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# Risk Factors for Child Sexual Abuse Victimization: A Meta-Analytic Review

Mark Assink, Claudia E. van der Put,  
and Mandy W. C. M. Meeuwse  
University of Amsterdam

Nynke M. de Jong  
Ambulant Center for Forensic Psychiatry De Waag, Den Haag,  
the Netherlands

Frans J. Oort, Geert Jan J. M. Stams, and Machteld Hoeve  
University of Amsterdam

Experiencing child sexual abuse (CSA) is a major public health problem with serious consequences for CSA victims. For effective assessment and (preventive) intervention, knowledge on risk factors and their effects is crucial. Here, the aim was to synthesize research on associations between (putative) risk factors and CSA victimization. In total, 765 (putative) risk factors were extracted from 72 studies, which were classified into 35 risk domains. A series of three-level meta-analyses produced a significant mean effect for 23 of the 35 risk domains ranging from  $r = .101$  to  $r = .360$ . The strongest effects were found for prior victimization of the child and/or its family members, such as prior CSA victimization of the child and/or siblings ( $r = .360$ ), prior victimization of the child other than child abuse ( $r = .340$ ), prior or concurrent forms of child abuse in the child's home environment ( $r = .267$ ), and a parental history of child abuse victimization ( $r = .265$ ). Other identified risks were related to parental problems (e.g., intimate partner violence,  $r = .188$ ), parenting problems (e.g., low quality of parent–child relation,  $r = .292$ ), a non-nuclear family structure (e.g., having a stepfather,  $r = .118$ ), family problems (e.g., social isolation,  $r = .191$ ), child problems (e.g., having a mental/physical chronic condition,  $r = .193$ ), and other child characteristics (e.g., being female,  $r = .290$ ). Moderator analyses suggested that contact CSA victimization may be better predicted than noncontact CSA victimization. It was concluded that an ecological perspective on preventing CSA victimization is necessary.

### **Public Significance Statement**

This review integrates research on risk factors for child sexual abuse (CSA) victimization. A substantial number of significant risks were identified. The results showed that the most important risks refer to prior (CSA and non-CSA) victimization of children and/or family members. Other relevant risks refer to different types of problems and characteristics of parents, the family system, and the child itself. Non-nuclear family structures also pose risk for CSA victimization.

**Keywords:** child maltreatment, child sexual abuse, meta-analysis, risk factor, sexual victimization

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In the past several decades, many scholars have provided meta-analytic evidence for the association between child sexual abuse (CSA) victimization and a variety of mental, physical, and behav-

ioral problems over the life course. These problems range from general symptoms of psychopathology (Neumann, Houskamp, Pollock, & Briere, 1996) to more specific problems, such as suicidal behavior (DeVries et al., 2014; Neumann et al., 1996), depression and anxiety (Beitchman, Zucker, Hood, DaCosta, & Akman, 1991; Lindert et al., 2014; Neumann et al., 1996), eating disorders (Smolak & Murnen, 2002), substance abuse (Neumann et al., 1996), risky sexual behavior in adolescence (Homma, Wang, Saewyc, & Kishor, 2012), and committing sexual offenses against children (Whitaker et al., 2008).

The detrimental effects for CSA victims as well as the economic costs attributable to CSA (see, for instance, Hankivsky & Draker, 2003; Saied-Tessier, 2014) make it imperative to prevent children from becoming a sexual abuse victim. For prevention strategies to be effective, clinicians must be able to accurately assess the risk

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Mark Assink, Claudia E. van der Put, and Mandy W. C. M. Meeuwse, Research Institute of Child Development and Education, University of Amsterdam; Nynke M. de Jong, Ambulant Center for Forensic Psychiatry De Waag, Den Haag, the Netherlands; Frans J. Oort, Geert Jan J. M. Stams, and Machteld Hoeve, Research Institute of Child Development and Education, University of Amsterdam.

Correspondence concerning this article should be addressed to Mark Assink, Research Institute of Child Development and Education, University of Amsterdam, P.O. Box 15780, 1001 NG, Amsterdam, the Netherlands. E-mail: [m.assink@uva.nl](mailto:m.assink@uva.nl)

for sexual victimization of a child, so that prevention is offered to those children identified as having a substantial risk for CSA victimization. In addition, clinicians must determine what exactly should be addressed in these preventive efforts to reduce the risk of future CSA (i.e., assessment of changeable risk factors, or care needs, that need to be addressed). In the assessment of risk and needs, knowledge about the effects of different risk factors for CSA victimization is necessary. Up to now, many studies have identified different risk factors for CSA, but a systematic quantitative overview of these risk factors was not available. Hence, the aim of the present review was to meta-analytically summarize associations between risk factors and child sexual abuse victimization.

