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Genetic Vulnerability to Patterns of Interpersonal Victimization and Associated Psychiatric Comorbidity

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Patrizia's research focuses on interpersonal violence and its relationship with mental health. During her doctoral studies, she explored the genetic mechanisms associated with the risk of experiencing maltreatment, as well as the neurocognitive mechanisms associated with the risk of perpetrating it. She has co-taught seminars in forensic neuroscience and a university course in behavior genetics.

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Åbo, Finland, 2019

ACADEMIC DISSERTATION

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Abstrakt

Interpersonell viktimisering är ett omfattande problem för barn och vuxna. Tidigare forskning har försökt identifiera de personliga kännetecken och omgivningsfaktorer som ökar risken att en individ viktimiseras. Trots det har man inte undersökt i vilken mån genetiska och omgivningsmässiga faktorer bidrar till risken för olika typer av viktimisering hos barn och vuxna, samt kopplingen mellan dessa. Man vet att barn som utsatts för misshandel eller misskötsel har högre risk att utveckla psykopatologi än andra. Ändå har man inte använt genetiskt informerade metoder för att studera flertalet samband för att undersöka om olika typers viktimisering i barndomen ökar risken för senare psykologiska besvär och kan förklara samvariationen mellan dem. Därför undersökte vi den genetiska och omgivningsmässiga etiologin bakom interpersonell viktimisering och samvarierande psykologiska besvär i ett nationellt representativt urval av vuxna finländare.

I Studie I uppskattade vi de genetiska och omgivningsmässiga effekterna på risken för viktimisering i barndomen. Vi fann att gener och den omgivning som delas mellan syskon ökade risken att utsättas för flera typer av viktimisering, speciellt sån där förövaren är en släkting. Den omgivning som inte delas mellan syskon ökade risken för specifika typer av viktimisering, i synnerhet sexuell misshandel. Vi fann också könsskillnader i förekomsten av och etiologin bakom viktimisering i barndomen. Detta gällde i synnerhet emotionell och sexuella misshandel. I Studie II undersökte vi genetiska och omgivningsmässiga effekter på risken att utsättas för sexuellt våld som vuxen och de fenotypiska och genetiska associationerna med viktimisering i barndomen. Vi fann att omgivningsmässiga orsaker, speciellt sådana utanför familjen, förklarade en stor del av risken att utsättas för sexuellt våld som vuxen, och bland dem var emotionell misshandel i barndomen den starkaste prediktorn. Att samma

etiologiska faktorer påverkade risken att utsättas för flera typer av viktimisering i barndomen och sexuellt våld som vuxen är också nämnvärt. I Studie III utforskade vi samvarians mellan psykiatrisk internalisering, externalisering och kroppsrelaterade psykologiska besvär, samt mindre studerade besvär såsom ilska och sexuella besvär. Jämte de kända dimensionerna av psykopatologi så identifierade vi också en ny dimension som förklarade samvariationen av kroppsrelaterade psyko-logiska besvär. Kroppsrelaterade psykologiska besvär var starkt kopplade till internalisering och var vanligare hos kvinnor och deltagare i medelåldern än hos män och unga vuxna. I Studie IV undersökte vi effekten av gener och tidiga missförhållanden på samvariationen mellan kroppsrelaterade psykologiska besvär och internalisering samt deras separata förekomst. Vi identifierade en etiologisk rutt för internalisering och en annan för kroppsrelaterade psykologiska besvär och anslutna symptom. Viktimisering i barndomen förklarade mer av internalisering än av kroppsrelaterade psykologiska besvär, och emotionell misshandel var den starkaste prediktorn. De observerade kopplingarna var små och förklarades främst av genetiska effekter för internalisering, och av både genetiska och omgivnings-mässiga effekter för kroppsrelaterade besvär.

Våra fynd visar att ens genetiska uppsättning påverkar risken för interpersonell viktimisering, speciellt under barndomen. Detta innebär att vissa individer är mer utsatta än andra till följd av kopplingen mellan deras genetiska uppsättning och omgivningarna de ärver, skapar och söker sig till. Försök att motverka viktimisering hos barn bör därför beakta individuella riskprofiler för att effektivt skydda särskilt utsatta individer. Med tanke på sexuellt våld och psykopatologi i vuxen ålder var emotionell misshandel och misskötsel speciellt skadliga. Det kan eventuellt bero på att föräldrar oftare bidrar till denna typ av viktimisering, samt att den är svårare att upptäcka och då ofta pågår längre än andra typer av viktimisering. Våra fynd kan också hjälpa förklara varför kvinnor har högre risk än män att viktimiseras:

Flickor kan ha högre risk att misshandlas emotionellt och sexuellt på grund av könsspecifika genetiska effekter medan kvinnor kan ha högre risk att utsättas för sexuellt våld eftersom deras livsmiljö är mer osäker. Slutligen så stöder våra studier antagandet om att kopplingen mellan viktimisering i barndomen och sexuellt våld i vuxen ålder förklaras av gemensamma omgivningsmässiga orsaker.

Abstract

Interpersonal victimization is a pervasive hardship for children and adults. Previous research has attempted to identify individual characteristics and environmental conditions that might predispose individuals to these experiences. Nonetheless, no research project has been designed to clarify the extent to which genetic and environmental factors contribute to the risk of, and to the associations between, multiple forms of victimization during childhood and adulthood. Moreover, victims of childhood abuse and neglect are more likely than non-victims to develop psychopathology. However, whether different types of childhood victimization confer risk for different mental health problems or, rather, for psychiatric comorbidity, has rarely been addressed in genetically controlled studies testing multiple associations simultaneously. Hence, we investigated the genetic and environmental etiology of interpersonal victimization and psychiatric comorbidity in a nationally representative sample of Finnish adults.

In **Study I**, we estimated genetic and environmental influences on the risk of childhood victimization. We found that genes and environments shared by siblings increased the risk for multiple victimization, especially by related individuals, while unshared environmental exposures increased the risk of specific adversities, particularly sexual abuse. We also found sex differences in the prevalence and etiology of childhood victimization, in particular emotional and sexual abuse. In **Study II**, we examined genetic and environmental influences on the risk of adult sexual victimization as well as its phenotypic and genetic associations with childhood victimization. We observed that environmental exposures, especially outside the family, largely explained the risk of adult sexual victimization. Childhood emotional abuse was the strongest predictor of adult sexual victimization. Notably, the same etiological factors influenced multiple childhood victimization and adult sexual victimization. In **Study III**, we explored the

patterns of psychiatric comorbidity of core internalizing, externalizing, and less investigated problems like sexual distress. Alongside the previously known dimensions of psychopathology, we identified a novel dimension accounting for the covariance of body-related problems. Body-related problems were strongly associated with core internalizing problems and affected women and middle-aged participants more than men and young adults, respectively. In **Study IV**, we investigated the impact of gene variants and early adversities on the comorbidity and distinctiveness of body-related and core internalizing mental health problems. We identified one etiological pathway for core internalizing and one for body-related and associated symptoms. Childhood victimization contributed to internalizing more than body-related problems, and emotional maltreatment was the strongest predictor of these outcomes. However, observed associations were small and explained by shared genetic effects for core internalizing, and by shared genetic and environmental effects for body-related problems.

Our findings suggest that gene variants may influence the risk of interpersonal victimization, especially during childhood. This means that some individuals are at higher risk than others, due to a correlation between their genetic make-up and the environments that they inherit, evoke, and seek out. As a result, preventive practices should account for individual risk profiles to effectively protect particularly vulnerable individuals. Emotional abuse and neglect were especially harmful in terms of later risk of adult sexual victimization and mental health problems. This might be because these forms of maltreatment are usually perpetrated by parents, are difficult to detect and, therefore, more often continuous stressors than isolated experiences. Our findings also help to explain why women experience elevated risk of victimization compared to men: Girls might be at increased risk of emotional and sexual abuse due to sex-specific genetic influences on traits exploited by perpetrators, and women might be at increased risk of sexual victimization because many environments are unsafe for them.

Lastly, our work supports the notion that the longitudinal associations observed between childhood victimization and adult sexual victimization and mental health problems might be accounted for by the same etiological factors.

1. Introduction

1.1. Interpersonal Victimization

Interpersonal violence is a serious social problem, encompassing different forms of maltreatment between family members, intimate partners, and unrelated individuals (Krug, Mercy, Dahlberg, & Zwi, 2002). It imposes a heavy burden on victims as well as on society as a whole, due to its impact on victims' well-being (Hillis, Mercy, & Saul, 2016; Peterson, DeGue, Florence, & Lokey, 2017; Sumner et al., 2015) and due to the costs associated with the identification and detention of violent offenders (McCollister, French, & Fang, 2010). Despite its historical decline (Finkelhor & Jones, 2012; Pinker, 2011), interpersonal violence has not been eradicated. In fact, average population-based prevalence estimates indicate that approximately 22% of children (Stoltenborgh, Bakermans-Kranenburg, Alink, & IJzendoorn, 2014) and 21% of adults (Krnjacki, Emerson, Llewellyn, & Kavanagh, 2015) still experience interpersonal victimization.

To inform policy makers, researchers attempt to identify the causes and consequences of interpersonal offending and victimization. Criminological research has identified a number of situational and individual risk factors for interpersonal offending. Examples of situational risk factors include economic disadvantage (VanderEnde, Yount, Dynes, & Sibley, 2012) and deviant peer affiliations (Beaver et al., 2008). Examples of individual factors include borderline (González, Igoumenou, Kallis, & Coid, 2016) and psychopathic personality traits (Hare & Neumann, 2008), which are associated with specific biological markers, such as low resting heart rate (Koenig, Kemp, Feeling, Thayer, & Kaess, 2016; Latvala et al., 2015). A smaller body of research has also identified situational and individual factors associated with the risk of becoming a victim (Sullivan, Wilcox, & Ousey, 2010). Social isolation, among situational factors (Hong & Espelage,

2012), and intellectual disabilities (Fisher, Baird, Currey, & Hodapp, 2016; Jones et al., 2012), among individual factors, have been shown to increase the risk of victimization. Nonetheless, possible biological precursors of interpersonal victimization have been overlooked. As a result, why victimization occurs non-randomly in the population, or, in other words, why certain individuals are more likely to be targeted by violent offenders than others, often repeatedly, remains unclear (Planty & Strom, 2007). In light of this, in the present thesis, we investigated the etiology and sequelae of interpersonal victimization, with the ultimate goal to inform effective preventive strategies and adequate mental health services for victims.

1.1.1. Childhood Victimization

Childhood victimization comprises the abuse and neglect of children under 18 years, resulting in "actual or potential harm to the child's health, survival, development or dignity in the context of a relationship of responsibility, trust or power" (World Health Organization, 2014, p. 82). Five main types of childhood victimization have been distinguished, namely emotional, physical, and sexual abuse, as well as emotional and physical neglect (Stoltenborgh et al., 2014). Previous research has shown that childhood victimization is a worldwide problem, exposing children to increased risk of short- and long-term harmful consequences (Norman et al., 2012). This problem is prevalent in low- as well as in high- income countries, including Finland (Kloppen, Mæhle, Kvello, Haugland, & Breivik, 2014), where child welfare systems should increase public awareness and promote good parenting. However, since these community-level preventive strategies do not appear to suffice, more research should focus on victim-specific risk factors to guide promising individual-level preventive strategies.

1.1.2. Adult Victimization

Similar to child maltreatment, adult victimization is alarmingly prevalent. It has been estimated that approximately one in five adults worldwide might be physically or sexually assaulted (Benjet et al., 2015). The present thesis focused on adult sexual victimization.

1.1.2.1. Adult Sexual Victimization

Adult sexual victimization is a violation of basic human rights and a major public health problem (World Health Organization, 2014). It has been estimated that, in western countries, approximately 36% of women and 17% of men might experience sexual victimization during their life (Smith et al., 2017). Victims of sexual violence have been found to be at increased risk of a host of harmful sequelae, including several psychological problems (Dworkin, Menon, Bystrynski, & Allen, 2017). Adult sexual victimization can be experienced in multiple ways. These include contact sexual violence, such as nonconsensual completed or attempted penetration (i.e., "rape"; Basile, Chen, Black, & Saltzman, 2007), touching (i.e., "sexual battery"; Muehlenhard, Peterson, Humphreys, & Jozkowski, 2017), and sexual acts resulting from coercion or incapacitation (McCauley, Ruggiero, Resnick, Conoscenti, & Kilpatrick, 2009). Sexual victimization also encompasses non-contact sexual violence, such as verbal harassment (i.e., unwanted sexual comments or advances; Krug et al., 2002). Albeit seldomly addressed in empirical studies, non-contact sexual acts might occur more frequently than other types of sexual victimization (Breiding, 2015; Smith et al., 2017). Despite the pervasiveness of sexual violence, research on its risk factors is insufficient, and prevention strategies remain largely ineffective (DeGue et al., 2014).

