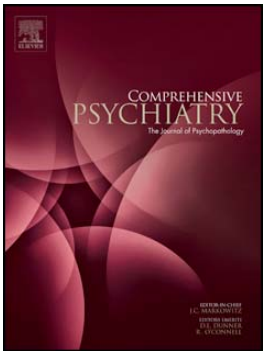
Age of onset of non-suicidal self-injury in Dutch-speaking adolescents and emerging adults : an event history analysis of pooled data



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PII: S0010-440X(17)30235-3
DOI: doi: 10.1016/j.comppsych.2017.10.007
Reference: YCOMP 51909

To appear in: *Comprehensive Psychiatry*

Please cite this article as: Gandhi Amarendra, Luyckx Koen, Baetens Imke, Kiekens Glenn, Sleuwaegen Ellen, Berens Ann, Maitra Shubhada, Claes Laurence, Age of onset of non-suicidal self-injury in Dutch-speaking adolescents and emerging adults: An event history analysis of pooled data, *Comprehensive Psychiatry* (2017), doi: 10.1016/j.comppsych.2017.10.007

Age of onset of non-suicidal self-injury in Dutch-speaking adolescents and emerging adults: An event history analysis of pooled data

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Highlights

- Non-suicidal self-injury (NSSI) is a major health concern in adolescents and young adults.
- Age-of-onset of NSSI was investigated in a pooled sample of adolescents and young adults.
- By age of 25 years, 21% of community sample engaged in at least one NSSI episode.
- The probability of age of onset of NSSI peaked around the age of 14-15 years.
- Depending the population studied, second peak in the age-of-onset was also observed.

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Abstract

Objective: Non-Suicidal Self-Injury (NSSI) has emerged as an important mental-health concern. However, epidemiological features like age of onset of NSSI have remained understudied. Therefore, the current study investigated the distribution of age of onset of NSSI in pooled sample of Dutch-speaking adolescents and emerging adults using event history analysis.

Method: Eleven datasets measuring age at first NSSI in community and clinical participants collected by researchers in the Dutch-speaking part of Belgium were pooled together. The final dataset consisted of 1973 community males, 1901 community females, and 505 clinical females. Discrete-time event history analysis was used to model the effect of gender and psychiatric disorders on the age of onset of NSSI.

Results: Twenty-one percent of adolescents from the community samples engaged in at least one episode of NSSI by the age of 25 years. Irrespective of the type of sample (community or psychiatric disorder), the probability of age of onset peaked around the age of 14-15 years. A second peak was observed around the age of 20 and 24 years in the community and psychiatric samples respectively.

Conclusions: Psychosocial interventions for prevention of NSSI should not only target adolescence through school mental health programs but also target emerging adults at the university level.

Keywords: Non-suicidal self-injury; Age of onset; Event history analysis; Adolescents; Emerging adults; Gender differences; Psychiatric disorders.

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1. Introduction

Non-suicidal self-injury (NSSI) is defined as repetitive, direct, and deliberate destruction of one's body tissue without an intention to die [1]. According to two recent reviews [2, 3], approximately 18% of adolescents, 13% of emerging adults, and 5% of adults have a history of at least one episode of NSSI. Although the prevalence of NSSI is generally known to be higher in adolescents with psychiatric disorders, it is especially elevated in individuals with borderline personality disorder (BPD) and eating disorders (ED). Approximately 70%-75% of patients with BPD [4] and 27%-33% of patients with EDs engage in NSSI [5]. A strong association between NSSI and various negative mental health outcomes such as low self-esteem, depression, anxiety, and suicide attempts has been established [6, 4]. Consequently, NSSI has emerged as an important public health concern.

In the recent years, significant advances have been made in understanding the factors that increase the vulnerability to NSSI. Nonetheless, more fundamental demographic question like the distribution of age of onset of NSSI (i.e., plot of first episode of NSSI as a function of age) has remained unanswered. Investigating the distribution of age of onset of NSSI can be useful in developing age-relevant prevention and intervention strategies that target individuals in the age group that are most vulnerable to NSSI [7]. Effective prevention strategies can be very relevant in the context of NSSI as early age of onset has been shown to be associated with greater NSSI frequency, the use of more diverse NSSI methods, and NSSI-related hospital visit [8]. Although, no existing research has systematically investigated the distribution of age of onset of NSSI, some conclusions can be drawn from a recent literature review of 27 longitudinal NSSI studies by Plener, Schumacher, Munz, and Groschwitz [9]. These authors reported that the prevalence of NSSI steadily increases from the age of

12 years and peaks between 14 to16 years of age; by the age of 18 years, the prevalence of NSSI appeared to decrease. Although Plener and colleagues [9] focused on the prevalence of NSSI and not on the age of onset, we expect the later to follow a similar trend given that the majority of individuals engage in NSSI for the first time during the ages of 12 -18 years of age [2]. However, further research is necessary to confirm this hypothesis.

Existing literature on NSSI has also indicated that gender and presence of psychiatric disorders like BPD and ED can influence the age of onset of NSSI [10, 11, 12]. A recent large-scale review by Bresin and Schoenleber [13] established gender as an important epidemiological factor that can influence NSSI. However, the extent of influence of gender on the age of onset of NSSI is still not clear. On one hand, individual studies have found that females had an earlier age of onset than males [10]. Yet, a meta-analysis by Bresin and Schoenleber [13] did not find a main effect of age on gender differences in NSSI. In light of these inconsistencies, further research is needed to confirm the effect of gender on the age of onset of NSSI. Similarly, the association between psychiatric disorders like BPD and ED on the age of onset of NSSI is far from clear as this issue has not been extensively studied. The handful of studies that do address the association between psychiatric disorders and age of onset of NSSI suggest that on an average, NSSI started approximately 1.4 years earlier in adolescents with BPD than in adolescents without BPD [11]. Conversely, mean age of onset of NSSI in in-patients with ED can range from 16.8-17.5 years which was later than the individuals without ED [14, 15]. However, more research is needed to corroborate these findings.

