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Common genetic contributions to high-risk trauma exposure and self-injurious thoughts and behaviors

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Abstract

Background. Prior research has documented shared heritable contributions to non-suicidal self-injury (NSSI) and suicidal ideation (SI) as well as NSSI and suicide attempt (SA). In addition, trauma exposure has been implicated in risk for NSSI and suicide. Genetically informative studies are needed to determine common sources of liability to all three self-injurious thoughts and behaviors, and to clarify the nature of their associations with traumatic experiences.

Methods. Multivariate biometric modeling was conducted using data from 9526 twins [59% female, mean age = 31.7 years (range 24–42)] from two cohorts of the Australian Twin Registry, some of whom also participated in the Childhood Trauma Study and the Nicotine Addiction Genetics Project.

Results. The prevalences of high-risk trauma exposure (HRT), NSSI, SI, and SA were 24.4, 5.6, 27.1, and 4.6%, respectively. All phenotypes were moderately to highly correlated. Genetic influences on self-injurious thoughts and behaviors and HRT were significant and highly correlated among men [$r_G = 0.59$, 95% confidence interval (CI) (0.37–0.81)] and women [$r_G = 0.56$ (0.49–0.63)]. Unique environmental influences were modestly correlated in women [$r_E = 0.23$ (0.01–0.45)], suggesting that high-risk trauma may confer some direct risk for self-injurious thoughts and behaviors among females.

Conclusions. Individuals engaging in NSSI are at increased risk for suicide, and common heritable factors contribute to these associations. Preventing trauma exposure may help to mitigate risk for self-harm and suicide, either directly or indirectly via reductions in liability to psychopathology more broadly. In addition, targeting pre-existing vulnerability factors could significantly reduce risk for life-threatening behaviors among those who have experienced trauma.

Non-suicidal self-injury (NSSI) is the intentional destruction of body tissue without suicidal intent (Nock & Favazza, 2009). The prevalence of NSSI among adolescents and young adults ranges from 13% to 23% (Ross & Heath, 2002; Jacobson & Gould, 2007). NSSI typically begins in adolescence (Ross & Heath, 2002; Whitlock & Knox, 2007), and women are more likely than men to report lifetime NSSI (Bresin & Schoenleber, 2015).

In contrast to NSSI, suicidal thoughts and behaviors (STBs) include intent to die [e.g. suicidal ideation (SI) and suicide attempts (SAs); Mann, 2002]. Approximately 13.5% of adults in a nationally representative survey reported lifetime SI and 4.6% reported lifetime SA (Kessler *et al.* 1999). Similar to NSSI, STBs often begin in adolescence (Nock *et al.* 2008; Darke *et al.* 2010), and women are more likely to attempt suicide than men (Nock *et al.* 2008). STBs are differentiated from NSSI in terms of the frequency and lethality of the behaviors, feelings of hopelessness, and attitudes about life (Guertin *et al.* 2001; Muehlenkamp & Gutierrez, 2004; Muehlenkamp & Gutierrez, 2007).

NSSI and STBs co-occur: 70% of inpatient and 50% of community adolescents who engage in NSSI report at least one lifetime suicide attempt (Nock *et al.* 2006; Muehlenkamp & Gutierrez, 2007). Co-occurring NSSI and STBs confer greater risk for negative outcomes than NSSI or STBs alone. In comparison to individuals who engage only in STBs, individuals who engage in both NSSI and STBs exhibit increased rates of suicidal ideation, plans, and attempts (Whitlock & Knox, 2007). They report using more lethal means to attempt suicide

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(Andover & Gibb, 2010) and endorse greater frequency of self-harm (Jacobson *et al.* 2008) and a higher likelihood of engaging in moderate-to-severe forms of NSSI (Lloyd-Richardson *et al.* 2007).

Several theories seek to explain the relation between NSSI and STBs (see, e.g. Selby *et al.* 2015). One theory suggests that NSSI and STBs are on a continuum, with NSSI at one end and completed suicide at the other (Linehan, 1986; Muehlenkamp, 2005). This is supported by many cross-sectional studies and several longitudinal analyses (e.g. Ribeiro *et al.* 2016; Willoughby *et al.* 2015) that have found NSSI to be predictive of STBs. Similarly, Joiner's theory of an acquired capability for suicide proposes that NSSI is a precursor to STBs and facilitates habituation to the fear and pain associated with self-harm and ultimately taking one's life (Joiner, 2005). This is supported by research suggesting that individuals who engage in NSSI report increased pain tolerance and suicidal capability (Hooley *et al.* 2010; Franklin *et al.* 2011). In addition, more frequent NSSI, using various methods of NSSI, and number of years spent engaging in NSSI have been found to predict the frequency and lethality of suicide attempts (Nock *et al.* 2006; Andover & Gibb, 2010).