1.2. How Could Genes Influence the Risk of Victimization?

Identifying the risk factors for child and adult victimization represents a crucial step towards its prevention. Past research has mainly explored the environmental conditions where the risk of victimization is increased. In particular, it has been consistently shown that children are most often victimized within their family environment (Gilbert et al., 2009; National Research Council, 2014; Stoltenborgh et al., 2014). This finding is somewhat intuitive, given that many forms of victimization entail, by definition, a relationship with, or dependence on, the perpetrator. Since parents and caregivers bear the responsibility to provide for their children, children cannot be physically neglected by strangers or individuals outside the family. Child sexual abuse is, however, an exception to this, as incestuous abuse is rarer than abuse perpetrated outside the home and by unrelated individuals (Sariola & Utela, 1996). This is probably due to the natural aversion against sex with biological family members (Kresanov et al., 2018; Lieberman & Antfolk, 2015). Environmental conditions where the risk of adult sexual victimization is increased have also been identified. These include, for example, disadvantaged urban environments (Decker et al., 2014; Hatch & Dohrenwend, 2007; Tyler, 2008) and institutional settings (Peterson, Voller, Polusny, & Murdoch, 2011; Wilson, 2018). Altogether, these findings suggest that both family- and community-level interventions are important for the prevention of interpersonal violence (DeGue et al., 2012).

To comprehensively understand the etiology of interpersonal victimization, not only situational risk factors, but also victim-specific risk factors should be considered (Hines & Saudino, 2008). In particular, gene variants might indirectly influence the risk of victimization. Importantly, this possibility does not imply that individuals are responsible for becoming a victim. In fact, perpetrators bear full moral and legal responsibility for their behavior. However, possible genetic influences on the risk of being targeted by a violent offender might partly explain why some individuals are

at higher risk compared to others, and why current environment-level violence prevention efforts, neglecting individual risk profiles, might be insufficient.

1.2.1. Gene-environment Correlation Mechanisms

The mechanisms via which genes might influence environmental exposures and experiences are referred to as gene-environment correlation mechanisms (*r*GE, Jaffee & Price, 2007). Three types of gene-environment correlation have been described in the literature: passive, evocative (or reactive) and selective (or active). Passive gene-environment correlation refers to the correlation between heritable traits and rearing environments. For example, parents with psychopathic traits, which are highly heritable (Larsson, Andershed, & Lichtenstein, 2006), might struggle to empathize with others (Seara-Cardoso, & Viding, 2014), and, as a result, expose their offspring to an emotionally neglecting environment. Evocative gene-environment correlation refers to the correlation between heritable traits and the behavior of other individuals in response to such traits. For example, callous-unemotional traits in children, also highly heritable (Viding, Jones, Paul, Moffitt, & Plomin, 2008), might increase the risk of harsh disciplinary responses from parents (Flom, White, & Saudino, 2019; Micalizzi, Wang, & Saudino, 2017), including corporal punishment (Jaffee et al., 2004). Similarly, adults with intellectual disabilities, largely genetic in origin (Vissers, Gilissen, & Veltman, 2015), might be perceived as defenseless, and, thus, might be taken advantage of by sexual offenders (Reyns & Scherer, 2018). Lastly, selective gene-environment correlation refers to the correlation between heritable traits and the environmental niches that individuals with such traits tend to select. For instance, sensation seeking, a highly heritable personality trait (Stoel, De Geus, & Boomsma, 2006), might increase the exposure to unsafe surroundings, where the risk of interpersonal victimization is increased (Averdijk, Ribeaud, & Eisner, 2019).

In line with the possibility that genes may influence the risk of child and adult interpersonal victimization, past research has demonstrated gene-environment correlation effects on the risk of experiencing stressful events like relationship, work, and financial difficulties (Bolinskey, Neale, Jacobson, Prescott, & Kendler, 2004). Since children are most often victimized by parents (e.g., Stoltenborgh et al., 2014), from whom they inherit both genetic make-up and rearing environment, gene-environment correlation effects on the risk of childhood victimization might be especially relevant (Hines & Saudino, 2002). Nonetheless, only a small number of studies have estimated, and supported, the genetic propensity to adversities, both in children (Kendler & Baker, 2006) and adolescents (Johnson, Rhee, Whisman, Corley, & Hewitt, 2013). Similarly, the possibility of gene-environment correlation effects on the risk of adult sexual victimization should be explored. In fact, research has shown that sexually motivated perpetrators preferentially seek out victims who present certain heritable traits, such as physical and mental disabilities (Fisher et al., 2016; Hughes et al., 2012; Vissers et al., 2015), neurodevelopmental disorders (Ohlsson Gotby, Lichtenstein, Långström, & Pettersson, 2018), and externalizing behavior (Daigle & Teasdale, 2018; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Vezina & Hebert, 2007). As a result, genetic factors might also, indirectly, influence the risk of adult sexual victimization. However, to the best of our knowledge, only two studies have been conducted to test this possibility, and have found support for it (Sartor et al., 2012; Stein, Jang, Taylor, Vernon, & Livesley, 2002).

1.2.2. Sex Differences in the Vulnerability to Victimization

Differences between women and men in the vulnerability to interpersonal victimization have been consistently observed. First, sex differences have been repeatedly estimated in the prevalence of some types of childhood victimization (Coêlho et al., 2018): Girls are more likely than boys to

experience sexual abuse (Kloppen et al., 2014; Stoltenborgh, Van Ijzendoorn, Euser, & Bakermans-Kranenburg, 2011), whereas boys are more likely than girls to experience physical abuse (Thompson, Kingree, & Desai, 2004). However, evidence of sex differences in the prevalence of other forms of childhood victimization, such as emotional abuse and neglect, is mixed (May-Chahal & Caws, 2005; Taillieu, Brownridge, Sareen, & Afifi, 2016). Second, sex differences have been identified in the prevalence rates of adult interpersonal victimization. Women are more likely to experience sexual violence than men, who are, instead, more likely than women to experience physical assault (Iverson et al., 2013; Tolin & Foa, 2008).

Despite the replicated evidence of sex differences in the prevalence of interpersonal victimization, the underlying reasons are poorly understood. The study of sex-specific risk factors seems especially compelling to determine why women and men are more likely to become victims of certain forms of abuse and neglect rather than others. In particular, before sex-specific risk factors are examined, the extent to which these factors are biological and environmental should be addressed. Conceivably, genetic and environmental risk factors might differ quantitatively and qualitatively between sexes. In other words, some risk factors might influence both women and men, but to a different extent, and, in addition, women and men might be subject to entirely different risk factors. For example, due to gender social expectations, women and men might be treated differently by others, or engage in partly different activities. As a result, they might be exposed to increased risk of interpersonal victimization in different environments. With respect to genetic risk factors, women and men might differ in the proportion of phenotypic variance explained by genetic variance and, additionally, this genetic variance might include the effect of different genes (Ratnu, Emami, & Bredy, 2017). As a result, heritable traits that are more prevalent in one sex than the other might expose them to specific types of victimization. For example, conduct problems, more common in men than

women, might put men at an increased risk of becoming involved in fights, and, thus, of being physically assaulted (Berkout, Young, & Gross, 2011). Nonetheless, no previous study has been designed to investigate sex differences in the extent of the genetic and environmental influences on interpersonal victimization.

1.3. Recurrent Victimization

Empirical research has indicated that prior exposure to maltreatment strongly predicts subsequent victimization (e.g., Fisher, Daigle & Cullen, 2010). Indeed, repeat victims account for the largest proportion of victimization rates (Sullivan et al., 2010). In particular, victimization during sensitive developmental stages might result in persisting biological changes, such as alterations in genome function (Dunn et al., 2019), that might increase the vulnerability to experiencing violence later in life (Ports, Ford, & Merrick, 2016). Indeed, childhood victimization has been associated with the long-term dysregulation of brain circuitries involved in emotional and stress responses (e.g., Dannlowski et al., 2012; McCrory, De Brito, & Viding, 2011). As a consequence, several studies have attempted to clarify which behavioral patterns, observed in survivors of childhood victimization, might increase their risk of recurrent victimization.

1.3.1. Childhood Victimization and Adult Sexual Victimization

Most previous studies addressing recurrent victimization have focused on sexual revictimization, showing that child sexual abuse survivors are at greater risk of adult sexual victimization compared to the general population (Walker, Freud, Ellis, Fraine, & Wilson, 2019). In the attempt to explain this finding, it has been proposed that child sexual abuse survivors might engage in more risky sexual behaviors, such as unprotected casual sex (Bensley, Van Eenwyk, & Simmons, 2000; Lemieux & Byers, 2008). Sexual

risk-taking, in turn, has been found to increase the risk of sexual revictimization (Messman-Moore, Walsh, & DiLillo, 2010; Van Bruggen, Runtz, & Kadlec, 2006). However, the impact of other childhood adversities on the risk of adult sexual victimization is poorly understood. The limited available evidence suggests that the association between child emotional and physical abuse and adult sexual victimization might be weaker (Desai, Arias, Thompson, & Basile, 2002; Kimerling, Alvarez, Pavao, Kaminski, & Baumrind, 2007; Zurbriggen, Gobin, & Freyd, 2010) or comparable (Messman-Moore et al., 2010; Widom, Czaja, & Dutton, 2008) to that between child sexual abuse and adult sexual victimization. Thus, research should address the impact of relatively overlooked types of childhood victimization, including multiple childhood victimization (Conway, Raposa, Hammen, & Brennan, 2018).

Importantly, although childhood victimization might constitute *per se* a risk factor for subsequent victimization, it is also possible that, for some individuals, the same risk factors may increase the risk of victimization in childhood as well as in adulthood. In this context, the continued exposure to such risk factors would explain, at least in part, the repeated victimization. In line with this, individual differences have been found to mediate the risk of violent revictimization (Clay-Warner, Bunch, & McMahon-Howard, 2016; Ellonen & Salmi, 2011). For instance, genetically influenced traits, such as mental illness, might increase the risk not only of victimization, but also of recurrent victimization (Policastro, Teasdale, & Daigle, 2015). Similarly, individuals who live in the proximity of sex offenders might be at risk of repeated sexual victimization (Leclerc, Wortley, & Smallbone, 2010; Schewe, Riger, Howard, Staggs, & Mason, 2006). Surprisingly, however, this alternative pathway has remained largely unexplored. In fact, to the best of our knowledge, no previous study has investigated whether overlapping genetic and environmental risk factors underlie the risk of interpersonal

victimization in childhood and adulthood, thus partly explaining the strong phenotypic association typically observed.

1.4. Childhood Victimization and Adult Mental Health

In addition to being positively associated with experiences of adult victimization, childhood victimization is associated with adult mental health problems. In fact, victims of childhood victimization are twice as likely as non-victims to develop subsequent psychiatric disorders (Herrenkohl, T. I., Hong, Klika, Herrenkohl, R. C., & Russo, 2012; McCrory et al., 2011; McLaughlin et al., 2010). The impact of some types of childhood victimization, particularly sexual abuse (Cutajar et al., 2010; Fergusson, McLeod, & Horwood, 2013, Hovdestad, Campeau, Potter, & Tonmyr, 2015), has been more widely investigated than the impact of other types of childhood victimization, such as emotional abuse and neglect (Jud, Fegert, & Finkelhor, 2016; Taillieu et al., 2016). Nonetheless, when investigated, emotional abuse and neglect have also been associated with adult mental health problems (McLaughlin et al., 2010; Norman et al., 2012). Moreover, although it has been demonstrated that different types of childhood victimization tend to co-occur (Curran, Adamson, Stringer, Rosato, & Leavey, 2016; Turner, Finkelhor, & Ormrod, 2010), multiple childhood victimization has been somewhat overlooked. In fact, most studies have either focused on single forms of childhood victimization, neglecting their covariance, or summarized multiple forms into cumulative scores, neglecting their relative effects (Keyes et al., 2012; McLaughlin & Sheridan, 2016; Westermair et al., 2018). Thus, more research should investigate the relative effect of multiple types of childhood victimization on the risk of adult mental health problems.