In addition to the lack of studies systematically investigating the distribution of age of onset of NSSI, the studies and the meta-analyses cited so far had one additional drawback: they were directly or indirectly based on the mean-statistic which did not consider the effects of censored data or the effect of time. This may lead to a biased estimation of the true value of the effects [16]. In light of these

limitations, the current study investigated the distribution of age of onset in pooled samples of Dutch-speaking adolescents and emerging adults using event history analysis. Use of event history analysis allowed us to develop probabilistic models that mapped the distribution of age of onset of NSSI while incorporating the effect of censoring, time, and the non-normal distribution of variables. As mentioned earlier, based on the work of Plener and colleagues [9], we expected that the onset of NSSI in community samples would increase around the age of 12, peak around the age of 14-15 years, and subsequently reduce around the age of 18 years. We additionally investigated the influence of gender and the presence of psychiatric disorder like BPD and EDs on the distribution of age of onset as a function of time. Because of a lack of previous research, a detailed hypothesis regarding the effect of differences in gender distribution of age of onset of NSSI could not be formulated. However, based on the work of Andover and colleagues [10], we expected that females would have an earlier age of onset than males.

2. Material and methods

2.1. Data collection and processing

Four researchers working in the area of NSSI in the Dutch-speaking part of Belgium were requested to provide published or unpublished data if they met the following inclusion criteria: 1) NSSI was clearly defined as deliberate, self-inflicted, destruction of body tissue without an intent to die; 2) The studies were conducted in the Dutch-speaking parts of Belgium or the Netherlands; and 3) The studies also collected data regarding age at first episode of NSSI. The sample could be either clinical or from a community sample. For each study, copies of the original questionnaires, manuscripts or dissertations describing the study methods were also requested. As the focus of the present work was on adolescents and emerging adults, individual cases were included in the study only if the age of the participants were less than or equal to 25 years of age. Data with missing values on the variables

relevant to the current study (presence/absence of NSSI, age of onset of NSSI, age, gender) were excluded from the final pooled dataset. Each individual study was approved by the ethics review board of the authors' university. Table 1. presents the characteristics of the 11 studies included in the current research. The pooled sample consisted of 4,379 participants.

All the datasets assessed the age of onset of NSSI by means of some version of the question "How old were you the first time you intentionally hurt yourself?" [17]. Finally, the pooled data were converted from a person-level data file to a person-period data file. Age was used as the time-scale such that each person entered the risk set at the age of one year and exited the study when they first engaged in NSSI (i.e., experienced the event) or if they reached the age at which they participated in the data collection process (i.e., the point of attrition). Additionally, the variables gender and the presence/absence of psychiatric disorders were included as time-invariant variables in the pooled dataset. Overall, the person-period data file had 60,853 data points (30,376 males, 30,477 females without psychiatric disorder, and 8627 females with psychiatric disorders (BPD or ED).

2.2. Analytical strategy

The analyses in the present work were performed using MLWin (version 2.36), SPSS (version 24). and STATA (version 13.1). To investigate heterogeneity, we considered the pooled data as a multi-level data with individual participants (level 1) nested within each dataset (level 2). We fitted a null frailty model (i.e., model without predictor variables but with random effects at the level of datasets) with presence/absence of NSSI as the outcome variable. In spite of being a dichotomous variable, the presence/absence of NSSI was modeled as a continuous variable to obtain the variance partitioning coefficient (VPC) using the Iterative Generalized Least Squares estimation procedure. The VPC represents the amount of variance (i.e., heterogeneity) explained at the level of datasets. This method of obtaining VPC is commonly referred to as the binary linear method. Note that use of the binary linear

method was necessary as VPC in multi-level non-linear models are not otherwise comparable [18]. A smaller VPC value indicated smaller heterogeneity.

In order to investigate the effect of gender and presence of psychiatric disorders on the distribution of age of onset of NSSI, the discrete-time event history analysis method outlined by Willet and Singer [19] was adopted. As noted by Willet and Singer [19], discrete-time event history analysis begins by computing the baseline or initial hazard model. A baseline hazard model does not differentiate between sample members (i.e., it does not have any covariates) and it aids in the examination of the main effect of time on the onset of the event. To visually inspect the main effect of age on the onset of NSSI, we plotted the temporal profile of risk of onset of NSSI. This plot was obtained by entering Age as the time-varying variable into the X-axis and presence/absence of NSSI in the Y-axis. The resulting graph represented the baseline hazard for experiencing NSSI during a given age. Additionally, by using the hazard function, we computed and plotted the cumulative survival function to investigate the probability of individuals who never experienced NSSI during the first 25 years of their lives.