Another theory suggests that the relation between NSSI and STBs reflects common risk factors, including psychiatric disorders and heritable influences (Nock *et al.* 2006; Sher & Stanley, 2009). Maciejewski *et al.* (2014) observed that the correlation between lifetime NSSI and SI was predominantly explained by shared genetic factors (76% for men, 62% for women). Durrett (2006), employing a sample of female twins, found that genetic influences explained 47% of the relation between NSSI and suicide attempts. However, no genetically informative studies have modeled NSSI, SI, and SA simultaneously. Estimating the genetic and environmental contributions common to all three outcomes can help to capture a greater range of severity in psychopathology and impairment and inform theoretical and etiologic models of the relations between NSSI and STBs.

Trauma exposure has been linked with NSSI and STBs (Turner *et al.* 2012; Devries *et al.* 2014; Dworkin *et al.* 2017; Liu *et al.* 2018). The extent to which these associations reflect causal mechanisms, however, remains unclear. Heritable factors have been found to play a role in exposure to certain types of traumatic events, including assaultive trauma (McCutcheon *et al.* 2009; Afifi *et al.* 2010). In addition, common genetic influences underlie trauma exposure and PTSD (Sartor *et al.* 2011) and other psychiatric conditions associated with self-harm and suicide, including depression (Koenen *et al.* 2008; Afifi *et al.* 2010; Sartor *et al.* 2012). Thus, genetic influences may increase vulnerability to both traumatic experiences and psychopathology.

However, genetically informed studies also indicate that trauma exposure may confer direct risk for self-injurious thoughts and behaviors. Studies of discordant twins, which control for family background factors that may confound associations between trauma and psychopathology, suggest that childhood sexual abuse is associated with risk for later SA (Nelson *et al.* 2002) and other mental health problems (Kendler *et al.* 2000), and trauma exposure and victimization in adolescence and young adulthood are related to risk for psychopathology (e.g. Brown *et al.* 2014; Schaefer *et al.* 2017). [It should be noted, however, that discordant twin studies of trauma exposure and STBs are limited, and other analyses (e.g. Dinwiddie *et al.* 2000) have obtained more modest or non-significant within-pair associations.]

Similar inferences regarding causality may be drawn from biometric models. If trauma exposure itself increases risk for self-injurious thoughts and behaviors, one would expect to observe the relation after controlling for common heritable and environmental influences. Thus, significant overlap in their unique environmental influences would provide evidence of a 'quasi-causal' relation (Turkheimer & Harden, 2014). [The term 'quasi-causal' is used because although within-twin-pair analyses control for a range of unmeasured risk factors, they may be confounded by variables that differ within pairs and relate to both the exposure and the outcome (Turkheimer & Harden, 2014).] To date, no research has examined the genetic and environmental overlap between trauma exposure and the full range of self-injurious thoughts and behaviors. Such work is necessary to further our understanding of the associations between traumatic experiences and life-threatening behaviors, and guide the development of appropriate assessment and treatment strategies.

This study employed data from two population-based twin samples to conduct a multivariate behavioral genetic analysis of high-risk trauma exposure and self-injurious thoughts and behaviors. First, we estimated the genetic and environmental contributions to high-risk trauma. Second, we estimated the shared and unique genetic and environmental contributions to NSSI, SI, and SA. Finally, we modeled high-risk trauma and self-injurious thoughts and behaviors simultaneously to examine the degree of overlap in their genetic and environmental influences. Significant genetic and/or shared environmental overlap would provide evidence that a common familial liability explains their association. A significant relation between their unique environmental influences would provide evidence that high-risk trauma exposure may exert some influence on the general risk for self-injurious thoughts and behaviors.

We proposed three hypotheses. First, we hypothesized that high-risk trauma exposure would be partly explained by genetic factors. Second, we hypothesized that NSSI, SI, and SA would share significant genetic liability. Finally, we predicted that there would be significant genetic overlap between high-risk trauma and self-injurious thoughts and behaviors.

