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**THE ROLE OF STRESSFUL LIFE EVENTS EXPERIENCED DURING
CHILDHOOD AND ADOLESCENCE IN THE DEVELOPMENT OF
MAJOR DEPRESSION**

Research paper

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Running heading: Stressful life events and depression

The role of stressful life events experienced during childhood and adolescence in the development of major depression

Abstract

Despite the fact that the role of stressful life events (SLE) in the development of major depression (MD) has been much studied, there are a few gaps in the literature. Little of the research has used longitudinal and, in particular, population representative samples, and examined the significance of both the total accumulation of events and the variety of events. This study investigated the potential role of up to 26 SLE-s recorded at age 18 in the occurrence of lifetime major depression as measured in a psychiatric interview conducted at age 25. I have used data of the longitudinal Estonian Children Personality Behaviour and Health Study, comprising two birth cohorts. Assessment of the total number of SLE-s revealed that experience of even only one traumatic event can increase the risk to meet criteria of lifetime diagnosis of major depression (OR=1.43, 95% CIs [1.25, 1.64] $p < .001$). The strongest association between MD with specific SLEs (ORs ranged from 2 to 4.5) was found for persistent severe worrying, accidents and traumas, poor or absent relationship with a separately living parent, suicidal attempts, suicidal behaviour and depression of a close relative. I also found that the greatest impact across abuse related SLE-s was elicited by emotional abuse in the family. Chronic and episodic SLE-s had independent effects on major depression, and there was no moderation effect between them. The impact of chronic SLEs on major depression was slightly larger compared to episodic events. I did not find any evidence for the association between SLE-s and MD for the group of events "Loss and parental separation". Neither could gender differences across both cohorts and all event groups be demonstrated.

Keywords: stressful life events, major depression, child development, gender, longitudinal study

Lapsepõlves ja noorukieas kogetud stressirohkete elusündmuste roll kliinilise depressiooni väljakujunemisel

Lühikokkuvõte

Vaatamata sellele, et stressirohkete elusündmuste rolli kliinilise depressiooni väljakujunemisel on palju uuritud, on antud teemat käsitlevas kirjanduses mõned lüngad. Vähesed uuringud on olnud longituudsed, tuginenud rahvastiku suhtes esindulikule valimile ning uurinud nii kõikide sündmuste akumulatsiooni kui ka nende erinevate variantide olulisust. Käesolevas töös uuritakse kuni 26 uuritavate 18-aastasena kirja pandud erineva stressirohke elusündmuse potentsiaalset rolli kliinilise depressiooni tekkele senise elu jooksul. Uurimismeetodiks on siinkohal psühhiaatriline intervjuu, mis viidi uuritavatega läbi 25-aastasena. Olen kasutanud longituudse ja kahest sünnikohordist koosneva Eesti Laste Isiksuse, Käitumise ja Tervise Uuringu andmeid. Stressirohkete elusündmuste koguarvu analüüs on näidanud, et isegi ühe traumaatilise sündmuse kogemine suurendab kliinilise depressiooni tekke riski (OR=1.43, 95% CIs [1.25, 1.64] $p < .001$). Tugev seos kliinilise depressiooni ja stressirohkete elusündmuste vahel (ORs vahemikus 2 kuni 4.5) leiti pideva ja tõsise muretsemise, õnnetuste ja traumade osas, halbade või puuduvate suhete puhul endast eraldielava vanemaga, enesetapukatsete ning lähedase sugulase suitsiidaalse käitumise ja depressiooni korral. Samuti avastasin, et suurim mõju väärkohtlemisega seotud sündmuste seas oli vaimsel vägivallal perekonnas. Kroonilistel ja episoodilistel stressirohketel elusündmustel oli kliinilisele depressioonile iseseisev mõju, ja nende vahel ei esinenud koosmõju depressioonile. Krooniliste stressirohkete elusündmuste mõju kliinilisele depressioonile oli kergelt suurem võrreldes episoodiliste sündmustega. Sündmuste grupi „kaotus ja vanemate lahkumine“ osas ei leidnud ma seost kliinilise depressiooniga. Samuti ei leidnud ma soolisi erinevusi kummaski kohordis ega ühegi sündmuste grupi osas.

Võtmesõnad: stressirohkeid elusündmused, kliiniline depressioon, lapse areng, sugu, longituudne uuring

Depression is the leading cause of disability worldwide, and is a major contributor to the global burden of disease (impact of health problems measured using the disability-adjusted-life-years). The median age of first onset of major depression is in the early-to-mid twenties, with risk being fairly low in the early years of life, rising during adolescence and through the middle-to-late twenties, and then decreasing in later years. The average age of onset of the condition appears to be decreasing (Andrade 2003). At its worst, depression can lead to suicide. The link between suicide and mental disorders, in particular depression, is well established. Suicide was the second leading cause of death among 15–29-year-olds globally in 2012 (World Health Organization). These facts underline the importance of studying the risk of mental disorders and particularly major depression in adolescents and young adults.

The World Health Organization (WHO) defines an adolescent as any person between ages 10 and 19. Adolescence is an important period of transition between childhood and adulthood. It is characterized by significant changes in the brain, hormonal secretion, and physical, cognitive, and socio-emotional development (Evans & Seligman, 2005). Adolescents who are cognitively unprepared for continual physical, psychological and social changes may face difficulties; hence adolescence may be a particularly stressful period. Andersen and Teicher (2008) found in their review that stress exposure during the development of the hippocampus and pre-frontal cortex in this developmental period may make adolescents more prone to depression.

Indeed stress has been studied intensively as an important risk factor for depression. Clear empirical link exists between stress and depression in both adults (Paykel, 2003, Hammen, 2005;) and adolescents (Ge et al., 1994; Adkins et al., 2009; Stroud et al., 2011, Vitiello, 2011). Despite the fact that stress is one of the most widely studied concepts in psychology, it is still difficult to give clear definition of stress. This lead to a variety of definitions of stress, with focus on different aspects. One of the most comprehensive models is the Biopsychosocial Model of Stress (Bernard & Krupat, 1994). According to this model, stress involves three components: an external (environmental conditions, stressors), an internal (neurological and physiological reactions to stress, state of the organism, strain) and the interaction between the external and internal components. The most widely used method for assessing stressful life experiences among children and adolescents is the self-report checklist, and most of these checklists are consistent with conceptualizations of environmental stressors (Grant et al., 2003). Grant and colleagues (2003) define stress as

"Environmental events or chronic conditions that objectively threaten the physical and/or psychological health or well-being of individuals of a particular age in a particular society".

Many studies have found an association between stressful life events and depressive disorders, including symptom severity, response to treatment, duration of episodes and recurrence (Ge et al., 1994; Hammen, 2005; Adkins et al., 2009, Monroe & Reid, 2009; Stroud et al., 2011). There are some differences among child-, adolescent-, and young-adult-onset depressions in the context of childhood and adolescence adversity, but generally experience of SLE significantly increases the risk of early-onset of depression (Jaffee et al., 2002; Hill et al., 2004; Shanahan et al., 2011).

Divergent methods have been used to assess SLE as leading to depression: total number of events experienced, event groups and specific individual events have been examined separately or together by different authors.

Although people are likely to experience a wide range of SLEs, the probability of occurrence of any specific SLE in an individual is likely to be low, making it difficult in most cases to interpret the role of single SLE. As a result, researchers tend to analyze cumulative scores derived from a SLE checklist to examine exposure of a large number of SLE-s. The association between aggregated stressful life events (regardless of types of stressors included in checklist) and depression among adolescents and young adults have been found in samples from Western (Burton, Stice, & Seeley, 2004; Rudolph, Flynn, Abaied, Groot, & Thompson, 2009; Slopen et al. 2011; Zhang, 2013) and Eastern societies (Ng & Hurry, 2011).

Kessler and colleagues (1997) found that the association of specific events with the onset of mood and anxiety disorders is diminished when controlling for other types of SLE. Kessler et al. (1997) and McCutcheon et al. (2010) suggest that the grouping of different types of SLE, rather than examining the unique effects of individual events, is an appropriate way of investigation of associations between childhood and adolescence adversity, and depression.

There are different approaches to grouping SLE-s, based either on their domain (school, family, peer and romantic relationships, sociodemographic conditions, health, natural disasters etc) or on the nature of stress (dependent - caused or worsened by individual own choices, thoughts, actions, etc; independent, personal and interpersonal events, episodic and chronic stressors). Family related or intrafamilial events is the most frequent group used in studies of SLE in children and adolescents. Developmental theories view adolescence as a period when young people begin to think for themselves and question aspects of their lives and of family relationships. Relationship between child and parents is becoming more equal,

the role of family is lessening, peer relationships becomes more central (Noller, Feeney, & Petersen, 2001). Despite of changing role of family during adolescence, family environment still plays one of the most important roles in the psychosocial development of adolescents. For example parents and family becomes a "secure base" from which to explore peer relationships and different roles and identities. Positive family environment during adolescence has been described as a predictive factor of successfully adaptive adult functioning across several domains, including social functioning and protection against major depression (Waldinger, Vaillant & Orav, 2007).

Loss events are also very often examined in studies of SLE in children and adolescents. Although loss events are usually investigated in the family context (loss of parents and close relatives), some researchers form a separate group for these events. This seems to be justified owing to the strong associations between loss events (especially parental death) and the risk for onset of depression (Mayer et al., 2009).

The association between childhood abuse and depression has also been investigated repeatedly, but there are a few gaps in literature. The majority of studies has examined effects of physical and sexual abuse, with fewer studies focusing on the role of emotional abuse (Alloy, Abramson, Smith, Gibb & Neeren, 2006; Liu, Alloy, Abramson, Iacoviello & Whitehouse, 2009). Emotional maltreatment may appear in many forms – a physically and/or emotionally uninvolved parent; parents who constantly bicker, yell, undermine and fight with each other in front of the child; perfectionist parents with unreasonably demanding expectations and critical observations. The source of emotional abuse can also exist outside the family (other adults, peers). In any case emotional abuse is generally characterized by repeated destructive actions: spurning, terrorizing, isolating, ignoring, exploiting, corrupting, etc (Brassard & Donovan, 2006). Finkelhor, Ormrod, & Turner (2007) found that different types of abuse often occur together. They concluded that future research should examine childhood abuse by taking into consideration the possible effects of different types of abuse, especially if they overlap. Indeed, review of the maltreatment literature has shown that the different forms of abuse are rarely examined in the same study with statistical controls for their overlap. So, for example, it is unclear whether reports of a significant association between childhood sexual abuse and depression is truly due to the sexual abuse or whether it is due to the co-occurrence of emotional abuse with the sexual abuse (Alloy et al., 2006). Shapero and colleagues (2014) have suggested that childhood emotional abuse may be particularly maladaptive, highlighting the need for more research targeted at emotional abuse specifically within a longitudinal framework.

