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Non-suicidal self-injury and life stress: A systematic meta-analysis and theoretical elaboration

Richard T. Liu, **Shayna M. Cheek**, and **Bridget A. Nestor**

Department of Psychiatry and Human Behavior, Alpert Medical School of Brown University, Bradley Hospital

Abstract

Recent years have seen a considerable growth of interest in the study of life stress and non-suicidal self-injury (NSSI). The current article presents a systematic review of the empirical literature on this association. In addition to providing a comprehensive meta-analysis, the current article includes a qualitative review of the findings for which there were too few cases (i.e., < 3) for reliable approximations of effect sizes. Across the studies included in the meta-analysis, a significant but modest relation between life stress and NSSI was found (pooled OR = 1.81 [95% CI = 1.49–2.21]). After an adjustment was made for publication bias, the estimated effect size was smaller but still significant (pooled OR = 1.33 [95% CI = 1.08–1.63]). This relation was moderated by sample type, NSSI measure type, and length of period covered by the NSSI measure. The empirical literature is characterized by several methodological limitations, particularly the frequent use of cross-sectional analyses involving temporal overlap between assessments of life stress and NSSI, leaving unclear the precise nature of the relation between these two phenomena (e.g., whether life stress may be a cause, concomitant, or consequence of NSSI). Theoretically informed research utilizing multi-wave designs, assessing life stress and NSSI over relatively brief intervals, and featuring interview-based assessments of these constructs holds promise for advancing our understanding of their relation. The current review concludes with a theoretical elaboration of the association between NSSI and life stress, with the aim of providing a conceptual framework to guide future study in this area.

Keywords

life events; meta-analysis; non-suicidal self-injury; self-harm

1. Introduction

Life stress has been identified as a non-specific risk factor for psychiatric illness. It figures prominently in etiological models of a variety of mental disorders, including schizophrenia

*Corresponding author. Richard T. Liu, Department of Psychiatry and Human Behavior, Brown University, Bradley Hospital, 1011 Veterans Memorial Parkway, East Providence, RI 02915 (rtliupsych@gmail.com).

(Walker, Mittal, & Tessner, 2008), substance use (Sinha, 2001), depression (Monroe & Harkness, 2005), and, of course, post-traumatic stress disorder (Brewin & Holmes, 2003). Consistent with several of these theoretical perspectives, life stress has been found to be associated with increased risk for psychosis (Beards et al., 2013), substance use and dependence (Enoch, 2011; Hyman & Sinha, 2009), and depression (Hammen, 2005).

Stressful life events have also been theoretically and empirically linked with risk for self-harm, particularly in the form of suicidal ideation and behavior (Liu & Miller, 2014; Mann et al., 2005). Considerably less studied in this regard is non-suicidal self-injury (NSSI), defined as the direct and deliberate destruction of one's own bodily tissue in the absence of any suicidal intent (Nock, 2010). Although it has traditionally received less empirical consideration than suicidal behavior, NSSI has been increasingly recognized over the last decade as an important and phenomenologically distinct clinical phenomenon in its own right (Muehlenkamp, 2005). Indeed, NSSI as a distinct syndrome has been included in DSM-5 as a disorder warranting further study (American Psychiatric Association, 2013). The relative neglect of NSSI in earlier clinical research stemmed, in large measure, from the view that it exists along with suicidal behavior on a continuum of deliberate self-harm, and, moreover, that it falls on the milder end of this spectrum (Brent, 2011a). There is emerging evidence, however, suggesting that this is not the case. That is, although NSSI and suicidal behavior do share some common correlates, they differ significantly in their functions, neurobiology, response to treatment, and long-term trajectory (Brent, 2011a; Mars et al., 2014; Muehlenkamp & Gutierrez, 2007; Muehlenkamp, 2005; Wichstrøm, 2009). Not only is NSSI a meaningfully distinct form of self-harm, there is some emerging evidence to indicate that it may be an even stronger predictor of suicidal behavior than a prior history of suicide attempts (Asarnow et al., 2011; Wilkinson, Kelvin, Roberts, Dubicka, & Goodyer, 2011), highlighting the clinical importance of this behavioral phenomenon.

Several researchers have emphasized the need for studies of suicide to move beyond the identification of general risk factors towards elucidating causal elements that lead individuals to engage in this specific form of self-harm, so as to inform intervention and treatment strategies (Brent, 2011b; Nock, 2009a). Given the current paucity of evidence-based treatments for NSSI (Whitlock, 2010), especially in adolescents (Nock, 2012), the need for research uncovering causal factors for this behavior also holds true. Arriving at a better understanding of the relation between life stress and NSSI may be particularly important in this regard. Specifically, insofar as stressful life events are a temporally delimited rather than trait-like risk factor, and, moreover, insofar as these stressful life events potentially precipitate occurrences of NSSI, documenting this relation may aid in advancing our conceptualization of *who* is generally at risk to include *when* they are at imminent risk for engaging in this behavior. Such knowledge is of potential clinical utility in formulating and timing intervention strategies, particularly with chronically high-risk individuals.

The principal aim of the present article was to present a systematic meta-analysis of the empirical literature on the association between life stress and NSSI. As our interest was in conducting a comprehensive review of the literature, our meta-analysis was supplemented by a qualitative review of findings for which there were too few cases (i.e., < 3) for reliable approximations of effect sizes. To provide an appropriate context in which to evaluate the

existing literature, we first began with a brief overview of the conceptualization and measurement of life stress. We then proceeded with a consideration of the relevance of life stress to conceptual models of NSSI. Following a comprehensive review of the current literature on life stress and NSSI, we ended with a discussion of methodological considerations and a theoretical elaboration of the relation between these two constructs, with the aim of providing a framework to guide future study in this area.

1.1. Conceptualization and measurement of life stress

As a risk factor for negative mental health outcomes, stress has been defined and studied in a variety of notably different ways (for a detailed review, see Cohen, Kessler, & Gordon, 1995). These include physiologic stress, especially as operationalized in terms of hypothalamic-pituitary-adrenal (HPA) axis reactivity and allostatic load (McEwen, 1998; Selye, 1936). A second frequently adopted approach to conceptualizing stress centers on psychological or subjective stress (e.g., degree of distress experienced as a result of the individual's cognitive appraisal of the threat or challenge posed by an event; Lazarus & Folkman, 1984). In contrast, the focus of yet another commonly observed perspective is on exposure to exogenous stimuli or events within the individual's environment (e.g., loss of a job, end of a friendship), independent of the individual's subjective interpretation of the events (Grant, Compas, Thurm, McMahon, & Gipson, 2004; Hammen, 2005).

Although studies of physiologic and subjective stress have contributed considerably to our understanding of risk for different forms of psychopathology (Gunnar & Quevedo, 2007; Park, 2010), the current review focuses exclusively on stress as defined within the third tradition, often termed "objective" stress (Hammen, 2005). Several researchers have commented on the existence of certain significant advantages of this conceptualization of "objective" stress relative to subjective stress (Grant et al., 2003; Hammen, 2005). In particular, a potential concern with operationalizing stress based on subjective or cognitive appraisals of the stressfulness of an event is that it risks confounding environmental events with the individual's underlying diathesis. That is, measures based on self-report of the perceived stressfulness of experienced life events, to varying degrees, reflect both objectively occurring life events and pre-existing vulnerabilities (e.g., cognitive traits or biological propensities), with the latter accounting for individual variability in subjective stress ratings of the same life event. Support for this view may be found in several cognitive theories of mental illness. For example, within the depression literature, negative cognitive appraisals of life events have been hypothesized to lead to greater experiences of distress (Abramson, Metalsky, & Alloy, 1989). Empirical support for the potential influence of individual diatheses on subjective appraisals of life stress is also evident in studies observing subjective ratings of life event severity and their association with neuroticism (Espejo et al., 2011), and a polymorphism in the promoter region of the serotonin transporter gene (Conway et al., 2012). Moreover, these pre-existing diatheses may exert an influence on subjective stress even in the absence of clear objectively occurring stressors, with ambiguous or non-threatening situations being more likely to be interpreted in a negative manner (Beevers, 2005; Mathews & MacLeod, 2005).

The importance of cleanly observing the distinction between life stress and diatheses in testing etiological models of negative mental health outcomes becomes clear as research on life stress in several fields advances from predominantly stress exposure models (i.e., the notion that life stress temporally precedes and elevates risk for mental illness) to more nuanced diathesisstress theories (i.e., the concept of mental illness as a product of pre-existing vulnerabilities and external stressors) or mediational models of psychopathology (Grant et al., 2003). With individual diatheses, in some measure, already incorporated in subjective stress ratings, evaluations of diathesis-stress models of psychopathology may be at greater risk for false negatives (i.e., Type II error); if the life stress variable is already a compound of objectively occurring stressors and the diathesis of interest, it stands to reason that an interaction between this variable and the diathesis is unlikely to account for a significantly greater amount of variance in the outcome. With assessments of mediators of the relation between life stress and mental illness, on the other hand, the concern instead is of an inflated association or even spurious effect (i.e., Type I error). Inasmuch as the life stress variable is confounded with the mediator of interest, the strength of the observed association between the two is likely to be exaggerated. Furthermore, the direct effect of the life stress variable on the outcome is likely to be spuriously reduced after accounting for this confounded mediator.