Methods

Participants

Participants were 9526 monozygotic (MZ) and dizygotic (DZ) twins from two cohorts of the Australian Twin Registry (Table 1). Cohort II completed a diagnostic interview based on the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA-OZ; Bucholz *et al.* 1994) between 1996 and 2000 (n = 6265 twins; Heath *et al.* 2001; Knopik *et al.* 2004). A separate sample, Cohort III, completed an interview based on the SSAGA-OZ in 2005–2009 (n = 3348 twins and 476 non-twin siblings; Lynskey *et al.* 2012). Also included were data from the Childhood Trauma Study (CTS; n = 3434), which includes twins from Cohort II identified by responses to screening questions indicating childhood abuse (n = 1532; Nelson *et al.* 2010). In 2003–2008, CTS participants completed interviews modified from the SSAGA-OZ and the Christchurch Trauma Assessment (Fergusson *et al.* 1989; Lynskey & Fergusson, 1997). Some members of Cohort II (n = 44 of the present sample) also provided data through the Nicotine Addiction Genetics Study, a multisite genetic study of tobacco use and related behaviors (Saccone *et al.* 2007).

The 9526 participants included in the analyses consisted of twins who provided data for at least one outcome. Individuals

Table 1. Sample characteristics

	Cohort II[a]	Cohort III	CTS	Full sample
Males	2255	1151	536	3942
Females	2450	2138	996	5584
Total *N*	4705	3289	1532	9526
Complete pairs	1901[b]	1204	644[b]	3959[b]
N twins from incomplete pairs	903[b]	881	244[b]	1608[b]
Monozygotic male twins	956	478	177	1611
Monozygotic female twins	1094	971	400	2465
Dizygotic same-sex male twins	773	368	165	1306
Dizygotic same-sex female twins	765	732	367	1864
Dizygotic opposite-sex	1117	740	423	2280
Mean age at interview (range)	29.9 (24–36)	31.8 (27–37)	37.2 (32–42)	31.7 (24–42)

[a]Indicates twins from Cohort II who completed the 1996–2000 assessment but did not participate in the Childhood Trauma Study.
[b]There were 210 twin pairs ($n = 420$ individual twins) from Cohort II in which one twin participated in the CTS assessment and their co-twin did not. These individuals are excluded from the totals for complete pairs and included in the totals for incomplete pairs within Cohort II and CTS. Thus, the *N*s derived when summing across the sub-samples differ from the *N*s reported for the full sample ($n_{pairs} = 3749$ *v.* 3959; $n_{subjects} = 2028$ *v.* 1608).

with missing data were included (see *Statistical analysis* section). CTS participants were independent of Cohort II participants in all analyses.

Procedure

The Cohort II assessment was administered by telephone using a paper interview, and the Cohort III and CTS assessments were administered by telephone using computer-assisted interviews. Trained lay interviewers were unaware of the status of the co-twin. Participants provided written and verbal consent. Study procedures were approved by the ethical review boards at the Washington University School of Medicine and the Queensland Institute of Medical Research.

Measures

Non-suicidal self-injury

All participants were asked if they had ever harmed themselves on purpose, other than times when they tried to take their own lives, and were asked what they had done. NSSI was coded as a dichotomous variable indicating whether an individual had ever engaged in any self-harm. Participants administered multiple assessments of NSSI were coded as positive if they endorsed NSSI on at least one assessment.

Suicidal ideation

All participants were queried, 'Have you ever thought about taking your own life?' In Cohort III and CTS, SI was also queried within the assessment of major depressive disorder (MDD). Because the Cohort II assessment of SI within the MDD module included passive wish for death, this item was not used. SI was coded as a binary variable indicating if individuals had ever reported ideation. Participants administered multiple assessments of SI were coded as positive if they endorsed ideation on at least one assessment.

Suicide attempt

Participants in all samples, regardless of their history of SI, were asked, 'Have you ever tried to take your own life?' SA was also queried during the assessment of MDD. SA was operationalized as a dichotomous variable indicating if individuals had ever tried to take their own life. Respondents who endorsed SA on any assessment were coded as positive.

High-risk trauma exposure

History of trauma exposure was assessed among all Cohort II participants. Trauma exposure was coded as a binary variable indicating whether an individual reported having ever experienced a high-risk traumatic event. CTS participants who reported high-risk trauma at either the 1996–2000 interview or the 2003–2008 interview were coded as positive. HRT was defined to include exposure to rape, sexual molestation, physical abuse during childhood, and serious neglect during childhood. Individuals who reported no trauma or traumatic experiences defined as low risk (e.g. fire, flood, natural disaster) were coded as unaffected. Traumatic events were classified empirically based on the relative risk of PTSD associated with each event (Sartor *et al.* 2012). Traumatic event exposure was not assessed in Cohort III participants and thus they were coded as missing for trauma exposure.