1.4.1. Psychiatric Comorbidity

Historically, mental health problems have been conceptualized and classified as discrete categories. In clinical practice, the co-occurrence of two or more mental health problems is, however, the norm rather than the exception (Krueger & Markon, 2006). This co-occurrence is referred to as comorbidity and represents a challenge to current diagnostic systems (Kotov et al., 2017). To understand the etiology and course of mental health problems, beyond traditional nosologies, transdiagnostic approaches have been developed (Eaton, Rodriguez-Seijas, Carragher, & Krueger, 2015). These approaches have suggested the existence of two broad dimensions of psychopathology: internalizing and externalizing (Kessler et al., 2011). The internalizing dimension captures the tendency to introject distress and experience symptoms commonly described as depressive and anxious. The externalizing dimension, on the other hand, captures the tendency to express distress with outward-directed behaviors, such as substance use and aggression. The internalizing and externalizing dimensions have also been shown to correlate, suggesting the presence of an overarching general factor of psychopathology, referred to as the "*p*" factor (Caspi et al., 2014; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017). In light of this evidence, when investigating the relationship between childhood victimization and adult mental health, not only multiple types of victimization, but also multiple psychopathological symptoms should be accounted for.

1.4.1.1. Body-related Mental Health

Body-related mental health problems, such as disordered eating and sexual distress, are regularly comorbid with core internalizing disorders, namely depression and anxiety (e.g., Forbes, Baillie, & Schniering, 2015a; Hudson, Hiripi, Pope, & Kessler, 2007; Laurent, & Simons, 2009). Accordingly, in the transdiagnostic literature, these and other body-related problems have emerged as sub-facets of the internalizing dimension (e.g., Forbes, Baillie, &

Schniering, 2015b; Forbush et al., 2017; Kotov et al., 2017). However, body-related symptoms might reflect a special way to express psychological distress. Indeed, these problems share pathological correlates, such as alterations of interoceptive responses (Jenkinson, Taylor, & Laws, 2018; Berenguer, Rebôlo, & Miguel Costa, 2019), and might also share etiological mechanisms, possibly explaining their frequent co-occurrence (Castellini, Lo Sauro, Ricca, & Rellini, 2017; Pinheiro et al., 2009). Therefore, the possibility that partly separate genetic and environmental pathways underlie body-related and core internalizing mental health problems should be addressed. Conceivably, body-related and internalizing problems might covary due to common risk factors and, at the same time, their separate occurrence might be explained by other, unique, risk factors.

1.4.1.2. Sex Differences in Body-related Mental Health

Sex differences in mental health problems have been systematically reported. Specifically, women and men have been shown to more likely experience internalizing and externalizing psychopathology, respectively (Eaton et al., 2012). Moreover, sex differences have been observed in the prevalence and severity of body-related problems. Compared to men, women have been found to experience higher prevalence and mean levels of disordered eating and body image dissatisfaction (Striegel-Moore et al., 2009) as well as sexual distress (Milhausen et al., 2014). However, no previous study has adopted a transdiagnostic approach to investigate sex differences in the strength of the association between body-related and other mental health problems. Possibly, sex differences in the patterns of comorbidity of body-related and other psychiatric disorders might explain why some patients but not others report body-related complaints alongside internalizing mental health conditions. Therefore, measurement invariance of the transdiagnostic structure of psychopathology should be tested.

1.4.1.3. Genetic Contribution to Comorbidity

Behavioral and molecular genetic approaches have demonstrated that genes strongly contribute to psychiatric comorbidity (Anttila et al., 2016; Waldman, Poore, van Hulle, Rathouz, & Lahey, 2016). In particular, core internalizing disorders like depression and anxiety have been found to share substantial genetic influences (Cerdá, Sagdeo, Johnson, & Galea, 2010; Kendler et al., 2011; Middeldorp, Cath, Van Dyck, & Boomsma, 2005). Moreover, internalizing disorders partly share genetic influences with disordered eating (Slane, Burt, & Klump, 2010) and sexual distress (Burri, Spector, & Rahman, 2012). Thus, the possibility of a common genetic pathway underlying core internalizing and body-related mental health problems should be investigated. Such a common genetic pathway would support their joint dimensional classification and contribute to explaining their comorbidity and their common features, such as low self-esteem. Moreover, a common genetic etiology would imply the existence of disorder-specific environmental risk factors, contributing to the separate occurrence of body-related and internalizing psychopathology.

1.4.2. Childhood Victimization: Specific or Unspecific Risk Factor?

Among the environmental exposures associated with psychiatric comorbidity, researchers have widely examined childhood victimization (Herrenkohl et al., 2012; McCrory et al., 2011). In spite of this, the existing body of research has not conclusively determined whether certain types of childhood victimization may lead to certain mental health problems (i.e., disorder-specific effect) or, conversely, whether the effect of childhood victimization is generalized (i.e., unspecific effect). For example, childhood victimization is strongly associated with core internalizing mental health problems (Cecil, Viding, Fearon, Glaser, & McCrory, 2017; Cutajar et al., 2010; Fergusson et al., 2013) and also moderately associated with disordered eating (Carr, Martins, Stingel, Lemgruber, & Juruena, 2013; Slevec &

Tiggemann, 2011) and sexual distress (Rellini & Meston, 2007; Stephenson, Hughan, & Meston, 2012). However, it remains unclear whether different types of childhood victimization predominantly increase the susceptibility to core internalizing or to body-related problems, possibly via their impact on different developmental mechanisms and, thus, contributing to their distinctiveness. Furthermore, if genes influence, at least partly, the risk of childhood victimization (Kendler & Baker, 2006), the possibility of genetic confounding of the association between childhood victimization and adult psychopathology should also be addressed. In fact, unless this possibility is ruled out, the causal role of childhood victimization in the onset of mental health problems remains controversial (Schaefer et al., 2017).

2. Aims and Expected Results

The overall aim of the present thesis was to investigate the etiology of, and associations between, child and adult victimization and mental health.

In **Study I**, we estimated the extent of genetic and environmental influences on childhood victimization. In particular, we aimed to clarify whether the extent of such influences varied as a function of the type of childhood victimization, the genetic relatedness between victim and perpetrator, as well as the sex of the victim. We expected to observe moderate genetic influences on the risk of each and multiple types of childhood victimization, as well as a strong overlap in the genetic and environmental influences on all types of childhood victimization but sexual abuse. We also predicted larger genetic and smaller unique environmental influences on victimization by genetically related compared to potentially unrelated individuals, as well as substantial sex differences in the etiology of childhood victimization, possibly including sex-limited genetic effects.

In **Study II**, we examined the extent of genetic and environmental influences on the risk of adult sexual victimization, and we investigated the phenotypic and genetic relationship between childhood victimization, in particular sexual abuse, and adult sexual victimization. We also inspected sex differences in the etiology of adult sexual victimization and in the extent of its genetic and environmental associations with childhood victimization. We hypothesized that genetic influences on adult sexual victimization would emerge, in addition to environmental influences. Moreover, we predicted that victims of child sexual abuse would be at increased risk of adult sexual victimization, compared to non-victims, but also that this form of abuse would have a weaker impact when accounting for multiple victimization. Also, we expected the association between childhood victimization and adult sexual victimization to be partly explained by common etiological factors. Lastly, we explored the possibility of

quantitative and qualitative sex differences in the etiology of adult sexual victimization.

In **Study III**, we estimated the latent dimensions underlying the comorbidity of multiple mental health problems, and we tested their invariance across sexes and age cohorts. In particular, we aimed to clarify whether body-related symptoms would be subsumed within the internalizing dimension, or, alternatively, cluster into a separate dimension. We predicted that body-related symptoms would constitute a separate dimension of psychopathology, and that their covariance with the internalizing and externalizing dimensions would be explained by a general tendency to experience any form of psychopathology, or "*p*" factor. Furthermore, we explored whether the emerged latent structure of psychopathology would be age- and sex- invariant, so that age and sex differences in the mean levels of the latent dimensions could be estimated.

In **Study IV**, we estimated the extent of the genetic and environmental sources of comorbidity of body-related and core internalizing mental health problems. We further inspected the phenotypic and genetic relationships between different types of childhood victimization and both body-related and core internalizing psychopathological outcomes. We hypothesized that body-related and internalizing symptoms would be influenced by a shared genetic pathway. Moreover, we expected all types of childhood victimization to show moderate and unspecific predictive effects, and that individual predictive values would be substantially reduced when accounting for multiple victimization. Lastly, we expected the relationship between childhood victimization and mental health to be causal, rather than mediated by genetic and environmental risk factors shared between them.

3. Materials and Methods

3.1. Participants

Participants were selected from the sample involved in the "Genetics of Sexuality and Aggression" project. This project consisted of two major data collections, approved by the Board for Research Ethics at Åbo Akademi University in accordance with the 1964 Declaration of Helsinki. Monozygotic (MZ) and dizygotic (DZ) twins and their siblings, who were native Finnish speakers, were identified from the Central Population Registry of Finland. The scope of the study, as well as the voluntary and anonymous nature of the participation, were explained upon first contact. Questionnaires covering a wide range of experiences, behaviors, and disorders were sent to those who consented to participate. Zygosity was determined with questionnaire items addressing physical resemblance (Sarna, Kaprio, Sistonen, & Koskenvuo, 1978), and validated through genotyping in a portion of the full sample who returned a saliva collection kit for DNA extraction (n = 775 twin pairs). The first data collection, carried out in 2005, recruited twins aged 33 to 43. Questionnaires were sent to 10000 individuals, followed by a reminder letter and new questionnaires. The response rate was 36% (N = 3558, of whom 2245 women and 1313 men). The second data collection, in 2006, recruited twins aged 18 to 33 and their siblings, aged 18 or above. Questionnaires were sent to 23577 individuals, followed by a reminder letter. Participants were also given the opportunity to complete the questionnaires via a secure web page. The response rate was 45% (N = 10524, of whom 6601 women and 3923 men). Thus, the overall response rate for both data collections was 41%. The full sample included 14082 individuals (8846 women and 5236 men), of whom 9614 twins (3273 MZ and 6341 DZ). More information on the data collection is presented in Johansson et al. (2012).

We analyzed data collected from 13024 individuals (8415 women and 4609 men) in **Studies I**, **III**, and **IV,** and from 12952 individuals (8367 women and 4576 men) in **Study II**. From the full samples, we selected 9562 twins (3248 MZ and 6314 DZ) in **Studies I** and **IV** and 9513 twins (3236 MZ and 6277 DZ) in **Study II** for quantitative genetic analyses. Across studies, samples were split by sex to inspect sex differences, or by age into three cohorts (18 – 25, 26 – 32, and 33 – 49) to analyze cohort effects. When data from the full samples was used, we regressed the measures of interest on age and sex to control for these possible sources of confounding. Furthermore, when conducting phenotypic analyses using the full sample, we implemented complex sampling correction, estimating robust standard errors, and scaled test statistics, to control for the multilevel nature of our data, namely, participants clustered into families (Muthén & Satorra, 1995).

3.2. Measures

3.2.1. Measures of Childhood Victimization

The Childhood Trauma Questionnaire, Short Form (CTQ-SF; Bernstein et al., 2003) was used to measure childhood victimization in **Studies I**, **II,** and **IV**. The CTQ-SF is a Likert-type scale that addresses childhood victimization by parents, unspecified family members or unspecified individuals. To complete the CTQ-SF, respondents rated the frequency of 25 instances of childhood victimization, categorized as physical abuse, emotional abuse, sexual abuse, emotional neglect and physical neglect, on a five-point scale. In **Studies I**, **II** and **IV**, we created composite scores corresponding to each sub-scale before statistical analyses. Furthermore, in **Study I**, we categorized item-level reports as addressing abuse or neglect perpetrated by parents and family members ("family-specific"), or by individuals potentially unrelated to the child ("family-unspecific"), based on

face validity. Then, we created two mean scores and standardized them for subsequent analyses. Also, in **Study II**, we created a dichotomous variable that categorized participants as victims or non-victims of severe childhood victimization. Specifically, we considered participants who scored 4 or 5 on any of the CTQ-SF items (i.e., "often true" or "very often true") as victims of severe childhood victimization, and participants who scored 3 or less ("sometimes true", "rarely true", or "never true") as non-victims of severe childhood victimization.

3.2.2. Measures of Adult Sexual Victimization

In **Study II**, we measured experiences of adult sexual victimization using an item created for our data collection based on the Sexual Experiences Survey (SES, Koss & Oros, 1982), but substantially simplified in its level of behavioral specificity. Participants responded yes or no to every given option to the question "Have you, as an adult (over the age of 15), become the target of sexual harassment or abuse?". Response options 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", coded as 2; "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. We assigned the highest score when multiple options were selected. We standardized raw scores into a z-score before statistical analyses.