### Risk Factors and Theories

One of the theories that has commonly been used in predicting risk for crime victimization of youth is the routine activities theory, which was first posed by Cohen and Felson (1979). Central to this theory is that children's activities lead to environments or situations where they may come into contact with potential offenders. Thus, by undertaking certain activities, children put themselves at risk for victimization. Specifically, this theory suggests that victimization occurs when adequate supervision of a victim's guardian is missing in situations where victims are in close proximity to individuals who are not only motivated to commit an offense, but also view the victim as an attractive target. In short, the routine activities framework is built around four concepts and states that crime victimization occurs in an (a) *interaction* of (b) a *potential offender* and (c) a *suitable target* in the absence of (d) *capable guardianship* (Tilley, Farrell, & Clarke, 2015). In this framework, the target suitability concept refers to the vulnerability of the individual to be victimized. Finkelhor and Asdigian (1996) argued that the routine activities theory cannot explain all forms of victimization, such as intrafamilial and acquaintance victimizations. They noted that family members and acquaintances may very well be perpetrators of child sexual abuse (Finkelhor, 1994), and that many children are (sexually) victimized without engaging in activities that put them in a risky environment. This means that an increased risk for victimization is not necessarily determined by routine activities of children.

In building on the concepts of the routine activities theory of Cohen and Felson (1979), Finkelhor and Asdigian (1996) focused on environmental factors that lead to victimization. From their perspective, the lack of guardianship, because of fighting and/or inattentive parents, is an environmental condition that puts a child at risk for victimization and not a problem in the routine activities of a child. They also focused on child characteristics that increase a child's vulnerability to victimization, as they may be congruent with the needs, motives, and/or reactions of offenders. As a result, Finkelhor and Asdigian reconceptualized Cohen and Felson's concept of target suitability and broke it down into three components. They stated that child characteristics may increase the risk for victimization, because (a) they compromise a child's capacity to resist or deter victimization, making the child an easier target (target vulnerability); (b) they represent a child's quality, possession, skill, or attribute that an offender wants to obtain, use, have access to, or manipulate (target gratifiability); and (c) they represent a child's qualities, possessions, skills, or attributes that arouse

anger, jealousy, or destructive impulses of the offender (target antagonism). Thus, according to Finkelhor and Asdigian, child sexual abuse may occur because environmental risks are present and/or offenders are drawn to, or react to, children with specific characteristics that are in line with an offender's motives.

Both the routine activities theory (Cohen & Felson, 1979) and later theoretical elaborations from Finkelhor and Asdigian (1996) put emphasis on environmental factors that contribute to victimization, and are therefore ecological in nature. However, the diversity in environmental risk factors covered by these theories is relatively low. A different theoretical framework built around a broader range of child characteristics and environmental factors that increase a child's risk for child abuse victimization, was posed by Belsky (1980), who based his theory on Bronfenbrenner's (1979) ecological perspective on child development. Belsky emphasized that the risk for child abuse victimization is influenced by the interplay of risk factors and protective factors at four different levels that vary in proximity to the child: (a) aspects of the history of each parent that is brought into the parenting role and the family (ontogenic development), (b) characteristics of the child and family (microsystem), (c) characteristics of parental employment, the community, and social support (exosystem), and (d) characteristics of a society's attitude toward children and maltreatment (macrosystem). Following Bronfenbrenner, Belsky noted that ecological influences can be proximal or more distal to the child. Proximal influences represent direct and immediate vulnerabilities to child abuse, whereas distal influences represent indirect and broad vulnerabilities affecting social systems surrounding the child. According to Belsky, child abuse is determined by the balance between risk and protective factors, and because these factors come in many different forms, he argued that there are many different pathways leading to child abuse.

After Belsky (1980) developed his ecological model, Cicchetti and Rizley (1981) elaborated that transactional influences must be taken into account when explaining child abuse. In their model, not only ecological or systemic influences are considered, but also the complex interplay (i.e., transactions) between child characteristics and environmental factors. They stated that each ecological system comprises potentiating and compensatory factors exerting influence on both the child and ecological systems surrounding the child. Potentiating factors increase the risk for child abuse, whereas compensatory factors decrease this risk. They also noted that potentiating and compensatory factors have a temporal dimension. Transient factors can fluctuate and may be temporary, whereas enduring factors represent more permanent or chronic characteristics.

