Child Maltreatment and Adult Sexual Assault Victimization:

Genetic and Environmental Associations

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Abstract

Despite the pervasiveness of adult sexual assault (ASA), evidence-based knowledge on the risk factors for sexual victimization is insufficient. Here, we investigated the etiology of ASA in a population-based Finnish twin sample. Specifically, we estimated the extent of the genetic and environmental influences on the risk of ASA, and we examined its phenotypic and genetic associations with five types of child maltreatment (CM). We found large unique environmental, but also small genetic influences on the risk of ASA, motivating further research on situational and behavioral conditions potentially exploited by sexually motivated perpetrators. The prevalence of ASA was highest among victims of severe child sexual abuse. However, when accounting for the co-occurrence of multiple types of CM, emotional abuse was the strongest predictor of ASA. We further examined, and could not entirely rule out, the possibility of common genetic and environmental pathways underlying CM and ASA. Lastly, we focused on sex differences. Emotional and physical abuse were the strongest predictors of ASA in women and men, respectively, and genetic influences on the risk of ASA were larger in women than men. However, such higher heritability did not reflect sex-limited genetic effects, but, rather, women's systematic exposure to environmental risk of ASA.

Keywords: Assault, Sexual Aggression, Violence; Genetics; Childhood, Adolescence, Adolescent Sexuality; Gender Differences; Epidemiology.

Child Maltreatment and Adult Sexual Assault Victimization: Genetic and Environmental

Associations

Sexual Assault is a violation of basic human rights and a major public health problem (World Health Organization, 2014). As indicated by nationally representative empirical studies (e.g., Breiding, 2015), as well as by social media coverage (e.g., The #MeToo movement), sexual assault is a widespread phenomenon, affecting different segments of the population. Indeed, it has been estimated that approximately 36% of women and 17% of men experience some form of sexual violence throughout their lifetime (Smith et al., 2017). These include criminal behaviors, such as nonconsensual completed or attempted oral, vaginal or anal penetration (or "rape"; Basile, Chen, Black, & Saltzman, 2007), unwanted touching (or "sexual battery"; Muehlenhard, Peterson, Humphreys, & Jozkowski, 2017), sexual acts obtained using coercion or incapacitation (McCauley, Ruggiero, Resnick, Conoscenti, & Kilpatrick, 2009) as well as non-contact acts, such as verbal harassment (i.e., unwanted sexual comments or advances; Krug, Mercy, Dahlberg, & Zwi, 2002), that are especially commonplace (Breiding, 2015; Smith et al., 2017). Given the extent of this problem, and its detrimental impact on victims (Dworkin, Menon, Bystrynski, & Allen, 2017), empirical research on its risk factors is vital.

Genetic and Environmental Contributions to the Risk of Adult Sexual Assault Victimization

Previous research has identified environmental conditions in which sexual assault is most likely to occur, such as disadvantaged urban environments (Decker et al., 2014; Tyler, 2008) as well as institutional settings (Peterson, Voller, Polusny, & Murdoch, 2011; Wilson, 2018). Exposure to unsafe environments, and, primarily, the presence of sexually motivated perpetrators, represent the necessary proximate risk factors for sexual victimization. However, it has also been shown that perpetrators might seek victims with specific traits, such as intellectual disabilities (Fisher, Baird, Currey, & Hodapp, 2016) and externalizing problems (Daigle & Teasdale, 2018; Vezina & Hebert, 2007), traits that are highly influenced by genes (Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Larsson, Andershed, & Lichtenstein, 2006; Vissers, Gilissen, & Veltman, 2016). As a result, the

risk of sexual victimization might, itself, be indirectly influenced by genes. The relative impact of genetic and environmental factors on traits and experiences can be estimated using twin designs (Boomsma, Busjahn, & Peltonen, 2002). Twin designs are based on the difference in genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twins reared together, and on the assumptions that shared environmental exposures (i.e., shared between co-twins) contribute equally to their traits, whereas unique environmental exposures (i.e., unshared) contribute to their differences. Only a few studies have investigated the risk of interpersonal victimization using twin designs. Such studies have estimated moderate-to-large unique environmental, but also small-to-moderate genetic influences on the risk of lifetime assaultive trauma, including sexual assault (Sartor et al., 2012; Stein, Jang, Taylor, Vernon, & Livesley, 2002). Shared environmental influences, instead, have been found to have small or no impact, similarly to other stressful life events (e.g., Bolinskey, Neale, Jacobson, Prescott, & Kendler, 2004; Johnson, Rhee, Whisman, Corley, & Hewitt, 2013). In this context, it is important to note that such genetic influences do not, in any way, imply that victims bear any moral responsibility, as perpetrators bear full moral and legal responsibility for their sexual transgressions. However, a better understanding of the genetic risk factors for sexual victimization might inform individual-level prevention strategies. In fact, a successful integration of individual-level and environment-level interventions might improve the effectiveness of sexual violence prevention programs (DeGue et al., 2014).

The Phenotypic Relationship Between Child Maltreatment and Adult Sexual Assault Victimization

Among distal predictors of adult sexual assault (ASA), child maltreatment (CM) has been investigated in samples of women (e.g., Lau & Kristensen, 2010; Miron & Orcutt, 2014), men (e.g., Aosved, Long, & Voller, 2011; Peterson, Beagley, McCallum, & Artime, 2019), as well as in mixed samples (e.g., Ports, Ford, & Merrick, 2016; Widom, Czaja, & Dutton, 2008). This body of research has consistently shown that CM victims are at heightened risk of ASA victimization later in life, compared to non-victims. The vast majority of previous studies have focused on sexual

revictimization (for a review, see Walker, Freud, Ellis, Fraine, & Wilson, 2019). Although less

extensively, the association between child physical abuse and ASA has also been addressed, and

found to be weaker (Desai, Arias, Thompson, & Basile, 2002; Kimerling, Alvarez, Pavao,

Kaminski, & Baumrind, 2007), or comparable (Messman-Moore, Walsh, & DiLillo, 2010) to the

association between child sexual abuse (CSA) and ASA. At present, hardly any evidence is

available on the impact of other types of CM, such as emotional abuse and neglect, which also

might predict ASA (Widom et al., 2008; Zurbriggen, Gobin, & Freyd, 2010). Moreover, different

types of CM tend to co-occur (Turner, Finkelhor, & Ormrod, 2010), and to have cumulative,

generalized effects (Conway, Raposa, Hammen, & Brennan, 2018; Wilkins, Tsao, Hertz, Davis, &

Klevens, 2014). Consequently, by investigating different types of CM in isolation, past research

might have inadequately represented their relative impact on the risk of ASA (Senn & Carey, 2010).

Therefore, more research should address the relationship between multiple types of CM and ASA.

By doing so, research will clarify whether CSA is, indeed, the strongest predictor of ASA,

compared to and in the presence of other types of CM.