Statistical analysis

Descriptive analyses were performed using both Mplus (version 7; Muthén & Muthén, 1998–2015) and SAS (version 9.4; SAS Institute Inc., Cary, NC). Survey analysis procedures were employed to obtain correct standard errors. Data were treated as clustered in all analyses, with the family number for each twin pair specified as the clustering variable.

Twin modeling

Biometric models were fitted directly to the raw data by the method of robust weighted least squares (WLSMV) using Mplus. Biometric modeling provides the opportunity to decompose the variance of a trait and the covariance among traits into additive genetic (A), dominant genetic (D), shared environmental (C), and non-shared environmental (E) components. D and C are confounded within the twin design and are estimated

in separate models. Univariate model fitting was conducted with high-risk trauma. We fit a common pathway model (CPM; online Supplementary Fig. S1) to the data for NSSI, SI, and SA. The CPM specifies genetic, shared environmental or dominant genetic, and unique environmental factors that are common to all outcomes and are mediated through a latent phenotype (indicated here as 'self-injurious thoughts and behaviors'). In addition, the model specifies genetic and environmental factors that are specific to all outcomes.

To evaluate the genetic and environmental overlap between high-risk trauma and self-injurious thoughts and behaviors, we fit a correlated factors model to the data (online Supplementary Fig. S2). In this model, the relation between outcomes is modeled in terms of correlations in the underlying genetic and environmental influences. These correlations indicate the degree to which the genetic and environmental influences on high-risk trauma exposure are the same as the genetic and environmental influences on self-injurious thoughts and behaviors. Consistent with the CPM, sources of variation in NSSI, SI, and SA are also modeled at the residual levels.

Evidence for sex differences was tested by comparing the fits of models that allowed parameter estimates for men and women to vary with the fits of models that equated estimates. Nested models were compared using the Satorra–Bentler scaled χ^2 difference test. Cohort/study was included as a covariate. All 9526 individuals who provided data for at least one phenotype were included in the multivariate models. WLSMV accommodates missing data, though under somewhat more restrictive missing data assumptions than full information maximum likelihood (Asparouhov & Muthén, 2010).

Results

Descriptive analyses

Table 2 displays the prevalence rates for HRT, NSSI, SI, and SA. Tetrachoric correlations between all phenotypes are reported in Table 3. Correlations among NSSI and STBs were large (*rs* = 0.55–0.88), and HRT was moderately correlated with self-injurious thoughts and behaviors (*rs* = 0.40–0.48). Nearly all individuals [430 (99.1%) of 434] who reported SA also reported SI.

The odds of reporting SI and SA were significantly higher among individuals who reported NSSI [ideation: odds ratio (OR) 8.20, 95% CI (6.26–10.76); attempt: OR 9.85 (7.34–13.22)], and risk for SA was significantly elevated among individuals with SI [OR 222.03 (82.76–595.70)]. Associations remained significant after adjusting for high-risk trauma (online Supplementary Table S1). The wide confidence limits indicate that the associations between SI and SA were not precisely estimated, likely due to the low prevalence of SA and the variables' collinearity. However, the lower bounds for all estimates were well above zero.

Twin correlations

Inspection of Table 4 reveals that: (1) the within-trait MZ correlations were larger than the DZ correlations, indicating genetic influences on all phenotypes studied; (2) nearly all cross-trait MZ correlations were larger than the corresponding DZ correlations, implicating genetic influences on (*a*) the covariation among NSSI, SI, and SA, and (*b*) the covariation between high-risk trauma exposure and self-injurious thoughts and behaviors; (3) the within-trait DZ correlations for SI were less than half the MZ correlations, suggesting dominant genetic effects; and (4) for SA, the within-trait DZ correlation was greater than half the MZ correlation among women and less than half the MZ correlation among men, suggesting shared environmental influences among women and dominant genetic influences among men.