3.2.3. Measures of Mental Health

In **Study III**, we measured psychopathology using eight Likert-type self-report scales. These included 1) two sub-subscales of the Brief Symptom Inventory, measuring depression and anxiety (Derogatis, 2001); 2) five representative items from the Eating Attitudes Test, for disordered eating (EAT-26, Garner, Olmsted, Bohr, & Garfinkel, 1982); 3) the gender-neutral

items of the Female Sexual Distress scale, for sexual distress (FSDS; Derogatis, Rosen, Leiblum, Burnett, & Heiman, 2002); 4) the body image sub-scale of the Derogatis Sexual Functioning Inventory, for body image dissatisfaction (Derogatis, 1997); 5) the Alcohol Use Disorder Identification Test (Saunders et al., 1993); 6) the lifestyle, interpersonal, antisocial and affective subscales of the Self-Report Psychopathy scale III, short form (SRP-SF; Paulhus, Hemphill, & Hare, 2002) for psychopathy (Hare, 2003); 7) the physical and verbal subscales of the Aggression Questionnaire, for aggression (Buss & Perry, 1992); and 8) the sub-scale of the State Trait Anger Expression Inventory II measuring trait anger (Spielberg, 1999). The first three scales were also used in **Study IV**.

3.2.4. Preliminary Data Handling

Raw data were prepared for statistical analyses by reverse-coding, imputing missing data, transforming non-normally distributed data using Log10 transformation (Kline, 2016), and creating composite scores corresponding to the scales or sub-scales of interest. To do so, we first ruled out extreme collinearity and singularity (Field, 2013), by ensuring that items from the same scale or sub-scale were positively correlated, and that the determinants of the correlation matrices were larger than .00001. Next, we performed dimension reduction by maximum likelihood factor analysis, extracting a single factor and saving the score derived using the Bartlett method. Overall, composite scores showed acceptable internal consistency.

3.3. Statistical Analyses

3.3.1. Structural Equation Modeling

Structural Equation Modeling (SEM) is a family of statistical techniques integrating regression and factor analysis. In **Study III**, SEM was used to

model relationships between observed and unobserved (latent) variables, also referred to as factors. These relationships were modelled based on the estimated correlations and covariances between observed variables. SEM consists of two components: a measurement model and a structural model. The measurement model, corresponding to factor analysis, serves the purpose of estimating one or more latent variables, onto which observed variables "load" (i.e., are regressed on). Regression coefficients (or factor loadings), residual variances, and error terms are estimated. The structural model, on the other hand, serves the purpose of linking the variables (latent and/or observed) through recursive and non-recursive relationships. Recursive relationships allow causation to flow in a single direction, whereas non-recursive relationships allow causation to flow in both directions.

3.3.1.1. Regression Analysis

To investigate the relationship among observed variables, we conducted regression analysis. Specifically, we used regression to predict the values of adult sexual victimization (**Study II**) as well as body-related and core internalizing psychopathology (**Study IV**) based on the values of each type of childhood victimization separately and together. We used linear regression in **Study IV** and ordered logit regression with maximum likelihood estimation, appropriate with continuous independent variables but ordinal dependent variables (Bauer & Sterba, 2011), in **Study II**.

3.3.1.2. Exploratory and Confirmatory Factor Analysis

In **Study III**, we performed factor analysis with the scope of identifying the latent phenotypic structure underlying multiple mental health problems. First, we performed exploratory factor analysis (EFA) with varimax rotation, a technique that loads small groups of items highly on each extracted factor. Refinement strategies included deletion of items with

factor loadings smaller than .32 or cross-loading on multiple items (Worthington & Whittaker, 2006). Next, we tested the exploratory factor structures by means of confirmatory factor analysis (CFA) with maximum likelihood estimation robust to non-normality. Model re-specification strategies involved removing items with standardized squared loadings smaller than .40. First-order and hierarchical analyses were performed. In first-order analyses, groups of observed variables load onto correlated latent variables whereas, in hierarchical analyses, additional levels of the latent structure can be estimated. Specifically, we used second-order models, where first-order latent variables loaded, in turn, onto a second-order factor, as well as bifactor models, where every observed variable loaded onto a general latent factor, and, additionally, groups of observed variables loaded onto un-correlated first-order latent variables explaining their residual shared variance.

3.3.1.3. Measurement Invariance Testing

In **Study III**, we investigated the measurement invariance of our best-fitting model of mental health across sexes and cohorts using nested model comparisons. First, we tested whether the retained factor structure was valid in each group by estimating baseline multi-group models with no equality constraints (i.e., configural invariance). Second, we tested whether the unit of measurement was consistent across groups by constraining the factor loadings to be equivalent (i.e., metric invariance). Third, we tested whether the starting values were consistent, by constraining the intercepts to be equivalent (i.e., scalar, or "strong", invariance). When this level of invariance was not achieved, latent comparisons were precluded and observed means were compared. However, we estimated partial scalar invariance to clarify which intercepts differed between groups. When, instead, scalar invariance was achieved, we compared group means on the latent variables, by performing a structured means analysis. Lastly, we tested

whether observed and latent variances and covariances were comparable between groups, by constraining them to be equivalent (i.e., strict invariance, or full uniqueness).

3.3.2. The Twin Design

To investigate the etiology of victimization and mental health, we adopted a quantitative genetic approach, and, more specifically, a classical twin design (Boomsma, Busjahn, & Peltonen, 2002). Using a twin design, researchers can estimate the extent of the genetic and environmental influences on traits and experiences. Genetic influences further comprise additive and non-additive influences. Additive genetic influences reflect the cumulative effect of multiple alleles or genes and correspond to the sum of their individual effects. Non-additive genetic influences, on the other hand, are a product of the interactions between alleles within the same gene (dominance) or between different genes (epistasis). The twin design is based on the difference in genetic relatedness between MZ and DZ twins reared together. In fact, MZs share 100% of their segregating DNA. DZs, instead, may share two, one, or no chromosomes with each other, and, therefore, they share on average 50% of their additive genetic influences and 25% of their dominant genetic influences. As a result, observing that MZs are more similar to each than DZs, on a particular trait or experience, is an indication that this particular trait or experience is subject to genetic influences. Several assumptions underlie the twin design. Most importantly, the "equal environments assumption", postulates that the environmental exposures shared between co-twins (e.g., family and school environments) contribute equally to a particular trait in both MZ and DZ twins. Therefore, shared environmental influences comprise all non-genetic influences that contribute to twin resemblance, irrespective of zygosity. Lastly, any environmental exposure that is not shared between co-twins will contribute to their individual differences. Based on these assumptions, we decomposed

the variance and covariance of the measures of interest into additive genetic (A), shared environmental (C), and unique environmental (E) sources of variance. Of note, the E component also enclosed non-systematic measurement error. Moreover, in **Study II**, genetic influences were further decomposed into additive and dominant (D).

3.3.2.1. Twin Models

Twin designs often employ SEM-based models to decompose the phenotypic variance and covariance of measured traits and experiences (see Neale & Cardon, 2013 for details on model estimation). In the present work, we estimated univariate and multivariate twin models using maximum likelihood estimation and bootstrapped standard errors (1000 resamples with replacement) and robust estimation when comparing nested models.

Univariate models were used to estimate the etiological influences on individual items or composite scores. In **Study I**, we estimated the magnitude of the genetic and environmental influences on factor and mean scores of childhood victimization, in the full sample as well as in sub-samples by sex. In **Study II**, we estimated the magnitude of the genetic and environmental influences on our item report of adult sexual victimization. Furthermore, in **Studies I** and **II**, we used univariate sex limitation models to clarify whether different sets of genes influenced the risk of victimization in women and men. To estimate these qualitative sex differences, we estimated a baseline "general sex limitation" model, and a nested "common effects" model. The former allowed etiological influences to differ between sexes, and estimated one additional component, corresponding to sex-specific sets of genes. The latter constrained the sex-specific component to zero, estimating only the genetic effects common to both sexes. Then, we conducted nested model comparisons to determine the presence of sex-limited genetic effects.

Multivariate models included correlated factors, independent pathway, and common pathway models, used to estimate the etiological influences on the covariance between items or composite scores. With correlated factors models (**Study IV**), the variance of each variable was decomposed into A, C and E components, and these components were allowed to correlate, respectively, with the A, C and E components of the remaining variables, thus estimating the extent of their etiological correlations. With independent pathway models (**Study IV**), the variance of each variable was also decomposed into A, C and E components, but, then, additional A, C and E components, common to all variables, were estimated. With common pathway models (**Studies I**, **II**, and **IV**), the A, C and E components common to all variables were mediated through one or more latent factors, onto which the observed variables loaded. Additionally, we used a covariance matrix decomposition, referred to as Cholesky decomposition, to further decompose the etiological influences estimated on the measures of childhood victimization into separate terms, predicting adult sexual victimization (**Study II**) and psychopathology (**Study IV**).

3.3.3. Criteria for Model Acceptance and Significance Testing

To test whether models fitted our data, and to determine which one fitted best (**Studies I** and **IV**), we employed indices of absolute and relative fit. Among the indices of absolute fit, we relied on the Root Mean Square Error of Approximation (RMSEA), with upper 90% CIs smaller than .08 indicating mediocre fit, and smaller than .06 indicating excellent fit (Hu & Bentler, 1999). Among the indices of relative fit, we relied on the Akaike Information Criterion (AIC; Akaike, 1987) and the Bayesian Information Criterion (BIC; Raftery, 1995), with lower values reflecting a better tradeoff between model fit and complexity, as well as on the Comparative Fit Index (CFI; Bentler, 1990), with larger values indicating better fit.

For significance testing, in **Studies I** and **II**, we used the Satorra-Bentler scaled χ^2 difference test (Satorra & Bentler, 2009). Additionally, in **Study III**, we compared nested models using the Cheung – Rensvold criteria, or ΔCFI-rule (Cheung & Rensvold, 2002), according to which a difference between nested models greater than or equal to .01 points is considered significant. Furthermore, in **Studies I**, **II**, and **IV**, we determined whether differences between group means, regression coefficients, and estimates of genetic and environmental influences were significant based on visual inspection of confidence intervals. To do this, we calculated 95% CIs around the estimates, based on bootstrapped standard errors, and inspected their overlap. In line with recommendations (Cumming & Finch, 2005), we considered no overlap, or CIs just touching, as indicative of significant differences. Moreover, the difference between individual coefficients or estimates was calculated and considered as an unstandardized measures of effect size (Kelley & Preacher, 2012). Standardized measures of effect size (Cohen's *d*; Cohen, 1988) were, instead, obtained for significant comparisons between group means.

3.4. Software and Open Data

We conducted statistical analyses using SPSS Statistics for Macintosh, versions 23.0 – 25.0 (IBM Corp., 2015 – 2017), Mplus, version 8 (Muthén & Muthén, 1998 – 2017), and *R* environment for statistical computing, version 3.3.2 (R Core Team, 2016). Specifically, we used the *R* package OpenMx 2.10.0 (Neale et al., 2015) in **Study I**; Hmisc, 4.1-1 (Harrell et al., 2018), MASS, 7.3.45 (Ripley et al., 2013), and umx, 2.8.5 (Bates, 2018) in **Study II**; data.table 1.10.0-4 (Dowle et al., 2017), nFactors 2.3.3 (Raiche, 2010), psy 1.1 (Falissard, 2012), and semPlot 1.0.1 (Epskamp, 2014) in **Study III**; lavaan.survey 1.1.3.1 (Oberski, 2014), and semTools 0.4-14 (Pornprasertmanit et al., 2013) in **Studies I** and **III**. We posted annotated scripts and output files at each study web page on Open Science Framework

(osf.io/akrjc for **Study I**, osf.io/7qxh3 for **Study II**, osf.io/myskx for **Study III**, and osf.io/67fyr for **Study IV**). In accordance with the decision of our Ethics Committee, we made the dataset available upon request.

4. Results

The sections below summarize the results of the four manuscripts enclosed in the present work. Relevant numeric information is available in the manuscripts, directly in the text or in tables, figures, and appendices.