1.2. Conceptualizations of the relation between life stress and NSSI

That life stress likely functions as a proximal predictor of NSSI is consistent with several theoretical conceptualizations of this behavior. Specifically, according to a four-function model of NSSI (Bentley, Nock, & Barlow, 2014; Nock & Prinstein, 2004, 2005), this behavior is reinforced by several distinct processes. These include two intrapersonal emotion regulation processes (negative reinforcement with the release of tension or decrease in negative affect following engagement in NSSI, and positive reinforcement, such as is involved in the need to feel pain or to act on feelings of guilt through self-punishment). Paralleling these two intrapersonal processes, two interpersonal functions underlying NSSI have also been identified (positive reinforcement, with NSSI serving as means of communicating the need for help and support, and negative reinforcement, such as the cessation of negative interpersonal interactions following NSSI). Such interpersonal functions may be especially pertinent in the case of individuals with impaired interpersonal problem-solving abilities and poor general communication skills (Hilt, Cha, & Nolen-Hoeksema, 2008; Nock & Mendes, 2008). A common element across all four functions is the implied precipitating presence of a form of distress, such as that experienced when confronted with an acute stressor, the primary difference across the four functions being the reason for engaging in NSSI to cope with this distress.

Prominent across several different conceptualizations of the pathogenesis of NSSI is the view that those who engage in this behavior suffer from a compromised ability to tolerate distress and to regulate their emotions, such as when they encounter a stressful life event (Klonsky, Oltmanns, & Turkheimer, 2003; Nock & Mendes, 2008). This view complements the four-function model of NSSI, the focus, however, being on the causal factors precipitating this behavior, rather than its contingent response. Support for this position comes from several studies documenting greater self-reported feelings of emotional distress

in those who engage in NSSI when they experience stress (Najmi, Wegner, & Nock, 2007; Nock, Wedig, Holmberg, & Hooley, 2008).

Although similarly not directly assessing life stress, several psychobiological studies of emotion regulation, distress tolerance, and laboratory stress induction tasks in relation to NSSI report findings also suggestive of the possibility that life stress may indeed have a central role in the etiology of this clinical phenomenon. In particular, one study found NSSI to be associated with heightened physiological reactivity (as indexed by skin conductance) in response to an experimentally induced stressor, as well as lower ability to tolerate the stressor (Nock & Mendes, 2008). In a more recent study, HPA axis dysregulation following an experimentally induced social stressor was found to be associated with NSSI (Kaess et al., 2012). Also indicative of the potential influence of life stress on the pathogenesis of NSSI, experimentally elicited recollection of recent negative interpersonal interactions appears to be associated with reduced distress tolerance among women who engage in NSSI (Gratz et al., 2011). In contrast, this diminished distress tolerance was not observed when induced recollection of interpersonal stressors was omitted.

Collectively, such findings have informed recent theoretical understandings of NSSI, with life stress more explicitly integrated into conceptual models of this behavior (e.g., Nock, 2009b, 2010). In particular, individuals at risk for NSSI may possess certain intrapersonal risk factors (e.g., a predisposition to experience diminished distress tolerance in response to stressful circumstances) and interpersonal vulnerabilities (i.e., poor interpersonal problem-solving and communication skills), which, when combined with life stress, particularly within interpersonal domains, may trigger dysregulated emotions. This emotion dysregulation, in turn, is resolved by engaging in NSSI as a means to alleviate feelings of distress, as a form of self-punishment, or as a method of interpersonal communication (Nock, 2009b, 2010).

2. Method

2.1. Search strategy and eligibility criteria

A systematic search of the literature was conducted in PsycINFO and MEDLINE to identify studies of potential relevance to the current review. The following search string was applied: (self-injur* OR mutilat* OR self-cut* OR self-harm*) AND (life stress* OR stressor* OR event OR events). The search results were limited to: (i) English-language publications and (ii) journal publications. This search strategy yielded a total of 1,379 articles, of which 1,080 were unique reports. Each unique search result was reviewed independently by at least two of the authors to determine eligibility. In cases where the eligibility of an article could not be ruled out based on the title and abstract, the full text was also examined. Discrepancies in coding article eligibility were rare (n = 7) and resolved by discussion among all three authors.

The study inclusion criteria were: (i) NSSI was defined as any intentional self-harm carried out without any suicidal intent; (ii) a well-defined time frame in the measure of NSSI was specified and consistent across all study participants; (iii) the assessment of life stress was standardized across study participants (e.g., studies that extracted life event data from

medical chart reviews were excluded); (iv) a well-defined time frame in the measure of life stress was specified and consistent across all study participants; (v) for reasons already mentioned above, analyses were included of objectively occurring life stress (i.e., studies that only assessed physiologic or subjective stress in relation to NSSI were excluded); (vi) so as to ensure adequate construct validity, and following the precedent of prior systematic reviews of life stress (e.g., Liu & Alloy, 2010; Liu & Miller, 2014), the measure of life stress must include a broad range of events (e.g., standard life events checklists or interviews) rather than only one to a few items (e.g., having a mid-term exam); and (vii) in the case of studies that included assessments of childhood maltreatment, this must not be the primary form of life stress examined in association with NSSI.[1] In the case of studies where more information on the measurement of self-harm or life stress was needed to determine study eligibility, every effort was made to obtain additional details in other publications describing the measure (e.g., other publications based on the same dataset). Details regarding the time frame assessed by the life stress measure in one study was obtained from its corresponding author (Baetens et al., 2014). Studies that included life stress only as a covariate in their analyses were considered for eligibility if these analyses included NSSI as a criterion variable.

Using these inclusion criteria, we excluded 955 reports based on their titles and abstracts. After this initial screen, an additional 102 were excluded based on a detailed full-text review, leaving a set of 23 studies satisfying the eligibility criteria (see Figure 1 for PRISMA flow chart). Studies were excluded based on full-text review because they (i) conflated NSSI with suicide attempts (n = 50); (ii) focused solely or primarily on childhood maltreatment in their assessment of life stress (n = 19); (iii) included assessments of life stress that did not comprehensively measure this construct (i.e., the measure was limited to only a few items; n = 8); (iv) provided insufficient information on how life stress was measured (n = 7); (v) utilized a measure that more accurately reflected constructs other than life stress (e.g., a scale assessing the severity of victimization experiences; n = 7); (vi) included only subjective stress in analyses of NSSI (n = 5); (vii) were inconsistent across participants in the time frame for which life stress was measured or the assessment time frame was not reported (n = 4); (viii) utilized a non-standardized measure of life stress (e.g., medical chart review; n = 1); and (ix) a corrigendum that did not result in any changes in the relevant findings (n = 1). Two additional studies were excluded at a later stage of the review, detailed below, for a final total of 104 excluded studies based on full-text review.

2.2. Data extraction

Of the 23 studies meeting eligibility criteria, nine featured overlapping samples from three datasets. In these cases, determination of which study to include in the meta-analysis was based, in descending order, on: (i) inclusion of sufficient reported data for meta-analysis; (ii) inclusion of a broader range of life stressors in analyses of the relation between life stress and NSSI; (iii) presentation of data on zero-order effects; (iv) use of longitudinal analysis; and (v) largest sample size for the relevant analysis. Two of the nine studies were excluded

[1]Studies focusing predominantly on childhood maltreatment were excluded, following the precedent of prior systematic reviews of life stress (e.g., Liu & Miller, 2014). Additionally, in the case of childhood sexual abuse, the relation with NSSI has been the subject of several prior systematic reviews (e.g., Klonsky & Moyer, 2008; Maniglio, 2011).

at this stage, as they presented findings on life stress and NSSI that did not differ substantively from those of other studies included in this review that drew from the same samples, leaving a final set of 21 studies included in the current review.

To assess for potential moderators, we extracted data for 11 study characteristics. These included four sample characteristics: (i) sample age group (i.e., youth versus adult), (ii) mean age of sample, (iii) sample clinical status (i.e., community versus clinical or high-risk sample); and (iv) percentage of female participants in the study sample. Data for seven study design characteristics were also extracted, including: (i) cross-sectional versus longitudinal design;[2] (ii) NSSI instrument (i.e., self-report versus interview); (iii) timeframe of NSSI assessment; (iv) coding of NSSI in the relevant analyses (dichotomous versus continuous); (v) life stress instrument (i.e., self-report versus interview); (vi) life stress type (i.e., traumatic stressors versus general/others); and (vii) timeframe of life stress assessment.