Biometric model fitting

Full models

Approximately two-thirds of the variation in HRT was attributable to genetic factors [A = 0.62 (0.34–0.90); online Supplementary Table S2]. Given the varying patterns of twin correlations across phenotypes, we fit ACE and ADE common pathway models to the data for NSSI, SI, and SA. Both models fit very well (RMSEA < 0.02). Proportions of variation in these outcomes attributable to common and specific genetic and environmental factors are displayed in online Supplementary Tables S3 and S4, and standardized path estimates are provided in online Supplementary Figs. S3–S6. Both models indicated that the common factor was 54–55% heritable in men and 51–52% heritable in women (for the ADE model, the sum of the A and D estimates yields the heritability coefficient). The remaining 45–46% (among men) and 48–49% (among women) of the variation was attributable to the non-shared environment. The majority of the genetic variation in SI and SA was attributable to common influences. For NSSI, however, specific genetic influences explained 33–36% of the variation among men and 46% of the variation among women (Tables S3 and S4).

Table 2. Prevalences of non-suicidal self-injury, suicidal ideation, suicide attempt, and high-risk trauma exposure

	Lifetime prevalence			
	Full sample (n = 9526)	Men (n = 3942)	Women (n = 5584)	Men/women differ?
Non-suicidal self-injury	535/9521 (5.6)	219/3939 (5.6)	316/5582 (5.7)	No (p = 0.84)
Suicidal ideation	2584/9521 (27.1)	1062/3942 (26.9)	1522/5579 (27.3)	No (p = 0.73)
Suicide attempt	434/9518 (4.6)	138/3940 (3.5)	296/5578 (5.3)	Yes (p < 0.0001)
	Cohort II (n = 6237)[a]	Men (n = 2791)	Women (n = 3446)	Men/women differ?
High-risk trauma exposure	1516/6215 (24.4)	621/2783 (22.3)	895/3432 (26.1)	Yes (p = 0.002)

[a]Includes 1532 twins who also participated in the Childhood Trauma Study.
Values are numbers (percentages).
All χ^2 tests have 1 df.

Table 3. Tetrachoric correlations between non-suicidal self-injury, suicidal ideation, suicide attempt, and high-risk trauma exposure

		Full sample ($n = 9526$)	
Phenotype	1	2	3
1. Non-suicidal self-injury	–	–	–
2. Suicidal ideation	0.56 (0.52–0.60)	–	–
3. Suicide attempt	0.55 (0.50–0.61)	0.88 (0.85–0.91)	–
4. High-risk trauma exposure	0.41 (0.34–0.48)	0.40 (0.36–0.44)	0.48 (0.42–0.54)
		Men ($n = 3942$)	
Phenotype	1	2	3
1. Non-suicidal self-injury	–	–	–
2. Suicidal ideation	0.47 (0.40–0.54)	–	–
3. Suicide attempt	0.51 (0.42–0.61)	0.85 (0.79–0.91)	–
4. High-risk trauma exposure	0.36 (0.27–0.46)	0.32 (0.25–0.38)	0.38 (0.28–0.49)
		Women ($n = 5584$)	
Phenotype	1	2	3
1. Non-suicidal self-injury	–	–	–
2. Suicidal ideation	0.61 (0.56–0.66)	–	–
3. Suicide attempt	0.58 (0.52–0.64)	0.90 (0.87–0.94)	–
4. High-risk trauma exposure	0.46 (0.37–0.54)	0.47 (0.41–0.52)	0.53 (0.46–0.61)

Correlations were estimated including individuals with missing data and controlling for cohort/sample.
Ninety-five percent confidence limits presented in the brackets.
Nearly all individuals [430 (99.1%) of 434] who reported suicide attempt also reported suicidal ideation, producing very high correlations between the phenotypes.

Reduced models

Shared environmental influences on high-risk trauma could be constrained to zero without a significant decrement in model fit ($\Delta\chi^2 = 1.07$, $df = 2$, $p = 0.59$). Similarly, common and specific shared environmental factors explained a negligible proportion of the variation in self-injurious thoughts and behaviors and could be dropped from the model (online Supplementary Table S5). Within the ADE CPM, A and D could be constrained to zero individually, but constraining them both to zero resulted in a significant decrement in fit (online Supplementary Table S5). We elected to fit A and E factors within the correlated factors model, as (1) the presence of dominant genetic variance in the absence of additive genetic variance is possible, but practically unlikely; (2) separate A and D estimates should be treated with caution, as they are highly confounded and imprecise when estimated simultaneously; and (3) within the AE model, A captures both additive and non-additive genetic variation. Indeed, the heritability coefficients obtained within the AE CPM were the same as those observed within the ACE and ADE models (men: $h^2 = 54\%$, women: $h^2 = 51\%$).