Based on the nature of stress and its duration, SLEs can be divided on episodic and chronic SLEs. Episodic SLEs - major incidents that have clearly delineated time represents (parental divorce, death of parent, trauma etc.). Chronic SLE - persistent difficult and demanding experiences in daily life (poverty, health problems, parental alcoholism etc) (Evans & Cohen, 1987, 574). Many SLE-depression studies using questionnaires and checklists have not clearly distinguished between the effects of chronic and episodic negative events (Caspi et al., 2003). Such an approach makes it difficult to fully explain mechanisms by which stressors impact on development of depression. There is a great amount of research on the effect of chronic stressors in relation to depression, but this is typically focused on a single domain (chronic illness or illness of a close family member, parent-child tensions, social-economic conditions etc) (Hammen, 2009). With such a limited focus the impact of all aspects of chronic stress across different areas of a person's life cannot be demonstrated. Until today it is not clear whether and to what extent the chronic and episodic SLE have independent associations with depression. Joint effects of chronic and episodic SLE also have not been sufficiently evaluated.

However, investigation of separate groups of SLE also seems reasonable and is justified in many cases, the role of single traumatic events should not be underestimated. Aggregating the stressful life events into a cumulative score creates a problem because it is often impossible to determine whether the harmful effects of stress involve a broad span of events or whether it is limited to a few types of events. The effect of SLEs is dose-dependent, but in some cases people, who become depressed, report that just one traumatic event had happened prior to they became depressed. Traumatic experiences that cause a high degree of threat and unpleasantness, such as the loss of a loved one, divorce, or serious health problem may be so affecting that they can serve as a triggering mechanism in predisposed individuals, resulting in the development of depressive disorder (Paykel, 2003; Hammen, 2005). Less traumatic single events can trigger recurrence of depression in people with lifetime history of depression. People who experience one SLE often experience several types of trauma (Breslau et al., 1998), but the fact, that depression may occur just after specific SLE, demonstrates the importance of research on single traumatic experiences. As stated in the previous paragraphs exploring of aggregated SLE and specific groups of SLE is a more appropriate way of research, however it is difficult to form such groups and analyze the results without prior knowledge of relative roles of individual events. Thus research should include complex analysis of aggregated and individual SLEs.

Researchers have also explored whether the association between SLE and depression is moderated by gender. Epidemiologic data from around the world indicate that compared to men, women are approximately twice as likely to develop depression (Nolen-Hoeksema, 2001). Gender differences begin to emerge during early adolescence and rise dramatically during middle adolescence, reaching the rates of adult population during late adolescence (Mezulis, Funasaki, Charbonneau & Hyde, 2010). Two models have been proposed to describe how stressors may explain gender differences in depression. The mediational stress exposure model postulates that girls experience more stressors than boys, and as a result, girls become more depressed. The results of numerous studies in adolescents provide empirical evidence for this model (Ge et al., 1994; Rudolph 2002; Hankin, Mermelstein & Roesch, 2007). The moderational stress reactivity model suggests that girls experience greater levels of depression than boys in response to equivalent levels of stress (Hankin et al. 2007). This model states that girls do not necessarily experience more stressors than boys, however girls are affected more than boys by the same stressors. This model has also been tested extensively, but findings are mixed. Some authors have found support to the notion that girls respond to stressors in a more depressogenic manner than boys (Achenbach, Howell, McConaughy & Stanger, 1995; Hankin et al. 2007; Calvete et al., 2011), while others have not found gender differences consistent with the moderational model (Larson & Ham, 1993; Leadbeater, Kuperminc, Herzog, & Blatt, 1999). Empirical evidence for both models suggests that gender differences do not emerge equally with all types of stressors. Girls experience more interpersonal stressors, which lead to a greater level of internalizing symptoms and, as a result, girls respond more often with depression to this type of stressors. Few authors (Hankin 2007; Telzer & Fuligni, 2011) have tested both models in one study in terms of the specific domains of stressors, and only Hankin (2007) found empirical support for both models. This work has suggested that the two models are not mutually exclusive and both may help to explain the emergence of gender differences in depression during adolescence.

Some Estonian authors also have investigated the relationship between SLE and depressiveness in adolescents, but these studies focused on specific events rather than on a wider range of SLE. The role of health problems, family relation variables, family structure, economic deprivation, bullying, fighting and the age of first sexual intercourse were explored (Samm et al., 2010; Heidmets et al., 2010; Mark et al., 2012). An advantage of these studies has been the use of large samples (up to 4389 participants), but all these studies are cross-sectional and self-report questionnaires were used to measure depressive symptoms. Self-

report questionnaires are easy to administer and can therefore be used in large community samples. Assessment of clinically significant depression is more resource demanding, because it should be conducted by a qualified practitioner trained in making the psychiatric diagnosis. Although most self-report questionnaires are validated using results of structured clinical interviews, clinical assessment of depression is considered the “gold standard” procedure (Steel, Dunlavy, Stillman & Pape, 2011).

In summary, stressful life events experienced during childhood and adolescence have consistently been associated with an increased risk for major depression in both adults and adolescents. Despite the fact that the role of SLE in the development of depressive disorder is well-studied, there are a few gaps in current literature. Up to date there are no studies which, simultaneously, were longitudinal, used population representative sample, clinical interview for assessment of major depression and examined the impact of total accumulation of events, various event groups and single events.

The current study has four goals:

- 1) to examine whether, and to what extent aggregated stressful life events are associated with lifetime diagnosis of major depression in a population-representative Estonian sample;
- 2) to examine which groups of stressful life events are associated with lifetime diagnosis of major depression;
- 3) to examine which single stressful life events are associated with lifetime diagnosis of major depression;
- 4) to investigate gender differences in the relationship between SLE and lifetime diagnosis of major depression.

Method

Sample

This study was carried out on the Estonian sample of the European Youth Heart Study (1998/99), which was subsequently incorporated into the longitudinal Estonian Children Personality Behavior and Health Study (ECPBHS). The rationale and procedure of sample formation have been described elsewhere (Harro et al., 2001, 2009; Laas et al. 2014). In brief, all schools of Tartu County, Estonia, that agreed to participate (54 of the total of 56) were included in the sampling, using the probability proportional to the number of students of the respective age groups in the school, and 25 schools were selected. In 1998–1999, all children from Grades 3 and 9 were invited to participate, and written informed consent was

received from 79% of the invited subjects and their parents. The total number of subjects in this sampling was 1,176, including 593 in the younger cohort (YC) and 583 in the older cohort (OC). The data for the present study were collected during the follow-ups of the cohorts. The follow-up studies for the younger cohort were in 2007 ($n=453$; $M_{Age}=18.3\pm 0.5$) and in 2014 ($n=437$; $M_{Age}=24.8\pm 0.6$). The follow-up studies for the older cohort were in 2001 ($n=479$, $M_{Age}=18.4\pm 0.7$) and 2008 ($n=541$, $M_{Age}=24.7\pm 0.7$). The present analysis is using data on SLE which was collected when participants were 18 years old and data on prevalence of lifetime major depression, when participants were 25 years old. Due to missing clinical information on depression or missing data on SLE in either follow-ups, the number of subjects in this analysis are $n=363$ (156 males and 207 females) for the younger cohort and $n=370$ (168 males, 212 females) for the older cohort. Combined data (total sample) of both cohorts was also formed and separately examined ($n=733$, 324 males, 419 females).

All participants were Caucasians. Participants and, in case of minor, their parents gave informed consent in all study waves, and the study procedure was approved by the Committee of Ethics of the University of Tartu, Estonia.

Assessment of Major Depression

Psychiatric assessment based on DSM-IV was carried out in both cohorts at age 25 by experienced clinical psychologists using the Mini-International Neuropsychiatric Interview - M.I.N.I.5.0.0 (Sheehan et al., 1998) adapted to use in Estonia at the Department of Psychiatry, University of Tartu (Shlik, Aluoja & Kihl, 1999). I have used lifetime prevalence of major depression in the current work.

Stressful Life Events

The history of stressful life events (SLE) was self-reported. The list of adverse life events varied across cohorts, but consisted up to 26 stressful experiences reported either present or absent. The list of stressful life events for both cohorts is shown in Appendix A.

The events were recorded as dichotomous variables. The events were then counted to form the cumulative score of experienced stressful life events. Events were also organized by groups on the basis of domain or duration of the stress: intrafamiliar, loss event and parental separation, violence and abuse, chronic and episodic SLE (Appendix A). Cumulative scoring was also used for groups.

There were not enough events to form a groups of events "Loss and parental separation" and "Violence and abuse" in the younger cohort, so these were analyzed only in

the older cohort. Violence and abuse events were divided into 4 groups. First group included all abuse events (physical, sexual and emotional abuse - PSEA) and the other three groups included combinations of abuse events: emotional and sexual abuse (SEA), physical and emotional abuse (PEA) and physical and sexual abuse (PSA). I have used a general definition "sexual abuse" for two types of stressors: rape attempt and sexual harassment.

Based on duration of stress, SLEs were also divided into episodic and chronic SLEs. I excluded from this analysis events of "risky sexual behaviour" and "sexual abuse " because it is difficult to decide to which group these events belong.

Statistical Analysis

Statistical analysis was performed for each cohort separately and also for the combined data of both cohorts - Total Sample (TS). One-way analysis of variance (ANOVA) with post-hoc Tukey test and the independent t-test were utilized in the statistical analysis of the mean number of SLE-s. Fisher's exact test and odds ratios were used in order to investigate gender differences in exposure to SLE. Logistic regression was used to investigate the associations between gender, depression and number of stressful life events. I fitted logistic regression models for major depression, that included gender, the cumulative score of SLE, and the gender and SLE score interaction. Using the regression coefficients from the model that included gender and the cumulative score of SLE, I estimated odds ratios for the main effects (a 95% confidence interval, CI). Using the regression coefficients from the model that included additionally gender and SLE score interaction, I estimated gender-specific odds ratios. Cumulative score was fitted models as a continuous variable, but participants with 5 and more SLEs were grouped into a single category, due to sparseness of data at high values. Participants with 3 and more events were grouped into a single category in group "Chronic SLEs", with 2 and more events in groups "Intrafamilial" and "Violence and Abuse", "Episodic SLEs", with 1 and more events in "Loss event and parental separation".

Cumulative score was also fitted models as a categorical variable, in order to analyze magnitude of SLE and to test for possible threshold effects. To check for overlap between different types of abuse, I have run separate model, which included all individual abuse events and gender. Simultaneous entry of variables was used for this model.

To check for interaction effect between chronic and episodic SLEs I have run logistic regression model which included both groups, their interaction and gender.

To investigate the impact of individual events I have run logistic regression model for each event and compared results of cohorts and total sample.

All models were assessed by the Homer-Lemeshow goodness-of-fit test, -2 Log likelihood, Cox & Snell R Square and Nagelkerke R Square tests. A p value < .05 was considered statistically significant. Statistical analyses were performed using SPSS 21.0.