2.3. Data analysis

All analyses were conducted with Comprehensive Meta-Analysis Version 3.3.070 (Biostat, 2014). The odds ratio (OR) was used as the primary index of effect size. In cases where ORs were not reported, they were derived whenever possible from available data reported in the study (e.g., means and standard deviations, correlation). All ORs were calculated such that values greater than 1 indicated a positive association between life stress and NSSI (i.e., life stress is associated with greater odds of engaging in NSSI). The overall weighted effect size was calculated by pooling ORs across all relevant studies. For all analyses, random-effects models were generated in preference to fixed-effects models, so as to account for the high expected heterogeneity across studies resulting from differences in samples, measures, and design. Heterogeneity across the studies was evaluated using the I^2 statistic. I^2 indicates the percentage of the variance in an effect estimate that is due to heterogeneity across studies rather than sampling error (i.e., chance). Low heterogeneity is indicated by I^2 values of around 25%, and moderate heterogeneity by I^2 values of 50%. Substantial heterogeneity that is due to real differences in study samples and methodology is indicated by an I^2 value of 75%, which suggests that the observed heterogeneity is more than would be expected with random error (Higgins, Thompson, Deeks, & Altman, 2003). In cases where high heterogeneity is observed, random-effects models are more appropriate than fixed-effects models in that they account for this heterogeneity by incorporating both sampling and study-level errors, with the pooled effect size representing the mean of a distribution of true effect sizes instead of a single true effect size. In contrast, fixed-effects models assume that a single true effect size exists across all studies and any variance detected is due strictly to sampling error. It thus estimates only within-study variance.

[2]Of the eight studies featuring a longitudinal design (Burke et al., 2015; Guerry & Prinstein, 2010; Hankin & Abela, 2011; Hasking et al., 2013; Liu et al., 2014; Voon et al., 2014a, 2014b; Yates et al., 2008), four included only analyses involving temporal overlap in the measurement of life stress and NSSI (Hankin & Abela, 2011; Liu et al., 2014; Voon et al., 2014b; Yates et al., 2008). These analyses were coded as cross-sectional in the current review. A fifth study included both cross-sectional and longitudinal analyses of life stress in relation to NSSI (Hasking et al., 2013). In this case, the cross-sectional, zero-order (i.e., unadjusted) effect was included in the meta-analysis, and the longitudinal, multivariate analysis of life stress in relation to first lifetime onset of NSSI was included in the supplemental qualitative review.

High heterogeneity is indicative of the need for moderator analyses to account for potential sources of this heterogeneity. Each potential moderator was first assessed individually, with the effect size at each level of the moderator estimated.

A common concern in conducting meta-analyses is the possibility of publication bias. That is, studies with small effect sizes or non-significant findings are less likely to be published, and thus may be more likely to be excluded from meta-analyses, resulting in a potentially inflated estimate of the overall effect size. To assess for the presence of a potential publication bias, the following publication bias indices were calculated: Orwin's fail-safe *N* (Orwin, 1983), Duval and Tweedie's trim-and-fill analysis (Duval & Tweedie, 2000), and Egger's regression intercept (Egger, Davey Smith, Schneider, & Minder, 1997). Orwin's fail-safe *N* is an indicator of the robustness of an overall effect size, calculating the number of studies with an effect size of 0 that would be required to reduce the overall effect size in a meta-analysis to non-significance. Duval and Tweedie's trim-and-fill analysis produces an estimate of the number of missing studies based on asymmetry in a funnel plot of the standard error of each study in a meta-analysis (based on the study's sample size) against the study's effect size. This analysis also calculates an effect size estimate and confidence interval, adjusting for these missing studies. It is important to note that this procedure assumes homogeneity of effect sizes, and thus, its results must be interpreted with a degree of caution in cases where significant heterogeneity is present. Finally, Egger's regression intercept also provides an estimate of potential publication bias using a linear regression approach assessing study effect sizes relative to their standard error.

3. Results

Of the final set of 21 studies, 13 were included in the meta-analytic review, yielding 14 unique effects.[3] The remaining studies were excluded from meta-analysis, but retained for qualitative review, because: (i) they featured samples included in other studies in the metaanalysis (n = 4); (ii) examined individuals with NSSI relative to a presumably more severe group (i.e., suicide attempters; n = 1); (iii) assessed life stress in an NSSI-only sample (i.e., compared single life-time engagement in NSSI to repeated NSSI in relation to life stress; n = 1); (iv) assessed life stress as an outcome, rather than predictor, for NSSI (n = 1); (v) or did not report enough data for meta-analysis (n = 1).[4]

Of the 21 final studies included in this review, 33% (n = 7) featured clinical or high-risk samples, 76% (n = 16) included a primarily or exclusively youth sample. Only 19% (n = 4) of the studies included longitudinal analyses. The time period covered by the assessment of NSSI ranged from 30 days to lifetime, with the latter being most common. The assessments of life stress covered periods ranging from 3 months to lifetime, with the latter being most frequently adopted. In terms of instruments used to assess NSSI, 24% (n = 5) of the studies adopted an interview, the rest using self-report measures. Similarly, a minority of studies featured interview-based assessments of life stress (19%, n = 4), the majority adopting self-report life events checklists. For a summary of study details, see Table 1.

[3]One study produced separate effect sizes for males and females.
[4]The authors were contacted but unable to produce the data required for inclusion in the meta-analysis.

3.2. Quantitative analyses

We first calculated the overall weighted effect size for the relation between life stress and NSSI. The weighted mean OR was 1.81 (95% CI = 1.49–2.21), $p < .001$, indicating that the odds of engaging in NSSI were 81.4% greater when exposed to a stressful life event. Heterogeneity was high ($I^2 = 95.67\%$), indicating the appropriateness of moderator analyses.

3.2.1. Moderator analyses: Sample characteristics—In our moderator analyses, the strength of the association between life stress and NSSI did not change as a function of the mean age of the study sample. This finding held true regardless of whether mean age was treated as a categorical ($p = .13$) or continuous variable ($b = -.02$, $p = .17$). Similarly, the association between life stress and NSSI was not moderated by the percentage of female participants in each study ($b < .01$, $p = .14$). Community samples were found to be associated with a larger effect (OR = 2.17 [95% CI = 1.26–3.73], $p < .01$), however, than were clinical or high-risk samples (OR = 1.16 [95% CI = 1.05–1.27], $p < .01$).

3.2.2. Moderator analyses: Study design characteristics—Larger effects were observed in studies featuring self-report measures of NSSI (OR = 2.16 [95% CI = 1.54–3.04], $p < .001$) relative to interview-based measures of this behavior (OR = 1.17 [95% CI = .88–1.55], $p = .29$). Additionally, assessing NSSI over a longer interval was associated with a stronger effect ($OR_{\text{NSSI over more than 12 months}} = 2.23$ [95% CI = 1.40–3.57], $p = .001$) than was measuring NSSI over the last year or less ($OR_{\text{NSSI over the past 12 months}} = 1.10$ [95% CI = 1.06–1.14], $p < .001$). In contrast, self-report measure of life stress did not differ significantly from interview-based assessments of this construct ($p = .07$). Similarly, the length of period covered by the assessment of life stress did not moderate the relation between this construct and NSSI ($p = .61$). The relation between life stress and NSSI also did not differ as a function of stressor type ($p = .32$).[5] A comparison of cross-sectional and longitudinal studies was not possible, as all studies included in the meta-analysis employed cross-sectional analyses. Similarly, all effect sizes included in the meta-analysis were for NSSI coded as a dichotomous variable, and the treatment of NSSI as a continuous versus categorical variable was therefore not suitable for moderator analysis. In sum, whereas larger effects were found in studies featuring self-report measures of NSSI as well as studies assessing NSSI over longer intervals, method of measuring of life stress and length of assessment interval for this construct did not moderate the strength of the association between life stress and NSSI.

3.2.3. Publication bias—In our assessment of potential publication bias, Orwin's fail-safe-N indicated that 74 unpublished studies with an OR of 1.0 would be required to reduce the weighted effect size for the relation between life stress and NSSI to 1.1 (our *a priori* trivial effect size), suggesting that the observed weighted effect size is relatively robust. Egger's regression test, however, revealed evidence of significant publication bias (intercept = 3.31, [95% CI = .35–6.28], $t = 2.44$, df = 12, $p < .05$). Additionally, the funnel plot of effect sizes was notably asymmetrical (Figure 2). When the trim-and-fill method was used to

[5]No differences between stressor subtypes were also found when comparisons were made between general stressors, family stressors, and traumatic stressors ($p = .10$).

correct parameter estimates for potential publication bias, the adjusted weighted OR was reduced from 1.81 (95% CI = 1.49–2.21) to 1.33 (95% CI = 1.08–1.63), a smaller, but still significant, effect.

3.3. Qualitative analyses

3.3.1. Prospective prediction of first lifetime onset of NSSI—Two studies drawn from the same sample examined life stress longitudinally in relation to first lifetime engagement in NSSI (Hasking, Andrews, & Martin, 2013; Voon, Hasking, & Martin, 2014a). Interestingly, these two studies reported contradictory findings, with one observing a significant relation between life stress and first onset of NSSI (adjusted $OR_{\text{baseline to 12-month follow-up}} = 1.10$ [95% CI = 1.06–1.17] and adjusted $OR_{\text{12-month to 24-month follow-up}} = 1.12$ [95% CI = 1.07–1.18]; Voon et al., 2014a) and the other failing to document this association (adjusted $OR_{\text{baseline to 12-month follow-up}} = 1.31$ [95% CI = .96–1.78]; Hasking et al., 2013). What may in large part account for these discrepant findings is that the latter study included more covariates than the former, particularly NSSI within the social network and thoughts of NSSI prior to engagement in this behavior. Even in the study reporting a positive association, it is worth noting that the observed effect size was very modest.