4.1. Study I: Genetic Vulnerability to Childhood Victimization

The **first aim** of **Study I** was to examine the etiology of the occurrence and co-occurrence of different types of childhood victimization. Beforehand, we examined the mean levels of childhood victimization in the full sample as well as by sex. Participants reported significantly higher mean levels of emotional neglect and emotional abuse, and significantly lower levels of sexual abuse, compared to other types of childhood victimization. Moreover, they reported significantly higher mean levels of family-specific compared to family-unspecific victimization. Women, compared to men, reported significantly higher mean levels of emotional and sexual abuse, but significantly lower mean levels of physical abuse. To obtain a first indication of genetic effects, we inspected the phenotypic cross-twin within-trait correlations. These were larger in MZs than DZs, suggesting additive genetic influences on each type of childhood victimization. Average DZ correlations were slightly larger than half the MZ correlations, suggesting shared environmental influences on each type of childhood victimization as well. MZ correlations were smaller than unity, suggesting unique environmental influences on each type of childhood victimization. Also, cross-twin cross-trait correlations were larger in MZs than in DZs, suggesting genetic influences common to different types of childhood victimization. With univariate models, we investigated whether the extent of the etiological influences varied depending on the type of abuse or neglect and the genetic relatedness with the perpetrator. We estimated modest additive genetic influences on physical abuse and neglect, and moderate influences on sexual

abuse and emotional abuse and neglect. Shared environmental influences were modest on physical neglect, small on physical abuse and emotional neglect, and non-significant on emotional and sexual abuse. We also estimated moderate unique environmental influences overall. The extent of the additive genetic influences did not differ significantly between family-specific and family-unspecific abuse and neglect. Nonetheless, shared environmental influences were significant only for family-specific victimization and unique environmental influences were significantly larger for family-unspecific compared to family-specific victimization.

To investigate the relative contribution of genetic and environmental factors to the risk of multiple victimization, we first ran a series of bivariate twin models between our composite scores of childhood victimization. Genetic and shared environmental correlations were positive and moderate-to-large, unique environmental correlations were positive and small. This suggested that genes and shared environments mostly contributed to the risk of multiple childhood victimization. Bivariate correlations were consistent across sexes. Next, with a common pathway multivariate model, we estimated one latent factor, accounting for the covariance between composite scores, and we estimated its etiological influences (see Figure 1). This model showed excellent fit. Compared to the univariate models, we estimated smaller additive genetic and larger unique environmental influences on all types of childhood victimization. This result indicated that genes largely influenced the susceptibility to multiple childhood victimization, while unique environmental factors largely influenced the likelihood of experiencing individual types of childhood victimization. Also, additive genetic influences were significantly smaller for emotional abuse and neglect, but only slightly smaller for sexual abuse, and unique environmental influences were significantly larger for all types of childhood victimization but sexual abuse. These results suggested the presence of shared influences underlying emotional maltreatment and other types of

victimization, as well as distinct influences underlying sexual abuse. Also, compared to the univariate models, no significant difference emerged in the extent of the shared environmental influences. These were small-to-zero for all types of childhood victimization, and slightly larger than zero for physical neglect. Thus, shared environmental factors did not appear to strongly influence individual types of childhood victimization.

The **second aim** of **Study I** was to clarify whether the impact of the genetic and environmental influences on childhood victimization varied as a function of the sex of the victim. In women, compared to men, we estimated comparable additive genetic and shared environmental influences on all types of victimization, and significantly smaller unique environmental influences on emotional abuse, emotional neglect, and sexual abuse. In light of these quantitative sex differences, we further explored the possibility of qualitative sex differences in the etiology of childhood victimization. To do so, for each type of childhood victimization, we estimated and compared a general sex limitation model and a common effects model. Nested model comparisons were close to significance for emotional and sexual abuse. For emotional abuse, sex-limited genetic influences were significantly larger and common genetic influences significantly smaller in women than men. For sexual abuse, sex-limited genetic influences emerged in women only, as the sex-limited genetic path was non-significant in men. If replicated, these findings might suggest that different sets of genes influence girls' and boys' risk of becoming a victim of emotional and sexual abuse, possibly via their influences on heritable traits that differ significantly between them.

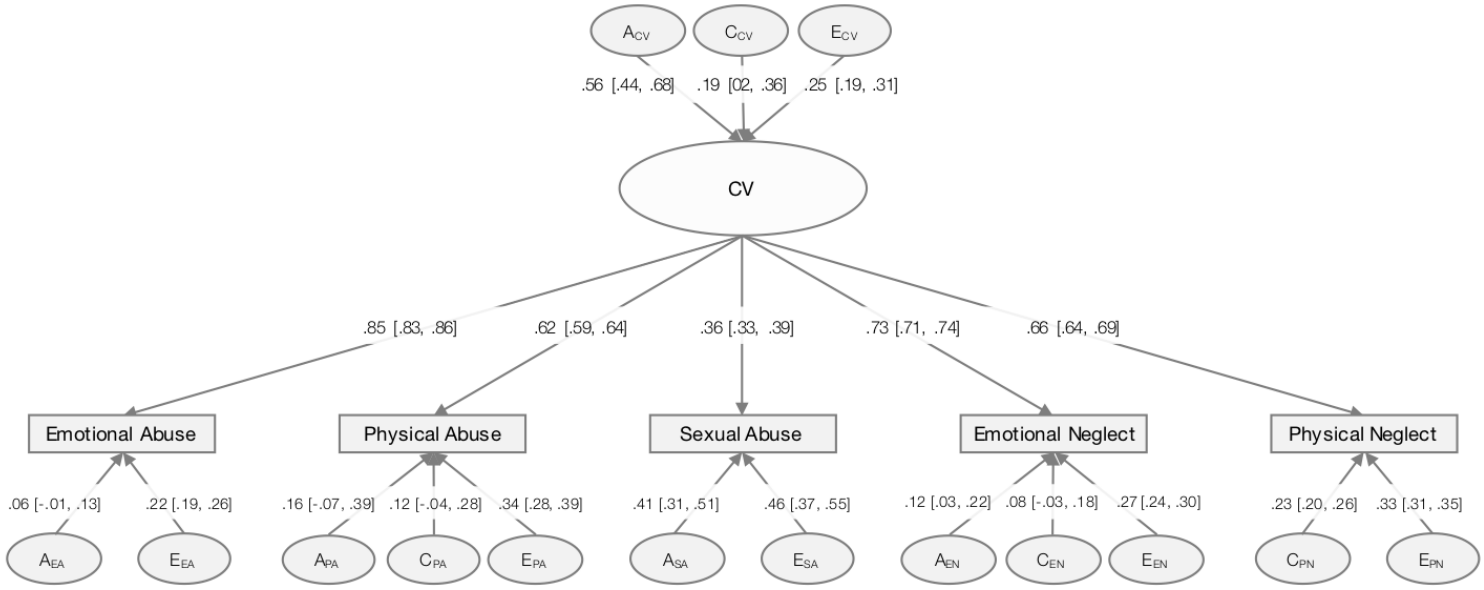

Figure 1. Common pathway twin model estimating the additive genetic (A), shared environmental (C) and unique environmental (E) sources of variance and covariance of emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect (**Study I**). CV = common pathway accounting for the variance and etiology shared between these types of childhood victimization. Squared standardized A, C, and E path coefficients (i.e., variance components) and standardized factor loadings are superimposed on their corresponding paths, confidence intervals are reported in brackets. Non-significant paths were fixed at zero and, thus, omitted.

4.2. Study II: Genetic Vulnerability to Adult Sexual Victimization

The **first aim** of **Study II** was to examine the etiology of adult sexual victimization. First, we inspected its prevalence and mean levels. More than two thirds of our participants reported no adult sexual victimization. Among the remaining participants, about one in ten reported having been touched or kissed against their will, and slightly less having experienced verbal or unspecified sexual harassment. Less than one in twenty reported complete or attempted forced penetrative sex, whether oral, vaginal or anal. We also estimated substantial sex differences, but no cohort effects. The prevalence of adult sexual victimization was more than three times higher in women than in men (36% vs. 11%). The prevalence of forced penetrative sex, in particular, was thirteen times higher (4% vs. 0.3%). Moreover, mean levels of adult sexual victimization were significantly higher in women than men.

Then, we explored possible genetic effects on the risk of adult sexual victimization by inspecting phenotypic cross-twin within-trait correlations. These were larger in MZs than DZs, consistent with additive genetic effects. However, DZ correlations were less than half the MZ's, suggesting dominant genetic rather than shared environmental influences. Thus, in two separate models, we decomposed the variance of adult sexual victimization into A, C, and E paths, as well as into A, D, and E paths. The ACE model indicated small additive genetic, no shared environmental, and large unique environmental influences on the risk of adult sexual victimization. The ADE model indicated small dominant and additive genetic influences and confirmed large unique environmental influences. These results indicated that environmental exposures not shared between siblings reared together

might influence the risk of being targeted by a perpetrator more than inherited traits.

The **second aim** of **Study II** was to explore the phenotypic and genetic relationship between childhood victimization, in particular child sexual abuse, and adult sexual victimization. The prevalence of adult sexual victimization was higher in victims of severe childhood victimization, irrespective of their sex and age. In particular, among victims of severe childhood victimization, the highest prevalence of adult sexual victimization was estimated for victims of severe child sexual abuse. Thus, frequency distributions suggested an association between childhood victimization, especially sexual abuse, and adult sexual victimization. Using regression models, we further clarified whether the risk of experiencing adult sexual victimization increased with a history of childhood victimization. When considered in isolation, all types of childhood victimization were positive statistically significant predictors of adult sexual victimization. Instead, when analyzed jointly, only emotional, sexual, and physical abuse remained positive statistically significant predictors. Importantly, emotional abuse emerged as the strongest predictor of adult sexual victimization, also when other types of childhood victimization were accounted for. The impact of child sexual abuse, instead, was considerably reduced when accounting for multiple types of childhood victimization.

Then, using bivariate and multivariate twin models with Cholesky decomposition, we examined the etiological relationship between childhood victimization and adult sexual victimization. Bivariate models indicated small-to-moderate genetic correlations between each type of childhood victimization and adult sexual victimization. Shared environmental correlations were estimated at unity, except for child sexual abuse, and unique environmental correlations were small. These results suggested that the same gene variants and environmental exposures, especially those shared between siblings, contributed to the small association between

childhood victimization and adult sexual victimization. The Cholesky decomposition supported the presence of significant additive genetic and unique environmental cross-paths only between child emotional and sexual abuse and adult sexual victimization. We could not detect any significant cross-paths for physical abuse, emotional neglect, and physical neglect. Next, with a multivariate common pathway twin model, we estimated a latent factor accounting for the variance and etiology common to all types of childhood victimization, and, again, we performed a Cholesky decomposition (see Figure 2). The decomposition supported a significant overlap in the additive genetic and unique environmental risk factors for multiple childhood victimization and adult sexual victimization. Therefore, these analyses showed that the same additive genetic and unique environmental risk factors for emotional abuse, sexual abuse, and multiple childhood victimization might also contribute to the risk of adult sexual victimization.

The **third aim** of **Study II** was to examine possible sex differences in the etiology of adult sexual victimization, as well as in the extent of its genetic and environmental association with childhood victimization. To do so, we first estimated regression models in women and men separately. For women, all types of childhood victimization were significant predictors of adult sexual victimization when considered individually, whereas, for men, sexual abuse and emotional neglect were not. Univariate coefficients were significantly larger for women for all types of childhood victimization but physical abuse. Thus, for women more than for men, all types of childhood victimization increased the risk of adult sexual victimization. Also, emotional abuse was the strongest predictor for women, while emotional as well as physical abuse were the strongest predictors for men. The multivariate model supported these findings. Emotional abuse was again the strongest predictor for women, although sexual abuse was also statistically significant, and physical abuse was the strongest and only significant

predictor for men. In other words, emotional and physical abuse especially increased the risk of adult sexual victimization for women and men, respectively.

Next, we estimated the twin models in women and men separately. The univariate results indicated significantly larger additive genetic and smaller unique environmental influences on the risk of adult sexual victimization in women compared to men. Therefore, gene variants appeared to influence women's risk of being sexually assaulted more than men's, presumably via their influences on other traits, while unique environmental exposures appeared to influence men's risk more than women's. Bivariate and multivariate findings indicated no significant difference between women and men in the extent of the etiological influences common to childhood victimization and adult sexual victimization. As an exception to this, the shared environmental influences common to multiple childhood victimization and adult sexual victimization were significantly larger in men than women, but, nonetheless, close to zero. Lastly, we clarified whether sex differences in the magnitude of the univariate estimates could be due to different sets of genes influencing the risk of adult sexual victimization in women and men. To do so, we estimated two sex limitation models, namely a general sex-limitation and a common effects model. Nested model comparison indicated that the sex-specific influences were not statistically significant, and, therefore, that the same genes influenced both women's and men's risk of being sexually assaulted. Nonetheless, an interesting finding emerged. In women, the sex-limited genetic path had a significantly larger impact on the risk of adult sexual victimization than the genetic path common to both sexes. Potentially, this finding explains why larger genetic influences emerge for women when sex-limited and common genetic effects are estimated jointly.