Next, logistic regression was used to fit the hazard profile to statistically evaluate the main effect of age on onset of NSSI. The baseline logit hazards were obtained by entering presence/absence of NSSI as the dependent variable and age as the independent (factor) variable. Data across the first five years of age was aggregated together in single category as the probability of onset of NSSI was almost zero in the first five years of age. This collapsed category was treated as the reference group. We also investigated if the fit of the basic hazard model improved by the addition of gender to the main effect of age. To investigate if the assumption of proportionality was violated (i.e., effect of gender on onset of NSSI changed with age), we also tested if models with interaction of age and gender had a better fit than the model only with main effect of gender and the baseline hazard model. The -2 log-

likelihood statistic test and information criteria (Akaike information criterion [AIC] and Bayesian information criterion [BIC]) were used to evaluate the improvement in the model fit. The appropriate model was subsequently interpreted. For the sake of brevity, the results of all the survival functions and the parameter estimated of the relevant logistic regression models used to fit the hazard functions are presented in an online appendix accompanying this manuscript.

The above-mentioned procedure was also followed for evaluating the effect of psychiatric disorders on the age of onset of NSSI. However, unlike the community samples, the general hazard and survival function for the combined samples of community and clinical females were less informative than the model including the interaction with the presence/absence of psychiatric disorder. Therefore, the general hazard and survival functions are only briefly discussed. As the clinical data did not have male participants, the effect of psychiatric disorders was investigated in females only.

3. Results

3.1. Heterogeneity among the datasets

The VPC estimate obtained using the null frailty model indicated that most of the variance in presence/absence of NSSI was situated at the individual level as only 2% of the variance existed at the level of the datasets (σ_u^2 [SE] = .0003 [.0001]). A low value of VPC indicated that the datasets were fairly homogenous. Given that use of random effects models offer limited advantage when VPC is less than .05 [20], we continued the analysis using the regular logistic regression models.

3.2. Influence of gender on distribution of age of onset

Figure 2 (Panel A) shows the distribution of age of onset of NSSI in the community sample. The graph indicates that the probability of onset of NSSI increased steadily through late childhood and it peaked at 14 years of age. Further, the probability of onset of NSSI decreased as the adolescents transitioned into emerging adulthood. A smaller second peak was also observed around the age of 20

years but NSSI was less likely to begin after the age of 21 years. Panel B of Figure 1 shows the general survival function for the community sample. From the plot, it is clear that up to 25 years of age, the cumulative probability of onset of NSSI was around .21 (=1-.79 [cumulative survival function at age 25]). The details of the generalized logistic regression model used to fit the hazard function can be accessed through the online appendix. The fitted logistic regression model indicated that the odds of age of onset of NSSI is consistently higher in all age groups as compared to the age group of 1-5 years. The odds of onset of NSSI was highest in the age groups of 14 years (77.33 higher odds as compared to the reference group) and 15 years (70.52 higher odds as compared to the reference group). The odds of onset of NSSI decreased around the age of 18 years before it increased to 24.62 at the age of 20 years as compared to the reference group.

Table 2 shows the comparison of the goodness of fit of the baseline model (only with the main effect of age) with the extended models that included gender (both main effect and interaction effect with age). Table 2 indicated that addition of interaction of gender and age (linear or categorical) to the baseline model significantly increased the overall fit of the model to the data. Model 3 was selected as the model best fitting the data as it was more parsimonious (indicated by low BIC value) than the interaction model 4. The parameters estimated for the interactive model are presented in the online appendix. The overall model indicated that interaction between gender and age was found to be significant ($\beta_{age(linear)*gender}$= .18, *SE* = .03, *z* = 31.68, *df* = 1, *p* = <.001) in the interaction model.

Figure 1 (Panel C) presents the plot of predicted probabilities of onset of NSSI against age using the interaction model 3. The hazard functions for males and females crossed at about 11 years. Before age 11, males had a marginally higher probability of onset of NSSI. However, after the age of 11, females consistently had a higher probability of onset of NSSI than males. Interestingly, the second peak in the age of onset of NSSI which was observed around the age of 20 years was much sharper for

females than males indicating the presence of a group of individuals with late onset of NSSI in females. Panel D of Figure 1 shows the gender differences in the survival function. The figure indicates that by the end of age 25, females (1- .74 [cumulative survival function at age 25] = .26) had a higher cumulative probability of engaging in at least one episode of NSSI than males (1-.83 [cumulative survival function at age 25] = .17).

3.3. Influence of psychiatric disorders on distribution of age of onset

Figure 2 (Panel A) shows the distribution of age of onset of NSSI in the community and clinical cohorts of females. Unlike the hazard function of the community sample, the hazard function of the combined female samples had a bimodal distribution of the probability of onset of NSSI with peaks around the ages 14 to 15 and a second peak at 24 years. The logistic regression model used to fit the hazard function shown in Figure 2 (Panel A) is presented in the online appendix. The relevant odds ratios are shown in the Panel A of Figure 2. The survival function (shown in Panel B of Figure 2) indicates that up to 25 years of age, the probability of having a first experience of NSSI was around .45 (1- .55 [cumulative survival function at age 25]). From Table 3, it is clear that adding the variable presence/absence of psychiatric disorder to the basic model (only with the main effect of age significantly reduced the value of the -2LL statistic hence indicating that the extended model had a better fit to the data than the basic model. Model 3 was again selected as the best model to represent the data as it was more parsimonious than the interactive model 4 (indicated by low BIC value). The parameters estimated for the interactive model 3 are presented in the online appendix and the model indicates that interaction between the presence or absence of psychiatric disorders and age was significant ($\beta_{age(linear)*disorder}$=.13, *SE* = .04, *z* = 14.09, *df* = 1, *p* = <.001).