Sex differences

When comparing the fits of models that allowed the parameter estimates for men and women to differ to models that constrained the estimates to be equal, there was evidence of a sex difference for high-risk trauma ($\Delta\chi^2 = 18.03$, $df = 3$, $p < 0.001$). Within the ACE and ADE CPMs, the paths from the latent factor to NSSI, SI, and SA could be equated across men and women, as could the common genetic and environmental factors. Constraining the variable-specific factors, however, resulted in a significant decrement in model fit (online Supplementary Table S6). Given the evidence for sex differences, correlated factors models were fit separately in men and women.

Correlated factors models

Table 5 displays the A and E estimates for HRT and self-injurious thoughts and behaviors, as well as the genetic and environmental correlations derived from the correlated factors models. All parameter estimates, including residual parameters, are depicted in online Supplementary Figs. S7 and S8. The genetic correlations between HRT and self-injurious thoughts and behaviors were large and significant in both men [$r_G = 0.59$ (0.37–0.81)] and women [$r_G = 0.56$ (0.49–0.63)], suggesting the covariation between these outcomes is partly attributable to shared heritable risk factors. After accounting for the familial overlap between trauma exposure and self-injurious thoughts and behaviors, we observed a significant unique environmental correlation among women [although the lower bound of the estimate was very close to zero; $r_E = 0.23$ (0.01–0.45)]. The unique environmental correlation was not significant among men [$r_E = 0.07$ (−0.10 to 0.23)].

Discussion

This report furthers our understanding of (1) the etiology and co-occurrence of NSSI, SI, and SA; and (2) the link between traumatic experiences and self-injurious thoughts and behaviors. We found that a significant proportion of the variation in NSSI, SI, and SA is attributable to genetic and unique environmental factors that are common to all three phenotypes. In addition, liability to self-injurious thoughts and behaviors results in part from genetic influences that are shared with high-risk trauma exposure. We

Table 4. Within-trait and cross-trait twin correlations for non-suicidal self-injury, suicidal ideation, suicide attempt, and high-risk trauma exposure

	Twin 2 phenotype							
	Monozygotic women ($n = 1094$ pairs)				Monozygotic men ($n = 661$ pairs)			
Twin 1 phenotype	1	2	3	4	1	2	3	4
1. Non-suicidal self-injury	0.61 (0.47–0.74)				0.56 (0.37–0.76)			
2. Suicidal ideation	0.36 (0.25–0.46)	0.59 (0.51–0.67)			0.35 (0.20–0.49)	0.39 (0.26–0.52)		
3. Suicide attempt	0.34 (0.21–0.47)	0.37 (0.25–0.48)	0.45 (0.27–0.62)		0.23 (0.01–0.46)	0.45 (0.28–0.61)	0.43 (0.09–0.77)	
4. High-risk trauma exposure	0.29 (0.13–0.44)	0.41 (0.32–0.50)	0.48 (0.35–0.61)	0.70 (0.62–0.79)	0.27 (0.09–0.45)	0.22 (0.10–0.34)	0.45 (0.27–0.63)	0.39 (0.23–0.56)
	Twin 2 phenotype							
	Dizygotic same-sex women ($n = 808$ pairs)				Dizygotic same-sex men ($n = 508$ pairs)			
Twin 1 phenotype	1	2	3	4	1	2	3	4
1. Non-suicidal self-injury	0.31 (0.08–0.54)				0.03 (−0.30 to 0.36)			
2. Suicidal ideation	0.16 (0.02–0.30)	0.12 (0.002–0.25)			0.12 (−0.04 to 0.29)	0.18 (0.03–0.33)		
3. Suicide attempt	0.19 (−0.05 to 0.43)	0.17 (0.02–0.32)	0.37 (0.13–0.61)		0.16 (−0.11 to 0.44)	0.23 (0.06–0.41)	0.02 (−0.41 to 0.44)	
4. High-risk trauma exposure	0.33 (0.18–0.49)	0.20 (0.09–0.31)	0.21 (0.04–0.38)	0.35 (0.20–0.50)	0.13 (−0.06 to 0.31)	0.17 (0.04–0.31)	0.30 (0.12–0.48)	0.30 (0.11–0.48)
	Male twin phenotype							
	Dizygotic opposite-sex ($n = 888$ pairs)							
Female twin phenotype	1	2	3	4				
1. Non-suicidal self-injury	0.09 (−0.17 to 0.34)							
2. Suicidal ideation	0.04 (−0.10 to 0.18)	0.14 (0.02–0.25)						
3. Suicide attempt	0.14 (−0.07 to 0.35)	0.01 (−0.13 to 0.16)	0.17 (−0.11 to 0.46)					
4. High-risk trauma exposure	−0.03 (−0.20 to 0.14)	0.14 (0.04–0.24)	0.21 (0.06–0.36)	0.17 (0.03–0.31)				

Twin correlations were estimated controlling for cohort/sample.
Also included in the analyses were data from 1608 single twins: 277 monozygotic women, 289 monozygotic men, 248 dizygotic same-sex women, 290 dizygotic same-sex men, and 504 dizygotic opposite-sex.
Ninety-five percent confidence limits presented in the brackets.