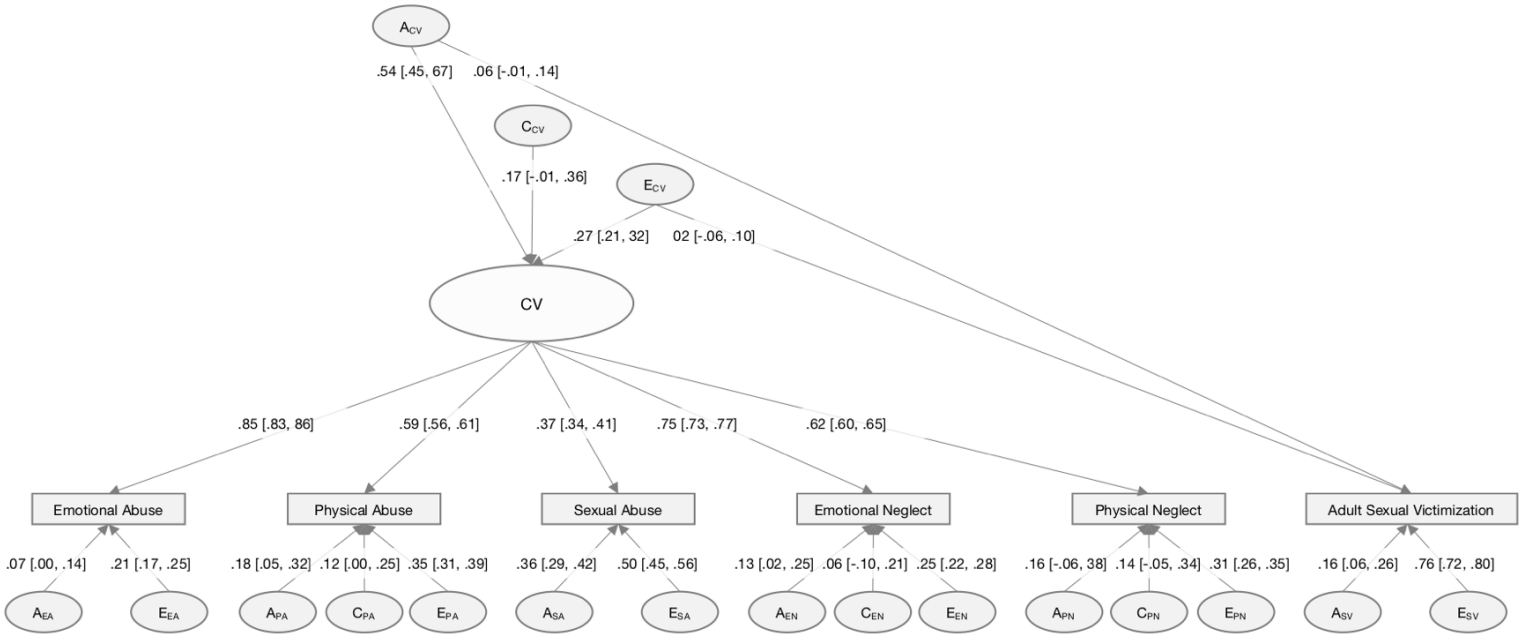

Figure 2. Common Pathway Cholesky twin model estimating the additive genetic (A), shared environmental (C) and unique environmental (E) sources of variance and covariance of different types of childhood victimization, as well as the overlap in the unique environmental influences on childhood victimization and adult sexual victimization (**Study II**). CV = common pathway accounting for the variance and etiology shared between emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Squared standardized A, C, and E path coefficients (i.e., variance components) and standardized factor loadings are superimposed on their corresponding paths, confidence intervals are reported in brackets. Non-significant paths were fixed at zero and, thus, omitted.

4.3. Study III: The Latent Sources of Psychiatric Comorbidity

The **first aim** of **Study III** was to expand the internalizing-externalizing meta-structure of psychopathology by including measures of anger, aggression, and body-related mental health problems. To evaluate and compare alternative latent structures, we conducted first-order and hierarchical exploratory and confirmatory factor analyses. Three latent factors emerged from the best-fitting first-order structure. Trait anger, aggression, alcohol use and psychopathy loaded onto a first factor, interpreted as the externalizing dimension. Depression, anxiety and sexual distress loaded onto a second, interpreted as the internalizing dimension. Disordered eating and body image dissatisfaction loaded onto a third factor, interpreted as a body-related dimension. Sexual distress and trait anger also cross-loaded onto the body-related dimension, suggesting a strong relationship between these and body-related symptoms. Among hierarchical structures, the bifactor model was retained as the most parsimonious explanation of our data (see Figure 3). Once we extracted a general "*p*" factor, sexual distress loaded more strongly onto the body-related than the internalizing factor. The variance of depression, sexual distress and trait anger were especially accounted for by the general "*p*" factor, suggesting that such symptoms potentially accompany any of these psychiatric conditions. Moreover, forcing the specific factors of internalizing, externalizing, and body-related psychopathology to be uncorrelated revealed that the "*p*" factor accounted for the covariance between body-related and the remaining problems, especially internalizing. Thus, body-related symptoms emerged as a separate dimension of psychopathology, rather than being subsumed within the internalizing dimension, and their covariance with other dimensions was explained by a broader vulnerability to psychopathology.

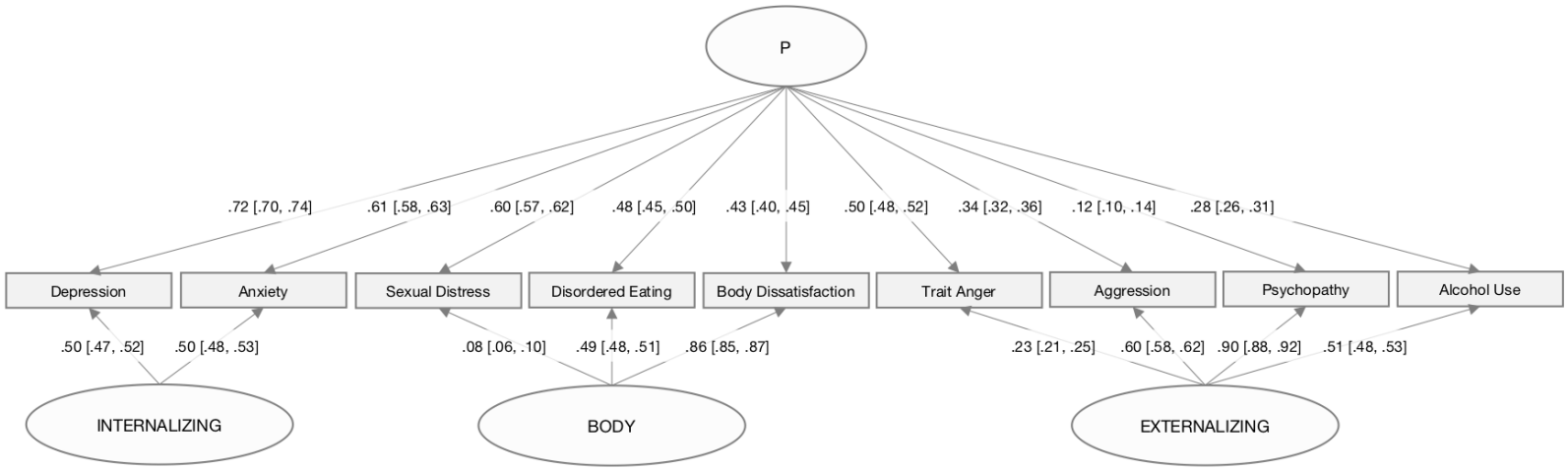

Figure 3. Bifactor model depicting the phenotypic sources of variance and covariance of core internalizing (depression and anxiety), externalizing (trait anger, aggression, alcohol use, psychopathy), and body-related (disordered eating, body image dissatisfaction, sexual distress) psychopathology (**Study III**). Standardized factor loadings are superimposed on their corresponding paths, confidence intervals are reported in brackets.

The **second aim** of **Study III** was to test the measurement invariance of the retained model of psychopathology and to compare sex and age groups on the latent dimensions. First, we tested invariance by sex. We were able to constrain the factor loadings on both general and specific factors to be equal across groups, but not the intercepts of the indicators. Additional analyses indicated that sexual distress was responsible for the non-invariance, meaning that sex differences in attitudes towards self-reporting sexual distress, such as social desirability and response style, might produce the observed differences in symptomatology, rather than true differences in the body-related dimension (Steinmetz, 2013). Residual variances and covariances of the specific factors, but not of the observed variables, were also sex-invariant. This result was unsurprising given that residual variances of the observed variables likely reflected measurement error. Moreover, since group comparisons on the latent means were precluded, we compared groups on the observed means, estimating higher levels of trait anger, internalizing, and body-related problems in women, and higher levels of aggression, alcohol use, and psychopathy in men. Next, we tested invariance across our three age cohorts (18 – 25, 26 – 32, 33 – 49). We were able to constrain the factor loadings on both general and specific factors, as well as the intercepts of the indicators, to be equal. Thus, we were also able to compare groups on the latent means. Participants aged 18 to 25 showed the highest levels of both internalizing and externalizing psychopathology, as well as of a general vulnerability to psychopathology ("*p*" factor). Instead, participants aged 33 to 49 showed the highest levels of body-related psychopathology. As with sex invariance, residual variances and covariances of the specific factors, but not of the observed variables, were age-invariant. In sum, measurement invariance testing supported the equivalence of our expanded bifactor model of psychopathology across cohorts, but not sexes.

4.4. Study IV: Childhood Victimization as a Source of Comorbidity

The **first aim** of **Study IV** was to clarify to what extent genetic and environmental factors contributed to the comorbidity of body-related and core internalizing mental health problems. To this end, we estimated a series of twin models, depicting different etiological pathways underlying these problems. In the best-fitting two-factor common pathway AE model, we estimated both etiological influences unique to each measure of body-related and internalizing psychopathology, as well as etiological influences common to them. We identified two latent factors: One factor accounted for the covariance and etiology of core internalizing problems, and a second factor accounted for the covariance and etiology of both body-related and internalizing problems. In other words, our results supported two separate additive genetic and unique environmental pathways: one specific to core internalizing problems, and one underlying body-related symptoms as well as the feelings of distress and fear associated with them.

The **second aim** of **Study IV** was to examine the relationship between different types of childhood victimization, separately and simultaneously, and both body-related and core internalizing psychopathological outcomes. To achieve this, we conducted a series of multivariate linear regressions. When considered separately, all types of childhood victimization were significantly and moderately associated with disordered eating, sexual distress, depression, and anxiety. Associations were, however, stronger for core internalizing than for body-related outcomes. Emotional abuse was the strongest predictor, sexual abuse the weakest. When all types of childhood victimization were considered simultaneously, only emotional abuse and emotional neglect remained statistically significant predictors, albeit with small effect sizes and R^2 values. Emotional abuse predicted core internalizing more strongly than body-related outcomes, whereas emotional

neglect predicted all outcomes to a similar extent. Moreover, a disorder-specific association emerged between physical neglect and anxiety. Altogether, our results highlighted the importance of accounting for multiple victimization, particularly for emotional abuse and neglect, as predictors of core internalizing and, to a smaller extent, body-related problems.

The **third aim** of **Study IV** was to clarify whether any significant association between childhood victimization, on the one hand, and body-related and core internalizing mental health problems, on the other, could be explained by shared genetic and environmental risk factors. To do so, we used a Cholesky decomposition which estimated the overlap in the additive genetic and unique environmental influences on a common factor of childhood maltreatment and the common factors underlying body-related and core internalizing psychopathology (see Figure 4). Additive genetic factors explained a moderate-to-large portion of the covariance of multiple childhood victimization and psychopathology, but a negligible portion of the variance in psychopathology. Unique environmental cross-paths explained the remaining portion of covariance between multiple childhood victimization and body-related problems and were non-significant for core internalizing problems. According to these findings, the same gene variants that might predispose a child to become a victim of child abuse and neglect might also influence the risk of developing body-related and internalizing mental health problems later in life. Similarly, the same unique environmental exposures might predispose to childhood victimization as well as adult body-related psychopathology. Albeit significant, common etiological pathways only explained a marginal portion of the variance in psychopathology, consistent with the weak phenotypic associations.