Results

Prevalence of Major Depression and Stressful Life Events

The rate of lifetime prevalence of major depression for the younger cohort was 26.7 % (21.1%, 33 cases, for males, and 30.9%, 64 cases for females) and 21.9 % for older cohort (n=370, 10.1%, 16 cases for males and 30.6%, 65 cases, for females). The lifetime prevalence rate for the older cohort was similar to comparable epidemiological studies (around 20 %), the rate for younger cohort was somewhat higher (de Graaf et al., 2012; Shim, Baltrus, Ye & Rust, 2011). The odds ratios of major depression were higher for females compared to males (the older cohort OR= 3.92, CIs [2.17;7.1], $p < .001$; the younger cohort OR= 1.67, CIs [1.03; 2.7], $p = .038$; total sample OR=2.406 [1.67; 3.48], $p < .001$).

There was a statistically significant difference between four groups (males and females with and without episode of MD) in the older cohort as determined by one-way ANOVA ($F(3, 366) = 7.26, p < .001$). A Tukey post-hoc test revealed that the mean number of stressful life events for females with an episode of MD (3.84 ± 3.04 min) was statistically significantly higher compared to females without MD (2.24 ± 2.36 min, $p < .001$) and males without MD (2.4 ± 2 min, $p < .001$). There were no statistically significant differences between other compared groups.

There was a statistically significant difference between groups in the younger cohort as determined by one-way ANOVA ($F(3, 359) = 5.86, p < .001$). A Tukey post-hoc test revealed that the mean number of SLEs for females without an episode of MD (1.22 ± 1.51 min) was statistically significantly lower compared to females with MD (1.83 ± 1.4 min, $p = .18$) and males with MD (2.15 ± 1.23 min, $p = .003$). The mean number of SLEs for males without an episode of MD (1.41 ± 1.19 min, $p = .29$) was statistically significantly lower compared to males with MD. There were no statistically significant differences between other compared groups.

The independent t-test revealed that the mean number of SLEs was not statistically significant different between males and females in both cohorts: the older cohort, males (2.73 ± 2.68); females (2.43 ± 2.04); $t(368) = 1.177, p = .24$; the younger cohort, males (1.56 ± 1.23); females (1.41 ± 1.49); $t(361) = -1.041, p = .29$. The independent t-test revealed that

the mean number of SLEs was statistically significant different between older cohort (2.6 ± 2.43) and younger cohort (1.48 ± 1.39), $t(731) = 7.673$, $p < .001$. The differences between cohorts possibly appeared because of different number of events included in SLE questionnaires and used for the current analysis: 14 for younger cohort and 20 older cohort. To see, if there is any difference between cohorts if same events were examined, I compared the mean number of 14 stressful life events included in both cohorts. The mean number of 14 SLE-s was not statistically significant different between the older cohort (1.42 ± 1.51) and the younger cohort (1.38 ± 1.29) $t(731) = 0.426$, $p = 0.671$.

The prevalence of stressful life events with rates of exposure to each event is presented in Table 1. Gender differences in exposure to specific events is presented as odds ratios (females compared to males). In both cohorts and the total sample similar statistically significant differences between males and females occurred in exposure to "physical abuse elsewhere", "accidents and traumas" (higher ORs for males) and "persistent severe worrying" (higher ORs for females). Rates of exposure to "suicidal attempts" between males and females were significantly different in the older cohort and total sample (higher ORs for females). In the older cohort (events are not included in the younger cohort's questionnaire) statistically significant differences occurred for "risky sexual behaviour" (higher ORs for males) and "sexual harassment, excluding family". "Sexual harassment, excluding family" OR was 16.2 CIs [3.84; 68.41], $p < .001$ with only 2 cases among men and 36 cases among women. Gender differences for the events "poor living conditions" (OR for females = 2.519; CIs [1.12; 5.64], $p = .021$) and "parental alcoholism" (OR= 3.83, CIs [1.1; 13.34], $p = .028$) became statistically significant only in total sample.

Table 1. Prevalence of stressful life events. Gender differences in exposure to individual events. Reference group: males.

SLE	Older cohort (n=370; 158 males; 212 females)				Younger Cohort (n=363; 156 males; 207 females)				Total Sample (n=733; 314 males; 419 females)						
	Prevalence ^a	OR ^b	95% CI		Prevalence	OR	95% CI		Prevalence	OR	95% CI				
parental death	4.30%	0.96	0.35	2.63	5.5%	1.81	0.68	4.83	4.9%	1.34	0.67	2.70			
parental divorce	27.4%	0.96	0.60	1.52	36.9%	1.05	0.68	1.63	32.0%	1.01	0.74	1.38			
poverty	11.1%	0.85	0.44	1.62	3.0%	0.42	0.12	1.46	7.1%	0.73	0.42	1.29			
poor living conditions	6.2%	2.22	0.85	5.77	3.1%	3.45	0.73	16.2	4.7%	2.52	1.12	5.64	*		
accidents and traumas	21.4%	0.46	0.28	0.77	***	25.4%	0.34	0.21	0.57	***	23.3%	0.40	0.28	0.57	***
physical abuse elsewhere	17.0%	0.32	0.18	0.56	***	16.9%	0.27	0.15	0.49	***	16.8%	0.36	0.26	0.51	***
physical violence family	26.8%	1.13	0.71	1.81		not included				not included					
physical punishment	16.5%	0.91	0.52	1.59		not included				not included					
emotional abuse elsewhere	3.9%	1.93	0.59	6.27	5.5%	0.59	0.24	1.46	4.6%	0.95	0.47	1.89			
emotional abuse family	21.8%	1.49	0.89	2.51		not included				not included					
parental alcoholism	3.8%	2.81	0.77	10.2	1.1%	no cases for males			2.5%	3.83	1.10	13.3	*		
persistent severe worrying	18.5%	1.85	1.05	3.25	*	16.3%	2.07	1.13	3.79	*	17.4%	1.95	1.29	2.94	***
suicidal attempts	4.7%	3.72	1.05	13.1	*	5.3%	1.65	0.61	4.45		5.0%	2.34	1.08	5.05	*
leaving home for several days	17.2%	1.26	0.72	2.21		13.4%	1.03	0.56	1.91		15.3%	1.15	0.76	1.74	
suicide/attempt of a close relative	5.2%	1.68	0.62	4.52		4.2%	0.84	0.30	2.38		4.7%	1.22	0.60	2.48	
depression of a close relative		not included				9.5%	1.62	0.77	3.44						
sexual abuse	10.5%	16.20	3.84	68.4	***										
rape attempt	10.1%	no cases for males													
single parent	3.2%	0.52	0.16	1.67											
risky sexual behaviour	15.1%	0.50	0.28	0.89	*										
poor relationship with separately living parent	14.4%	0.97	0.54	1.74											

*p < .05, **p < .01, ***p < .0001.

a Column Prevalence shows rates of exposure to individual event among males and females together. Percentages are not additive.

b Gender difference in exposure to individual event presented in odds ratios (reference group - males).

Total Life Events

The associations between lifetime diagnosis of major depression, gender and cumulative score of stressful life events are shown in Table 2. Model 1 (a,b,c) included only gender. Females odds ratio (OR) of having depression compared to males ranged from 1.67 in younger cohort to 3.92 in the older cohort.

Model 2 (a,b,c) included gender and cumulative score of SLE. Odds ratio for gender did not changed very much. Cumulative score was significantly associated with higher odds of major depression in both cohorts and the total sample. The strongest association have been found in the younger cohort with (OR=1.77; CIs [1.39; 2.25], $p < .001$).

Model 3 (a,b,c) included gender, cumulative score of SLE and interaction between gender and SLE. Interaction effects in all models were statistically insignificant. In model 3a (the older cohort) effect of SLE also became insignificant, however ORs of SLE in model 3b (younger cohort) and 3c (total sample) did not changed very much. Effects of gender and SLEs were most stable in total sample, changes of the effect sizes in older cohort possibly occurred due to lack of the statistical power (very high p values).

In model 4 (a,b,c) the number of stressful life events was included as a categorical variable. The overall effect of SLE (participants with 1,2,3 and 4+ events taken together) was statistically significant in both cohorts and total sample. In the older cohort odds ratio of participants, who reported 1, 2, 3 or 4+ SLE were higher compared to participants with no events reported, however difference was statistically significant only for participants with 4+ events (OR= 2.9; CIs [1.35; 6.21], $p = .01$). In the younger cohort ORs were not significant only for participants with 1 event recorded ($p = .15$). In the total sample the overall and separate effects of SLE were statistically significant: OR of major depression was higher (1.77 CIs [1.1; 2.86], $p = .02$) for participants who reported just one SLE as compared to participants with no history of SLE events. The results of analysis of model 4 has shown that the ORs of depression increase in magnitude with the number of events and any threshold effect did not occur.

I also tested interactions between the number of SLE recorded (categorical variable) and gender and did not find evidence for a gender difference in response to stressful life events for depression (data not shown).

Table 2. Odds of major depression in relation to gender, stressful life events, and the interaction between gender and stressful life events. Total events and groups.

		Older cohort (n=370, (158 m, 212 f) (a)				Younger cohort (n=363) (156 m, 207f) (b)				Total Sample (n=733) (314 m; 419 f) (c)			
Total events		OR	95% CI			OR	95% CI			OR	95% CI		
Model 1 ref. males	Gender	3.92	2.17	7.11	***	1.67	1.03	2.71	*	2.41	1.66	3.48	***
Model 2	Gender	4.10	2.25	7.48	***	1.93	1.17	3.20	*	2.60	1.79	3.80	***
	Score SLE	1.29	1.09	1.53	**	1.77	1.39	2.25	***	1.43	1.25	1.64	***
Model 3	Gender	1.87	0.66	5.36		2.33	0.86	6.36		2.24	1.21	4.14	*
	Score SLE	1.01	0.72	1.41		1.91	1.26	2.90	*	1.35	1.05	1.72	*
	Gender*SLE	1.40	0.94	2.07		0.89	0.54	1.49		1.09	0.81	1.47	
Model 4 Nr of events; ref. 0 events	1	1.20	0.50	2.88		1.72	0.82	3.61		1.77	1.10	2.86	*
	2	1.77	0.71	4.44		3.65	1.75	7.62	***	2.32	1.37	3.92	***
	3	1.44	0.56	3.72		5.12	2.32	11.3	***	2.46	1.29	4.70	*
	4+	2.90	1.35	6.21	*	-	-	-		5.11	2.72	9.61	***
		(d)				(e)				(f)			
Intrafamilial group		OR	95% CI			OR	95% CI			OR	95% CI		
Model 2	Gender	3.96	2.18	7.19	***	1.65	1.01	2.69	*	2.40	1.66	3.47	***
	Score SLE	1.39	1.03	1.88	*	1.46	1.07	1.99	*	1.25	1.00	1.57	*
Model 3	Gender	1.99	0.81	4.92		2.17	1.04	4.53	*	2.22	1.34	3.69	***
	Score SLE	0.85	0.46	1.57		1.82	1.07	3.10	*	1.16	0.78	1.75	
	Gender*SLE	1.92	0.95	3.91		0.72	0.37	1.38		1.11	0.68	1.81	
Model 4 ref. 0 events	1	1.41	0.72	2.78		1.30	0.76	2.22		1.00	0.67	1.48	
	2+	1.94	1.06	3.55	*	2.20	1.18	4.11	*	1.70	1.08	2.68	*
		(g)				(h)				(i)			
Episodic SLE		OR	95% CI			OR	95% CI			OR	95% CI		
Model 2	Gender	3.91	2.15	7.09	***	1.79	1.09	2.92	*	2.54	1.75	3.69	***
	Score SLE	1.45	1.04	2.02	*	1.58	1.12	2.24	*	1.48	1.16	1.89	***
Model 3	Gender	3.39	1.45	7.91	***	1.74	0.79	3.84		2.59	1.48	4.54	***
	Score SLE	1.25	0.62	2.56		1.55	0.88	2.74		1.50	0.98	2.31	.06
	Gender*SLE	1.20	0.54	2.69		1.03	0.50	2.11		0.98	0.58	1.64	
Model 4 ref. 0 events	1	1.73	0.97	3.08	.06	1.45	0.86	2.46		1.53	1.05	2.24	*
	2+	1.97	0.98	3.96	.06	2.60	1.28	5.31	*	2.15	1.29	3.58	***

*p < .05. **p < .01. ***p < .001

Model 4: Bold if the overall effect is significant.