Common Risk Factors for Child Maltreatment and Adult Sexual Assault Victimization

The mechanisms underlying the association between CM and ASA are only partly

understood (Benjet et al., 2015; Messman-Moore & Long, 2003). Investigations of potential causal

pathways have suggested a link between adverse childhood experiences, dysregulation of brain

systems involved in emotional and stress responses, and a host of behaviors potentially increasing

the risk of ASA (Noll & Grych, 2011; Ullman, Peter-Hagene, & Relyea, 2014; Walsh, Galea, &

Koenen, 2012). For instance, CM has been found to increase the likelihood of engaging in risky

sexual behaviors, including early (Fergusson, McLeod, & Horwood, 2013; Thibodeau, Lavoie,

Hébert, & Blais, 2017; Wilson & Widom, 2008) and unprotected (Bensley, Van Eenwyk, &

Simmons, 2000; Lemieux & Byers, 2008) sexual intercourse. In turn, sexual risk-taking has been

found to increase the likelihood of ASA (e.g., Fargo, 2009; Messman-Moore et al., 2010; Van

Bruggen, Runtz, & Kadlec, 2006). However, to our knowledge, no previous study has investigated

the extent to which genetic and environmental risk factors common to CM and ASA might explain

their association. For instance, social isolation or continued proximity to a sexually motivated

perpetrator might confer environmental risk for repeated victimization (Casey & Nurius, 2005;

Leclerc, Wortley, & Smallbone, 2010; Schewe, Riger, Howard, Staggs, & Mason, 2006). Similarly,

heritable traits that are somewhat stable from childhood throughout adulthood and exploited by

perpetrators might confer genetic risk for repeated victimization (e.g., intellectual disabilities;

Fisher et al., 2016). As a result, the possibility of an overlap in the genetic and environmental

influences on CM and ASA should be addressed.

Sex Differences in the Etiology of Adult Sexual Assault Victimization

Sex differences in the prevalence of ASA have been established. Specifically, previous

studies have consistently reported higher prevalence of ASA in women, compared to men (e.g.,

19% vs. 6%, in Black, Basile, Breiding, & Ryan, 2014; 49% vs. 23%, in Breiding, 2015; 56% vs.

21%, in de Haas, van Berlo, Bakker, & Vanwesenbeeck, 2012). Therefore, sex differences might

exist in the risk factors for ASA. First, women and men might be exposed to different genetic risk

factors. Genetic sources of sex differences might include the effect of genes differently expressed in

women and men, and the effect of genes on sex chromosomes (Ratnu, Emami, & Bredy, 2017). In

line with this possibility, sex-specific genetic effects have been estimated on the risk of CSA

(Pezzoli, Antfolk, Hatoum, & Santtila, 2018). Therefore, the possibility of sex-specific genetic

effects on the risk of ASA should also be addressed. Second, women and men might be exposed to

different environmental risk factors. In fact, since men engage in sexually harassing or aggressive

behaviors more than women (Taylor & Gassner, 2010; Tharp et al., 2013), women might be

exposed to the risk of ASA in a wider variety of social environments, compared to men (Butchart,

Phinney, Check, Villaveces, 2004; Campbell, Dworkin, & Cabral, 2009; Ellsberg et al., 2015).

Accordingly, the literature on male ASA has indicated higher prevalence rates within specific,

institutional settings (e.g., prison or military), compared to community settings (for a review, see

Peterson et al., 2011). Third, sex differences in the etiology of ASA might be influenced by sex

differences in other domains, such as CM. Accordingly, one study accounting for multiple types of CM has found a significantly stronger impact of CM on the risk of ASA in women than men (Widom et al., 2008). Thus, research should clarify whether sex differences in the strength of the association between CM and ASA reflect sex differences in the genetic and environmental risk factors common to CM and ASA.

The Current Study

The current study was aimed at investigating the prevalence and etiology of ASA in a large population-based sample. Specifically, we sought to address four main research questions:

RQ1. What is the prevalence of ASA in the Finnish population?

H1. Consistent with past studies, we predicted higher prevalence of non-contact than contact ASA, and higher prevalence of any form of ASA in women, compared to men;

RQ2. To what extent do environmental and, possibly, genetic factors influence the risk of ASA?

H2. Expanding past research, we explored the possibility that genetic factors, in addition to environmental factors, could influence the risk of ASA. Based on genetically informed studies of other forms of interpersonal victimization, we expected small-to-moderate genetic, no shared environmental, and moderate-to-large unique environmental influences on ASA;

RQ3. What is the phenotypic and genetic relationship between CM and ASA?

H3a. Consistent with past studies, we expected higher prevalence of any form of ASA in victims of severe CM, compared to non-victims of severe CM. We considered individuals who experienced at least one type of CM often or very often as victims of severe CM, and the remaining participants as non-victims of severe CM;

H3b. Consistent with past studies, we predicted that CSA would significantly predict ASA. Moreover, expanding past research, we examined the association between multiple types of CM, namely emotional abuse, physical abuse, emotional neglect, and physical neglect, in addition to CSA. Given the limited available evidence, we could not predict whether any type of CM, other than physical abuse, would show a meaningful association with ASA, but we

expected the association between CSA and ASA to be substantially weakened, once

accounting for multiple CM;

H3c. Expanding past research, we investigated whether the association between CM and ASA

would be, at least in part, explained by genetic and environmental risk factors common to

them. Based on previous evidence of large unique environmental influences on child and

adult interpersonal victimization, we predicted a significantly greater overlap in the unique

environmental influences, compared to the genetic influences, on the risk of CM and ASA;

RQ4. Does the etiology of ASA differ between women and men?

H4a. Consistent with past studies, we predicted significant sex differences in the strength of

the phenotypic associations between different types of CM and ASA. However, given the

paucity of previous evidence, we could not predict whether sex differences would emerge for

some types of CM rather than others, nor the direction of such differences;

H4b. Expanding past research, we estimated quantitative sex differences in the genetic and

environmental influences on ASA. Based on previous evidence of women's more

homogeneous exposure to environmental risk of ASA, compared to men, we predicted that

the impact of genetic factors on the risk of ASA would be significantly greater for them;

H4c. Expanding past research, we also explored the possibility of qualitative sex differences

in the genetic influences on ASA. Based on recent evidence of sex-specific effects on CSA,

we hypothesized that sex-specific effects on ASA would also be observed;

H4d. Expanding past research, we inspected sex differences in the extent of the genetic and

environmental overlap between CM and ASA. Given the absence of empirical research, we

could not predict whether the extent of genetic, environmental, or both genetic and

environmental overlap would differ between sexes, nor the direction of such differences.

Methods

Participants

Our sample comprised 12952 Finnish individuals, recruited through the Central Population Registry of Finland, in 2005 and 2006, to participate in the Genetics of Sexuality and Aggression project of the Åbo Akademi University (Turku, Finland). The University's Ethics Committee approved the research plans in accordance with the 1964 Declaration of Helsinki. To analyze sex differences, we split the sample into two groups by sex. Of note, since the Finnish language does not differentiate between sex and gender, we could not isolate participants who self-identified differently from the assigned sex at birth. To analyze differences across cohorts, we split the sample into three groups, comprising individuals aged 18 to 25, 26 to 32, and 33 to 49, respectively. To conduct quantitative genetic analyses, we selected a subset of the entire sample, comprising 9513 twins. Subsampling was based on zygosity, determined with questionnaire items addressing physical resemblance (Sarna, Kaprio, Sistonen, & Koskenvuo, 1978) and validated through genotyping in a portion of the sample ($n = 775$ twin pairs). To address sex differences at the quantitative genetic level, we further split the twin sample in two sub-samples by sex. Sample size, sex, age, and zygosity for the full sample and each sub-sample are reported in Table 1. For more information on procedural aspects pertaining to the data collection as well as on the demographic characteristics of our sample, please refer to Johansson et al. (2013).