Figure 2 (Panel C) presents the plot of predicted probabilities of onset of NSSI against age using model 3. The plot indicated that the probability of onset of NSSI was virtually the same for the females

with and without psychiatric disorders before the age of 9 years. After age 9, the probability of females with psychiatric disorders engaging in NSSI was higher than the hazard function for the females from the community samples. As observed in the community samples, after the peak around 14-15 years, the hazard of onset of NSSI decreased till the age of 18 years. However, after age 19, the probability of onset of NSSI in the females from the clinical samples increased again until it peaked around the age of 24. It should be noted that the peak at the age of 24 years may be an outcome of a methodological artefact. Tall peaks towards the end of the measurement waves are common in discrete event history analysis because presence of even a single case in already small number of cases can artificially inflate the hazard ratio. Therefore, the second peak must be interpreted with caution. Panel D of Figure 2 shows the differences in the survival function of females with and without psychiatric disorders. It is also clear from Figure 2 that till the age of 25 years, the probability of females from the psychiatric samples engaging in first episode of NSSI was higher (1- .36 [cumulative survival function at age 25] = .64) than females from the community samples (1 - .74 [cumulative survival function at age 25] = .26). The median survival rate for the females with psychiatric disorder reached around 19 years of age.

4. Discussion

With about 21% adolescents (about 26% females and 17 % males) engaging in at least one episode of NSSI by the time they reach the age of 25 years, our analysis indicated that NSSI may be a significant mental health issue in our Dutch-speaking samples of community adolescents and emerging adults. We found that although the first episode of NSSI could occur as early as 6 years, the probability of first NSSI episode increased dramatically after the age of 9 years. The age of onset of NSSI peaked around 14 years of age. Our findings also indicated that before the age of 9 years, males had a somewhat higher probability of engaging in NSSI as compared to females. The interaction model used in the current research did not permit us to investigate if the gender differences in the onset of NSSI

observed in the first 9 years were statistically significant. In any case, these findings contradicted some earlier research [10] that found females to have an earlier age of onset than males. Although the peak in the age of onset of NSSI for both males and females was around the age of 14 – 15 years, in line with earlier studies [13] we observed that females had a significantly higher probability of engaging in at least one episode of NSSI than males in this age group.

The cumulative survival function differentiating between community and psychiatric samples indicated that about 64% females with psychiatric disorder engaged in at least one episode of NSSI before the age of 25 years. About 50% of females with a diagnosis of a psychiatric disorder engaged in at least one episode of NSSI before the age of 19 years. The comparison of the age of onset of NSSI between females with and without psychiatric disorders indicated that before the age of 9 years, the probabilities of onset of NSSI between these cohorts were virtually similar. After 9 years of age, the probability of onset of NSSI increased exponentially in females with psychiatric disorders. The onset of NSSI peaked for females at the age of 14 to 15 years and the probability of onset of NSSI was more than twice in the clinical population as compared to the community population. This finding was not surprising as NSSI is known to be more prevalent in individuals with psychiatric disorders. A second peak in the first episode of NSSI was also observed in the clinical population at a much later age of 24 years.

The results of the age of onset of NSSI can be more insightful if juxtaposed with the onset of other psychiatric disorders. For example, onset of NSSI coincides with the onset of disorders like conduct disorder (9-14 years), impulse-control disorders (13-21 years), and mood disorders (13.9 -15.1 years) [21] indicating that NSSI may be an outcome of a combination of impaired impulse control and emotional dysregulation issues. Greater impulsivity and emotional dysregulation is common in adolescents as the areas in the brain associated with the reward system (e.g., caudate nucleus) and

emotional regulation (e.g., amygdala) develop before the areas associated with the development of the regulatory systems (e.g., prefrontal cortex) [22]. Without effective regulatory capabilities, adolescents with high impulsivity may indulge in immediate affect regulation strategies like NSSI even when the behavior is ultimately self-destructive [23]. Once the regulatory system matures, individuals may be better equipped to control impulses and may use more healthy ways to regulate affect. This may partially explain the lower prevalence of NSSI in adults as compared to adolescents [24]. The aforementioned neuro- regulatory mechanisms often fail to develop in individuals with BPD [25] and ED [26] and, therefore, these individuals may continue to use NSSI as a means of regulating emotions. This fact is reflected by the higher probability of onset of NSSI even when the females suffering from psychiatric disorders reach the age of 25 years.

The bimodal distribution of the age of onset of NSSI has also been reported in other psychiatric disorders like anxiety disorder [27], agoraphobia [28], eating disorders [29], obsessive compulsive disorder [30], and schizophrenia [31]. Although the reasons for the second peak in the age of onset of NSSI are not entirely clear, hypotheses regarding the late onset of NSSI can be formulated using the existing research on factors that increase vulnerability to psychiatric disorders in emerging adults. The most parsimonious explanation for the late onset of NSSI may be the presence of a recall bias. That is, emerging adults only remember the most recent instance of NSSI and fail to recollect earlier episodes that occurred during their adolescent years. Apart from the recall bias, a possibility of a "spill-over" of adolescent psychopathology has also been raised [32]. That is, any form of developmental issues (e.g., disturbances in identity formation) or mental illness during adolescence can present itself in form of a secondary psychopathology, like NSSI during emerging adulthood. The effect of adolescent mental health issues may get expressed more potently as most of these psychiatric disorders rarely get treated [33]. Developmentally important issues like disturbances in identity formation may also help in

explaining the observed bi-modal distribution of onset of NSSI. The process of identity formation is one of the key concerns in both adolescence and emerging adulthood [34, 35]. In individuals with significant identity disturbances, NSSI may serve as a means to either develop a new sense of self (self-injurer, self-cutter, etc.) or as a means of managing negative affect associated with identity disturbances [36]. Interestingly, association between identity disturbances and NSSI has been demonstrated to be stronger in patients with EDs and BPD [37].

