but if the DZ correlation is smaller than half the MZ correlation, D is estimated.

Prior to genetic modelling, we tested for the effects of age, sex, and zygosity on the thresholds of NSSI and suicidal ideation and then included these as fixed effects in the thresholds model as necessary. Subsequently, we fitted models to determine the relative influence of A, C or D, and E. We examined the significance of the genetic and environmental contributions to the phenotypes of interest.
environmental influences by testing whether dropping relevant parameters from the baseline model led to a significant decrease in model fit. For ease of interpretation, the bivariate model was transformed into a correlated factors model\(^\text{42}\) (see Figure 1).

**Results**

**Descriptive Statistics**

Table 1 shows prevalences of and odds ratios (ORs) between NSSI and suicidal ideation for the overall sample as well as separately for males and females. The overall prevalence of NSSI and suicidal ideation was 4.7% and 26.5%. Individuals that engaged in self-harm were much more likely to report suicidal ideation, OR(95% CIs)=8.4 (6.8–10.3).

**Preliminary Analyses**

Before determining the variance components, the effects of sex, age, and zygosity, on the thresholds were tested using an \(\alpha\)-level of 0.01. We did not find a significant age effect on suicidal ideation, but we did on NSSI \((\Delta \chi^2_1=39.65, p<0.001)\), showing that younger participants reported lifetime NSSI more often. We did not find a significant sex effect on the thresholds for NSSI nor suicidal ideation, implying there were no differences in the prevalences between males and females. Moreover, levels of NSSI did not significantly differ between MZ and DZ twins in either sex, and neither did the level of suicidal ideation differ between MZ and DZ females. However, males from opposite-sex twin pairs showed a higher prevalence of suicidal ideation than same-sex male twins \((\Delta \chi^2_1=9.10, p=0.003)\), so we did not equate this threshold with the threshold of same sex male pairs in subsequent modelling. We accounted for sex and age effects in subsequent modelling.

Table 2 displays polychoric twin pair correlations for each zygosity group. For both variables, the MZ twin pair correlations were higher than the DZ twin pair correlations, suggesting genetic influences on both traits. Given that the DZ twin pair correlations for both variables were less than half the MZ twin pair correlations, non-additive genetic influences could be expected for both NSSI and suicidal ideation. Therefore, D (and not C) was estimated in the univariate genetic models.

**Genetic model fitting**

Table 3 depicts the A, D, and E estimates as obtained from the univariate model. While the estimates of broad-sense heritability \((H^2; \text{including additive and non-additive genetic influences})\) were significant for both males and females for either trait, the separate A and D estimates were not significant for NSSI, and for males the D estimate was not significant for suicidal ideation. It should be noted that separate A and D estimates should be treated with caution; A and D are highly confounded as they predict similar (but not identical) patterns of twin pair correlations. Therefore, when A and D are estimated simultaneously in one model, the estimates are imprecise and their relative magnitude can be biased depending on the extent of non-additive genetic effects (dominance and epistasis). However, broad-sense heritability is quite robustly estimated with a classical twin design using only twins reared together\(^\text{39,43}\). For this reason, we only estimated A and E in the bivariate model, where A will have captured both the additive and non-additive genetic influences.
Figure 1 depicts the parameter estimates of the bivariate model, separately for males and females. Specifically, the figure shows proportions of variance in NSSI and suicidal ideation accounted for by genetic (heritability, $h^2$) and residual influences as well as the genetic and residual correlations. Parameter estimates could not be equated between males and females ($\Delta \chi^2 = 15.84, p = 0.001$). To test the significance of each path in Figure 1, genetic and environmental parameters were dropped from the bivariate AE model (separately for males and females) and model fit was compared using an $\alpha$-level of 0.05 (Table 4). Results showed that the genetic influences on NSSI ($A=0.37$ for males and 0.59 for females) and suicidal ideation ($A=0.41$ for males and 0.55 for females) were significant, see models 5 and 6 (Table 4). Moreover, the phenotypic correlation between NSSI and suicidal ideation (0.49 for males and 0.61 for females) as well as the genetic and residual correlations were significant, see models 2, 3, and 4, respectively (Table 4).