Table 1

Sample size, sex, age and zygosity information for the full sample and each sub-sample.

	n	Age			Zygosity		
		Min – Max	*M*	*SD*	MZ	DZ	Other
Full sample	12952	18 – 49	29.23	6.83	3236	6277	3439
Full sample, women	8376	18 – 49	29.12	6.87	2155	3912	2309
Full sample, men	4576	18 – 48	29.42	6.74	1081	2365	1130
Full sample, cohort 1	4467	18 – 25	21.90	2.18	1183	2162	1122
Full sample, cohort 2	4219	26 – 32	28.71	1.93	988	1959	1272
Full sample, cohort 3	4263	33 – 49	37.42	3.04	1063	2155	1045
Twin sample	9513	18 – 43	29.26	6.99	3236	6277	–
Twin sample, women	6067	18 – 43	29.05	7.01	2155	3912	–
Twin sample, men	3436	18 – 43	29.64	6.93	1081	2365	–

Note: *n* = sample size; *M* = mean; *SD* = standard deviation; Other = includes non-twin siblings and unidentified twins.

Materials

One item was used to measure ASA. This item, created for the purpose of our data collection, was inspired by the Sexual Experiences Survey (SES, Koss & Oros, 1982), but substantially simplified in its level of behavioral specificity. Participants were asked to choose one or more given alternatives to answer the question: "Have you as an adult (over the age of 15) become the target of sexual harassment or abuse?". Possible responses included "No", coded as "0"; "I have been verbally harassed" and "other", coded as "1"; "I have been touched or kissed against my will" and "There has been an attempt to force me to engage in oral, vaginal, or anal sex", jointly coded as "2"; and, lastly, "I have been forced to engage in oral sex", "I have been forced to engage in vaginal sex", and "I have been forced to engage in anal sex", coded as "3". In case participants gave multiple answers, only the highest score was assigned. Before phenotypic and genetic analyses, we standardized the raw scores to the ASA item into a z-score.

To measure CM, we used the Childhood Trauma Questionnaire Short Form (CTQ-SF; Bernstein et al., 2003). The CTQ-SF includes five subscales, each comprising five items, scored on a five-point scale ranging from "never true" to "very often true". Each subscale measures a different

type of CM: CSA, emotional abuse, physical abuse, emotional neglect, and physical neglect. Responses to the CTQ-SF were prepared for statistical analyses by imputing missing data with the Expectation-Maximization procedure (1.5% of responses, $SD = 1.9$, min = 1.2, max = 2.2), and by reverse-coding all emotional neglect and two physical neglect items, so that higher scores indicated higher levels of CM. To obtain descriptive statistics, we created a dichotomous score, which categorized participants based on whether they had experienced severe CM or not. Specifically, participants who scored 4 or 5 on any of the CTQ-SF items (i.e., "often true" or "very often true"), were categorized as victims of severe CM. Participants who scored 3 or less ("sometimes true", "rarely true", or "never true") were categorized as non-victims of severe CM. To conduct all subsequent phenotypic and genetic analyses, we created five composite scores corresponding to the CTQ-SF sub-scales. Starting from the individual items of each sub-scale, we obtained dimension reduction using a maximum likelihood factor analysis, extracting a single factor, and saving the score derived using the Bartlett method. Items from the same sub-scale were positively correlated (r_{RANGE}: CSA = .50 – .73, emotional abuse = .38 – .59, physical abuse = .28 – .55, emotional neglect = .44 – .66, physical neglect = .15 – .36), but the determinants of the correlation matrices were considerably larger than .00001 (CSA = .06, emotional abuse = .19, physical abuse = .25, emotional neglect = .11, physical neglect = .61). Thus, we ruled out extreme collinearity and singularity (Field, 2013). Composite scores showed good internal consistency (Cronbach's alpha: CSA = .90; emotional abuse = .83; physical abuse = .76; emotional neglect = .86, physical neglect = .62). Since the distributions of CSA and physical abuse were positively skewed (Skewness = 5.63, $SE = .02$ and Kurtosis = 35.40, $SE = .04$ for CSA; Skewness = 3.64, $SE = .02$ and Kurtosis = 16.86, $SE = .04$ for physical abuse), we transformed these two scores using Log10 transformation, obtaining acceptable distributions (Skewness = 3, $SE = .02$ and Kurtosis = 8.17, $SE = .04$ for CSA; Skewness = 1.34, $SE = .02$ and Kurtosis = 1.17, $SE = .04$ for physical abuse; Kline, 2016). Before all analyses involving the entire sample, we regressed out sex and age from the scores of ASA and CM. From the same variables, we regressed out age only, before analyses exploring sex differences.

Statistical Analyses

To address our first research question on the prevalence of ASA, we estimated its prevalence and mean levels in the entire sample, as well as in sub-samples by sex and age. To address our second research question on the etiology of ASA, we estimated two univariate twin models. The first, ACE, model, decomposed the variance of the ASA score into additive genetic (A), shared environmental (C), and unique environmental (E) influences. The second, ADE, model, estimated dominant genetic (D), instead of shared environmental (C) influences. Of note, in twin models, measurement error variance is included in the E term. To address our third research question on the phenotypic and genetic relationship between CM and ASA, we began by inspecting its prevalence and mean levels in victims and non-victims of severe CM. Then, we estimated the phenotypic correlations between the five types of CM and the z-score of ASA, and we regressed it on each composite score of CM, separately (univariate regressions) as well as together (multivariate regression). Since our predictors were continuous variables, and our outcome an ordinal variable, we used ordered logit regression with maximum likelihood estimation, considered adequate with multilevel data (i.e., participants were nested within families), and a small number of categories in the ordinal outcome (Bauer & Sterba, 2011). Then, we examined the extent of the genetic and environmental influences common to each type of CM, separately (bivariate twin models), as well as together (common pathway twin model). With a series of bivariate twin models, we decomposed the A, C, and E terms, influencing each type of CM, into additional terms, influencing ASA (Cholesky decomposition) and we estimated the genetic, shared environmental and unique environmental correlations between each type of CM and ASA (r_G, r_C, and r_E, respectively). Lastly, with a common pathway twin model, we estimated one latent factor, accounting for the etiology common to all types of CM, and we decomposed its A, C, and E terms into additional terms, influencing ASA. To address our fourth research question on sex differences in the etiology of ASA, we repeated phenotypic and genetic analyses in women and men separately. Once we determined quantitative sex differences in the genetic and environmental influences on ASA, we

explored the possibility of qualitative sex differences, using sex limitation models. For details on how to estimate all of these twin models, see Neale and Cardon (2013).