3.3.2. NSSI recurrence—Two cross-sectional studies provided preliminary support for the view that life stress is positively associated with recurrent NSSI. In one study (Manca, Presaghi, & Cerutti, 2014), higher rates of life stress were observed for frequent than for infrequent engagement in NSSI ($Z = 5.03$, $p < .01$). A degree of caution should be observed in interpreting this finding, however, as artificial dichotomization of continuous variables can lead to loss of measurement precision (Ruscio & Ruscio, 2002), an increased risk for spurious findings (i.e., Type I error; MacCallum, Zhang, Preacher, & Rucker, 2002; Maxwell & Delaney, 1993), as well as loss of statistical power and attendant risk for Type II errors (Cohen, 1983; MacCallum et al., 2002). Addressing this concern, a second study (Voon, Hasking, & Martin, 2014b) found evidence of a positive association between life stress and NSSI treated as a continuous variable ($r = .33$, $p < .01$). Importantly, this finding held when this relation was assessed longitudinally over a 12-month period ($r = .19$, $p < .001$; Hasking et al., 2013).

3.3.3. NSSI versus suicidal ideation and behavior—Two cross-sectional studies included direct comparisons of individuals who engaged in NSSI and had no history of suicidality with suicidal counterparts (Baetens, Claes, Muehlenkamp, Grietens, & Onghena, 2011; Tuisku et al., 2009). One of these studies reported no difference between the two groups (OR = .55 [95% CI = .22–1.40]; Tuisku et al., 2009). This finding should be interpreted with some caution, however, given its relatively small sample size ($n_{\text{NSSI only}} = 21$, $n_{\text{NSSI and suicidality}} = 52$), which limits its ability to detect a significant effect. In contrast, the other study, featuring a larger sample (n = 236), found adolescent suicide attempters were more likely to have experienced life stress ($t = -4.28$, $p < .001$, $r_{\text{effect size}} = .26$), particularly academic problems, parental separation or divorce, an illness in the family, suicidality in social network, and the death of friend (Baetens et al., 2011). A third cross-sectional study compared individuals with only NSSI to those who also had attempted

suicide in terms of traumatic life events, finding higher rates of interpersonal events in the case of the latter (partial η^2 = .06–.16; Zetterqvist, Lundh, & Svedin, 2013). In summary, whereas one study did not find any difference between individuals who engage in NSSI and suicidal counterparts in terms of life stress, two other studies observed higher rates of life stress among suicide attempters relative to those who engaged in NSSI.

3.3.4. Diathesis-stress and mediation—Four studies tested diathesis-stress models of NSSI (Guerry & Prinstein, 2010; Hasking et al., 2013; Voon et al., 2014a; Voon, Hasking, & Martin, 2014c). Life stress was found in one study to interact with depressogenic cognitions to predict NSSI 9 to 18 months later (Guerry & Prinstein, 2010). Life stress was similarly observed to interact with exposure to peer NSSI to predict engagement in this behavior over a 12-month period (Hasking et al., 2013). In a recent cross-sectional study, life stress interacted separately with rumination and emotion regulation in predicting NSSI (Voon et al., 2014c). These interactions did not predict first onset of NSSI, however, in a longitudinal study drawn from the same sample (Voon et al., 2014a). Voon et al. (2014c) also found life stress to be associated with NSSI through the mediational effect of psychological distress. A fifth study (Christoffersen, Møhl, DePanfilis, & Vammen, 2015) also found cross-sectional support for a mediational model, with reduced social support mediating the relation between traumatic life stress and NSSI. Caution should be taken in interpreting these findings, however, given concerns regarding the validity of cross-sectional tests of mediation (Lindenberger, von Oertzen, Ghisletta, & Hertzog, 2011; Maxwell & Cole, 2007). To summarize, three studies found support for a diathesis-stress model of NSSI, whereas one did not. Two studies identified potential mediators of the relation between life stress and NSSI, albeit with their cross-sectional design limiting inferences regarding the temporal nature of these associations.

3.3.5. NSSI and stress generation—An interesting possibility evaluated, in part, in a recent study (Burke, Hamilton, Abramson, & Alloy, 2015) is that NSSI may share a transactional relation with life stress. That is, not only may life stress increase risk for NSSI (i.e., a stress exposure model of psychopathology), but NSSI, or more precisely characteristics associated with risk for this behavior, may confer risk for later life stress (i.e., stress generation; Hammen, 1991, 2006). More specifically, in applying the stress generation hypothesis to NSSI, one would predict individuals who engage in this behavior to experience prospectively higher rates of life stress that are at least in part dependent on their own characteristics, beliefs, and behaviors (i.e., dependent stress, such as the end of a relationship), but also not to differ in their rates of life stress that occurs outside their influence (i.e., independent stress, such as the death of a relative). Consistent, in part, with the stress generation hypothesis, this study found adolescents with a lifetime history of NSSI prospectively experienced greater levels of interpersonal stress (β = .33, p < .001), but not non-interpersonal stress (β = .11, *ns*) over a six-month period. Furthermore, this relation was moderated by sex, such that this stress generation effect was observed in girls (t = 4.07, p < .001) but not boys (t = −.45, *ns*). Although only one study provided a partial assessment of the stress generation hypothesis in relation to engagement in NSSI, its findings suggest that future research in this area is warranted.

4. Discussion

The current review aimed to present a systematic quantitative and qualitative analysis of the association between life stress and NSSI. The results were largely consistent in providing support for a stress exposure model of NSSI. In the meta-analysis, the pooled odds of engaging in NSSI were approximately 80% greater after experiencing life stress than when life stress was absent, a relatively modest effect size. After adjusting for publication bias, the weighted odds of engaging in NSSI were reduced to 33% higher when experiencing life stress relative to the absence of life stress. In qualitative analyses, evidence of this relation was stronger for NSSI recurrence than first onset of this behavior. Again, in both cases, the magnitude of this relation generally appeared to be fairly modest. These qualitative findings, however, are based on a notably small number of studies, some drawn from the same sample, and thus their findings should be regarded as preliminary, awaiting replication in future research.

Several possibilities exist to account for the small effect sizes observed across these studies. First, the time period covered by life stress instruments tended to be quite long, with the vast majority cases involving single-time-point assessments spanning 12 months or longer. Such lengthy recall periods for life stress are problematic because recollection of major life events tends to fade after a year in adults (Paykel, 1997) and approximately seven months in adolescents (Monck & Dobbs, 1985), and less severe events are often forgotten over briefer intervals (Brown & Harris, 1982). If relatively more distal life stress is of pathogenic relevance to NSSI, it may not be adequately captured with the long recall periods employed by most studies, and assessments at multiple time-points over briefer intervals may be recommended. Additionally, the life stress that has been implicated in the pathogenesis of other stress-related forms of mental illness, such as depression (Hammen, 2005) and suicidal ideation and behavior (Liu & Miller, 2014), has generally been found to be much more proximally related to their occurrence. For example, life stress occurring in the three months prior to depressive onset appears to be most etiologically relevant to this disorder (Harkness, Bruce, & Lumley, 2006). Furthermore, one recent study found support for acute life stress as a precipitant of suicidal behavior, with suicide attempters being more likely to experience life stress in the 24 hours prior to their attempt than during hours 24 to 48 prior to attempt (Bagge, Glenn, & Lee, 2013). If proximal, relative to distal, life stress is similarly more relevant to the occurrence of NSSI, the ability to detect this relation is likely to be diluted substantially by the presence of less etiologically relevant and distal life stress captured with measures employing lengthy recall periods. Although the length of period covered by life stress instruments was not found significantly to account for effect size heterogeneity across studies in moderator analyses, this may in part be due to the dichotomization of this moderator variable as 12 months or less versus over 12 months, necessitated by the fact that only two studies in the meta-analysis featured life stress assessments of less than 12 months. Dichotomization at 12 months may not have provided sufficient sensitivity to detect moderation by length of period covered by the life stress instrument if many studies at both levels of this moderator variable employed too long of a recall period. Future research utilizing shorter recall periods with greater temporal resolution in the assessment of life stress is required to ascertain to what degree this may indeed be the case.

A second potential explanation for the small observed overall effect size for the relation between life stress and NSSI is the relative lack of resolution in the *types* of life stress assessed in the existing literature. More specifically, most studies examined life stress as a general construct, with traumatic events being most common among the studies that evaluated stressor subtypes (four studies included in the meta-analysis and five studies overall). Insofar as certain stressor subtypes are involved in the pathogenesis of NSSI, and insofar as other stressor subtypes are not, the consideration of all stressors within a unitary construct is a significant concern, as it is likely to diminish the magnitude of the observed association between life stress and NSSI. It is worth mentioning within this context that evidence of specificity of stressor subtypes has been noted for other forms of mental illness. For example, there is some evidence that dependent life stress may be more depressogenic than independent stress (Hammen, Marks, Mayol, & DeMayo, 1985; Kendler, Gardner, & Prescott, 2002, 2006). Moreover, interpersonal stress seems particularly relevant to the etiology and maintenance or recurrence of depression, anxiety, and suicidal ideation and behavior (Hammen, 2005; Heimberg, Brozovich, & Rapee, 2010; Liu & Miller, 2014). As will be detailed below, there is good reason to suspect that stressor content may be similarly important for understanding risk for NSSI.