From the estimates in Figure 1, we calculated (see 44) the extent to which the phenotypic correlation could be attributed to genetic factors: For males this calculation is $(0.61*0.95*0.64)/(0.61*0.95*0.64+0.79*0.20*0.77)=0.76$, showing that overlapping genetic factors accounted for 76% of the phenotypic correlation between NSSI and suicidal ideation, with the remaining 24% accounted for by correlated residual influences. For females, 62% of the phenotypic variance was due to common genetic factors, and 38% to residual influences.

**Discussion**

We examined genetic and environmental influences on NSSI and suicidal ideation as well as on the covariance between both behaviours using data from 10678 twins. Lifetime prevalences of NSSI and suicidal ideation were 4.7% and 26.5% and endorsing NSSI was related to an increased risk of suicidal ideation OR(95% CIs)=8.4 (6.8–10.3). Results of the genetic analyses showed that NSSI and suicidal ideation were moderately heritable. Moreover, both behaviours were highly correlated ($r=0.49$ for males, 0.61 for females) and the majority of the phenotypic correlations was due to overlapping genetic influences (76% for males, 62% for females), while overlapping residual influences (including non-shared environmental influences and measurement error) accounted for the remainder. This implies that NSSI and suicidal ideation are partly influenced by the same biological mechanisms.

These findings are consistent with prior findings of suicidal self-injury, indicating that approximately half of the variance in suicidality is explained by genes and the other half by residual environmental influences, whereas shared (family) environmental influences do not play a substantial role. Previous twin studies on NSSI were based on a small sample size or only examined females and showed inconsistent results; Jang et al. (1996) did not find significant genetic influences on NSSI, while Durrett found that the variance in NSSI was largely accounted for by genetic factors, consistent with our findings.

Most importantly, we examined the overlap in genetic and environmental influences on NSSI and suicidal ideation. Consistent with previous studies, we found a high phenotypic correlation between both behaviours. Furthermore, this comorbidity was largely...
driven by overlapping genetic factors and to a smaller degree by overlapping residual influences, which is in accordance with the findings from an unpublished study by Durret. More generally, our results are consistent with other studies that also found high phenotypic correlations between disorders from the same spectrum (e.g., depression and anxiety), as well as high genetic, considerably lower unique environmental, and very low/absent shared environmental correlations between these highly comorbid disorders. On the other hand, disorders from different spectra show substantially lower phenotypic correlations and a much lower genetic correlation. Findings from these studies indicate that genetic influences that are overlapping between NSSI and suicidal ideation may also partly underlie vulnerability to other mental problems such as depression and anxiety. Liability to NSSI and suicidal ideation as well as other highly correlated disorders from the same spectrum may be influenced largely by the same underlying genetic/biological factors, but the exact disorder that develops among vulnerable individuals within the spectrum may be more dependent on unique environmental influences.

Future research should further investigate which genetic and environmental influences underlie vulnerability in NSSI and suicidal ideation. Previous research has identified some potential biological influences. For instance, meta-analyses of molecular genetic studies have shown that polymorphisms in the tryptophan hydroxylase gene (TPH) and the serotonin transporter gene promoter (5-HTTLPR), both of which play important roles in serotonin functioning, are linked to suicidal self-injurious behaviours. Studies on the molecular underpinnings of NSSI are rare, but also implicate dysfunctions in the serotonin system. Serotonin may play a role, because it is linked to impaired emotion regulation and impulsivity, which are in turn associated with self-injurious behaviours. Other studies point to a possible influence of endogenous opioids, which play a role in disordered pain and reward, for both suicide and NSSI.