Table 2 continues. *Odds of major depression in relation to gender, stressful life events, and the interaction between gender and stressful life events. Groups of chronic, episodic and abuse events.*

		Older cohort (n=370; 158 m. 212 f) (j)				Younger cohort (n=363) (156 m, 207f) (k)				Total Sample (n=733; 314 m; 419 f) (l)			
Chronic SLE		OR	95% CI		OR	95% CI		OR	95% CI		OR	95% CI	
Model 2	Gender	4.09	2.24	7.48	***	1,80	1,09	2,96	*	2.55	1.75	3.71	***
	Score SLE	1.43	1.16	1.78	***	1,84	1,37	2,48	***	1.67	1.35	2.08	***
Model 3	Gender	1.94	0.78	4.85		2,00	0,98	4,10		2.20	1.31	3.70	***
	Score SLE	1.00	0.65	1.53		2,00	1,23	3,24	*	1.48	1.01	2.16	*
	Gender*SLE	1.63	0.99	2.69		0,88	0,48	1,62		1.20	0.76	1.92	
Model 4 Nr of events; ref. 0 events	1	1.65	0.79	3.43		1,93	1,09	3,42	*	1.70	1.13	2.56	*
	2	1.69	0.75	3.77		3,37	1,85	6,14	***	2.79	1.79	4.35	***
	3	3.17	1.62	6.20	***	-	-	-		-	-	-	
Chronic and Episodic SLE		OR	(m) 95% CI			OR	(n) 95% CI			OR	(o) 95% CI		
Model 1	Gender	4.04	2.21	7.39	***	1,89	1,14	3,12	*	2.62	1.80	3.83	***
	Chronic SLE	1.38	1.09	1.74	*	1,78	1,32	2,40	***	1.58	1.27	1.98	***
	Episodic SLE	1.18	0.82	1.71		1,46	1,02	2,09	*	1.32	1.03	1.69	*
Model 2	Gender	3.90	2.13	7.15	***	1,95	1,17	3,23	*	2.64	1.81	3.85	*
	Chronic SLE	1.16	0.84	1.62		2,38	1,51	3,76	***	1.69	1.22	2.34	*
	Episodic SLE	0.78	0.39	1.56		1,97	1,19	3,26	*	1.42	0.99	2.03	
	Chronic*Epis.	1.27	0.91	1.75		0,71	0,47	1,07		0.92	0.68	1.24	
Older cohort only													
		Psychical & Sexual abuse (p)				Psychical & Emotional abuse (r)				Sexual & Emotional abuse (s)			
		OR	95% CI			OR	95% CI			OR	95% CI		
Model 2	Gender	3.97	2.19	7.19	***	4.09	2.25	7.44	***	3.44	1.88	6.29	***
	Score SLE	1.25	0.93	1.69		1.38	1.02	1.86	*	1.58	1.12	2.22	**
Model 3	Gender	2.65	1.22	5.77	*	3.21	1.42	7.27	**	2.64	1.31	5.32	*
	Score SLE	0.83	0.43	1.60		1.11	0.60	2.04		0.87	0.32	2.39	
	Gender*S LE	1.71	0.81	3.58		1.33	0.66	2.68		2.01	0.68	5.92	
Model 4 Nr of events; ref. 0 events	1	1.53	0.80	2.91		1.55	0.81	2.97		1.38	0.75	2.55	
	2+	1.53	0.83	2.80		1.86	1.02	3.42	*	2.64	1.29	5.42	**

*p < .05. **p < .01. ***p < .001

Model 4: Bold if the overall effect is significant.

Groups of Stressful Life Events

To investigate associations between SLE groups and depression, I used the same procedures as for analysis of total life events. Results for "Intrafamilial" group were very similar to results of total life events. The cumulative score of SLE was significantly associated with higher odds of depression in both cohorts and total sample, however ORs were smaller compared to results of total events, except for the older cohort, where ORs increased from 1.29 to 1.41. For example, in the total sample, ORs of having depression for participants with 2+ events recorded (intrafamilial group), were similar to ORs for participants with 1 event recorded (total events). In models (3d,3e,3f) interactions between gender and SLE were statistically insignificant with the smallest p value ($p=.07$) in the older cohort. It should be noted that in the older cohort and the total sample the effect of SLE also became insignificant in model 3. In both cohorts and total sample the ORs for lifetime diagnosis of major depression increased in magnitude with the number of events (Models 4 d, e, f), however the overall effect of SLE in older cohort was not statistically significant, with $p=.10$. In the total sample and the younger cohort, odds ratios of depression for participants who reported 1 or 2+ SLE were higher compared to participants with 0 events reported; however, this difference was statistically significant only for participants with 2+ events.

Groups of chronic and episodic SLE were first analyzed separately using the above described procedures. The cumulative scores of episodic and chronic SLE (separately) were significantly associated with higher odds for major depression. This was true for both cohorts. In the older cohort (Model 2g and 2j) the odds ratio for both groups was almost similar (episodic, $OR=1.45$, CIs [1.04; 2.02], $p=.03$ and chronic, $OR=1.43$, CIs [1.16; 1.78], $p<.0001$). In the total sample (Model 2i and 2l) the difference between effects of chronic and episodic SLE-s was minimal, (episodic, $OR=1.48$, CIs [1.16; 1.89], $p<.0001$ and chronic, $OR=1.67$, CIs [1.35; 2.08], $p<.0001$). In the younger cohort the difference between effects of chronic and episodic SLE-s was slightly bigger (episodic, $OR=1.58$ CIs [1.12; 2.24], $p=.01$ and chronic $OR=1.84$ CIs [1.13; 2.48], $p<.0001$). It should be noted that according to Models 3 and 4 the association between chronic SLE and depression was more stable (less of insignificant associations) and stronger (higher ORs) compared to episodic SLE. In the next stage I examined chronic and episodic SLE-s by including them both in a common model (1m,1n, 1o) in addition to gender. In the older cohort the effect of chronic SLE-s on depression was higher (and statistically significant) compared to the effect of episodic SLE-s (that was statistically insignificant). The pattern of associations was similar for the total

sample and the younger cohort, and ORs were in both cases statistically significantly higher. In Models 2m, 2n and 2o, the interaction between chronic and episodic SLE-s was also added but the interaction effects were not significant.

The groups of events "Loss and parental separation " and "Abuse and violence" were studied only in the older cohort. For the group of events "Loss and parental separation" (Appendix B) I have used the median split and divided subjects into groups with no events reported and 1-3 events reported. The main effect of SLE-s and the interaction between SLE-s and gender were not statistically significant. I did not run Model 4, because the number of stressful life events was already included as a categorical variable.

Violence and abuse events were divided into 4 groups. One group included all abuse events and the other three included combinations of abuse events: sexual and emotional, psychological and sexual, psychological and emotional. Model 2r (event group psychological and emotional abuse - PEA) revealed a statistically significant effect with higher odds of major depression for subjects with more SLE (OR=1.37, CIs [1.02; 1.86], $p=.037$). The main and interaction effects in model 3r was not statistically significant, as in all previous models included interaction between gender and SLE. When I entered cumulative score of SLE as a categorical variable (model 4r), association between SLE and major depression (the overall effect) was not significant with p value = .106 . Similar results were found for sexual and emotional abuse group (SEA) with only difference in Model 4(s). Likewise in PEA group, difference was statistically significant only for participants with 2+ events (OR= 2.64, CIs [1.29; 5.42], $p= .008$), however the overall effect of SLE on depression was also statistically significant with $p=.029$. Results of group which included all abuse events - PSEA (Appendix B) were also similar to results of PEA group. Effect of SLE on depression (model 2t) was almost significant $p=0.06$ and became significant in model 3t with $p=0.03$. In PSA (psychological and sexual abuse) group no significant effects of SLE on depression were found.

Main effect of SLE on MD became statistically insignificant once interaction effect was added to 3p, 3r and 3s models (except for PSEA group).

I also assessed model, which included all individual abuse events and gender (Appendix C). Among abuse events, only effect of emotional abuse in the family was significant (OR=2.03, CIs [1.05; 3.92], $p=.035$).

Individual Events

I have run separate models for each event to investigate the associations between individual events and lifetime diagnosis of major depression (Table 3). Models included stressful life events and gender. Persistent severe worrying (ORs ranged from 2.89 to 3.42, $p < .001$) and suicidal attempts (ORs ranged from 3.13 to 4.54, $p < .001$) were found to be significantly associated with depression in both cohorts and total sample. Accidents and traumas (ORs ranged from 1.89 to 2.42, $p < .001$) were associated with depression in the younger cohort and total sample. Emotional abuse in the family (OR=1.99, [1.12; 3.54], $p=.02$) and poor or absent relationship with separately living parent (OR=2.47, [1.28; 4.77], $p=.01$) were also associated with depression, but these events were only included in the older cohort's SLE list. Depression of a close relative (OR=2.62, [1.27; 5.4], $p=.01$) was only included in SLE list of the younger cohort and was also associated with lifetime prevalence of major depression. Committed suicide or suicide attempt of a close relative were included in checklist of both cohorts, but effect of this event became significant only in total sample (OR=2.25, [1.09; 4.61], $p=.03$).

I also have run models which additionally included interaction between event and gender. Interaction effects was not statistically significant for any model. Main effect of SLE on MD became statistically insignificant once interaction effect was added to models (with only few exceptions).

Table 3.

Odds ratio of major depression in relation to gender, individual stressful life events and the interaction between gender and individual stressful life events.