We used maximum likelihood estimation and bootstrapped standard errors (1000 resamples with replacement) to obtain genetic and environmental estimates. Furthermore, we used robust estimation for nested sex limitation models (i.e., with the same variables, but different parameter constraints), and we compared them using the Satorra-Bentler scaled χ^2 difference test (Satorra & Bentler, 2010). To compare group means, regression coefficients, and heritability estimates, we computed 95% CIs around the estimates, and checked for overlap in the CIs. No overlap, or CIs just touching, indicate significance (Cumming & Finch, 2005). We further calculated standardized measures of effect size (*d*; Cohen, 1988) for significant comparisons between group means, and unstandardized measures of effect size (*ES*; Kelley & Preacher, 2012) for significant comparisons between individual coefficients. SPSS Statistics for Macintosh, 23.0 (IBM Corp., released 2015) was used for preliminary data handling. Analyses were performed in Mplus 8 (Muthén & Muthén, 1998-2017), and *R* environment for statistical computing, 3.3.2 (*R* Core Team, 2016), packages Hmisc, 4.1-1 (Harrell, 2018), MASS, 7.3.45 (Ripley et al., 2013), and umx, 2.8.5 (Bates, 2018). Annotated scripts and output files are available on Open Science Framework (osf.io/7qxh3). For reasons of confidentiality, the dataset is available upon request.

Results

Phenotypic Results

Descriptives. To test our hypothesis H1, we estimated the prevalence of ASA (Table 2) and its mean levels in the full sample as well as in sub-samples by sex and age. Most respondents (73%, $n = 7031$) reported no victimization. The remaining 27% ($n = 2616$), reported at least one form of ASA. Contrary to what expected, a slightly larger proportion reported having been touched or kissed against their will (12%, $n = 1140$), than having been verbally harassed or subject to some "other" form of sexual assault (10%, $n = 996$). A smaller proportion reported having been forced to engage in oral, vaginal or anal sex (3%, $n = 288$). The remaining participants reported that someone

attempted to force them to engage in penetrative sex (2%, n = 192). Prevalence differed considerably between women and men. More than three times more women than men reported at least one form of ASA (36%, n = 2243 women, vs. 11%, n = 373 men), and thirteen times more women than men reported having been forced to engage in penetrative sex (4% vs. 0.3%). Also, women reported significantly higher mean levels on the ASA item, compared to men (M = 1.77, SD = 1.34, vs. M = 1.18, SD = .058; p < .01, d = .05). Instead, no significant difference emerged between cohorts, in neither the prevalence nor the mean levels of ASA.

To test our hypothesis H3a, we estimated the prevalence of ASA (Table 2) and its mean levels (Table 3) in victims and non-victims of severe CM. In total, 42% of our participants (n = 5428, 43% of women and 40% of men) reported at least one severe experience of CM. Consistent with our prediction, the prevalence of ASA was higher in victims of severe CM, compared to non-victims of severe CM, across sexes and cohorts. Specifically, among victims of severe CM, 34% (n = 1299, 44% of women and 14% of men) experienced any form of ASA, compared to the 23% (n = 1317, 30% of women, 9% of men) of non-victims. In particular, among victims of CM, victims of severe CSA reported the highest prevalence of ASA (41%), followed by victims of severe emotional abuse (28%), physical abuse (26%), emotional neglect (24%), and physical neglect (22%).

Table 2

Prevalence of adult sexual assault victimization

	ASA response	Women		Men		Cohort 1		Cohort 2		Cohort 3	
		n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
	Never	7031	73	4065	64	2966	89	3068	72	2949	73	1014	77
	Verbal/Other	996	10	829	13	167	5	484	11	428	11	84	6
	Touched/Kissed	1140	12	950	15	190	5.7	541	12	459	11	140	11
	Attempted	192	2	185	3	7	0.2	77	2	85	2	30	2
	Forced	288	3	279	5	9	0.3	113	3	121	3	54	4
	Any ASA	2616	27	2243	36	373	11	1215	28	1093	27	308	23
CM_V	Never	2542	66	1454	56	1088	86	1047	64	1063	67	432	70
	Verbal/Other	460	12	379	15	81	6	220	14	195	12	45	7
	Touched/Kissed	551	14	465	18	86	7	251	15	217	14	83	14
	Attempted	105	3	102	4	3	0	41	2	47	3	17	3
	Forced	183	5	177	7	6	1	74	5	71	4	38	6
	Any ASA	1299	34	1123	44	176	14	586	36	530	33	183	30
CM_{NV}	Never	4489	77	2611	70	1878	90.5	2021	76.3	1886	77	582	82
	Verbal/Other	536	9	450	12	86	4.1	264	10	233	9	39	6
	Touched/Kissed	589	10	485	13	104	5	290	11	242	10	57	8
	Attempted	87	2	83	2	4	0.2	36	1.3	38	2	13	2
	Forced	105	2	102	3	3	0.2	39	1.4	50	2	16	2
	Any ASA	1317	23	1120	30	197	9	629	24	563	23	125	18

Note: Cohort 1 = participants aged 18 – 25; Cohort 2 = participants aged 26 – 32; Cohort 3 = participants aged 33 – 49; CM_V = victims of severe child maltreatment; CM_{NV} = non-victims of severe child maltreatment; ASA = Adult Sexual Assault; Never = no abuse reported; Verbal/Other = "I have been verbally harassed" and/or "other"; Touched/Kissed = "I have been touched or kissed against my will"; Attempted = "There has been an attempt to force me to oral sex" and/or "vaginal sex" and/or "anal sex"; Forced = "I have been forced to oral sex", and/or "vaginal sex", and/or "anal sex"; Any ASA = cumulative prevalence.

Table 3

Descriptive statistics of adult sexual assault victimization by child maltreatment history

| | Women | | | | Men | | Cohort 1 | | Cohort 2 | | Cohort 3 | |
	CM_V	CM_{NV}	CM_V	CM_{NV}	CM_V	CM_{NV}	CM_V	CM_{NV}	CM_V	CM_{NV}	CM_V	CM_{NV}
n	3841	5806	2577	3731	1264	2075	1633	2650	1593	2449	615	707
M	1.75*	1.44	2.01*	1.60	1.23*	1.16	1.77*	1.44	1.73*	1.45	1.76*	1.39
SD	1.36	1.01	1.53	1.16	0.66	0.53	1.33	0.97	1.34	1.04	1.47	1.02
SE	0.02	0.01	0.03	0.02	0.02	0.01	0.03	0.02	0.03	0.02	0.06	0.04
Var	1.84	1.01	2.34	1.35	0.44	0.28	1.78	0.95	1.79	1.07	2.15	1.04
CI_L	1.71	1.41	1.95	1.56	1.19	1.14	1.71	1.40	1.66	1.41	1.64	1.32
CI_U	1.79	1.47	2.07	1.64	1.27	1.18	1.83	1.48	1.80	1.49	1.88	1.46
d		0.27		0.39		0.07		0.29		0.24		0.30

Note: Cohort 1 = participants aged 18 – 25; Cohort 2 = participants aged 26 – 32; Cohort 3 = participants aged 33 – 49; CM_V = victims of severe child maltreatment; CM_{NV} = non-victims of severe child maltreatment; *M* = Mean, *SD* = Standard Deviation, *SE* = Standard Error, Var = variance; CI_L = 95% Confidence Interval around the mean, lower bound; CI_U = 95% Confidence Interval around the mean, upper bound; * = statistically significantly larger at *p* < .01; *d* = standardized measure of effect size.