Finally, comparative analysis of onset of internalizing disorders and NSSI highlights the fact that gender differences observed in NSSI are also observed in depression and anxiety. A surge in female gonadal hormones may play a role in the development of depression, anxiety, and possibly also in NSSI. This assertion is partly supported by the fact that the onset of these disorders closely follows the average age of menarche in Flemish and Dutch female adolescents (i.e., 13.13 ± 1.3 years) [38, 39]. There is increasing evidence to suggest that in females, estrogen and progesterone may modulate mood through their influence on GABA, 5-HT and/or dopamine systems – the neurotransmitters implicated in depression and anxiety [40]. Further research should also consider researching the influence of gonadal hormones on NSSI behavior in females through their ability to influence mood. Overall, the distribution of age of onset of NSSI in community and clinical samples seems to be an outcome of a complex interaction of biological and psychosocial processes.

Although the present study was one of the first to systematically investigate the distribution of age of onset of NSSI and the effect of gender and presence of psychiatric disorders on the first episode of NSSI, it is plausible that a number of limitations may have influenced the results obtained. First, whereas the pooling of datasets considerably increased the overall sample size, the individual datasets in the present study were collected by means of convenience sampling. Therefore, the resulting findings may not be representative of the general Dutch-speaking population. Second, information regarding the

distribution of age of onset of NSSI was based on retrospectively collected data. Due to the limitations in the way autobiographical memories are accessed, the use of self-report questionnaires to investigate the age of onset of NSSI may not be sufficient to develop an accurate picture of the age of onset of NSSI [41]. Long-term longitudinal studies may be required to confirm our findings. Given that such studies can be expensive and difficult to conduct, researchers can consider using tools like event history calendar (see [42]) as they may aid in forming a more accurate picture of retrospective data like age of onset of NSSI. Third, from Table 1, it is clear that presence/absence of NSSI was investigated either by using a single item with a forced Yes/No response or a checklist of self-harming behaviors. Although the use of different assessment methods does not lead to epidemiological inconsistencies in the estimation of the prevalence of NSSI [2] and its age of onset, yet, some measurement error may be inevitably introduced in the analyses. Interview techniques like clinically informed event history calendar can again assist in developing an accurate picture of epidemiology of NSSI. Fourth, the current study probabilistically modelled the age of onset of NSSI. Further research exploring the association between the age of onset other features of NSSI like frequency, severity, and methods used to harm oneself is also required. Fifth, the bimodal distribution of age of onset, which has been observed in many psychiatric disorders [27-31], was also evident in NSSI. Whereas the first peak in the onset of NSSI was observed around the age of 14-15 years of age in both community and clinical samples, it is not clear why the second peak observed in the age of onset of NSSI was observed earlier in the community samples (20 years) and later in the clinical samples (24 years). Further research may be necessary to understand the theoretical and clinical significance of the aforementioned finding. Finally, as mentioned earlier, interpretation of the hazard probabilities towards the tail end of the hazard function should be interpreted with caution as these probabilities may be over-inflated because of the drop in the overall number cases in the denominator.

In spite of these limitations, the current study is one of the first to probabilistically model the distribution of age of onset of NSSI. Our findings re-iterate the fact that NSSI is highly prevalent in the Dutch-speaking countries. The present study also highlights the fact that mental interventions targeting adolescents in schools may not be sufficient. From a public health perspective, the early period of emerging adulthood may also be seen as an important age bracket to address untreated mental health issues that originate during childhood and/or adolescence. Consequently, universities, technical schools, or other higher educational institutes can serve as gateways to identify and treat individuals with a higher risk of late onset of NSSI engagement.