A third possibility is that life stress and NSSI do indeed share only a modest association. Giving weight to this possibility is the fact that only a small minority of individuals who experience life stress engages in NSSI. The implication of this fact is that a stress exposure model of NSSI may provide an incomplete account of the role of life stress in the onset and recurrence of this behavior. Instead, it may be that only individuals who possess pre-existing diatheses for NSSI engage in this behavior when confronted with life stress (i.e., diathesis-stress models of NSSI). There is a notable paucity of studies to date evaluating diathesis-stress models of NSSI, with some preliminary support existing only for life stress interactions with depressogenic cognitive styles (Guerry & Prinstein, 2010) and some more mixed support for emotion regulation and rumination (Voon et al., 2014a, 2014c). Although not a diathesis-stress study *per se*, a fourth report found life stress to interact with external interpersonal factors in the form of peer NSSI to predict future NSSI (Hasking et al., 2013). Reflecting the preliminary state of the empirical literature in this area is the fact these four studies were based on only two samples. Additional research is therefore needed, particularly with theoretically derived diatheses, to advance our understanding of the potential applicability of diathesis-stress models to the etiology and recurrence of NSSI.

It is important to note that these possible explanations for the modest relation between life stress and NSSI need not be mutually exclusive. Rather, some or all of these explanations may to some degree be involved in accounting for the observed weighted effect size. It may be, for example, that proximal life stress precipitates engagement in NSSI, but only when interacting with a pre-existing diathesis, and only for specific subtypes of life stress.

In addition to the timeframe of life stress instrument, other findings that emerged in the moderator analyses warrant discussion. First, the magnitude of the association between life stress and NSSI was significantly larger in community samples than in clinical or at-risk samples. This may be a function of the fact that, as previously mentioned, life stress is a non-specific risk factor for mental illness (Beards et al., 2013; Enoch, 2011; Hammen, 2005;

Hyman & Sinha, 2009). In community samples, consisting mostly of healthy controls, NSSI is, in large measure, being compared to the absence of psychopathology. In contrast, clinical or at-risk samples involve comparisons to controls for whom there is a higher prevalence of psychopathology, and thus more exposure to life stress, than may reasonably be expected for healthy controls within community samples.

Second, moderator analyses yielded a significantly larger pooled effect size for studies using a self-report measure of NSSI than ones featuring interview-based assessments. Although this finding may initially seem counterintuitive, given the greater methodological rigor of empirically validated interview-based measures (Guerry & Prinstein, 2010), it may to some degree be due to the possibility that self-report measures of NSSI are less precise and consistent than interview-based assessments in capturing experiences of deliberate self-harm in a manner that observes the researchers' definition of this construct. Of particular importance in the present context is the possible endorsement of false positives on self-report measures of NSSI. More specifically, it is possible that acts of self-harm that would fit the standard research definition of suicide attempts (i.e., self-harm conducted with non-zero intent to die; Asarnow et al., 2011) may instead be endorsed as NSSI by respondents on self-report measures, particularly if the degree of suicidal intent was low (i.e., most of the intent in engaging in the self-harm behavior was *not* to die). Given the aforementioned preliminary evidence that suicidal behavior is associated with higher rates of life stress than is NSSI, the potential misclassification of suicide attempts as NSSI on self-report measures may reasonably be expected to result in a stronger association with life stress than would be the case with interview-based assessments.

The finding that the relation between life stress and NSSI was weaker when NSSI was assessed over the last 12 months than when measured over longer spans of time was particularly curious. Given the aforementioned study reporting preliminary evidence for the possible relevance of stress generation to NSSI (Burke et al., 2015), and given the cross-sectional design utilized in all studies included in the meta-analysis, one potential explanation for this finding is that both stress exposure and stress generation processes may be reflected across these studies in their analyses of the association between life stress and NSSI. In contrast to measures of lifetime occurrence of NSSI, assessments limited to the past 12 months likely would not identify a proportion of individuals with a lifetime history of NSSI. Inasmuch as such individuals are categorized in studies using such measures as not having engaged in NSSI, and inasmuch as such individuals experience higher rates of dependent stress over the past 12 months (i.e., stress generation), their inclusion in the control condition would likely reduce the strength of the observed association between life stress and NSSI. This is less likely to occur in studies utilizing a lifetime measure of NSSI. Importantly, this should not be taken to imply that measures of lifetime occurrence of NSSI are preferable to measures over briefer intervals. On the contrary, in studies that assess NSSI only over the lifetime, it is impossible to determine the temporal relation between life stress and NSSI, a consideration of particular importance to the degree that stress generation is indeed phenomenologically relevant to NSSI.

4.1. Design considerations

As has been touched upon above, the empirical literature on the relation between life stress and NSSI is qualified by several important methodological limitations. Although the existing studies in this area are valuable for the insight they provide into this relation, the adoption of several methodologically rigorous design features in future research is essential to advance our understanding of the association between life stress and NSSI. In particular, it would be important to conduct multi-wave assessments of both life stress and NSSI over relatively brief intervals (e.g., six months) so as to facilitate accurate recall of both. Such an approach would allow for finer temporal resolution and a cleaner assessment of the temporal relation between life stress and NSSI. One could evaluate, for example, the extent to which life stress over the preceding six months, after covarying the occurrence of NSSI during the same time period, prospectively predicts engagement in this self-harm behavior over the following six months. Such clean temporal separation between these two constructs is particularly important for determining whether the life stress is not merely a correlate but, in fact, a risk factor for NSSI (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer et al., 1997; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

Moreover, the use of exacting multi-wave designs is necessary for what may be termed second generation research, moving beyond tests of a basic stress exposure model of NSSI toward a more nuanced understanding of its relation to life stress, particularly in terms of mediational models elucidating the potential pathways through which life stress exerts its pathogenic effect. As noted above, only two studies to date have examined mediational pathways underlying the relation between life stress and NSSI, both featuring cross-sectional analyses (Christoffersen et al., 2015; Voon et al., 2014c). Future research in this area on mediational relations is important for its potential to identify targets for clinical intervention.

Another major design feature that is notably rare in the existing literature are interview-based assessments of life stress, particularly contextual threat life stress interviews, which are considered the gold standard in life stress research (Dohrenwend, 2006; Hammen, 2005; Kessler, 1997; Monroe, 2008; Paykel, 2001). Only one study of NSSI to date (Yates, Carlson, & Egeland, 2008) included an interview-based contextual threat approach to measuring life stress, albeit over a 64-month period. With its ability to facilitate precise dating of the occurrence of individual life events, especially when assessed over relatively brief periods, the adoption of this approach is necessary for achieving the fine degree of resolution required to delineate the temporal parameters of the relation between etiologically relevant life stress and NSSI. This methodology would make possible, for example, the ability to identify the time-point at which life stress becomes relatively less related to onset or recurrence of NSSI (e.g., whether life stress in the three months immediately prior to NSSI are of especial relevance to its occurrence).

Contextual threat approaches to measuring life stress are also unique in the degree of resolution they provide regarding the content of individual stressors, as they involve eliciting a rich narrative of the context and consequences surrounding each event (Brown & Harris, 1978). Such rich contextual information is particularly important for allowing an accurate identification of multiple potential stressor subtypes into which each event may be

categorized. The event “changed schools”, for example, could be classified as a dependent (e.g., if the child was expelled from a prior school) or independent stressor (e.g., if it is a consequence of the child’s parents moving to a new job in a different city). Another event, “got into a car accident”, could potentially be coded as a financial event, a legal event, an interpersonal event (e.g., if it results in an argument between an adolescent driver and his or her parents), a physical health event if the individual was injured, and a loss event. Such details are not possible with alternative approaches to measuring life stress, for which there are therefore not insubstantial challenges to evaluating hypotheses regarding the specificity of stressor subtypes in relation to NSSI.

Finally, for reasons already mentioned above, it would be important to use empirically validated interview-based assessments of NSSI, thereby ensuring that this behavior is cleanly differentiated from suicide attempts and that any observed association with life stress is not, in part, reflective of the presence of suicidal behavior. This methodological approach has the added advantage of making possible relatively accurate recording of recent occurrences of NSSI (e.g., dates and number of days engaged in this behavior over the past 30 days), especially when used in conjunction with calendar aids typical of contextual threat approaches to life stress assessment, facilitating analyses of the temporal relation between recent life stress and this self-harm behavior.

4.2. A conceptual model of life stress and NSSI

There is a marked need for theory-driven research to advance our knowledge of the association between life stress and NSSI. Drawing on the existing theoretical and empirical literature, we present a preliminary conceptual model of this association with the hope that it may serve as a useful aid for future work in this area. It should be noted, however, that it is beyond the scope of the current review to present a comprehensive explanatory model of life stress in relation to NSSI, and a detailed consideration of several potentially relevant factors (e.g., neurobiological processes) are therefore necessarily omitted. Additionally, in highlighting proximal interpersonal life stress, we do not mean to imply that other forms of proximal life stress are not etiologically relevant to NSSI. Rather, the proposed model is meant to serve as an initial and parsimonious organizational framework from which future studies could draw, with selective emphasis placed the types of life stress and associated diatheses that appear most relevant to risk for NSSI. Particular emphasis was also placed on hypothesized associations for which current theoretical and empirical support is strongest.