Table 5. Standardized parameter estimates and genetic and environmental correlations from the correlated factors model

	A	E	r_G	r_E
Phenotype		Men		
High-risk trauma exposure	0.65 (0.54–0.77)	0.76 (0.66–0.86)	0.59 (0.37–0.81)	0.07 (−0.10–0.23)
Self-injurious thoughts and behaviors	0.74 (0.64–0.85)	0.67 (0.55–0.79)		
Phenotype		Women		
High-risk trauma exposure	0.82 (0.73–0.91)	0.57 (0.44–0.71)	0.56 (0.49–0.63)	0.23 (0.01–0.45)
Self-injurious thoughts and behaviors	0.70 (0.62–0.78)	0.71 (0.63–0.79)		

A, additive genetic; E, unique environment; r_G, genetic correlation; r_E, unique environmental correlation.
A and E path estimates can be squared to obtain the proportion of variation in each outcome that is attributable to genetic and unique environmental influences, respectively.
Ninety-five percent confidence limits presented in the brackets.

observed a significant (though modest) unique environmental correlation in females, suggesting that HRT may confer some risk for self-injurious thoughts and behaviors among women.

Univariate heritability estimates

Present findings align with prior literature implicating heritable factors in trauma exposure, with higher heritability estimates observed for more severe events (e.g. assault; Afifi *et al.* 2010; Ehlers *et al.* 2013; Stein *et al.* 2002). In addition, results are broadly consistent with previous studies documenting genetic influences on NSSI and suicidality (Durrett, 2006; Maciejewski *et al.* 2014; Dutta *et al.* 2017; Statham *et al.* 1998). The DZ twin-pair correlation obtained in Durrett's (2006) analysis of NSSI (r = 0.37) was not as small as those observed in the present analysis; however, differences may be due to variability in participant ages across studies.

Self-injurious thoughts and behaviors

Much of the variation in SI and SA was explained by genetic influences shared with NSSI. This aligns with theories that the association between NSSI and STBs arises in part from a common inherited liability (Nock *et al.* 2006; Sher & Stanley, 2009). What factors comprise these genetic influences? Our understanding may be furthered by extending the Research Domain Criteria model to suicidal behavior (Glenn *et al.* 2017). Two vulnerability factors with which NSSI and suicidality have been well associated are impulsivity and negative emotion dysregulation (Nock *et al.* 2009; APA, 2013; Bresin *et al.* 2013; Klonsky & May, 2014; Hamza *et al.* 2015; Glenn *et al.* 2017). As GWAS of suicidal behaviors to date have failed to identify genome-wide significant associations (Mullins *et al.* 2014; Galfalvy *et al.* 2015), future GWAS focusing on these endophenotypes may have more success.

Prior studies, however, report relations between NSSI and STBs even after adjusting for psychopathology dimensions related to impulsivity and negative affect (Klonsky *et al.* 2013). Therefore, it seems unlikely that biological influences on impulsivity and negative emotion dysregulation can entirely account for the co-occurrence of self-injurious thoughts and behaviors. Indeed, a significant proportion of the covariation among NSSI and STBs in the present study was explained by unique environmental influences, which were not entirely attributable to high-risk traumatic events. Prior research on self-injurious thoughts and behaviors suggests additional candidates, including types of victimization not assessed in this study [e.g. intimate partner violence (Levesque *et al.* 2010; Vaughn *et al.* 2015) and peer victimization and bullying (Fisher *et al.* 2012; van Geel *et al.* 2014)], as well as other adverse life experiences (Skegg, 2005).

Although the majority of genetic variance in SI and SA was explained by common factors, 33–36% (among men) and 46% (among women) of the variation in NSSI was attributable to unique genetic influences. NSSI is associated with a range of emotional disorders (e.g. Cox *et al.* 2012; Bentley *et al.* 2015) and is a diagnostic criterion for borderline personality disorder (American Psychiatric Association, 2013). Some have called for creating a separate diagnostic category for NSSI (e.g. Selby *et al.* 2015) and cite this seeming ubiquity across categories, as well as evidence for NSSI's incremental validity in predicting poorer outcome, as support for a cross-diagnostic dimension of psychopathology that deserves study in its own right. Our data cannot resolve this question; however, they do suggest that some of the heritable risk factors for NSSI are distinct from those for STBs.