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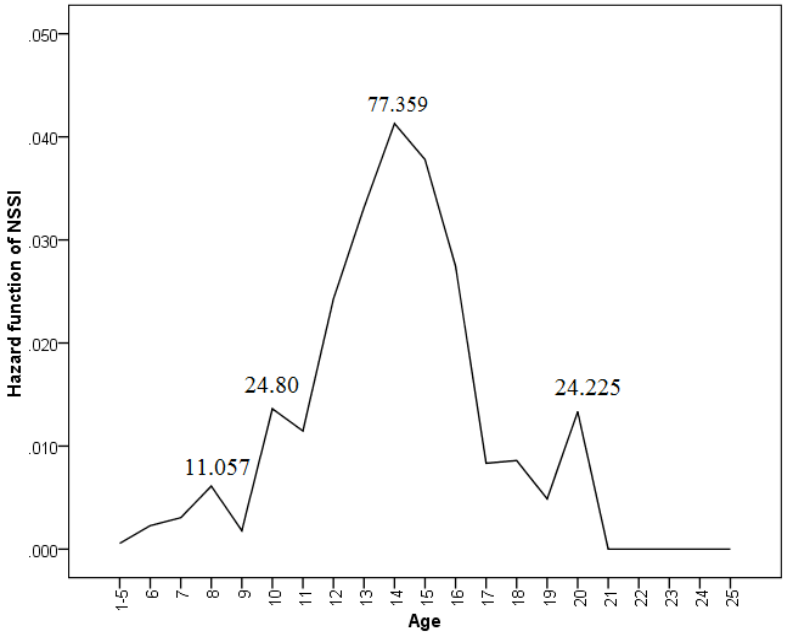
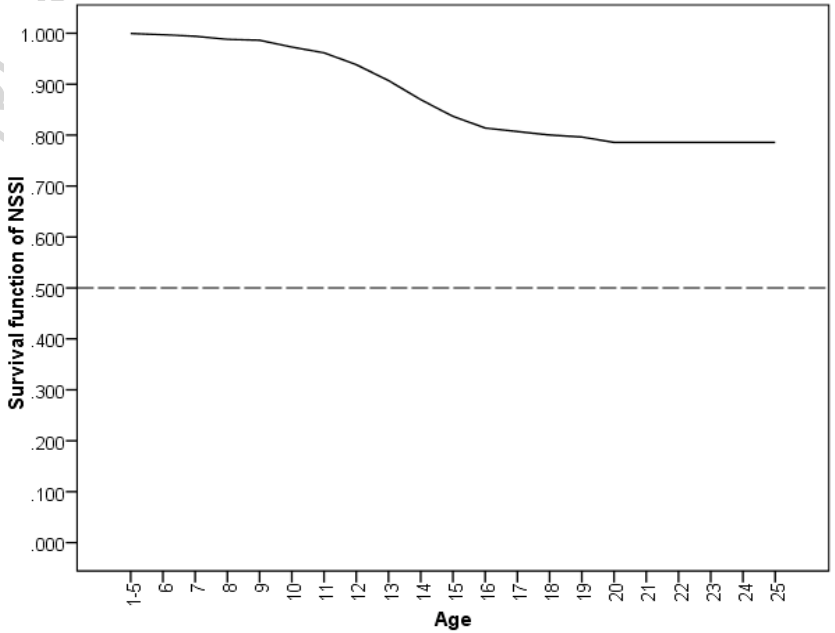
Figure 1. General hazard (Panel A) and survivor function (Panel B) for the age of onset of NSSI (in years) in adolescents and emerging adults. Numbers in the Panel A represents the odds of engaging in NSSI as compared to the reference category (1-5 years). The logistic regression used to obtain these odds is available as online appendix. Panel C shows the hazard and Panel D shows the survivor function for the age of onset of NSSI taking into account the influence of gender. The probabilities used to plot the survival function are available in the online appendix accompanying this manuscript.

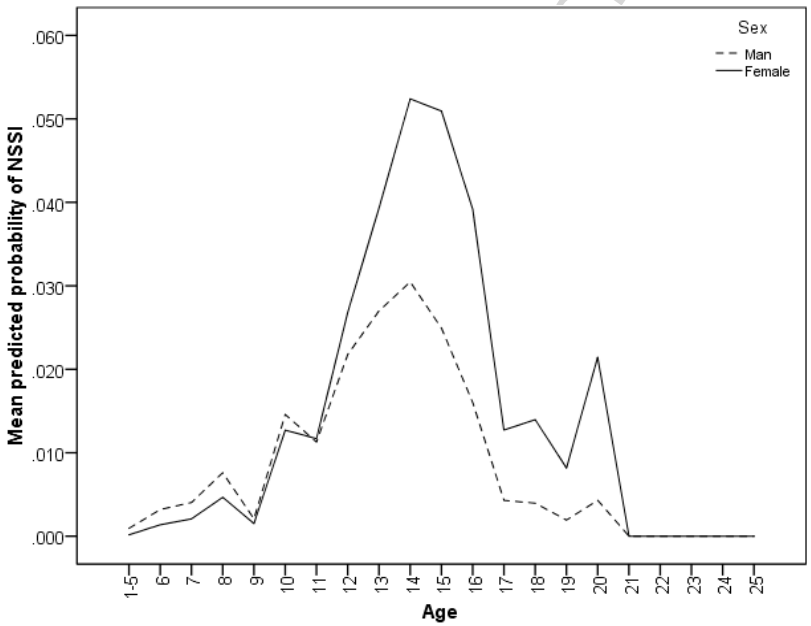
A.



B.



C.



D.

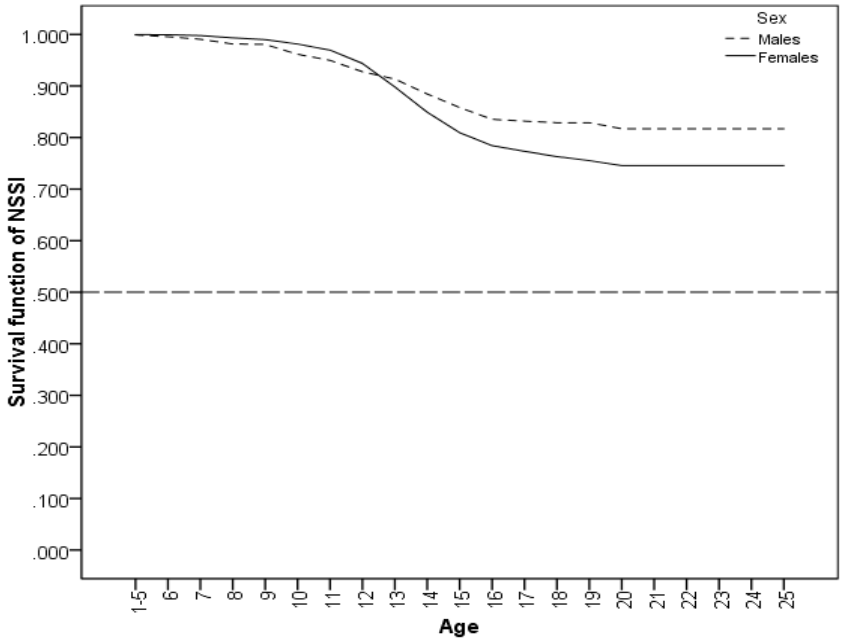
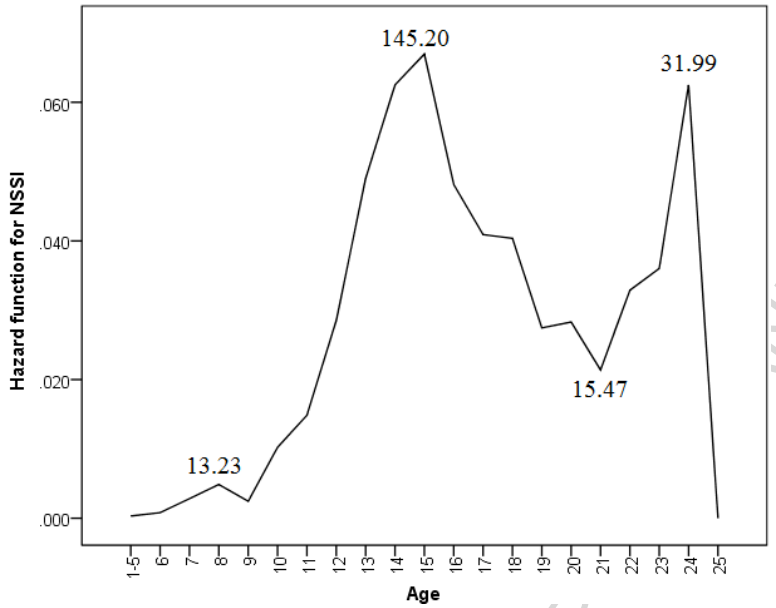