Regressions. To test our hypothesis H3b, we first inspected the zero-order correlations between each type of CM and ASA. Emotional abuse showed the largest coefficient ($r = .23$), followed by CSA ($r = .18$), physical abuse ($r = .17$), emotional neglect ($r = .16$), and physical neglect ($r = .12$). Then, we tested the proportional odds (or parallel regression) assumption of ordered logit regression. The assumption held for all predictors, meaning that, for a one unit increase in the log odds scale in each predictor, the odds of reporting an ASA score of 1, vs. the odds of reporting a score of 2 or 3, corresponded to the odds of reporting an ASA score of 1 or 2, vs. the odds of reporting a score of 3. Next, we estimated the contribution of each type of CM on ASA separately, with a series of univariate ordinal logistic regressions (Table 4). When considered in isolation, all types of CM were positive and statistically significant predictors. Emotional abuse was the strongest predictor, followed by CSA, emotional neglect, and physical abuse and neglect. For a one unit increase in each predictor, the odds of reporting more severe ASA were more than one time greater. Then, we ran a multivariate ordinal logistic regression including all predictors in the model simultaneously. Despite accounting for other types of CM, emotional abuse remained the strongest predictor, followed by CSA, physical abuse, and emotional neglect. The coefficient for physical neglect became negative. Notably, only CSA, emotional, and physical abuse remained statistically significant predictors. For a one unit increase in each significant predictor, the odds of reporting more severe ASA were slightly smaller than those observed in the univariate models.

To test our hypothesis H4a, we also ran the univariate and multivariate regressions in women and men separately (Table 4). For women, all predictors in the univariate models were significant. For men, only emotional and physical abuse, as well as physical neglect to a smaller extent, were significant. Statistically significant sex differences emerged in the size of all univariate coefficients, except for physical abuse, and indicated a stronger impact of those types of CM on the likelihood of experiencing ASA in women. For women, emotional abuse was the strongest predictor, followed by CSA, physical abuse, emotional and physical neglect. For men, emotional and physical abuse showed the largest and approximately equivalent coefficients, followed by CSA, physical and

emotional neglect. Again, the proportional odds ratios were larger than one. The multivariate results confirmed the univariate trends: Emotional abuse was the strongest predictor of ASA in women, although CSA was also statistically significant, and physical abuse was the strongest and only significant predictor in men. The proportional odds ratios were again larger than one, except for physical neglect in men, and larger for women compared to men, except for physical abuse.

Table 4

Univariate and multivariate regression coefficients

	Univariate Models								Multivariate Models							
	β	SE	CI_L	CI_U	p	OR	CI_L	CI_U	β	SE	CI_L	CI_U	p	OR	CI_L	CI_U
Entire N																
EA	.44	.02	.40	.48	<.01	1.55	1.49	1.62	.33	.03	.27	.39	<.01	1.39	1.32	1.48
CSA	.34	.02	.30	.38	<.01	1.41	1.35	1.46	.22	.02	.18	.26	<.01	1.25	1.19	1.30
PA	.27	.02	.23	.31	<.01	1.31	1.26	1.36	.06	.02	.02	.11	<.01	1.07	1.02	1.11
EN	.29	.02	.25	.32	<.01	1.33	1.28	1.38	.04	.03	-.01	.09	.15	1.04	.99	1.09
PN	.22	.02	.18	.26	<.01	1.24	1.20	1.29	-.03	.02	-.08	.02	.26	.97	.93	1.02
Women																
EA	.35	.03	.30	.40	<.01	1.42	1.34	1.50	.25	.04	.17	.33	.18	1.28	1.18	1.39
CSA	.29	.03	.23	.34	<.01	1.33	1.26	1.40	.18	.03	.13	.24	.22	1.20	1.13	1.27
PA	.24	.03	.19	.30	<.01	1.27	1.21	1.35	.06	.04	-.01	.13	.01	1.06	.99	1.13
EN	.24	.03	.19	.30	<.01	1.27	1.21	1.34	-.00	.04	-.08	.07	.24	1.00	.92	1.08
PN	.23	.03	.18	.29	<.01	1.26	1.20	1.33	.06	.04	-.01	.12	.64	1.06	.99	1.13
Men																
EA	.16*	.05	.07	.26	<.01	1.18	1.07	1.30	.09	.07	-.04	.22	.18	1.10	.96	1.25
CSA	.11*	.06	-.00	.22	.06	1.11	1.00	1.24	.07	.06	-.04	.19	.22	1.07	.96	1.20
PA	.16	.04	.08	.24	<.01	1.17	1.09	1.27	.12	.05	.03	.22	.01	1.13	1.03	1.24
EN	.04*	.04	-.04	.12	.37	1.04	.96	1.13	-.06	.05	-.17	.04	.24	.94	.84	1.04
PN	.08*	.04	.00	.16	.05	1.08	1.00	1.17	.02	.05	-.07	.12	.64	1.02	.93	1.13

Note: β = Standardized ordered log-odds regression coefficients; *SE* = Standard Error; CI_L = 95% Confidence Interval around the estimate, lower bound; CI_U = 95% Confidence Interval around the estimate, upper bound; *OR* = Odds Ratio; EA = (Child) Emotional Abuse; CSA = Child Sexual Abuse; PA = (Child) Physical Abuse; EN = (Child) Emotional Neglect; PN = (Child) Physical Neglect. * = statistically significantly smaller at $p < .01$, unstandardized measures of effect size: EA = .19; CSA = .13; EN = .20; PN = .15.

Genetic Results

Model fit indices for the estimated twin models are reported in Table 5, whereas squared

standardized A, C, and E path and cross-path coefficients are reported in Table 6.

Table 5

Model fit indices, twin models

	$\chi2$	df	p	CFI	RMSEA	90%CI	AIC	BIC
Univariate ACE (ASA)	28.03	6	< .01	.50	.04	.03 – .06	18705.58	18731.40
Univariate ADE (ASA)	20.03	6	< .01	.57	.03	.02 – .05	18704.21	18730.03
Bivariate 1 (EA – ASA)	42.64	17	< .01	.97	.02	.01 – .03	44262.96	44337.73
Bivariate 2 (PA – ASA)	40.14	17	< .01	.97	.02	.02 – .03	44531.55	44606.31
Bivariate 3 (CSA – ASA)	72.43	17	< .01	.89	.03	.02 – .04	44847.36	44922.12
Bivariate 4 (EN – ASA)	55.06	17	< .01	.96	.03	.02 – .03	44351.88	44426.66
Bivariate 5 (PN – ASA)	78.97	17	< .01	.94	.03	.03 – .04	44369.53	44444.30
Common Pathway Model	1012.60	146	< .01	.95	.04	.04 – .05	136584.67	136815.86
General Sex Limitation	16.19	16	.44	.99	.00	.00 – .03	17093.01	17151.11
Nested Sex Limitation	20.24	18	.32	.93	.01	.00 – .03	17093.90	17139.09

Note: ACE = Twin model estimating additive genetic (A), shared environmental (C) and unique

environmental (E) effects. ADE = Twin model estimating dominance (D) instead of shared

environmental (C) effects. ASA = Adult Sexual Assault; EA = (Child) Emotional Abuse; CSA =

Child Sexual Abuse; PA = (Child) Physical Abuse; EN = (Child) Emotional Neglect; PN = (Child)

Physical Neglect; Nested Sex Limitation = Common Effects Model; *df* = Degrees of Freedom; *CFI*

= Comparative Fit Index; *RMSEA* = Root Mean Square Error of Approximation; *CI* = Confidence

Intervals; *AIC* = Akaike Information Criteria; *BIC* = Bayes Information Criteria. Scaling correction

factors: General Sex Limitation = 2.23, Nested Sex Limitation = 2.03.