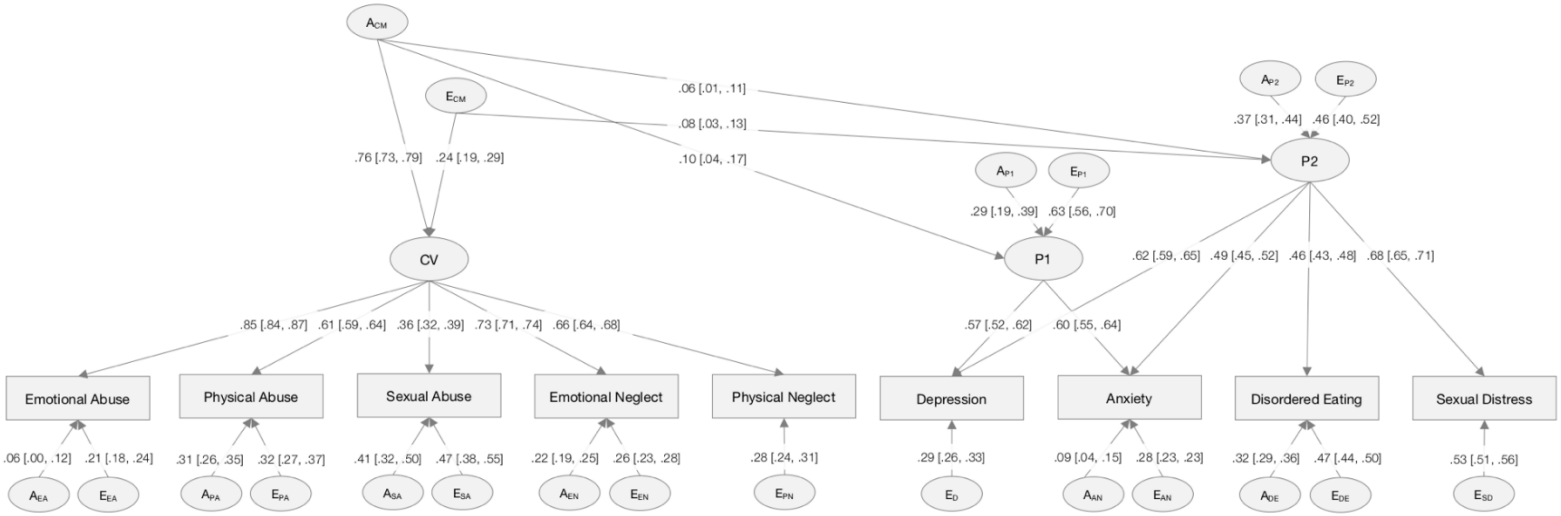

Figure 4. Common Pathway Cholesky model estimating the overlap in the additive genetic (A) and unique environmental (E) influences on childhood victimization, on the one hand, and body-related (disordered eating and sexual distress) and core internalizing (depression and anxiety) psychopathology, on the other hand (**Study IV**). CV = Common pathway accounting for the variance and etiology shared between emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect; P1 = common pathway accounting for the covariance and etiology of core internalizing problems; P2 = common pathway accounting for the covariance and etiology of body-related and internalizing problems. Squared standardized A, C, and E path coefficients (i.e., variance components) and standardized factor loadings are superimposed on their corresponding paths, confidence intervals are reported in brackets. Non-significant paths were fixed at zero and, thus, omitted.

5. Discussion

5.1. Interpersonal Victimization: Prevalence and Forms

Past research has shown that interpersonal victimization is a pervasive problem (Benjet et al., 2015), with harmful outcomes for victims (McCrory et al., 2011; Dworkin et al., 2017). The present thesis focused on child abuse and neglect as well as on adult sexual assault. Using a genetically informed design in a large representative sample, we retrospectively addressed interpersonal victimization and we analyzed its prevalence, risk factors, and sequelae.

Broadly in line with evidence from recent meta-analyses (e.g., Norman et al., 2012, Stoltenborgh et al., 2014), **Study I** showed that emotional abuse and neglect are the types of childhood victimization most frequently experienced by children, sexual abuse the least frequently experienced. Emotional abuse and neglect are, almost by definition, perpetrated by someone in a close relationship with the child. Indeed, most of our items inquired emotional maltreatment by parents and family members. Consistent with past reviews (e.g., Gilbert et al., 2009), thus, our results demonstrated that children are mostly victimized within their family environment. Moreover, sexual abuse emerged as the type of child victimization least frequently experienced even though it has been the most extensively addressed by previous research (Stoltenborgh, Bakermans-Kranenburg, Alink, van IJzendoorn, 2012). Even if all types of childhood victimization should be studied and, crucially, prevented, our findings indicate that more empirical and public attention should be devoted to emotional maltreatment.

Also, in accordance with previous nationally representative studies (Breiding, 2015; Krug et al., 2002; Smith et al., 2017), **Study II** indicated that approximately one in four adults experience sexual victimization, and one

in twenty experience attempted or completed forced penetration, whether oral, vaginal, or anal. Since forced sex represents a serious threat for victims' health (Dworkin et al., 2017), its prevention is vital. It is even more common that individuals who experience some form of sexual violence are touched or kissed against their will or verbally harassed. Thus, our results support public appeals for increased prevention of all forms of sexual abuse (e.g., #MeToo movement), including acts that may not constitute sex crimes.

5.2. The Etiology of Interpersonal Victimization

While studies investigating the possible outcomes of interpersonal victimization abound, evidence on its risk and protective factors is largely inconclusive (Collin-Vézina, Daigneault, & Hébert, 2013; Turner, Finkelhor, & Ormrod, 2009, DeGue et al., 2014). Previous investigations have mainly focused on environmental exposures that increase the risk of interpersonal violence, such as underprivileged urban settings (Decker et al., 2014) and problematic family backgrounds (Finkelhor, Ormrod, Turner, & Holt, 2009). However, environmental exposures, especially involving genetically related individuals, may be subject to genetic influences, a phenomenon known as gene-environment correlation (Jaffee & Price, 2007). In spite of this, the present work was among the first to estimate the extent of genetic and environmental influences on the risk of interpersonal victimization.

Study I demonstrated that genes might influence the risk of childhood victimization. In particular, a correlation between inherited gene variants and environmental exposures shared between siblings raised together might increase the risk of multiple victimization. Instead, environmental exposures unique to a child might increase the risk of unique forms of victimization, especially by genetically unrelated individuals. These findings can be interpreted as evidence of intrafamilial intergenerational recursive effects (Hines & Saudino, 2002; Berlin, Appleyard, & Dodge, 2011; Pittner

et al., 2019). In other words, some forms of maltreatment might be more prevalent in some families than in others because of a correlation between the traits that individuals in such families share, and the environments that they inherit, evoke, and shape. For example, in families where more individuals suffer from impulse control issues, partly due to their genetic make-up and genetic similarity, it seems more likely that conflicts will escalate into verbal or physical aggression. Thus, interventions supporting high-risk families should be a priority in violence prevention agendas. Also, our results suggest that specific genetic and environmental influences, unshared between siblings, underlie the risk of experiencing child sexual abuse. Thus, familial factors, including exposure to other forms of childhood victimization, may not contribute to the risk of child sexual abuse as much as individual traits and contexts that perpetrators take advantage of. As a result, to protect vulnerable children, such traits and contexts should be identified (Seto, 2019).

Moreover, **Study II** demonstrated that unique environmental exposures exert the largest influence on adults' risk of being targeted by sexually motivated perpetrators. As a result, future research needs to clarify where adult sexual victimization is most likely to occur (Jewkes, Fulu, Roselli, & Garcia-Moreno, 2013; Tharp et al., 2013). At the same time, however, small genetic and shared environmental influences also emerged, suggesting that some traits or behaviors, partly under genetic and shared environmental control, might increase the risk of becoming a victim. This finding fits with the limited available evidence that individuals with certain traits, such as mental health problems (Fisher et al., 2016; Krnjacki et al., 2015), might be preferentially targeted by sex offenders. As a consequence, it seems especially important that individuals with high-risk traits be protected from additional environmental risk factors. Of note, the large unique environmental influences estimated on adult sexual victimization might also, in itself, reflect *r*GE as well as gene-environment interaction

effects (GxE, i.e., the expression of genetic effects dependent upon environmental factors and vice versa; Dick, 2011). Indeed, *r*GE and GxE could accumulate over time, as individuals select into increasingly compatible environments and respond to environments based on their genotype (Beam, Turkheimer, Dickens, & Davis, 2015; Plomin & Asbury, 2005). In the classical twin design, if genetic effects differ based on environmental exposures unshared between siblings raised together, unique environmental estimates will be inflated (e.g., Medland & Hatemi, 2009). Thus, increased unique environmental effects on experiences in adulthood compared to childhood might reflect the actual greater impact of unshared exposures on individual variation or, instead, increased *r*GE and GxE effects.

5.3. Sex Differences in the Etiology of Interpersonal Victimization

Past research has shown that sex differences exist in the prevalence of various forms of interpersonal victimization. In particular, women are more likely than men to experience sexual assault both during childhood (Stoltenborgh et al., 2011) as well as adulthood (Breiding, 2015). This also emerged from **Studies I** and **II**. Conversely, men are more likely than women to experience physical assault both during childhood (Thompson et al., 2004) and adulthood (Iverson et al., 2013). Accordingly, in **Study I**, we found that men were more likely than women to report a history of child physical abuse. Nonetheless, it was less clear whether sex differences extended to emotional abuse and neglect (English, Thompson, White, & Wilson, 2015; Stoltenborgh et al., 2012). In the present work, we found higher prevalence of emotional abuse in girls than boys, but comparable prevalence rates of emotional and physical neglect. Importantly, the reasons for such sex differences were poorly understood. As a consequence, we

further examined sex differences in the extent of the genetic and environmental risk of interpersonal victimization.

In **Studies I** and **II**, we estimated higher heritability of childhood victimization as well as adult sexual victimization in women. With respect to childhood victimization, **Study I** further showed that the higher heritability reflected genetic influences specific to women on the risk of emotional and sexual abuse. Specifically, sex-specific sets of genes were found to influence the risk of child emotional abuse for women more than men, and the risk of child sexual abuse for women exclusively. Thus, sex-specific genetic effects might explain why girls experience such types of child maltreatment more than boys. In other words, perpetrators of emotional and sexual abuse might preferentially take advantage of girls, and, in particular, of those girls who present certain heritable sex-specific traits. For example, sex offenders might pursue pubertal more than pre-pubertal girls (Bergen, Antfolk, Jern, Alanko, & Santtila, 2013), whose onset of puberty is influenced by sex-specific genes (Stringer, Polderman, & Posthuma, 2017). With respect to adult sexual victimization, **Study II** suggested that the increased heritability might reflect increased environmental homogeneity (Johnson, Turkheimer, Gottesman, & Bouchard, 2009) for women compared to men, rather than sex-specific genetic effects. In other words, since women are more persistently exposed to the risk of adult sexual victimization across multiple environments (Ellsberg et al., 2015), individual differences in this risk might be influenced by genetic and unshared environmental factors. For instance, attitudes that condone predatory behaviors by heterosexual men might expose all women to a comparable risk of sexual assault (Butchart, Phinney, Check, Villaveces, 2004). Individual differences will, in turn, depend on the specific traits and environments that offenders exploit to perpetrate sexual violence.

5.4. The Sequelae of Childhood Victimization

Childhood victimization has been shown to predict subsequent victimization (Miron & Orcutt, 2014) and psychiatric vulnerability (Kessler et al., 2010). However, past literature has largely failed to clarify whether childhood victimization constitutes a causal risk factor, or, instead, whether the same risk factors underlie childhood victimization and later adversities (Clay-Warner et al., 2016). For instance, although widely assumed, the causal association between child victimization and mental illness has rarely been empirically addressed (Schaefer et al., 2017). Moreover, past research has not conclusively determined whether different types of childhood victimization have disorder-specific or unspecific effects on different mental health problems (Cecil et al., 2017). In fact, most studies have focused on individual associations (Armour, Elklit, & Christoffersen, 2013). Thus, in **Studies II** and **IV**, we examined the impact of different types of childhood victimization, separately as well as together, on the risk of adverse outcomes, including adult sexual victimization, body-related, and internalizing mental health problems.

Emotional maltreatment emerged as the strongest predictor of all these adverse outcomes. This result was somewhat surprising given the limited empirical attention dedicated, until recently, to the characterization (Glaser, 2011), consequences (English et al., 2015), and prevention (Baker, Brassard, Schneiderman, Donnelly, & Bahl, 2011) of emotional maltreatment. Indeed, at present, possible behavioral and biological connections between emotional maltreatment and adult sexual victimization (Zurbriggen et al., 2010) as well as mental health problems (Cecil et al., 2017) remain largely speculative. While most past research has focused on the sequelae of child sexual abuse (Hovdestad et al., 2015), our findings suggest that the impact of this form of abuse on the risk of adult sexual victimization and psychopathology is considerably reduced when accounting for multiple childhood victimization. This does not mean that

child sexual abuse is not deleterious *per se*. It means, instead, that alternative risk pathways to adult adversities might have remained uninvestigated. For instance, the disorder-specific association between childhood physical neglect and adult anxiety is worth investigating further. Also, effect sizes for all types of childhood victimization on all adult outcomes were small-to-moderate, suggesting that multiple risk factors, including but not limited to childhood victimization, contribute to adult sexual victimization and psychopathology. Importantly, we also provided empirical evidence that the link between childhood victimization and such outcomes could be explained by genetic and environmental risk factors common to them. In particular, the same gene variants and unique environmental exposures might influence the risk of recurrent victimization as well as of psychopathology following multiple childhood victimization. However, these shared etiological effects only explained small amounts of variance in all measured outcomes. These results demonstrate not only that individual differences in lifetime risk of victimization and psychopathology are partly due to genetic backgrounds, but also that the associations between such adverse experiences and health outcomes are partly under genetic control.