SLE	Older cohort (n=370; 158 males; 212 females)				Younger cohort (n=363;156 males; 207 females)				Total Sample (n=733; 314 males; 419 females)			
	OR	95% CI	p		OR	95% CI	p		OR	95% CI	p	
gender	3.92	2.17	7.11	<.001	1.67	1.03	2.71	0.04	2.41	1.66	3.48	<.001
parental death	1.75	0.56	5.46	0.33	1.66	0.56	4.93	0.36	1.53	0.74	3.17	0.25
event*gender	0.29	0.03	2.72	0.28	a				0.48	0.11	2.16	0.34
parental divorce/separation	1.00	0.56	1.77	1.00	0.80	0.49	1.32	0.39	0.91	0.62	1.31	0.60
event*gender	3.85	0.74	20.16	0.11	1.20	0.43	3.38	0.73	1.61	0.70	3.69	0.26
poverty	1.96	0.93	4.12	0.08	1.77	0.87	3.60	0.11	1.70	0.91	3.15	0.09
event*gender	2.40	0.40	14.5	0.34	3.08	0.91	10.44	0.07	1.53	0.42	5.64	0.52
poor living conditions	1.99	0.81	4.89	0.13	1.99	0.81	4.89	0.13	1.98	0.97	4.05	0.06
event*gender	a				a				1.08	0.18	6.63	0.94
accidents and traumas	1.28	0.68	2.43	0.44	2.42	1.41	4.17	<.001	1.89	1.26	2.84	<.001
event*gender	0.47	0.12	1.78	0.27	1.67	0.56	4.94	0.36	0.87	0.38	1.98	0.74
physical abuse elsewhere (only OC)	1.39	0.68	2.85	0.37	1.74	0.93	3.24	0.08	1.59	0.99	2.53	0.053
event*gender	1.92	0.43	8.66	0.40	0.98	0.28	3.47	0.97	1.23	0.48	3.13	0.66
physical violence in the family	1.29	0.74	2.27	0.37								
event*gender	1.45	0.37	5.63	0.59								
physical punishment in the family	1.28	0.66	2.50	0.47								
event*gender	2.36	0.42	13.28	0.33								
emotional abuse elsewhere	2.52	0.80	7.85	0.11	0.71	0.23	2.19	0.55	1.34	0.62	2.91	0.45
event*gender	a							a	5.62	0.59	53.4	0.13
emotional abuse in family	1.99	1.12	3.54	0.02								
event*gender	1.16	0.29	4.67	0.83								

a Impossible to estimate the coefficients of interaction between event and gender, because the presence of at least one cell with a frequency equal to 0

Table 3 continues.

Odds ratio of major depression in relation to gender, individual stressful life events and the interaction between gender and individual stressful life events.

SLE	Older cohort (n=370; 158 males; 212 females)				Younger cohort (n=363;156 males; 207 females)				Total Sample (n=733; 314 males; 419 females)			
	OR	95% CI		p	OR	95% CI		p	OR	95% CI		p
parental alcoholism ^a	2.04	0.66	6.26	0.21	1.140	.353	3.678	.827	1.66	0.62	4.40	0.31
serious concerns	3.42	1.89	6.19	<.001	2.89	1.61	5.18	<.001	3.05	2.02	4.60	<.001
event*gender	1.39	0.33	5.80	0.65	0.94	0.27	3.34	0.92	1.25	0.50	3.14	0.64
suicidal attempts	4.54	1.61	12.75	<.001	3.13	1.22	7.99	0.02	3.81	1.90	7.61	<.001
event*gender	a				0.71	0.10	5.28	0.74	0.78	0.16	3.76	0.76
leaving home for several days	1.32	0.69	2.50	0.40	0.79	0.41	1.53	0.48	1.27	0.81	2.02	0.30
event*gender	1.96	0.35	10.83	0.44	0.40	0.10	1.53	0.18	0.83	0.31	2.21	0.71
committed suicide (or attempt) of a close relative	1.29	0.45	3.70	0.63	1.29	0.45	3.70	0.58	2.25	1.09	4.61	0.03
event*gender	a				0.22	0.02	1.98	0.18	0.48	0.11	2.08	0.33
depression of a close relative		not included			2.62	1.27	5.40	0.01				
event*gender					1.27	0.27	6.07	0.77				
sexual harassment elsewhere	1.99	0.96	4.11	0.062								
rape attempt ^a	1.75	0.83	3.67	0.14								
single parent ^a	2.43	0.66	8.92	0.18						not included		
risky sexual behaviour	1.39	0.68	2.84	0.36								
event*gender	1.92	0.40	9.34	0.42		not included						
poor relationships with separately living parent	2.47	1.28	4.77	0.01								
event*gender	2.21	0.47	10.45	0.32								

a Impossible to estimate the coefficients of interaction between event and gender, because the presence of at least one cell with a frequency equal to 0

Discussion

I investigated the potential role of up to 26 SLE-s recorded at age 18 in the occurrence of lifetime major depression as measured in a psychiatric interview conducted at age 25. I used data of longitudinal Estonian Children Personality Behaviour and Health Study (ECPBHS), comprising two birth cohorts, which are population based samples. I found that individuals with more SLE experienced during childhood and adolescence have higher risks of lifetime episode of major depression. Association between the number of SLE and MD is dose-dependent and the threshold effect did not occur. According to results of the total sample, each additional SLE increases the risk of lifetime episode of major depression by 1.43 in odds compared to individuals with no history of SLE. Results are consistent with previous studies, where cumulative score of SLE are used to investigate relationship between stress and depression among adolescents and young adults (Adkins et al., 2009; Zhang 2013). There are several works that also used ECPBHS samples to investigate relationship between depression and SLE (Oreland et al., 2010; Kurrikoff et al., 2012; Kurrikoff et al., 2013; Laas et al., 2014). Mentioned works were related to genetic studies, so investigation of independent effects of SLE was not the main focus, but merely noting the effect of total number of SLEs on depression. In contrast to current study, the authors mostly used data from self-report questionnaires to assess depression. Only one work used data of psychiatric interview. Laas and colleagues (2014) found that SLE was associated with higher rates of affective disorders (OR=1.85, CIs [1.13; 3.01], $p=.018$) (only data of older cohort were available). In current study, OR=1.27, CIs [1.07; 1.5], $p=.006$ (older cohort), but only major depression was investigated.

Results of analysis of stressors belonging to intrafamilial group were similar to results of analysis of total number of events and also consistent with previous studies (Mayer et al., 2009). It is obvious that family plays an important role in the development of child and adolescent, providing psychological support and care for healthy growth. Family instability reduces the ability of parents to provide sufficient support and as a result children see family environment as less secure and it induces emotional arousal in the child. According to the family systems theory, in the case of unresolved conflict, parents tend to involve children in the conflict to release some anxiety and tension between them (Wang & Crane, 2001). Caught in between feuding parents places high psychological demands on the unprepared child or adolescent, who can feel responsible for the parents relationship; this can lead to self blame if the parents relationship become worse. Unable to relief stress in the family, children become

more vulnerable to internalizing symptoms and engaging in risky behaviour that increase risk of triggering depression.

The analysis of "Chronic SLE" and "Episodic SLE" groups has shown that impact of chronic SLEs on probability of lifetime major depression was slightly greater compared to episodic SLEs. Such results occurred in both cases: when groups were evaluated separately and together (included in common regression model). The small difference between independent effects of chronic and episodic SLEs suggests that both groups of stressors are important in understanding mechanisms of developing depression. Previous research has shown mixed results. McGonagle and Kessler (1990) reported that participants who had experienced chronic stressors exhibited more severe depressive symptoms than did individuals who had experienced an acute (episodic) stressful life events. The opposite pattern occurred in studies of Muscatell et al. (2009) and Hammen (2009). Rojo-Moreno et al. (2002) stated that chronic difficulties are equally important to the genesis of depressive disorders as severe life events. Interaction effect between two groups was also tested, but results were insignificant. It is consistent with study of Turner and Turner (2005) who concluded that the effects of chronic and episodic stress may be independent of each other, both may have an impact, whereas neither moderates the other's effects. However other studies found that chronic SLEs can moderate effect of episodic SLEs on depression. Ormel et al. (2005) and Hammen (2009) concluded that chronic SLEs increase the depressive consequences of episodic SLEs. The opposite results were found in studies of McGonagle and Kessler (1990) and Cairney et al. (2003). Authors of these work suggested that chronic SLEs decrease the negative effect of episodic SLEs on depression, arguing that "saturation" results from ongoing difficulty such that episodic SLE have less impact. To conclude there are limited studies in this field and different authors use different approaches and different stress domains in their studies, which makes it difficult to compare results. Standardized measurement and assessment of SLEs should be used in future research to make results more comparable. Also deeper research on functional relationships between chronic and episodic stressors is needed.

The analysis of "Violence and abuse" groups (only the older cohort) has revealed that effect of SLE were significant only in groups, included emotional abuse (PEA, SEA, PSEA). The largest effect size of SLE (in OR) with also lowest p value was found in group included sexual and emotional abuse. Individual events analysis has shown that all abuse events increased risk for MD, but only effect of event "emotional abuse in the family" was

statistically significant (OR=1.99, CIs [1.12; 3.54], $p=0.02$). It should be noted that effects of events "sexual harassment, excluding family" and "physical abuse elsewhere" were close to a marginally significant level ($p=.06$ and $p=.053$) with OR=1.99 and 1.59 respectively. It should be also noted that in the current work, event psychological abuse or violence in the family did not specify who was the direct victim of violence: child, one of the parents, or other family members. Previous studies have shown that psychological abuse to a child as a direct victim increases the risk for MD to a greater extent compared to the effect of violence in the family, with a child as a witness (McCutcheon et al., 2010).

Results of current work are consistent with findings of Alloy and colleagues (2006). In their review of empirical research in this field, they suggested that although there are fewer studies of the role of childhood emotional abuse, the evidence for relationship between emotional abuse and depressive symptoms and diagnoses is more consistent than for physical and sexual abuse. They also assumed that the studies of childhood psychological and sexual abuse are rarely controlled for the overlap with childhood emotional abuse; thus, even positive findings may be attributable to the emotional abuse that often accompanies psychological and sexual abuse. In the current work, control for overlap between different types of abuse was done by running a logistic regression model, which included all individual abuse events and results were consistent with analysis of groups and individual events. Emotional abuse in the family was the only event with statistically significant effect on depression after controlling for other types of abuse events. It is consistent with attachment theory, which states that attachment figures (usually parents) help to develop a sense of security and build positive mental representations of self and others. In the case of emotional abuse in the family, attachment figures, who are supposed to be supportive helping to relieve distress and maintain self-worth for a child, instead themselves become sources of stress, insecurity and ruin to the positive self-model of a child. It is catastrophic for psychologically unprepared individuals, who have learned to cope with stress relying on help of attachment figures. As a result, a negative model of oneself and other could be formed, reducing resilience in coping with SLE and making person more vulnerable to psychological break down in times of crisis and developing psychopathology, including MD (Mikulincer & Shaver 2012). More recent studies also confirmed the stronger association between emotional abuse and major depression compared to psychological and sexual abuse (Liu et al., 2009; Shapero, 2014).