It is important to note that although we fit a common pathway model to the data for self-injurious thoughts and behaviors, other models may offer alternative advantages or insights. For instance, SA may be considered contingent upon prior SI. Two-stage models, such as causal–common–contingent models (e.g. Edwards *et al.* 2011), could help to capture this data structure and allow for exploration of the progression between NSSI and STBs.

Correlated factors model

To our knowledge, this is the first study to examine the genetic overlap between trauma exposure and self-injurious thoughts and behaviors. However, our finding of significant genetic covariance is consistent with results from genetically informative studies of traumatic experiences and psychiatric disorders, including depression and PTSD (Koenen *et al.* 2008; Afifi *et al.* 2010; Sartor *et al.* 2012). Because trauma is an exposure rather than a direct behavior, shared genetic influences could reflect heritable characteristics that influence vulnerability to both victimization and psychopathology [e.g. personality traits such as neuroticism and antisociality (Jang *et al.* 2003; Parslow *et al.* 2006) and emotion regulation difficulties]. In addition, 'common' genetic factors may operate through phenotypic causality, in which heritable features influence exposure to traumatic events and the experience of being traumatized makes an individual more likely to engage in self-injurious behaviors (or *vice versa*).

We observed a significant (albeit modest) unique environmental correlation between HRT and self-injurious thoughts and behaviors among women. This suggests that in females, high-risk

trauma may confer some direct risk for self-harm and suicide. This aligns with a prior discordant twin analysis of childhood sexual abuse and SA (Nelson *et al.* 2002) and several twin studies of victimization and other psychopathology (e.g. Kendler *et al.* 2000; Brown *et al.* 2014; Schaefer *et al.* 2017).

We did not observe a significant unique environmental correlation among men. This may, however, have been attributable to reduced statistical power, as our sample comprised fewer men than women and the prevalence of trauma exposure was lower among men. In addition, strong causal conclusions cannot be drawn solely from the present findings, as (*a*) they concern cross-sectional data, and (*b*) within-twin-pair associations may be confounded by twin-specific environmental differences that relate to both trauma exposure and self-injurious thoughts and behaviors. However, regardless of the extent to which associations reflect causal factors, prevention of trauma exposure should remain a top clinical and public health priority. The deleterious consequences of maltreatment and victimization for health and development are well documented (Keyes *et al.* 2012; Schaefer *et al.* 2017). Minimizing individuals' exposure to trauma may help reduce liability to a broad range of psychopathology (Schaefer *et al.* 2017), which may also reduce risk for life-threatening behaviors.

Limitations

Findings should be interpreted in the context of some limitations. First, the sample consisted of Australian Caucasians, which restricts generalizability. Second, data were collected retrospectively and thus prone to retrospective biases. Relatedly, we could not determine the temporal ordering of behaviors; in particular, whether traumatic experiences preceded NSSI and STBs. These issues limit causal inference. Third, although we differentiated between low- and high-risk trauma, we could not conduct fine-grained analyses of different types of high-risk traumatic events (e.g. childhood maltreatment *v.* later-life victimization). This represents an important goal for future research. In addition, operationalizing trauma exposure dimensionally (e.g. according to the number or severity of experiences) may increase statistical power to detect potential within-twin-pair associations with self-injurious thoughts and behaviors. Finally, the CTS design oversampled for families in which twins reported childhood maltreatment. To the extent that the relation between trauma and self-injurious thoughts and behaviors in this high-risk group differs from that in the general population, generalizability may be limited. However, we also included trauma data from the community-based full Cohort II sample, which helps to reduce concerns about possible ascertainment bias.

Conclusions

Individuals engaging in NSSI are at increased risk for SI and SA, and common heritable factors contribute significantly to these associations. Future research should investigate the mechanisms underlying this shared genetic vulnerability, as well as environmental factors that modify expression of this liability and risk for progression from NSSI to completed suicide. Preventing trauma exposure may help to mitigate risk for self-harm and suicide, either directly or indirectly via reductions in liability to psychopathology more broadly. In addition, targeting pre-existing vulnerability factors could significantly reduce risk for life-threatening behaviors among those who have experienced trauma.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291718001034.

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