Previous studies have also identified some potential unique environmental influences. For instance, studies have shown that early traumatic childhood experiences (i.e., abuse and neglect), peer victimization and bullying, and intimate partner violence and abuse are associated with an increased risk of self-injurious behaviours. Studies that directly compare NSSI and suicidal behaviours to investigate the differential effect of unique environmental contributions are however rare, although studies suggest that compared with NSSI, suicidal behaviours are associated with more stressful life events and greater sexual and emotional abuse.

The present study is not without limitations, most of which are concerned with the classical twin design. For instance, one assumption of the classical twin design is that there are no effects of gene-environment correlation or interaction; not modelling these influences may lead to biased estimates. Another limitation is that C and D cannot be modelled simultaneously and that simultaneously estimating A and D influences leads to imprecise estimates. Lastly, an important limitation is that we only used single item responses to determine lifetime NSSI and suicidal ideation: This could have led to miss-estimation of the...
prevalences. However, the prevalence of NSSI in our sample (4.7%) is consistent with prevalences reported in previous adult population samples2,3. Furthermore, for a subsample of the individuals that endorsed the NSSI item (N=240), data were available regarding the specific self-injurious behaviours they had endorsed, showing that severer forms of self-injurious behaviours (e.g., cutting was endorsed 64%, burning 23%) were reported more frequently than moderate forms (e.g., scratching oneself, punching oneself, punching a hard object were endorsed about 10% each). Note that due to a lack of power we were unable to run analyses on this subgroup only. Our prevalence for suicidal ideation is relatively high potentially because the question did not distinguish between brief and sustained suicidal ideation. Given our crude assessment, it is likely that some of the non-shared environmental variance in and covariance between our measures is due to measurement error, which could have resulted in an overestimation of E and underestimation of A influences.

Despite these limitations, the present study made an important contribution to the current debate about the relationship between NSSI and suicidal ideation. We showed that both behaviours are substantially influenced by genetic and residual environmental factors. Furthermore, we found that the substantial correlation between NSSI and suicidal ideation is largely driven by overlapping genetic factors, suggesting that the two behaviours share similar biological underpinnings. Overlapping residual influences also explain part of the covariance between the two traits. An important goal for future research is to investigate which overlapping and specific genetic and environmental influences underlie the vulnerability in NSSI and suicidal ideation.

Acknowledgments

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References


44. Falconer, DS.; Mackay, TFC. Introduction to Quantitative Genetics. 4. Harlow, Essex, UK: Longmans Green; 1996.


Figure 1.
Correlated factor model. Graphical presentation of the parameter estimates and proportions of variance in non-suicidal self-injury (NSSI) and suicidal ideation accounted for by additive genetic (A) and residual environmental influences (E). $h^2$ (heritability) is the percentage of variance accounted for by genetic factors. The double-headed arrows represent the genetic and residual correlations, indicating the degree to which the same genetic or residual (including non-shared environmental) factors are influencing the two traits. The residual
correlation for males is significant at $p=0.049$, while all other estimates are significant at $p < 0.001$. 

Table 1

Frequencies (%) of and Odds Ratios (ORs) between lifetime non-suicidal self-injury (NSSI) and suicidal ideation

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSSI (n=10674)</td>
<td>206 (4.7%)</td>
<td>300 (4.8%)</td>
<td>506 (4.7%)</td>
</tr>
<tr>
<td>Suicidal ideation (n=10668)</td>
<td>1175 (26.5%)</td>
<td>1650 (26.4%)</td>
<td>2825 (26.5%)</td>
</tr>
<tr>
<td>OR (95% CIs) (n=10664)</td>
<td>5.97 (4.43–8.06)</td>
<td>10.91 (8.23–14.47)</td>
<td>8.39 (6.84–10.29)</td>
</tr>
</tbody>
</table>

Note. ORs as obtained from Mplus6 accounting for sample nonindependence.
Table 2

Polychonic twin pair correlations (95% confidence intervals) for lifetime non-suicidal self-injury (NSSI) and suicidal ideation for each zygosity group, estimated in Mx (corrected for age and sex effects)