We hypothesize that both relatively distal and more proximal life stress are involved in the etiology and recurrence of NSSI. Importantly, however, we also hypothesize that a degree of specificity exists regarding the stressor subtypes that function as distal and proximal risk factors for this behavior; relatively distal stressors most relevant to risk for NSSI are likely to include both interpersonal stressors as well as those involving physical pain or fear for physical safety, whereas relevant proximal stressors are more likely to be interpersonal in nature. Furthermore, we posit that the precise nature of the relation between these stressors and NSSI differs appreciably, with more distal life stress affecting risk for NSSI through mediational pathways, whereas more proximal life stress functioning as precipitants moderating the relation between pre-existing diatheses and NSSI.

More specifically, and starting with more distal factors, interpersonal stressors, as well as physically painful and fear-provoking ones, may indirectly increase risk for future engagement in NSSI through the development of specific diatheses that have been linked to this behavior. We hypothesize that some specificity exists between distal stressor subtypes and individual diatheses. In particular, and drawing on the interpersonal theory of suicide (Joiner, 2005; Van Orden et al., 2010), we posit that distal stressors involving physical pain and fear for physical safety are likely to lead to increased tolerance for the pain involved in self-harm. According to this theory, through multiple experiences of physically painful and fear-provoking experiences over time, individuals may habituate to the pain and fear associated with self-injurious behavior, what has been termed the acquired capability for suicide. Consistent with this theory, painful and fear-provoking experiences have been associated with the acquired capability for suicide (Anestis & Joiner, 2012; Franklin, Hessel, & Prinstein, 2011). This process may be similarly applicable to the etiology of NSSI. Importantly, this potential mechanism may be specific to physical rather than psychological distress tolerance, as individuals who engage in NSSI have been found to demonstrate greater physical pain tolerance than those who do not engage in selfinjury (Franklin et al., 2011; Gratz et al., 2011; St. Germain & Hooley, 2013), but lower psychological distress tolerance (Nock & Mendes, 2008; Nock, 2010). Although several studies included in the current review have found a higher rate of traumatic life events in individuals who engage in NSSI (Christoffersen et al., 2015; Layne et al., 2014; Paul, Schroeter, Dahme, & Nutzinger, 2002; Ray-Sannerud, Bryan, Perry, & Bryan, 2015; Zetterqvist et al., 2013), the degree to which increased tolerance for physical pain underlies the relation between physically painful or fear-provoking life stress and NSSI remains to be empirically determined.

The aforementioned lower tolerance for psychological distress, or emotion dysregulation, has consistently been found to be a prominent feature of NSSI within the research literature (Adrian, Zeman, Erdley, Lisa, & Sim, 2011; Gratz & Roemer, 2008; Gratz et al., 2011; Kaess et al., 2012; Nock & Mendes, 2008; Nock et al., 2008; Sim, Adrian, Zeman, Cassano, & Friedrich, 2009). It may also potentially function as a mechanism linking distal interpersonal stressors, as well as painful and fear-provoking stressors, to NSSI. That painful and fear-provoking life stress may potentially be involved in the development of emotion dysregulation is supported by a recent study in which children with a history of traumatic stressors, particularly ones involving physical pain and fear for safety, exhibited attenuated amygdala-anterior cingulate cortex connectivity, a pathway associated with emotion regulation (Pagliaccio et al., 2015). Several studies have also implicated interpersonal stressors, particularly ones involving victimization or social rejection, in the disruption of the normative development of emotion regulation skills (Baumeister, DeWall, Ciarocco, & Twenge, 2005; McLaughlin, Hatzenbuehler, & Hilt, 2009; McLaughlin & Hatzenbuehler, 2009). The possibility that emotion dysregulation may serve as a mediating mechanism between distal interpersonal stressors, as well as painful and fear-provoking stressors, and NSSI is in need of empirical validation.

Pathogenic cognitive processes may be yet another potential mediational pathway linking distal interpersonal stressors and NSSI. Individuals who engage in NSSI have been found to possess a more self-critical cognitive style (Glassman, Weierich, Hooley, Deliberto, & Nock, 2007), and negative self-views are frequently reported as a trigger for engagement in NSSI

(Nock, Prinstein, & Sterba, 2009). Whether distal interpersonal stressors may be an antecedent to the negative cognitive patterns relevant to the etiology and maintenance of NSSI is an intriguing possibility awaiting future investigation. There is substantial theoretical and empirical support, however, for the role of negative interpersonal interactions in the development of dysfunctional cognitive styles. According to Cole's (1990, 1991) competency-based model of depression, for example, children can become at risk for developing negative self-schemata through repeated experiences of interpersonal stress, particularly in the form of negative feedback from others (e.g., parents, teachers, and peers). The role of these negative interpersonal experiences in the formation of negative self-views has received empirical support (Cole, Martin, & Powers, 1997; Tram & Cole, 2000). Similarly, Rose and Abramson (1992) have put forth the view that repeated life stress may lead a child eventually to develop a negative cognitive style characterized, in part, by the tendency to infer negative self-characteristics in response to future negative events. Importantly, this is particularly the case for interpersonal conflicts involving verbal victimization from peers and adults, because, in such cases the negative self-inference is often directly supplied by the other individual and thus has a higher probability of being internalized as part of the negative interpersonal experience. The role of verbal peer victimization in the development of negative cognitive styles has been documented (Gibb, Abramson, & Alloy, 2004). This effect of negative interpersonal experiences on the development of negative self-schemata is specific to early childhood, as cognitive schemata are malleable during this period of development, but appear to solidify into more stable and trait-like thinking patterns during the transition to adolescence, corresponding with changes in cognitive development (e.g., concrete or formal operational stage; Cole, 1991; Garber & Flynn, 2001; Hankin, 2008; Nolen-Hoeksema, Girgus, & Seligman, 1992; Rose & Abramson, 1992; Tram & Cole, 2000; Turner & Cole, 1994).

With the increased stabilization of relevant diatheses in adolescence and adulthood, their relation with life stress and NSSI is hypothesized to transition from one of mediation to moderation. That is, proximal life stress may moderate the relation between diatheses for NSSI and the prospective occurrence of this behavior. Such a transition from a mediational to moderational relation with life stress has been observed for other forms of psychopathology (e.g., depression; Cole & Turner, 1993; Turner & Cole, 1994). Importantly, we hypothesize that the life stress involved in moderational relations with diatheses for NSSI is likely to be particularly interpersonal in nature. This may be especially true in the case of NSSI enacted for reasons of social negative reinforcement, as a means of terminating negative interpersonal interactions. Peer victimization experiences have, indeed, been associated with social negative reinforcement of NSSI, as well as social positive reinforcement of this behavior, in individuals with a pre-existing diathesis (Hilt et al., 2008). Given the increasing importance of social relationships in adolescence (Brown, 1990), and corresponding increases in social sensitivity (Somerville, 2013) and emotional reactivity to interpersonal stressors (Larson & Ham, 1993; Rudolph & Hammen, 1999), interpersonal stressors may have greater potential to elicit negative affect relative to other stressors subtypes and thus motivate intrapersonal functions of NSSI (i.e., intrapersonal positive and negative reinforcement), as conceptualized within the four-function model of this behavior (Bentley, Nock, & Barlow, 2014; Nock & Prinstein, 2004, 2005), particularly in this age

group. These intrapersonal functions, in turn, may increase risk for subsequent NSSI in response to similar future stressors. Partially suggestive of this possibility, in one study, NSSI history was associated with emotion dysregulation (and physical pain tolerance), but only when interpersonal distress was elicited (Gratz et al., 2011). Furthermore, as previously mentioned, another study found evidence of a cognitive diathesis-interpersonal stress interaction in predicting the prospective occurrence of NSSI (Guerry & Prinstein, 2010).

Finally, an intriguing possibility that potentially adds a layer of complexity to the association between life stress and NSSI is that it may be a dynamic one. In particular, the relation between the two phenomena may change over time, such that the level of life stress generally required to precipitate NSSI may be greater for its first onset than for its recurrence (i.e., stress sensitization; Monroe & Harkness, 2005; Post & Weiss, 1998). This may, in part, be because of the self-reinforcing functions of this behavior (Bennum & Phil, 1983; Nock & Prinstein, 2004; Nock, 2010). Moreover, NSSI, itself, could lead to increased physical pain tolerance and acquired capability for suicide (Gratz et al., 2011; Joiner, Ribeiro, & Silva, 2012; Joiner, 2005), and this increased diathesis, in turn, may lower the threshold for life stress required to precipitate subsequent recurrences of NSSI, in a manner consistent with a psychological scarring effect (i.e., scar hypothesis; Lewinsohn, Steinmetz, Larson, & Franklin, 1981).

4.3. Summary

The last few years have seen a marked growth of interest in delineating the relation between life stress and NSSI. Indeed, of the 21 studies included in the current review, 15 were published in the last five years alone. Across these studies, there appears to be a significant but modest relation between life stress and NSSI. Several methodological limitations characterize much of the existing literature, however, with the frequent adoption of cross-sectional analyses involving temporal overlap between measures of life stress and NSSI, in particular, constraining interpretability of the precise nature of the association between these two phenomena (e.g., the degree to which life stress may be a cause, concomitant, or consequence of NSSI). Future theoretically informed studies employing a multi-wave design, assessing life stress and NSSI over relatively brief intervals, and utilizing interview-based assessments of these constructs, hold particular promise for advancing our understanding of their association. Finally, in addition to highlighting these considerations, the current review proposes a preliminary conceptual model of this association which we hope may serve as a guide for future research in this area.