Furthermore, **Study IV** indicated that genetic risk factors strongly contribute to psychiatric comorbidity. In particular, we identified a genetic liability specific to core internalizing disorders, consistent with past research (e.g., Cerdá et al., 2010), as well as a novel genetic pathway accounting for the additive genetic and unique environmental influences on body-related problems and to their correlated symptoms of distress and fear. These partly separate etiological pathways might explain why some but not all patients suffering from core internalizing disorders also report body-related symptoms. Although the possibility of transdiagnostic genetic variants has been supported by genomic research (Anttila et al., 2016), molecular approaches have not yet solved the "missing heritability" problem, that is, the gap between the heritability of psychopathology and other complex

traits estimated by family studies and the heritability of such traits explained by specific loci on the genome (Maher, 2008). Alongside intermediate phenotypes, dominant genetic and epigenetic effects, sex-specific genetic effects might be hiding in this gap (Gilks, Abbott & Morrow, 2014). Nonetheless, past genetic research has largely ignored the link between sex-related genetic variation and sex differences in health (Short, Yang, & Jenkins, 2013). Thus, it was unclear whether epidemiological sex differences in mental health reflected sex differences in the etiological dimensions underlying them. In **Study III**, we addressed this possibility at the phenotypic level by testing whether the internalizing, externalizing, and body-related dimensions of psychopathology were consistent across sexes. While the internalizing and externalizing dimensions were sex invariant, in line with previous research (Eaton et al., 2012), body-related problems were not. This result indicated that sex differences in other traits, such as attitudes towards self-reporting body-related problems, might explain sex differences in symptom levels, although these may not reflect true differences at the latent, etiological level.

5.5. Implications for Prevention and Treatment

While past studies have investigated genetic and familial risk factors for interpersonal violence perpetration (e.g., Långström, Babchishin, Fazel, Lichtenstein, Frisell, 2015; Pettersson, Larsson, & Lichtenstein, 2015), research on the interplay between genes and environments in the vulnerability to interpersonal victimization was scarce. Presumably, researchers were concerned that searching for individual characteristics increasing the risk of victimization would be perceived as victim blaming (Hines & Saudino, 2008). Similarly, primary prevention has typically consisted of knowledge-building educational programs (Harden, Buhler, & Parra, 2016). The effect size of these programs in terms of prevention, however, has been found to be small (DeGue et al., 2014). Their enduring

popularity might be due to the limited scientific evidence on individual risk factors, but, also, to concerns related to placing the responsibility of prevention in the hands of potential victims (Collin-Vézina et al, 2013). In this thesis, we undertook the challenge of exploring victims' genetic risk for different forms of interpersonal victimization, for their associations, and for their mental health consequences. Besides advancing the scientific knowledge on abuse and neglect, our findings have implications for their prevention as well as the treatment of its victims.

First, the present work indicated that genes might play a role in the etiology of childhood victimization, and, to a smaller extent, of adult sexual victimization. This explains, at least in part, why interpersonal victimization is neither randomly nor evenly distributed in the population, or, in other words, why certain individuals are more vulnerable to victimization than others. Notably, this result suggested that interpersonal violence might especially affect individuals raised in problematic families, due to a correlation between genetic and environmental vulnerabilities. For example, if one or both parents possess a genetic predisposition to antisocial behavior, including violent behavior (Tielbeek et al., 2017), their offspring might inherit this genetic susceptibility, and, in addition, they might witness violent behaviors within their family (Hines & Saudino, 2004). A similar situation might have, at least, two adverse aftermaths. First, these children might already be at risk of violent behavior themselves, due to both genetic and environmental influences. Second, any challenging behavior on their behalf might enhance parental stress and exacerbate parental emotional outbursts. Given the complexity of these recursive intergenerational effects, we propose that high-risk individuals and their families require tailored interventions. In fact, an unfavorable genetic background can be counterbalanced by a protective environment. Moreover, interventions for high-risk individuals might be especially effective in the context of global public health efforts that promote appropriate parenting practices, such as

community-wide parenting support. By promoting appropriate nurturing responses, and correcting sub-optimal and maladaptive practices, these interventions might help parents deal with, for example, sensitive developmental stages like puberty and critical periods like divorce. Thus, based on the results of the present work, we support prior calls for a public health approach to the prevention of child victimization (e.g., Herrenkohl, Higgins, Merrick, & Leeb, 2015).

Second, if different forms of interpersonal victimization are influenced by different risk factors, different interventions might be necessary to address them. For example, according to our results, emotional abuse and neglect represent the most detrimental forms of childhood victimization in terms of subsequent sexual victimization, internalizing and body-related mental health problems. However, early signs of childhood emotional abuse and neglect might be more difficult to detect than signs of physical abuse and neglect (Wolfe & McIsaac, 2011). As a result, identifying children at high risk of emotional maltreatment, and preventing continued maltreatment and its outcomes, might be especially challenging. More broadly, individuals who are routinely in contact with children, such as teachers and child health practitioners, should be systematically trained to recognize and respond promptly and adequately to the signs of emotional maltreatment. In addition, our results showed that child sexual abuse might be influenced by risk factors unshared with other types of childhood victimization. However, insofar, the prevention of child sexual abuse has mainly involved offender management, which does not prevent child exploitation before it happens (Collin-Vézina et al., 2013). Thus, more efforts should be directed to preventing individuals who are sexually attracted to children from offending in the first place.

Third, our findings support the impact of childhood victimization on adult psychopathological outcomes. Therefore, professionals who work with victims of childhood abuse and neglect should take into account their

patients' heightened risk of mental health problems, including but not limited to post-traumatic stress disorder (PTSD; Ehring et al., 2014). A history of childhood victimization might also contribute to psychiatric comorbidity, especially of internalizing disorders. As a result, diagnostic approaches focusing on etiological mechanisms, including neurobiological alterations associated with victimization history, might be more effective for subsequent treatment than approaches based solely on observed symptoms. To this aim, attempts to integrate the results of initiatives like the National Institute of Mental Health's Research Domain Criteria with the results of the childhood victimization literature seem especially promising (Stover & Keeshin, 2018). Nonetheless, effect sizes were small and associations were explained by common etiological factors. In other words, a large portion of the variance in body-related and core internalizing psychopathology remained unexplained after accounting for a history of childhood victimization. This result is consistent with the fact that not all individuals who suffer from mental health problems have a history of childhood abuse and neglect (e.g., Rehan, Antfolk, Johansson, Jern, & Santtila, 2017). Despite this, child victimization has been among the lifetime adversities most widely investigated as candidate risk factors for psychopathology. Our findings indicate that other experiences potentially relevant to mental illness should also be explored. Moreover, our results demonstrate an overlap in the genetic vulnerability to childhood victimization and psychopathology. This finding suggests that individuals with unfavorable genetic predispositions might be at risk of both adverse life experiences and mental health problems. However, a predisposition should not be interpreted as a pre-destination, as it may be compensated by protective factors.

5.6. Limitations and Recommendations for Future Research

Our findings should be interpreted in the context of their limitations. Some limitations pertain to variables that we have failed to analyze. First, we did

not comprehensively assess the characteristics of the perpetrators of both childhood victimization and adult sexual victimization. In particular, while item measures of childhood victimization referred to either parents, family members, or undefined individuals, no information was collected for adult sexual victimization. Thus, we could not appreciate the exact relationship between victims and perpetrators. As a result, future research should differentiate between degrees of genetic similarity as well as between intimate partner and stranger violence, to clarify whether these are affected by different risk factors. Second, we did not measure the age at which victimization occurred nor the age of onset of mental health problems. Past research has shown that interpersonal victimization predicts mental illness (Schaefer et al., 2017), but also that mental illness predicts interpersonal victimization (Turner et al., 2009). Thus, future genetically informed designs should address the direction of causation. Third, we did not control for pertinent covariates, such as parental psychopathology and socioeconomic status, that have been associated with increased risk of victimization (Finkelhor et al., 2009) and mental health problems (Matthews & Gallo, 2011). In fact, despite the modest response rates to our data collections (36% and 45%, respectively; Johansson et al., 2012), we might not have included the most disadvantaged groups of the population due to selection bias.

Other limitations pertain to our Finnish population-based twin sample. Since heritability is a population statistic, we cannot rule out that different results might be obtained in different populations. In particular, genomic research has shown that Finns differ significantly from other European populations in terms of genetic variation (Lek, 2016). How this translates into differences in the genetic and environmental risk of interpersonal victimization and psychopathology remains, therefore, an empirical question. Also, cultural aspects distinctive of the Finnish population might have influenced the mean levels on these variables and,

conceivably, their etiology. Furthermore, our sample was ethnically homogeneous. Past studies have shown that minority ethnic communities in developed countries experience more victimization (Lauritsen, Rezey, & Heimer, 2013) and untreated mental health problems (Waheed, Hughes-Morley, Woodham, Allen, & Bower, 2015) compared to individuals of European ancestry. Moreover, although the representativeness of our twin sample has been illustrated in previous studies (Johansson et al., 2012; Rehan et al., 2017), we cannot exclude that traits specific to twins and their families might have influenced our results. For instance, the simultaneous parental investment might be increased for parents of young twins than age-discrepant siblings, potentially increasing parental stress, a risk factors for child victimization (Yokoyama, Oda, Nagai, Sugimoto, & Mizukami, 2015). Nonetheless, twins have not been found to differ significantly from non-twins in terms of victimization rates (Barnes & Boutwell, 2013) and health variables (Andrew et al., 2001).

Lastly, some limitations pertain to our measurement tools. First, we exclusively employed retrospective self-reports. While past research has supported the agreement between prospective and retrospective accounts of child victimization and mental health (Reuben et al., 2016; Scott, McLaughlin, Smith, 2012) as well as between self-reports and clinical ratings (Morey, Krueger, & Skodol, 2013), we believe that future research should focus on intermediate phenotypes. Second, although we measured multiple types of childhood victimization and adult sexual victimization, we were unable to analyze other forms of lifetime victimization, such as adult physical assault. Similarly, not all mental health problems have been addressed. Moreover, we did not measure interpersonal violence *perpetration*, which has also been associated with child victimization history (Hines & Saudino, 2002) and mental health problems (Tharp et al., 2013). Therefore, the present work suggested several avenues for future research

that could replicate and extend our findings as well as overcome their limitations.

5.7. Conclusions

Previous research has shown that genetic and environmental vulnerabilities can predispose to the perpetration of interpersonal violence. Instead, less was known about the genetic and environmental vulnerabilities to becoming a victim of interpersonal violence. Since environment-level prevention programs have not eradicated violence from modern societies, research on individual-level risk profiles represented a promising strategy. Also, interpersonal victimization has been shown to confer susceptibility to a host of mental health problems. However, whether this effect is causal and disorder-unspecific remained unclear. Since different adversities and mental health problems tend to co-occur and to share risk factors, they should be concurrently examined, possibly using genetically controlled designs.

In present thesis, we have demonstrated that both genetic *and* environmental factors influence the risk of various forms of childhood victimization and adult sexual victimization, with substantial etiological differences between women and men. These results contribute to explaining why some individuals are at increased risk of interpersonal victimization, including recurrent victimization. We have also explained the implications of these findings for the conceptualization, prevention, and management of interpersonal violence. Moreover, we have provided support for a modest and transdiagnostic effect of childhood victimization on mental health problems, as well as for disorder-specific associations. We have further illustrated that putatively distinct mental health problems reflect broad latent dimensions of psychopathology, with implications for diagnosis and treatment, and that both gene variants and environmental exposures,

including childhood victimization, contribute to these dimensions as well as to their correlation.

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Patrizia Pezzoli

Genetic Vulnerability to Patterns of Interpersonal Victimization and Associated Psychiatric Comorbidity

The present thesis investigated the etiology and sequelae of interpersonal victimization in a large representative Finnish twin sample. Results indicated that genes, in addition to environmental vulnerabilities, explain why some individuals experience victimization more likely than others. This work also highlighted the perniciousness of childhood emotional maltreatment and illustrated the etiological sources of sex differences in the risk of victimization and psychiatric comorbidity.

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