Alloy and colleagues (2006) also stated that only few studies have explicitly considered the role of maltreatment from non-relatives, which would allow for the

unconfounding of abuse experiences with genetic risk and the effects of a general negative family environment. In the current work, among all abuse events, only OR of emotional abuse in the family were significant. More detailed analysis of two types of abuse (family and elsewhere) was not done.

Results of analysis of the group of events "Loss and parental separation" has shown that there is no significant effect of such SLEs on depression. It is inconsistent with well documented association between loss events and MD (Speisman 2006; Kendler et al., 2002). However, previous results for parental divorce and separation are mixed; many studies have not found strong relationship of parental divorce during childhood or adolescence with depression in adulthood. These studies have shown that effect of divorce and separation on a child or adolescent depends greatly on quality of future relationship with separately living parents. The poor or absent relationship with separately living parents mediates the effects of divorce or separation on MD (Reiter et al., 2013). After closer look at analysis of individual events and prevalence rate of events, results of group of events "Loss and parental separation" can be explained. Event parental divorce and separation had the prevalence rate 27.3 %, which is approximately equal to sum of prevalence rates of all other events included in group. Event "poor or absent relationship with separately living parent" had the prevalence rate 14.4 % and significantly increase risk of MD. It means that approximately half of participants who experienced parental divorce or separation had normal relationship with separately living parent and possibly the negative impact of divorce was not so great for them. Unfortunately, in the current study, interaction effect between these events was not significant (data not shown). Divorce has also greater impact on females and the negative role of divorce in the psychological development of a child or adolescent decreases with the age. In the present study, the difference between males and females in rate of exposure to divorce and separation was not statistically significant. Data about age of the participants, when they experienced parental separation, is missing. In conclusion, "parental divorce and separation" odds ratio were equal to 1 with p value also close to 1. These numbers together with high prevalence rate of divorce, probably affect results of "Loss and parental separation" group in general.

I already discussed some significant results of individual events analysis (emotional abuse in the family, poor or absent relationship with separately living parent). The other events that are significantly associated with increased risk of MD: persistent severe worrying,

suicidal attempts, accidents and traumas, depression of a close relative, committed suicide or suicide attempt of a close relative.

Suicidal attempts were found to be significantly associated with diagnosis of depression. This was true for both cohorts. The link between depression and suicidality is well established. The vast majority of adolescents with a lifetime history of suicide attempts (96.1%) meet lifetime criteria for at least 1 mental disorder included in DSM-IV, with depression being the most prevalent one, up to 75.1 % (Nock, Green & Hwang, 2013). Suicidal behaviour of close relative also increase risk for lifetime prevalence of MD. First, as was discussed above, parental or close relative death is a good independent predictor of lifetime MD (Speisman, 2006). Second, a death through suicide increases the negative impact of loss event on children. Additionally to coping with unexpected death, children also have to deal with the way their relative has died. Third, close relatives with suicidal behaviour are at high risk of having MD. The current study and numerous previous studies found evidence for heritability of depression. Twin studies suggest a heritability of 40% to 50%, and family studies indicate a twofold to threefold increase in lifetime risk of developing MD among first-degree relatives (Lohoff, 2010). Last, suicidal behaviour of close relatives increases risk of suicidal attempts among adolescents and young adults via imitation and modelling (Burke et al., 2010). Taken together, suicidal behaviour of close relative are highly associated with depression among adolescents and young adults via different pathways.

The current study has shown that individuals with history of accidents and traumas are twice as likely to experience major depression during their lifetime. The findings from previous studies support this result and relationship seems to be bidirectional (Patten, Williams, Lavorato & Eliasziw, 2010). The fact that depression can increase risk of being injured should be considered in different areas of life, especially including activities that are recognised to pose a high level of risk to the health and safety of people.

Event "Persistent severe worrying" was associated with MD in both cohorts and total sample, with p value less than .001 and ORs ranged 2.89-3.42. It is indeed a strong relationship, but not surprising, as this event is associated with anxiety disorders. Participants who were marked as having this event, answered that they have lots of severe concerns, they constantly worry about. Different forms of this question is often used in anxiety disorders checklists, especially in checklists for generalized anxiety disorder (GAD). It is well documented that there is a strong relationship and high comorbidity rate between depression and anxiety disorders. In longitudinal studies (adolescents and young adults) of Moffitt and

colleagues (2007), cumulatively 72% of lifetime anxiety cases had a history of depression and 48% of lifetime depression cases had anxiety.

Analysis of gender differences has shown that the mean number of SLE was not statistically significant different between males and females in both cohorts. However, in both cohorts women had higher ORs to experience MD. The results do not support the mediational stress exposure model, which postulates that girls experience more stressors than boys, and as a result, girls become more depressed. It should be noted that empirical evidence for mediational model suggests that gender differences emerges mostly across interpersonal stressors (e.g., peer, romantic, and family relationships) highlighting the important role of social relations for girls (Hankin et al., 2007). In the current work, SLE were not subdivided into groups of interpersonal and non-interpersonal events, because there were not enough details to make such division. Males are more likely than females to become depressed following achievement related stressors. The list of SLE-s did not contain such type of stressors. Including a wider range of stressor should be considered in future studies investigating gender difference in relationship between SLE and MD.

Analysis of the prevalence of SLEs in the samples has shown that there are significant gender differences in exposure to some events. Analysis of individual events has shown that a majority of these events were also associated with lifetime diagnosis of major depression. However, interaction effects of SLE and gender were not significant for any regression model; p values for interactions were high. It is inconsistent with majority of previous studies (Rudolph, 2002; Hankin et al., 2007). However, some studies failed to find gender differences in association between SLE and MD. For example, Turner and Lloyd (2004) in their adolescents' study tested total number of events and did not find a mediating effect of gender.

I did not include gender and event interaction for sexual harassment elsewhere, because there were 36 cases for females and only 2 for males. Individuals with a record of sexual harassment were twice as likely to experience MD than individuals without such experience; however, p values were only close to significant level ($p=.062$). Although this cannot be confirm, there is a strong assumption that the association between depression and sexual abuse is mediated by gender. It is consistent with previous research and with the mediational stress exposure model.

To conclude no gender differences were found for these types of SLEs in assessed cohorts, with exception for one event.

Limitations

A few limitations should be acknowledged. The most important limitation is low frequencies of some individual events: 7 out of 20 events in the older cohort ; 7 out of 13 events in the younger cohort have prevalence rate in the range 4-6%, which is a low rate for sample size N=370, especially for investigation of interaction effects. Despite the fact that population based samples were used, low frequencies could reduce the power of our regression analyses by masking significant effects. Among individual events with statistically significant association with MD, only two, "suicidal attempts" and "committed suicide or suicide attempt of close relative", have low prevalence rate. Such association is consistent with past research and it can be assumed that association is indeed strong, in despite of low frequencies, these events became significant. Still, low frequencies of some individual events set limitations to my findings.

Another limitation is the fact that control for variables apart for gender in logistic regression models. First the participants were same age (+-1 year), same race and nationality. When SLE were recorded, participants were school students, they did not have job and were not married. Economic status was not used as a control variable as poverty and living conditions were included in the list of SLE. Participants were also interviewed at age 25, but mentioned control variables could not be used, because there is no information about time of first onset of depressive episode. No information available also on duration and severity of the depressive episode, only present/absent coding of lifetime diagnoses of major depressive disorder. Current episodes were also recorded, but there were only few individuals who met such criteria; therefore, this information was not useful for analysis. SLE were recorded at age 18, but the occurrence of each event is not precisely dated. Because of missing information, even approximate timing between SLE occurrence and onset of the depressive episode cannot be determined. It is important, as previous work suggest that negative effect of some SLE-s decreasing with age. Because of absent data, bidirectional relationship between depression and SLE also couldn't be investigated. Future studies should use methods, which allow to examine bidirectional link between SLE and MD. The moderational stress reactivity model could not be test, because information about level of stress caused by particular event was not recorded. This approach is likely to have obscured more nuanced differences in SLE and depressive episodes across participants.

Conclusion

In conclusion, the assessment of the total number of SLE-s and SLE groups of events has revealed that experience of even only one traumatic event during adolescence and childhood can increase the risk to meet criteria of a lifetime diagnosis of major depression by age 25. The strongest association between MD and SLEs was found for the following events: persistent severe worrying, accidents and traumas, poor or absent relationship with separately living parent, emotional abuse in the family, suicidal behaviour of participant or his close relative and depression of a close relative. The effect of SLEs of intrafamilial group on major depression was with same direction as in case of total number of events, but effect size was smaller. I also found that the greatest impact across abuse SLE-s was elicited by emotional abuse in the family. All abuse forms contain elements of emotional abuse and it should be considered in future research. Chronic and episodic SLE-s have independent effects on major depression, and there was no moderation effect between them. The impact of chronic SLEs on major depression was slightly larger compared to episodic events. Previous research on this topic has shown mixed results. More research on functional relationships between chronic and episodic stressors is needed.

There was no evidence for the association between SLE-s and MD for the group of events "Loss and parental separation", as results were not significant and it is inconsistent with previous research. A possible explanation for these results is the different prevalence rates of events included in group, increasing the role of particular event on the overall effect. In this case, it was divorce and previous studies have had mixed results regarding the effect of parental divorce on the developing of major depression among adolescents and young adults. No evidence was found that relationship between SLE and major depression is mediated by gender. It is inconsistent with the majority of past research; however, some previous studies also failed to find such relationship.

To summarize, the current work, with its unique combination of sample and methodology, contributed to the empirical knowledge about the role of stressful life events on development of major depression. It also has shown that employing different methods of examining SLE in the same study, namely assessment of total number of events, groups of events and individual events, provide more detailed information for sufficient analysis and grounded conclusions.

References

- Achenbach, T. M., Howell, C. T., McConaughy, S. H., & Stanger, C. (1995). Six-year predictors of problems in a national sample of children and youth: I. Cross-informant syndromes. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 336–347.
- Adkins, D. E., Wang, V., Dupre, M. E., van den Oord, E., & Elder, Jr., G. H. (2009). Structure, and Stress: Trajectories of depressive symptoms across adolescence and young adulthood. *Social Forces*, *88* (1), 31-60. doi: 10.1353/sof.0.0238.
- Alloy L.B., Abramson L.Y., Smith J.B., Gibb B.E., Neeren A.M. (2006). Role of parenting and maltreatment histories in unipolar and bipolar mood disorders: Mediation by cognitive vulnerability to depression. *Clinical Child and Family Psychology Review*, *9*, 23–64.
- Andrade, L., Jorge J., Berglund, P., Bijl, R. V., De Graaf, R., Vollebergh, W., ... Wittchen, H. U. (2003) . The epidemiology of major depressive episodes: results from the International Consortium of Psychiatric Epidemiology (ICPE) surveys. *International Journal of Methods in Psychiatric Research*, *12* (1): 3–21 doi: 10.1002/mpr.138
- Bernard, L. C., & Krupat, E. (1994) . Health Psychology: Biopsychosocial Factors in Health and Illness. New York: Harcourt Brace College Publishers.
- Brassard, M. R. , Donovan, K. L. (2006) . Defining psychological maltreatment: The interface between policy and research. In. M. M. Freerick, J. F. Knutson, P. K. Trickett, S. M. Flanzer (Eds.), *Child abuse and neglect: Definitions, classifications, a framework for research* (pp. 151–197). Baltimore: Paul H. Brookers Pub. Company.
- Breslau N., Kessler R. C., Chilcoat H. D., Schultz L. R., Davis G. C. , & Andreski P. (1998) . Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. *Archives of General Psychiatry*, *55*, 626–632.