Figure 2. General hazard (Panel A) and survivor function (Panel B) for the age of onset of NSSI (in years) in females with and without psychiatric disorders. Panel C shows the hazard and Panel D shows the survivor function for the age of onset of NSSI taking into account the presence of psychiatric disorders. The probabilities used to plot the survival functions in panel D of the figure 2 are available in the online appendix accompanying this manuscript.

A.



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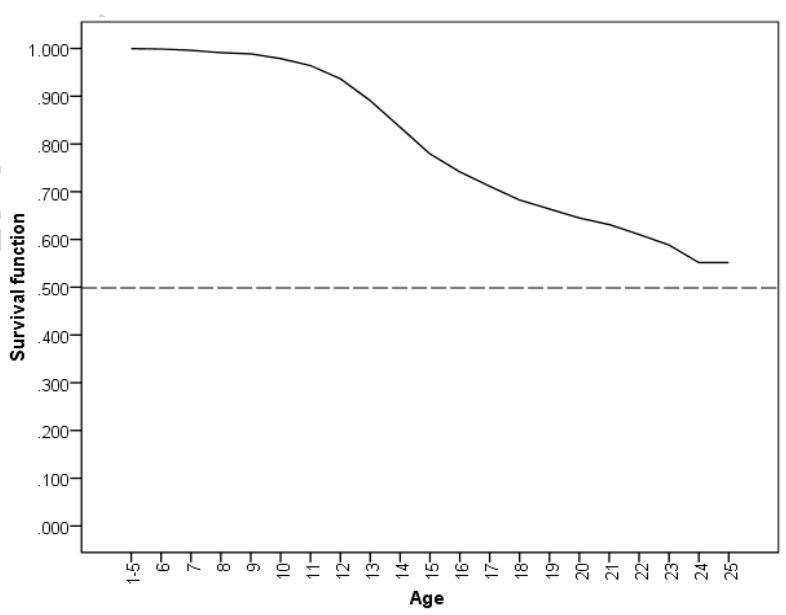

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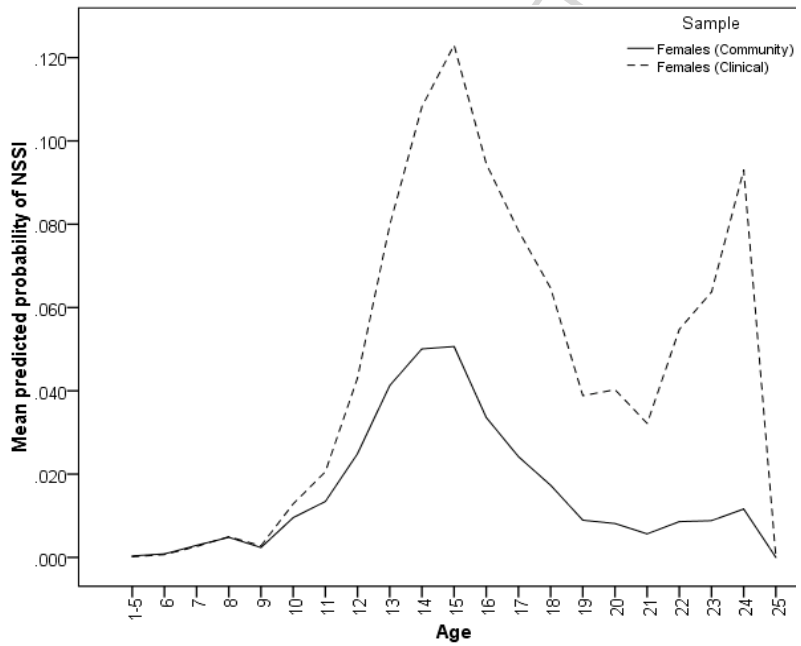