Table 6

Standardized additive genetic, shared environmental, and unique environmental path and cross-path coefficients

	A^2	A^2 SE	A^2 p	A^2 CI_L	A^2 CI_U	C^2	C^2 SE	C^2 p	C^2 CI_L	C^2 CI_U	E^2	E^2 SE	E^2 p	E^2 CI_L	E^2 CI_U
						Univariate Paths									
ASA	.22	.04	.00	.14	.30	.00	.00	.76	.00	.00	.78	.02	.00	.74	.82
						Cholesky Cross-Paths									
EA – ASA	.03	.06	.00	-.08	.14	.01	.10	.38	-.18	.20	.02	.04	.00	-.05	.09
PA – ASA	.03	.09	.05	-.14	.20	.00	.08	.42	-.14	.15	.01	.04	.05	-.07	.08
CSA – ASA	.03	.05	.00	-.07	.12	.00	.09	.99	-.17	.17	.01	.04	.00	-.06	.08
EN – ASA	.02	.07	.07	-.12	.15	.01	.08	.26	-.14	.16	.00	.03	.11	-.06	.07
PN – ASA	.01	.10	.31	-.19	.21	.01	.07	.17	-.13	.15	.00	.04	.17	-.07	.07
CM – ASA	.03	.07	.01	-.11	.17	.01	.08	.20	-.15	.17	.02	.04	.00	-.06	.10

Note: A^2 = Additive genetic; C^2 = Shared Environmental, E^2 Unique Environmental; *SE* = Standard Error; *p* = two-tailed *p* value for the estimate; CI_L = Confidence Interval around the estimate, lower bound; CI_U = Confidence Interval around the estimate, upper bound; ASA = Adult Sexual Assault; EA = (Child) Emotional Abuse; CSA = Child Sexual Abuse; PA = (Child) Physical Abuse; EN = (Child) Emotional Neglect; PN = (Child) Physical Neglect; CM = Latent factor of Child Maltreatment.

Univariate Twin Models. To test our hypothesis H2, we first obtained an indication of the sources of variation in ASA by comparing the magnitude of the phenotypic MZ and DZ cross-twin within-trait correlations. Correlations were larger in MZ than DZ twins (*r* = .22, and *r* = .08, respectively), suggesting additive genetic effects. DZ correlations were less than half the MZ correlations, indicating dominant genetic rather than shared environmental influences. Thus, in two separate models, we decomposed the variance of ASA into A, C, and E components, as well as into A, D, and E components. The ACE model indicated that 22% of the variance in ASA could be explained by additive genetic factors, and the remaining 78% by unique environmental factors. As could be expected based on the pattern of MZ-DZ correlations, no shared environmental influences were estimated. The ADE model confirmed that the largest proportion of the variation in ASA

(76%) could be accounted for by unique environmental sources. Moreover, this model indicated larger dominant genetic than additive genetic influences on the risk of ASA ($D^2 = 15\%$, $A^2 = 9\%$).

To test our hypothesis H4b, we further estimated the two univariate models in women and men separately. A substantial difference between same-sex and opposite-sex DZ twins emerged in the cross-twin within-trait correlations. Correlations were positive in same-sex DZ twins ($r = .16$ in women, and $r = .07$ in men), and negative in opposite-sex DZ twins ($r = -.02$), suggesting that genetic factors influencing ASA in women could be, at least in part, qualitatively different from the those influencing ASA in men. The ACE model indicated that, for women, 28% of individual differences in the likelihood of experiencing ASA could be explained by additive genetic factors, 1% by shared environmental factors, and the remaining 71% by unique environmental factors. For men, we estimated no additive genetic effects, and, instead, only shared and unique environmental effects ($C^2 = 5.5\%$, $E^2 = 94.5\%$). As predicted, women showed significantly larger additive genetic influences on ASA compared to men ($p < .01$; $ES = .28$), who, on the contrary, showed significantly larger unique environmental influences ($p < .01$; $ES = .23$). No dominant genetic effect emerged when we estimated the ADE model separately in the two sex groups.

Bivariate Twin Models. To test our hypothesis H3c, we first inspected the etiological sources of covariation between each type of CM individually and ASA, by comparing the magnitude of the MZ and DZ cross-twin cross-trait correlations. For all types of CM, we observed larger MZ than DZ correlations, indicative of genetic influences common to CM and ASA. We then estimated the additive genetic, shared and unique environmental correlations between each type of CM separately and ASA. All types of CM showed small-to-moderate positive genetic correlations ($r_G = .37$ for emotional abuse, .36 for CSA and physical abuse, .28 for emotional neglect, and .22 for physical neglect). One negative shared environmental correlation emerged between CSA and ASA ($r_C = -.33$), while the remaining shared environmental correlations were estimated at unity, implying identical shared environmental influences on child and adult victimization. Unique environmental correlations were small ($r_E = .16$ for emotional abuse, .12 for CSA, .08 for physical

abuse, .06 for emotional neglect, and .05 for physical neglect). We detected a statistically

significant overlap with ASA in the etiological factors only for CSA ($A^2 = 3\%$, $E^2 = 1\%$) and

emotional abuse ($A^2 = 3\%$, $E^2 = 2\%$). The remaining cross-paths were non-significant. Thus,

bivariate models suggested that the shared etiology between CM and ASA was consisting of

common additive genetic and unique environmental influences on child sexual and emotional abuse

and ASA. We also performed the Cholesky decompositions in women and men separately (H4d)

and found no significant sex differences in the etiological cross-paths.

Common Pathway Model. To test our hypothesis H3c, we further estimated one latent

factor accounting for the covariance between all available types of CM. Then, we decomposed the

A, C, and E terms predicting this latent factor of CM into additional A, C, and E terms predicting

ASA. When accounting for the covariance between multiple types of CM, the shared additive

genetic and unique environmental etiology of CM and ASA was small but significant ($A^2 = 3\%$, E^2

$= 2\%$). To test our hypothesis H4d, we also tested the common pathway model in women and men

separately. Again, no statistically significant sex difference emerged in the extent of the additive

genetic and unique environmental overlap between the latent factor of CM and ASA. Only the

extent of the shared environmental overlap differed significantly between sexes, but it was very

marginal nonetheless ($C^2 = 0.8\%$ in women, $C^2 = 1.2\%$ in men).