- Burke, A. K., Galfalvy, H., Everett, B., Currier, D., Zelazny, J., Oquendo, M. A., ... Brent, D. A. (2010). Effect of Exposure to Suicidal Behavior on Suicide Attempt in a high-risk sample of Offspring of Depressed Parents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*(2), 114–121.
- Burton, E., Stice, E., & Seeley, J. (2004). A prospective test of the stress-buffering model of depression in adolescent girls: No support once again. *Journal of Consulting and Clinical Psychology*, *72* (4), 689-697. doi:10.1037/0022-006X.72.4.689. 689.
- Cairney, J., Boyle, M., Offord, D. R., & Racine Y. (2003) . Stress, social support and depression in single and married mothers. *Social Psychiatry and Psychiatric Epidemiology*, *38*, 442–449.
- Calvete, E., Camara, M., Estevez, A., & Villardon, L. (2011). The role of coping with social stressors in the development of depressive symptoms: Gender differences. *Anxiety, Stress & Coping*, *24* (4), 387–406.
- Caspi, A., Sugden, K., Moffitt, T. Taylor, A., Craig, I.W., Harrington, H., McClay, J. ... Poulton, R. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science*, *301*, 386– 389.
- Daley, S. E., Hammen, C., Davila, J., & Burge, D. (1998). Axis II symptomatology, depression, and life stress during the transition from adolescence to adulthood. *Journal of Consulting and Clinical Psychology*, *66*, 595-603
- De Graaf, R., ten Have, M., van Gool, C., van Dorsselaer, S. (2012) . Prevalence of mental disorders and trends from 1996 to 2009. Results from the Netherlands Mental Health Survey and Incidence Study-2. *Social Psychiatry and Psychiatric Epidemiology*, *47*, 203–213.
- Evans, G. W. & Cohen, S. (1987) . Environmental stress. In. Stokols, D. , & Altman, I. (Eds.), *Handbook of environmental psychology* (pp. 571-610). New York: Wiley.
- Ge, X., Lorenz, F. O., Conger, R. D., Elder, G. H. Jr., & Simons, R. L. (1994). Trajectories of stressful life events and depressive symptoms during adolescence. *Developmental Psychology*, *30* (4), 467-483. doi:10.1037/0012-1649.30.4.467.

- Grant, K. E., Compas, B. E., Stuhlmacher, A. F. , Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003) . Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin*, *129* (3),447-466.
doi: 10.1037/0033-2909.129.3.447.
- Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology*, *100*, 555–561.
- Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology*, *1*, 293–319. doi:10.1146/annurev.clinpsy.1.102803.143938.
- Hammen, C. (2006). Stress generation in depression: Reflections on origins, research, and future directions. *Journal of Clinical Psychology*, *62*(9), 1065–1082.
doi:10.1002/jclp.20293.
- Hammen, C., Kim E.Y., Eberhart N.K., & Brennan P.A. (2009). Chronic and Acute stress and the prediction of major depression in women. *Depression and Anxiety*, *26*(8), 718–723. doi:10.1002/da.20571.
- Hankin, B. L., Mermelstein, R., & Roesch, L. (2007). Sex differences in adolescent depression: Stress exposure and reactivity models. *Child Development*, *78* (1), 279-295. doi: 10.1111/j.1467-8624.2007.00997.x.
- Harro, M., Eensoo, D., Kiive, E., Merenäkk, L., Alep, J., ... Oreland, L. (2001). Platelet monoamine oxidase in healthy 9- and 15-year-old children: The effect of gender, smoking, and puberty. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *25*, 1497–1511.
- Harro, J., Merenäkk, L., Nordquist, N., Konstabel, K., Comasco, E., & Oreland, L. (2009). Personality and the serotonin transporter gene: Associations in a longitudinal population-based study. *Biological Psychology*, *81*, 9–13.
- Harro J., Kiive E. (2011). Droplets of black bile? Development of vulnerability and resilience to depression in young age. *Psychoneuroendocrinology*, *36* (3), 380-392.

- Heidmets, L., Samm, A., Sisask, M., Kõlves, K., Aasvee, K., Värnik, A. (2010). Sexual behavior, depressive feelings, and suicidality among Estonian school children aged 13 to 15 years. *The Journal of Crisis Intervention and Suicide Prevention, 31*(3), 128–136.
- Hill J., Pickles A., Rollinson L., Davies R., Byatt M. (2004). Juvenile versus adult-onset depression: Multiple differences imply different pathways. *Journal of Research in Psychiatry and the Allied Sciences, 34*, 1483–1493.
- Jaffee S. R., Moffitt T. E., Caspi A., Fombonne E., Poulton R., Martin J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression. *Archives of General Psychiatry, 59*, 215–222.
- Kendler K. S., Karkowski-Shuman L. (1997). Stressful life events and genetic liability to major depression: genetic control of exposure to the environment? *Psychological Medicine, 27*, 539–547
- Kendler K.S., Sheth K., Gardner C.O., Prescott C.A. (2002). Childhood parental loss and risk for first-onset of major depression and alcohol dependence: The time-decay of risk and sex differences. *Psychology and Medicine, 32*, 1187–1194.
- Kercher, A., Rapee, R. M., & Schniering, C. A. (2009). Neuroticism, life events and negative thoughts in the development of depression in adolescent girls. *Journal of Abnormal Child Psychology, 37*, 903-915
- Kessler R.C., Davis C.G., & Kendler K.S. (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychological Medicine, 27*, 1101–1119.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions' of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*(6), 593-602.
- Kurrikoff T., Lesch K-P., Kiive E., Konstabel K., Herterich S., Veidebaum T., Reif A., & Harro J. (2012). Association of a functional variant of the nitric oxide synthase 1 gene with personality, anxiety, and depressiveness. *Development and Psychopathology 24*(4), 1225-1235.

- Kurrikoff, T., Hiio K., Täht K., Harro J., Veidebaum T. (2013). The 5-HTTLPR genotype and depressiveness link: contribution of aspects of environment and gender. *Psychiatry Research*, 209 (1), 126-127.
- Laas K., Reif, A., Akkermann, K., Kiive E., Domschke K., Lesch K-P., Veidebaum T., & Harro J. (2014). Interaction of the neuropeptide S receptor gene Asn107Ile variant and environment: contribution to affective and anxiety disorders, and suicidal behaviour. *Neuropsychopharmacology*, 17(4), 541-552.
- Larson, R., & Ham, M. (1993). Stress and “storm and stress” in early adolescence: The relationship of negative events with dysphoric affect. *Developmental Psychology*, 29, 130–140.
- Leadbeater, B. J., Kuperminc, G. P., Hertzog, C., & Blatt, S. J. (1999). A multivariate model of gender differences in adolescents’ internalizing and externalizing disorders. *Developmental Psychology*, 35, 1268–1282.
- Lohoff, F. W. (2010). Overview of the Genetics of Major Depressive Disorder. *Current Psychiatry Reports*, 12(6), 539–546. doi:10.1007/s11920-010-0150-6
- Mayer, L., Lopez-Duran, N. L., Kovacs, M., George, C., Baji, I., Kapornai, K., ... Vetró, Á. (2009). Stressful Life Events in a Clinical Sample of Depressed Children in Hungary. *Journal of Affective Disorders*, 115(1-2), 207–214. doi:10.1016/j.jad.2008.08.018
- Mark, L., Samm, A., Tooming L-M., Sisask, M., Aasvee, K., Zaborskis, A., Zemaitiene, N., Värnik, A. (2012). Suicidal ideation, risk factors, and communication with parents: an HBSC study on school children in Estonia, Lithuania, and Luxembourg. *The Journal of Crisis Intervention and Suicide Prevention*. doi 10.1027/0227-5910/a000153.
- McCutcheon, V. V., Heath, A. C., Nelson, E. C., Bucholz, K. K., Madden, P. A. F., & Martin, N. G. (2010). Clustering of Trauma and Associations with Single and Co-Occurring Depression and Panic Attack over Twenty Years. *Twin Research and Human Genetics : The Official Journal of the International Society for Twin Studies*, 13(1), 57–65. doi:10.1375/twin.13.1.57
- McGonagle, K. A., & Kessler, R. C. (1990). Chronic stress, acute stress, and depressive symptoms. *American Journal Of Community Psychology*, 18(5), 681-706.

- Mezulis, A. H., Funasaki, K. S., Charbonneau, A. M., & Hyde, J. S. (2010). Gender differences in the cognitive vulnerability-stress model of depression in the transition to adolescence. *Cognitive Therapy and Research, 34*, 501–513.
- Mikulincer, M. & Shaver, P. R. (2012). An attachment perspective on psychopathology. *World Psychiatry, 11(1)*, 11–15
- Moffitt T., Harrington H, Caspi A. (2007). Depression and Generalized Anxiety Disorder: Cumulative and Sequential Comorbidity in a Birth Cohort Followed Prospectively to Age 32 Years. *Archives of General Psychiatry, 64(6)*, 651-660.
doi:10.1001/archpsyc.64.6.651.
- Monroe, S. M., & Reid, M. W. (2009). Life stress and major depression. *Current Directions in Psychological Science, 18*, 68–72. doi:10.1111/j.1467-8721.2009.01611.x.
- Muscattell, K. A., Slavich, G. M., Monroe, S. M., & Gotlib, I. H. (2009) . Stressful Life Events, Chronic Difficulties, and the Symptoms of Clinical Depression. *The Journal of Nervous and Mental Disease, 197(3)*, 154–160.
<http://doi.org/10.1097/NMD.0b013e318199f77b>
- Ng, C. S. M., & Hurry, J. (2011). Depression amongst Chinese adolescents in Hong Kong: An evaluation of a stress moderation model. *Social Indicators Research, 100 (3)*, 499-516. doi: 10.1007/s11205-010-9626-3.
- Nock M.K., Green J., & Hwang I., (2013). Prevalence, Correlates, and Treatment of Lifetime Suicidal Behaviour Among Adolescents: Results From the National Comorbidity Survey Replication Adolescent Supplement. *JAMA Psychiatry, 70(3)*,300-310.
doi:10.1001/2013.jamapsychiatry.55.
- Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Science, 10 (5)*, 173–176. doi: 10.1111/1467-8721.00142
- Noller, P., Feeney, J., & Petersen, C. (2001). *Personal relationships across the lifespan*. London: Psychology Press.
- Oreland L., Nordquist N., Hallman J., Harro J., Nilsson K.W. (2010). Environment and the serotonergic system. *European Psychiatry 25(5)*, 304-306.