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Highlights

- We conducted a meta-analysis and narrative review of the link between stress and NSSI
- A significant but modest relation was found between life stress and NSSI
- Cross-sectional design limitations render the temporal nature of the relation unclear
- A preliminary conceptual model of this association is presented to guide future study

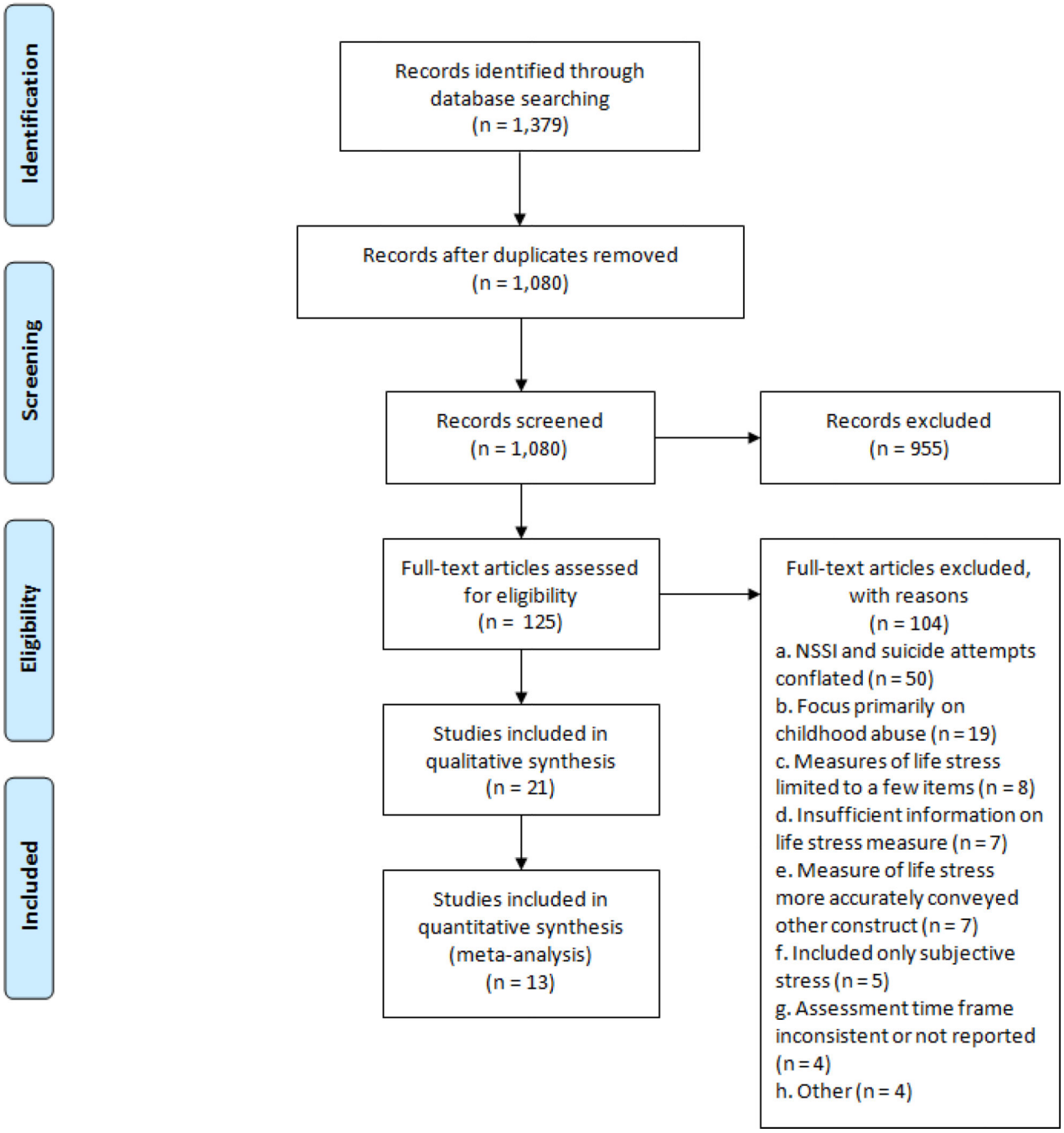

Figure 1.
PRISMA flow chart of literature search.

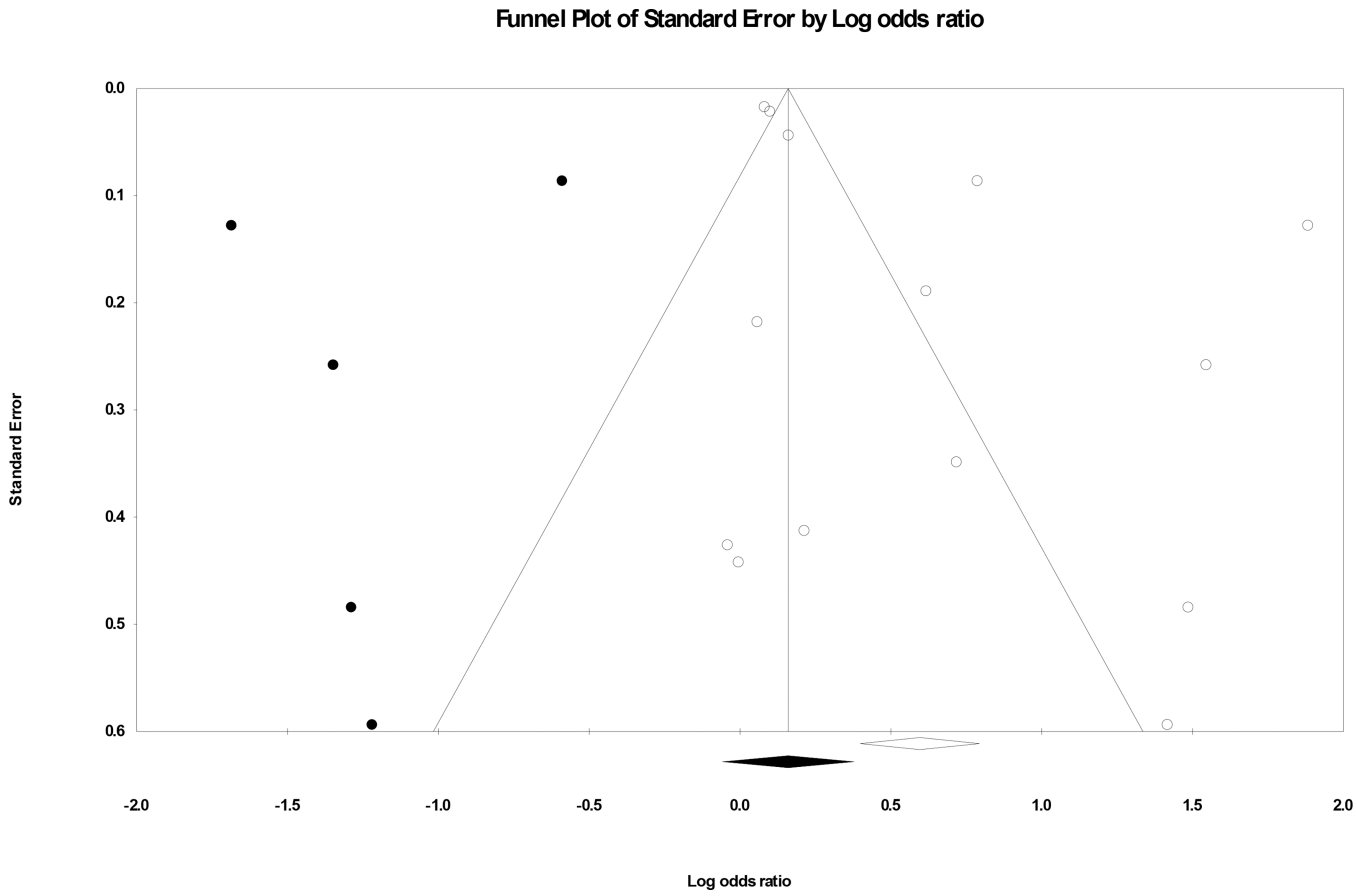

Figure 2.

Funnel plot for effect sizes in the meta-analysis. The vertical line indicates the weighted mean effect. Open circles indicate observed effects for actual studies, and closed circles indicate imputed effects for studies believed to be missing due to publication bias. The clear diamond reflects the unadjusted weighted mean effect size, whereas the black diamond reflects the weighted mean effect size after adjusting for publication bias.