Sex Limitation Models. In light of the sex differences emerged in the heritability of ASA,

we further tested our hypothesis H4c. To do so, we estimated two sex limitation models: a "general

sex limitation" and a nested "common effects" model (see Table 7). With the general sex limitation

model, we decomposed the A, C and E variance components of ASA, allowing them to differ

between sexes, and estimated an additional component, reflecting sex-specific genetic effects. With

the common effects model, we constrained the sex-specific component to zero, estimating only

genetic influences common to women and men. Model fit comparison indicated that the sex-

specific component was not statistically significant, and, thus, that the same genes influenced the

risk of ASA in both sexes. In line with the univariate model, the sex limitation model indicated

significantly larger unique environmental influences on ASA in men, but, in contrast with the univariate model, it did not indicate significantly larger additive genetic influences in women. Inspecting the estimates from the general sex limitation model clarified that, in women, the sex-specific genetic component had a significantly larger impact on the risk of ASA than the common genetic component ($ES = .28$). This might explain why larger genetic influences emerged for women when sex-limited and common genetic effects were estimated jointly. Overall, these results indicated that unique environmental factors mainly contributed to sex differences in the risk of ASA.

Table 7

Standardized estimates, sex-limitation models

Model	Sex	A^2	A^2 SE	A^2 p	A^2 CI_L	A^2 CI_U	A'^2	A'^2 SE	A'^2 p	A'^2 CI_L	A'^2 CI_U	C^2	C^2 SE	C^2 p	C^2 CI_L	C^2 CI_U	E^2	E^2 SE	E^2 p	E^2 CI_L	E^2 CI_U
GSL	W	.00	.06	.85	-.12	.12	.28	.12	<.01	.04	.52	.01	.17	.61	-.32	.34	.71*	.03	<.01	.65	.77
	M	.00	.11	.85	-.22	.22	.00	.16	.99	-.31	.31	.06	.13	.06	-.19	.31	.94	.03	<.01	.88	1.00
CE	W	.02	.08	.06	-.14	.18	-	-	-	-	-	.20	.04	<.01	.12	.28	.78*	.02	<.01	.74	.82
	M	.08	.15	.05	-.21	.37	-	-	-	-	-	.01	.09	.29	-.17	.19	.91	.04	<.01	.83	.99

Note: GSE = General Sex Limitation model; CE = Common Effects model; W = Women; M = Men; A^2 = Additive genetic; A'^2 = Sex-limited genetic effects; C^2 = Shared Environmental, E^2 Unique Environmental; SE = Standard Error; p = two-tailed p value for the estimate; CI_L = 95% Confidence Interval around the mean, lower bound; CI_U = 95% Confidence Interval around the mean, upper bound. * = sex difference significant at $p < .01$, unstandardized measures of effect size: .23 (GSL), .13 (CE).

Discussion

The Prevalence of Adult Sexual Assault Victimization in the Finnish Population

The first aim of the present investigation was to estimate the prevalence of different forms of ASA victimization in a representative sample of the Finnish population (Johansson et al., 2013). In our sample, the prevalence of any form of ASA (27%) was comparable to that in other population-based Western samples (e.g., 22.5% in Breiding, 2015; 26.7% in Smith et al., 2017), and considerably larger than those obtained from police statistics (e.g., 0.005% to 0.05% of the European population between 2007 and 2011; Aebi et al., 2014, p. 27). This result probably reflects both under-reporting to the authorities (Lonsway & Archambault, 2012) as well as the fact that not all forms of ASA measured here constituted sex offences as legally defined. We further addressed the prevalence of distinct forms of ASA. Our results were in contrast with previous studies that indicated higher prevalence of non-contact than contact ASA (Breiding, 2015; Smith et al., 2017). In fact, more participants reported having been touched or kissed against their will, than having been verbally harassed. On the other hand, the cumulative prevalence of attempted or completed forced sex (5%) was in line with the results of the WHO Report on Violence and Health with respect to the Finnish population (5.9%; Krug et al., 2002). Despite being the least prevalent type of ASA, forced sex represents a serious threat for the health of the victim (Dworkin et al., 2017). Thus, investigating its prevalence and risk factors is crucial to improve prevention. In addition, our results point to the need for increased empirical and public attention to putatively milder forms of ASA, such as verbal harassment and unwanted touching or kissing. In fact, such forms of ASA might be especially widespread in the population. However, if not sufficiently acknowledged, they cannot be successfully prevented. Furthermore, we investigated whether the prevalence and mean levels of ASA differed between women and men. Consistent with previous evidence (e.g., Black et al., 2014; Breiding, 2015; de Haas et al., 2012), the estimated prevalence of any form of ASA was considerably higher in women, compared to men (36% vs. 11%). This result was especially marked for forced penetrative sex, arguably the most intrusive form of ASA (4% vs. 0.3%). Several factors

might contribute to the higher rates of victimization in women, including sexual strategies, implicit norms surrounding sexual socialization, and sexist ideology (Buss & Schmitt, 2016; Butchart et al., 2004; Campbell et al., 2009). As a result, the need for evidence-based prevention of gendered violence is compelling (Ellsberg et al., 2015).

Unsafe Environments Influence the Risk of Adult Sexual Assault More than Victim's Heritable Traits

The second aim of the present study was to explore the extent to which genetic and environmental factors influenced the risk of ASA victimization. In fact, compared to the available empirical evidence on the genetic and environmental risk factors for sexual perpetration (e.g., Frisell, Lichtenstein, & Långström, 2011; Långström, Babchishin, Fazel, Lichtenstein, & Frisell, 2015, Pittner et al., 2019), evidence on the genetic and environmental risk factors for sexual victimization was particularly scarce (Sartor et al., 2012, Stein et al., 2002). Our univariate genetic results suggested that unique environmental factors might exert the largest influence on the risk of ASA, whereas additive and dominant genetic factors, as well as features of the rearing environment, might have significantly smaller impact. As a result, to improve prevention, more research should investigate the environmental conditions where the risk of ASA might be increased (Jewkes, Fulu, Roselli, & Garcia-Moreno, 2013; Tharp et al., 2013) and evaluate the effectiveness of environment-level prevention practices (DeGue et al., 2012). In addition, our findings suggest that systematic research on the behavioral profiles that might be preferentially targeted by sexually motivated perpetrators is also needed, as it might inform individual-level prevention strategies.