<table>
<thead>
<tr>
<th></th>
<th>MZ females (1154 pairs)</th>
<th>MZ males (693 pairs)</th>
<th>DZ females (932 pairs)</th>
<th>DZ males (594 pairs)</th>
<th>DZ opposite-sex (1038 pairs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSSI</td>
<td>0.62 (0.47–0.75)</td>
<td>0.49 (0.22–0.70)</td>
<td>0.12 (−0.18–0.39)</td>
<td>−0.10 (−0.47–0.26)</td>
<td>0.18 (−0.10–0.43)</td>
</tr>
<tr>
<td>Suicidal ideation</td>
<td>0.59 (0.51–0.66)</td>
<td>0.42 (0.29–0.54)</td>
<td>0.14 (0.02–0.25)</td>
<td>0.22 (0.08–0.35)</td>
<td>0.14 (0.03–0.24)</td>
</tr>
</tbody>
</table>
Table 3

Estimates (and 95% CIs) of the proportion of variance in non-suicidal self-injury (NSSI) and suicidal ideation

<table>
<thead>
<tr>
<th>SS</th>
<th>Males</th>
<th>Females</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.11 (0.00–0.61)</td>
<td>0.19 (0.00–0.71)</td>
<td>0.43 (0.33–0.53)</td>
<td>0.13 (0.01–0.38)</td>
</tr>
<tr>
<td>D</td>
<td>0.35 (0.00–0.67)</td>
<td>0.43 (0.00–0.74)</td>
<td>0.00 (0.00–0.38)</td>
<td>0.45 (0.15–0.61)</td>
</tr>
<tr>
<td>H² (A+D)</td>
<td>0.46 (0.18–0.68)</td>
<td>0.62 (0.47–0.74)</td>
<td>0.43 (0.31–0.53)</td>
<td>0.58 (0.50–0.66)</td>
</tr>
<tr>
<td>E</td>
<td>0.54 (0.32–0.82)</td>
<td>0.38 (0.26–0.53)</td>
<td>0.57 (0.47–0.69)</td>
<td>0.42 (0.34–0.50)</td>
</tr>
</tbody>
</table>

Note. A = additive genetic influences, D = nonadditive genetic influences, E = residual environmental influences. H² (A + D) represents broad-sense heritability (in **bold**).
### Table 4

Goodness-of-fit statistics for bivariate models of non-suicidal self-injury (NSSI) and suicidal ideation

<table>
<thead>
<tr>
<th>Model</th>
<th>Males versus</th>
<th>Males Δdf</th>
<th>Males Δχ²</th>
<th>Males p</th>
<th>Females versus</th>
<th>Females Δdf</th>
<th>Females Δχ²</th>
<th>Females p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Full model</td>
<td>1</td>
<td>2</td>
<td>160.53</td>
<td>&lt; 0.001</td>
<td>1</td>
<td>2</td>
<td>352.71</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>2 Test significance of phenotypic correlation between NSSI and suicidal ideation; Drop genetic and residual environmental cross-paths</td>
<td>1</td>
<td>1</td>
<td>27.72</td>
<td>&lt; 0.001</td>
<td>1</td>
<td>1</td>
<td>58.76</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>3 Test significance of the genetic correlation between NSSI and suicidal ideation; Drop genetic cross-path</td>
<td>1</td>
<td>1</td>
<td>3.87</td>
<td>0.049</td>
<td>1</td>
<td>1</td>
<td>28.81</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>4 Test significance of the residual correlation between NSSI and suicidal ideation; Drop residual environmental cross-path</td>
<td>1</td>
<td>1</td>
<td>29.74</td>
<td>&lt; 0.001</td>
<td>1</td>
<td>1</td>
<td>77.19</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>5 Test significance of the genetic influences on NSSI; Drop genetic influence on NSSI</td>
<td>1</td>
<td>1</td>
<td>65.21</td>
<td>&lt; 0.001</td>
<td>1</td>
<td>2</td>
<td>165.90</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>6 Test significance of the genetic influences on suicidal ideation; Drop genetic influence on suicidal ideation</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>