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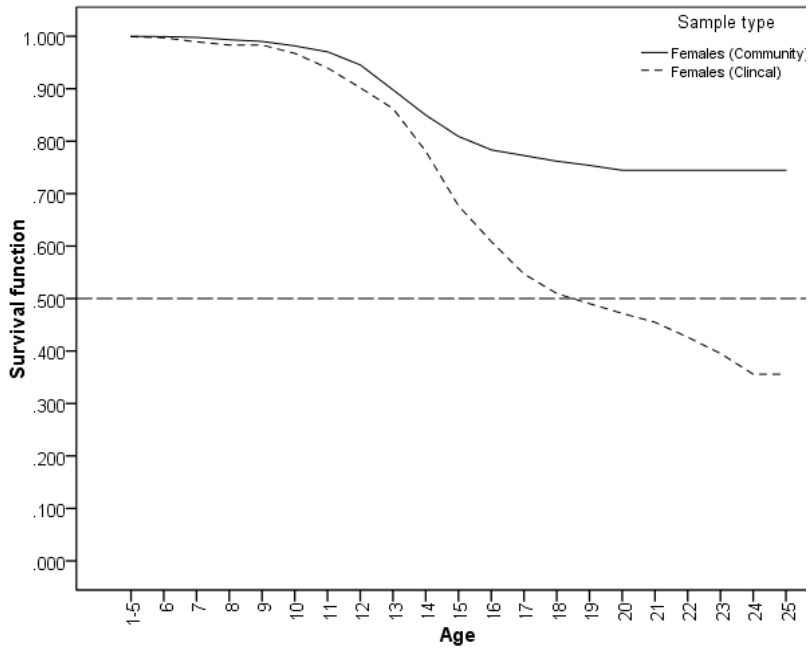

Table 1. Details of the datasets included the current study. For more information regarding the datasets and the procedure adopted to collect the data can be found in the referenced manuscripts[1].

	Study	Population type	Year of data collection	*N:* Original dataset (NSSI cases)			Cases selected for analysis (Cases of NSSI)			Assessment of NSSI
				Males	Females	Total	Males	Females	Total	
1	Dataset 1 [43]	Community	2011	166 (20)	192 (28)	358 (48)	164 (19)	160 (27)	324 (46)	Checklist
2	Dataset 2 [44]	Community	2012	395 (95)	137 (26)	532 (121)	395 (95)	136 (25)	531 (120)	Single-item
3	Dataset 3*	Community	2012	246 (39)	375 (68)	651 (107)	240 (25)	294 (62)	534 (87)	Single-item
4	Dataset 4 [45]	Community	2012	511 (125)	408 (99)	1013 (224)	451 (81)	372 (74)	823 (155)	Checklist
5	Dataset 5*	Community	2013	163 (8)	218 (7)	397 (15)	55 (2)	68 (5)	123 (7)	Single-item
6	Dataset 6 [46]	Community	2014	220 (26)	447 (157)	705 (183)	215 (21)	406 (109)	621 (130)	Single-item
7	Dataset 7 [47]	Community	2015	194 (23)	207 (43)	401 (66)	192 (21)	202 (40)	394 (61)	Single-item
8	Dataset 8 [35]	Community	2015	261 (20)	267 (55)	528 (75)	261 (20)	263 (52)	524 (72)	Single-item
Total							1973 (284)	1901 (394)	3874 (678)	
9	Dataset 9# [48]	Clinical (ED)	2002-2005	0	127 (53)	127 (53)	0	94 (47)	94 (47)	Single-item
10	Dataset 10# [49]	Clinical (BPD)	2002-2009	0	488 (209)	488 (209)	0	361 (172)	361 (172)	Single-item
11	Dataset 11# [50]	Clinical (ED)	2013-2016	0	140 (111)	140 (111)	0	50 (45)	50 (45)	Single-item
Total							0	505 (264)	505 (264)	

[1] The data collection procedure for the published datasets can be accessed from the referenced manuscript.

* The data collection procedure for the published datasets community datasets were almost similar to the procedure described by Gandhi and colleagues (35)

In case of the clinical samples, the sample sizes mentioned in the present study may differ from the sample size mentioned in the corresponding manuscripts. This is because the data were collected in psychiatric units for more than a single study (each with proper ethics approval). Therefore, the manuscripts referred in the table above included patients who only had data available on the relevant variables.

Table 2. Goodness-of-fit and information criteria of models with main effect and interaction effect of gender

Model	Model Specification	-2LL	*df*	DID#	Δ*df*	$\chi^2_{(critical\ .05)}$	***AIC***	***BIC***
1	General specification	6500.90	20	-	-	-	6532.90	6677.07
2	General specification + Sex	6480.16	21	20.74	1	3.84	6514.16	6667.32
3	General specification + Sex + Age(Linear) * Sex	6444.25	22	56.65	2	5.99	6480.45	6642.42
4	General specification + Sex + Age(Categorical) * Sex	6408.31	41	92.59	21	32.67	6470.31	6749.53

Difference in Deviance

Table 3. Goodness-of-fit and information criteria of models with main effect and interaction effect of presence or absence of psychiatric disorders.

Model no	Model Specification	-2LL	*df*	DID[#]	Δ*df*	$\chi^2_{(critical\ .05)}$	AIC	BIC
1	General specification	5592.48	20	Reference		-	5632.48	5803.95
2	General specification + Population type	5503.14	21	89.34	1	3.84	5545.14	5725.18
3	General specification + Population type + Age(Linear)* Population type	5486.92	22	105.56	2	5.99	5530.92	5719.52
4	General specification + Population type + Age(Categorical)* Population type	5446.26	41	146.22	21	32.67	5516.26	5815.67

Difference in Deviance