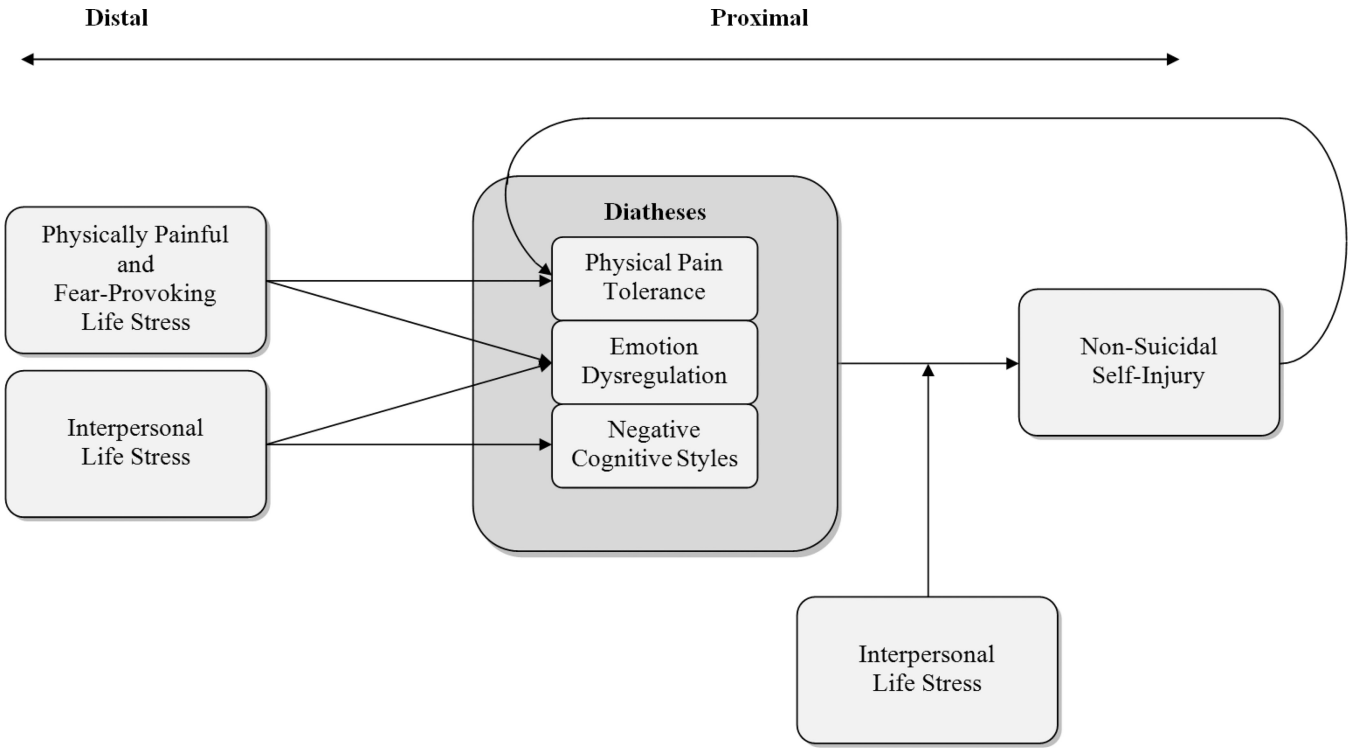

Figure 3.
Conceptual model of moderation and mediational associations between life stress and non-suicidal self-injury.

Table 1

Study characteristics.

Study Author(s) (year)	N[1]	% Female	Mean Age	Sample	Prospective Analysis	NSSI Measure	NSSI Time Frame	Life Stress Measure	Life Stress Time Frame	Life Stress Type
Baetens, Claes, Muehlenkamp, Grietens, & Onghena (2011)	236	81.4	15.1	Community	No	Questionnaire (SSM)	Lifetime	Questionnaire (SSM)	12 months	General
*Baetens et al. (2014)	1439	54.8	12.0	Community	No	Questionnaire (SSM)	Lifetime	Questionnaire (SSM)	Lifetime	Family
*Bennun (1983)	40	85.0	23.3	Clinical	No	Interview (SSM)	Lifetime	Interview (SSM)	Lifetime	General
Burke, Hamilton, Abramson, & Alloy (2015)	110	73.0	18.7	Community	Yes	Questionnaire (FAFSI)[5]	Lifetime	Interview (LES & LEI)	6 months	General
*Cerutti, Manca, Presaghi, & Gratz (2011)	234	50.4	16.5	Community	No	Questionnaire (DSHI)[5]	Lifetime	Questionnaire (LSC-R)	Lifetime	General
Christoffersen, Møhl, DePanfilis, & Vammen (2015)	2980	47.8	24.5	Community	No	Interview (SSM)	Lifetime	Questionnaire (SSM)	Lifetime	Trauma
*Garrison, Addy, McKeown, & Cuffe (1993)	444	56.0	13.4	Clinical	No	Interview (K-SADS)	12 months	Questionnaire (CLES-A)	12 months	General
Guerry & Prinstein (2010)[a]	143	72.0	13.5	Clinical	Yes	Questionnaire (SSM)[5]	3 months	Questionnaire (LE-C)	9 months	Social
*Hankin & Abela (2011)	97	61.0	12.6	Community	No[3]	Questionnaire (FASM)[5]	2.5 years	Questionnaire (ALEQ)	3 months	General
*Hasking, Andrews, & Martin (2013)[b]	1973	71.72	14.9	Community	Yes/No[4]	Questionnaire (SHBQ)[5]	Lifetime	Questionnaire (ALES)	Lifetime	General
*Layne et al. (2014)	3785	62.7	15.3	Clinical	No	Questionnaire (ISFP)	30 days	Interview (THP)	Lifetime	Trauma
*Liu et al. (2014)[a]	110	70.9	14.3	Clinical	No[3]	Questionnaire (SSM)[5]	3 months	Questionnaire (LE-C)	9 months	General
Manca, Presaghi, & Cerutti (2014)	205	52.8	16.2	Community	No	Questionnaire (R-NSSI-Q)[5]	Lifetime	Questionnaire (LSC-R)	Lifetime	General
*Paul, Schroeter, Dahme, & Nutzinger (2002)	376	100.0	24.3	Clinical	No	Questionnaire (SSM)[5]	Lifetime	Questionnaire (TLEQ)	Lifetime	Trauma
*Ray-Sannerud, Bryan, Perry, & Bryan (2015)	422	28.1	36.3	Community	No	Questionnaire (SITBI-SR)[5]	Lifetime	Questionnaire (LE-C)	Lifetime	Trauma

Study Author(s) (year)	N[1]	% Female	Mean Age	Sample	Prospective Analysis	NSSI Measure	NSSI Time Frame	Life Stress Measure	Life Stress Time Frame	Life Stress Type
*Tuisku et al. (2009)	103	82.0	16.4	Clinical	No	Interview (K-SADS)	6 months	Questionnaire (LE-C)	12 months	General
Voon, Hasking, & Martin (2014a)[b]	2328	70.7	13.9	Community	Yes	Questionnaire (SHBQ)[5]	12-Month	Questionnaire (ALES)	12 months	General
Voon, Hasking, & Martin (2014b)[b]	2507	68.0	13.9	Community	No	Questionnaire (SHBQ)[5]	Lifetime	Questionnaire (ALES)	12 months	General
Voon, Hasking, & Martin (2014c)[b]	2637	68.0	13.9	Community	No[3]	Questionnaire (SHBQ)[5]	Lifetime	Questionnaire (ALES)	12 months	General
*Yates, Carlson, & Egeland (2008)	155	51.6	26.0	Community	No[3]	Interview (SIBQ)[5]	Lifetime	Interview (SSM)	64 months	Family
*Zetterqvist, Lundh, & Svedin (2013)	2185	56.0	16.0[2]	Community	No	Questionnaire (SITBI-SF-SR& FASM)[5]	Lifetime	Questionnaire (LYLES)	Lifetime	Trauma

Note:

*Articles marked with an asterisk were included in the meta-analysis.

a, b Studies with identical superscripts were drawn from the same sample.

[1]The number of participants included in relevant analyses, rather than entire study sample, is presented.

[2]As the mean age was not reported, the median value of the sample age range is presented here.

[3]These studies featured a longitudinal design but employed cross-sectional analyses of the relation between life stress and NSSI.

[4]This study presented both longitudinal and cross-sectional findings for the relation between life stress and NSSI. The bivariate findings (cross-sectional) were included in the meta-analysis, and the multivariate finding (longitudinal) in the narrative review.

[5]These measures included multi-query assessments of different forms of NSSI to determine its occurrence during the time period under consideration. In some studies, however, data were dichotomized for analysis.

ALEQ = Adolescents Life Events Questionnaire; ALES = Adolescent Life Events Scale; CASE = Child and Adolescent Self-Harm in Europe; CLES-A = Coddington Life Events Scale for Adolescents; DSHI = Deliberate Self-Harm Inventory; FAFSI = Form and Function Self-Injury Scale; FASM = Functional Assessment of Self-Mutilation; ISFP = Indicators of Severity of Functional Problems; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; LE-C = Life Events Checklist; LEI = Life Events Interview; LES = Life Events Scale; LSC-R = Life Stressor Checklist-Revised; LYLES = Linköping Youth Life Experiences Scale; R-NSSI-Q = Repetitive Non-Suicidal Self-Injury Questionnaire; SHBQ = Self-Harm Behavior Questionnaire; SIBQ – Self-Injurious Behavior Questionnaire; SITBI-SF-SR – Self-Injurious Thoughts and Behaviors Interview – Short Form – Self Report; SITBI-SR – Self-Injurious Thoughts and Behaviors Interview – Self Report; SSM = study-specific measure; THP = Trauma History Profile; TLEQ = Traumatic Life Events Questionnaire.