Child Maltreatment and Adult Sexual Assault Victimization: New Associations and Distinct Risk Pathways

The third aim of the present study was to investigate the phenotypic and genetic association between CM and ASA. Past research has indicated that victims of CM are at heightened risk of later victimization (e.g., Miron & Orcutt, 2014; Widom et al., 2008; Werner et al., 2016). In line with this evidence, we found higher prevalence and mean levels of ASA in participants who experienced

at least one instance of severe CM, compared to non-victims of severe CM, irrespective of their sex and age. However, the majority of previous studies have focused exclusively on sexual revictimization (Kimerling et al., 2007; Werner et al., 2016), and have hypothesized a continuity from CSA to ASA via unsafe sexual conduct (Messman-Moore et al., 2010). Instead, based on evidence that different types of CM tend to co-occur (Turner et al., 2010) and to have cumulative and generalized harmful effects (Conway et al., 2018; Wilkins et al., 2014), we addressed the relationship between multiple types of CM and ASA. Among victims of CM, victims of severe CSA did also report the highest prevalence of ASA. However, accounting for multiple types of CM explained the variance in ASA better than CSA alone. Most interestingly, emotional abuse emerged as the stronger predictor of ASA compared to any other type of CM, including CSA. Emotional abuse has been shown to represent a worldwide, and often underestimated, problem (Stoltenborgh, Bakermans-Kranenburg, Alink, & van Ijzendoorn, 2012). Indeed, it has hardly ever been examined as a risk factor for ASA victimization (Zurbriggen et al., 2010). Our findings suggest that therapeutic interventions for emotional abuse survivors should account for their increased vulnerability to subsequent victimization. Furthermore, previous literature has failed to clarify whether similar risk factors might influence victimization in childhood and adulthood (Sartor et al., 2012). As a result, why some individuals tend to be repeatedly victimized remains unclear (Benjet et al., 2015). In particular, to the best of our knowledge, no previous study has investigated whether the association between CM and ASA could be partly explained by overlapping genetic and environmental influences. In fact, both the risk of experiencing sexual abuse as a child (Pezzoli et al., 2018) and as an adult (e.g., Stein et al., 2002) has been shown to be influenced by genetic factors. Our results indicated significant additive genetic and unique environmental influences common to CM and ASA. A meaningful overlap with ASA in such etiological influences emerged for two types of CM, namely CSA and emotional abuse, as well as for multiple victimization. These results indicated that the associations between child emotional and sexual abuse and ASA might be explained by risk factors common to them. Instead, other types of CM might per se represent risk

factors for subsequent interpersonal violence, through direct or indirect influences on victims'

vulnerability to unfavorable environments, where the risk of ASA victimization is increased.

Different Factors Influence the Risk of Adult Sexual Assault Victimization in Women and Men

The fourth aim of the present study was to explore the possibility of sex differences in the

etiological influences on ASA, as well as in its phenotypic and genetic associations with CM. First,

we clarified that sex differences in the prevalence of ASA might originate from differences in the

relative impact of genetic and environmental risk factors. In fact, our univariate genetic results

indicated that the risk of ASA might be influenced by genetic factors significantly more in women,

and by unique environmental factors significantly more in men. Furthermore, our study was the first

to test the hypothesis of a sex-limited expression of this genetic vulnerability. The sex-limitation

analyses, however, indicated that the same sets of genes might influence the risk of ASA in both

sexes. Therefore, our findings did not suggest that sex-specific heritable traits increase women's

susceptibility to ASA. Rather, the larger heritability estimates might result from the more

persistently unsafe surroundings to which women are exposed (i.e., from their more homogeneous

environment; Johnson, Turkheimer, Gottesman, & Bouchard, 2009). Second, among victims of

CM, we found that women and men were exposed to a proportionate risk of ASA. However, we

also found that emotional abuse most strongly predicted ASA in women, and physical abuse in

men. This result suggests that emotional and physical abuse, the types of CM most frequently

reported by women and men, respectively (Stoltenborgh, Van Ijzendoorn, Euser, & Bakermans-

Kranenburg, 2011; Thompson, Kingree, & Desai, 2004), might also have the strongest long-term

influence on their risk of ASA. Lastly, we investigated, and ruled out, the possibility of sex

differences in the genetic and environmental risk factors common to CM and ASA. Thus, future

research should explore alternative sex-specific risk pathways underlying repeated victimization.

Limitations

Despite the multitude of impactful findings, the current study was not without limitations. Firstly, we employed a single item to measure ASA, rather than the full Sexual Experiences Survey. Although we were still able to address different forms of ASA, the behavioral specificity of our item was substantially decreased compared to the original survey. As a result, we cannot assume that comparable results would be obtained using more detailed and comprehensive scales. For instance, we were not able to measure the age at which ASA had occurred. Thus, we could not corroborate past research suggesting that sexual victimization during adolescence is associated with an increased risk of sexual revictimization in early adulthood (Humphrey & White, 2000), possibly acting as a mediator between CSA and ASA (Arata, 2002). Also, we did not measure characteristics of the perpetrator, such as sex and sexual orientation. Thus, we could not substantiate previous evidence that ASA is mostly perpetrated by heterosexual men (Tharp et al., 2013; Turchik & Edwards, 2012). Similarly, we did not account for the sexual orientation of the victim, which also represents a risk factor for ASA (Johnson, Matthews, & Napper, 2016). Most importantly, we were not able to discriminate between intimate partner violence and non-partner violence, and between genetically related and unrelated perpetrators. Since ASA perpetrated by an intimate partner results less often in a complaint (Novisky & Peralta, 2015), more studies on this form of ASA in population-based surveys are needed. Moreover, while CSA tends to be perpetrated by individuals outside of the family (Gilbert et al., 2009), the extent to which ASA might be perpetrated by genetically related and unrelated individuals is less clear. In addition, despite Finland is known to be among the Countries with lowest wealth inequality (Pickett & Wilkinson 2015), we were not able to adjust for socioeconomic characteristics of the respondent, which have been linked to a wide range of health sequelae (e.g., Matthews & Gallo, 2011). Lastly, despite the modest response rates to two data collections (36% and 45%, respectively; Johansson et al., 2013), we cannot rule out that other vulnerable sections of the population were not included due to selection bias (Tarczon, & Quadara, 2012).

Conclusion

In the current study, we investigated the prevalence and etiology of ASA in a large sample of Finnish twins and their siblings, including both women and men, aged 18 to 49. The estimated prevalence of any form of ASA in our sample, and that of forced penetrative sex in particular, was comparable to other population-based studies in Western countries. However, in contrast with previous research, we found higher rates of unwanted touching or kissing than verbal harassment. Women were found to experience significantly higher mean levels of ASA than men, whereas means did not vary substantially between different cohorts. Our quantitative genetic results indicated that environmental factors, mostly operating outside of the family environment, have the largest impact on the risk of ASA. However, small genetic influences were also estimated, and motivate further research on behavioral profiles that might be preferentially targeted by sexually motivated perpetrators. Then, we addressed the phenotypic and genetic relationship between different types of CM and ASA. Victims of severe CM, especially CSA, were found to also report higher levels of ASA. However, among five different types of CM, emotional abuse emerged as the strongest predictor of ASA. Thus, our findings encourage more research on the detrimental impact of emotional abuse. We further investigated whether the association between CM and ASA could be explained by an overlap in their risk factors. Our findings were only partially in contrast with this possibility. In fact, additive genetic and unique environmental factors did fully explain the modest covariance of ASA with CSA, emotional abuse, and multiple victimization, while no substantial etiological overlap emerged with the remaining types of CM, suggesting that both shared and unique factors might influence the risk of interpersonal victimization in childhood and adulthood. Lastly, we identified marginal sex differences in the strength of the association between CM and ASA, but significant sex difference in the extent of the etiological influences on ASA. Specifically, we observed significantly larger genetic influences in women than men, and we determined that such larger influences did not reflect sex-limited genetic effects, but, rather, the more persistent environmental risk for women.

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