- Ormel, J. , Oldehinkel, A. J. , & Brilman, E. I. (2001) . The interplay and etiological continuity of neuroticism, difficulties, and life events in the etiology of major and subsyndromal, first and recurrent depressive episodes in later life. *The American Journal of Psychiatry*, *158*, 885–891.
- Paradis, A. D., Giaconia, R. M., Reinherz, H. Z., Beardslee, W. R., Ward, K. E., & Fitzmaurice, G. M. (2011). Adolescent Family Factors Promoting Healthy Adult Functioning: A Longitudinal Community Study. *Child and Adolescent Mental Health*, *16*(1), 30–37. doi:10.1111/j.1475-3588.2010.00577.x
- Patten S.B., Williams J.V., Lavorato D.H., & Eliasziw M. (2010). Major depression and injury risk. *Canadian Journal of Psychiatry*, *55*, 313–318.
- Paykel E.S. (2003). Life events and affective disorders. *Acta Psychiatrica Scandinavica*, *108*,61–66.
- Reiter S.F., Hjörleifsson S., Bredablik H.J, & Meland E. (2013). Impact of divorce and loss of parental contact on health complaints among adolescents. *Journal of Public Health*, *35*(2), 278-85. doi: 10.1093/pubmed/fds101
- Rojo-Moreno, L., Livianos-Aldana, L., Cervera-Martínez, G., Dominguez-Carabantes, J. A. & Reig-Cebrian, M. J. (2002) . The role of stress in the onset of depressive disorders. A controlled study in a Spanish clinical sample. *Social Psychiatry and Psychiatric Epidemiology*, *37*(12), 592-8.
- Rudolph K. D, & Hammen C. (1999). Age and gender as determinants of stress exposure, generation, and reactions in youngsters: A transactional perspective. *Child Development*,*70*, 660–677.
- Rudolph K.D., Hammen C., Burge D., Lindberg N., Herzberg D., & Daley S.E. (2000). Toward an interpersonal life-stress model of depression: The developmental context of stress generation. *Development and Psychopathology*, *12*, 215–234.
- Rudolph, K. D. (2002). Gender differences in emotional responses to interpersonal stress during adolescence. *Journal of Adolescent Health*, *30S*, 3–13. doi:10.1016/S1054-139X(01)00383-4

- Rudolph, K. D., Flynn, M., Abaied, J. L., Groot, A., & Thompson, R. (2009). Why is past depression the best predictor of future depression? Stress generation as a mechanism of depression continuity in girls. *Journal of Clinical Child & Adolescent Psychology*, 38 (4), 473-485. doi: 10.1080/15374410902976296.
- Stroud, C., Davila, J., Hammen, C., Vrshek-Schallhorn, S. (2011). Severe and nonsevere events in first onsets versus recurrences of depression: Evidence for stress sensitization. *Journal of Abnormal Child Psychology*, 120(1), 142-54. doi: 10.1037/a0021659.
- Samm, A., Tooding, L-M., Sisask, M., Kõlves, K., Aasvee, K., Värnik, A.(2010). Suicidal thoughts and depressive feelings among Estonian schoolchildren:effect of family relationship and family structure. *European Child & Adolescent Psychiatry*, 19(5):457-468.
- Shanahan, L., Copeland, W. E., Costello, E. J., & Angold, A. (2011). Child-, Adolescent-, and Adult-Onset Depressions: Differential Risk Factors in Development? *Psychological Medicine*, 41(11), 2265-2274. doi:10.1017/S0033291711000675
- Sheehan D.V, Lecrubier Y., Sheehan K.H., Amorim P., Janavs J., Weiller E., Hergueta E, ... Dunbar G. (1998). The Mini International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-VI and ITS-10. *Journal of Clinical Psychiatry*, 59, 22-33.
- Shim, R., Baltrus, P., Ye, J., Rust G., (2009). Prevalence, Treatment, and Control of Depressive Symptoms in the United States: Results from the National Health and Nutrition Examination Survey (NHANES), 2005-2008. *The Journal of the American Board of Family Medicine*, 24 (1), 33-3 doi:10.3122/jabfm.2011.01.100121
- Shih, J. H., Abela, J. R. Z., & Starrs, C. (2009). Cognitive and interpersonal predictors of stress generation in children of affectively ill parents. *Journal of Abnormal Child Psychology*, 37,195-208.
- Shlik J, Aluoja A, Kihl E. (1999). MINI 5.0.0. Mini rahvusvaheline neuropsühhiaatriline intervjuu DSM –IV. Estonian version of MINI international neuropsychiatric interview.

- Slopen, N., Williams, D. R., Fitzmaurice, G. M., & Gilman, S. E. (2011). Sex, stressful life events, and adult onset depression and alcohol dependence: are men and women equally vulnerable? *Social Science & Medicine Journal* 73 (4): 615-622
- Speisman B. (2006). The association between early parental loss and adulthood depression. Mind matters. *The Wesleyan Journal of Psychology*, 1,19–27.
- Steel, J. L., Dunlavy, A. C., Stillman, J., & Pape, H. C. (2011). Measuring depression and PTSD after trauma: Common scales and checklists. *Injury*, 42(3), 288–300.
doi:10.1016/j.injury.2010.11.045
- Telzer E. H, Fuligni A.J. (2013). Positive daily family interactions eliminate gender differences in internalizing symptoms among adolescents. *Youth and Adolescence*, 42(10), 1498-511 doi: 10.1007/s10964-013-9964-y
- Turner, R. J., & Lloyd, D. A. (2004). Stress burden and the lifetime incidence of psychiatric disorder in young adults: racial and ethnic contrasts. *Archives of General Psychiatry*, 61(5), 481-488.
- Turner, H. A., & Turner, R. J. (2005) . Understanding variations in exposure to social stress. *Sociology of Health & Illness*, 9, 209–240.
- Vitiello B. (2011). Prevention and treatment of child and adolescent depression: challenges and opportunities. *Epidemiology and Psychiatric Sciences* 20, 37-43.
- Waldinger R. J., Vaillant G.E., Orav E.J. (2007). Childhood sibling relationships as a predictor of major depression in adulthood: A 30-year prospective study. *American Journal of Psychiatry*, 164, 949–954.
- Wang, L., & Crane, D. R. (2001). The relationship between marital satisfaction, marital stability, nuclear family triangulation, and childhood depression. *The American Journal of Family Therapy*, 29, 337-347
- World Health Organization. (2015). Depression. Fact sheet N°369.
<http://www.who.int/mediacentre/factsheets/fs369/en/>
- Zhang, X. (2013). Stress, coping, and depression in adolescents: A longitudinal analysis of data from National Longitudinal Study of Adolescent Health (PhD dissertation). University of Nebraska. <http://digitalcommons.unl.edu/cehsdiss/185>

Appendix A.

Table 4. *Stressful life events included in different groups.*

Stressful life events	Total events			Intafamilial group			Loss and separation			Chronic			Episodic		
	OC	YC	TS	OC	YC	TS	OC	YC	TS	OC	YC	TS	OC	YC	TS
parental death	+	+	+	+	+	+	+						+	+	+
parental divorce	+	+	+	+	+	+	+						+	+	+
poverty	+	+	+	+	+	+				+	+	+			
poor living conditions	+	+	+	+	+	+				+	+	+			
accidents and traumas	+	+	+										+	+	+
poor health	+	+	+							+	+	+			
physical abuse elsewhere	+	+	+							+	+	+			
physical violence family	+			+						+					
physical punishment	+			+						+					
emotional abuse elsewhere	+	+	+							+	+	+			
emotional abuse family	+			+						+					
parental alcoholism	+	+	+	+	+	+				+	+	+			
child neglect	+	+		+	+	+				+	+	+			
hunger	+	+	+	+	+	+				+	+	+			
persistent severe worrying	+	+	+							+	+	+			
suicidal attempts	+	+	+										+	+	+
leaving home for several days	+	+	+	+	+	+				+	+	+			
suicide or attempt of a close relative	+	+	+	+	+	+	+						+	+	+
depression of a close relative		+			+						+				
unemployed parent		+			+						+				
sexual abuse family	+			+						Excl.					
sexual abuse elsewhere	+									Excl.					
rape attempt	+												+		
single parent	+			+			+			+					
risky sex behaviour	+									Excl.					
poor relationship with separately living parent	+			+			+			+					

Appendix B

Table 5.

Odds ratio of major depression in relation to gender, stressful life events and their interaction. Groups "Psychical, sexual, emotional abuse" and "Loss and parental separation", included only in the older cohort.

Psychical, sexual and emotional abuse (t)		OR	95% CI		p	Loss and parental separation (v)		OR	95% CI		p
Model 1 ref. Males	Gender	3.92	2.17	7.11	***	Gender	3.92	2.17	7.11	***	
Model 2	Gender	0.25	0.14	0.46	***	Gender	3.95	2.18	7.16	*	
	Score SLE	1.32	0.99	1.76	.06	Score SLE	1.31	0.78	2.20		
Model 3	Gender	0.35	0.15	0.81	*	Gender	3.26	1.53	6.96		
	Score SLE	1.43	1.03	1.99	.03	Score SLE	0.92	0.32	2.68		
	Gender*SLE	0.70	0.35	1.39		Gender*SLE	1.60	0.47	5.42		
Model 4 Nr of events; ref. 0 events	1	1.42	0.72	2.82							
	2+	1.73	0.97	3.08	.06						

*p < .05. **p < .01. ***p < .001

Model 4: Bold if the overall effect is significant.

Appendix C.

Single abuse events included in the common logistic regression model.

Hosmer and Lemeshow Test			
Step	Chi-square	df	Sig.
1	1,387	6	,967

Model Summary			
Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	337,454 ^a	,094	,145

Variables in the Equation								
	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I. for EXP(B)	
							Lower	Upper
Gender	1,159	,337	11,814	1	,001	3,187	1,64	6,17
Psychical abuse in the family	-,155	,366	,179	1	,673	,857	,418	1,75
Psychical punishment	-,157	,425	,136	1	,712	,855	,371	1,96
Psychical abuse elsewhere	-,209	,417	,252	1	,615	,811	,358	1,83
Emotional abuse in the family	,851	,346	6,050	1	,014	2,341	1,18	4,61
Emotional abuse elsewhere	,527	,651	,655	1	,418	1,693	,473	6,06
Sexual harrassment in the family	,054	1,373	,002	1	,968	1,056	,072	15,5
Sexual harrassment elsewhere	,674	,441	2,338	1	,126	1,962	,827	4,65
Rape attempt	,173	,438	,155	1	,693	1,189	,503	2,80
Constant	-2,281	,305	56,057	1	,000	,102		

Käesolevaga kinnitan, et olen korrektselt viidanud kõigile oma töös kasutatud teiste autorite poolt loodud kirjalikele töödele, lausetele, mõtetele, ideedele või andmetele.

Olen nõus oma töö avaldamisega Tartu Ülikooli digitaalarhiivis DSpace.

/Ilja Polozov/