A more recent model is the ecological, transactional, and developmental model of child sexual abuse as proposed by Bolan (2001). Her model builds upon the ecological and transactional theoretical approaches, but she added that the risk for CSA can only be grasped in the context of a child's developmental stage. Bolan posed that children's developmental (cognitive, affective, or physiological) maturity is related to the risk of abuse, and that depending on age, children are differentially accessible or vulnerable to potential offenders. Bolan's model suggests that at any point in time, a child's level of risk for sexual abuse is determined by the transactional interaction between risk factors in all systemic environments and a child's developmental stage and history. In this view, risk is conceived to be dynamic and may increase and

decrease over time, as a child's developmental stage and environmental context change. Bolan also stressed that the risk for sexual abuse is in particular determined by factors in the macro- and exosystem. Based on the notions of Reiss (1981) and Cicchetti and Lynch (1993), she implied that the critical influence of the macro- and exosystem is expressed through a family's internalized representational model of beliefs, values, and assumptions of the community and society in which it resides. In other words, risk factors present in ecological systems that are more proximate to the child originate at ecological systems that are more distal to the child.

In the theories described above, risk factors play a critical role in understanding the risk for child abuse victimization. Throughout the years, many different scholars were guided by these theories and identified a large number of different risk factors for CSA victimization. These risk factors vary greatly in nature. Some factors pertain to child characteristics (e.g., being female, having behavioral or physical health problems), others pertain to environmental factors, including parental factors (e.g., substance abuse, being unemployed, and being violent) and characteristics of the community in which the child and the family reside (e.g., high rate of crime and violence in the neighborhood and poor social family support). Further, given the transactional nature of several theories, risk factors pertaining to interactions between the child and the environment (e.g., low rate of social interaction between child and parents) have also been examined.

Primary studies on risk factors often show a wide variation in effect magnitude of the factors. For instance, many studies have found that a child being female is a relatively strong risk factor for child sexual abuse (with correlations  $> .500$ ; e.g., Bouvier et al., 1999; Fergusson, Lynskey, & Horwood, 1996), but others have found that being female is hardly associated with CSA (with correlations  $< .100$ ; e.g., Mohler-Kuo et al., 2014; Turner, Vanderminden, Finkelhor, Hamby, & Shattuck, 2011), or that being female may even have protective effects (with negative correlations; e.g., Tang, 2002). In a meta-analysis, such divergent findings can be brought together in estimating a single effect for each risk factor. In doing so, more insight may be gained in the risk factors that contribute most to CSA victimization, leading to a better understanding of its etiology. Additionally, from primary studies alone, it is difficult to understand how different methods and samples affect the strength of risk factors. A meta-analysis can quantify the effects of these differences between primary studies on the magnitude of risk factors.

### Risk Factors and Treatment

In child welfare, many clinical professionals are tasked daily with predicting whether children will become victims of child abuse (especially in the near future). Therefore, many studies in the child abuse literature are aimed at improving the prediction of child abuse victimization by identifying one or more predictors that clinical professionals can use in detecting and assessing the risk for victimization. However, knowledge of risk factors is not only essential in determining *who* should receive (preventive) treatment, but also in determining *what* care needs must be met to reduce the risk for victimization. A theoretical framework for delivering effective treatment in which risk factors play a critical role is the risk-need-responsivity (RNR) model (Andrews & Bonta, 2010; Andrews, Bonta, & Hoge, 1990; Bonta & Andrews,

2007, 2010), which is a criminological theory designed for guiding effective rehabilitation programs aimed at reducing recidivism of (juvenile) criminal offenders.

The RNR model comprises three principles of which two are relevant in light of the present study: the risk principle and the need principle. The former states that an intervention's intensity should match an offender's risk of recidivism, implying that treatment of high intensity should be offered to individuals at high risk for recidivism, and treatment of low intensity (or no treatment at all) should be offered to low-risk individuals. The latter principle states that interventions should target risk factors for recidivism that are dynamic (i.e., changeable in treatment) and present in the offender or the offender's environment. Forensic treatment services adhering to these principles are much more effective than services ignoring these principles, which has consistently been proven in multiple comprehensive meta-analytic reviews (e.g., Andrews et al., 1990; Dowden & Andrews, 1999a, 1999b; Koehler, Lösel, Akoensi, & Humphreys, 2013). As described in the previous section, the most common theories for sexual abuse victimization are fundamentally based on the presence of risk factors in different ecological systems. Therefore, it can be hypothesized that applying the risk and need principles of the RNR model to (preventive) treatment strategies is equally effective in child welfare as it is in criminal justice.

For applying the risk and need principles in child welfare, instruments for risk and needs assessment are required. Risk assessment instruments assess static and dynamic risk factors and produce a risk for child abuse victimization informing clinical professionals on who should be treated with what intensity. Needs assessment instruments assess only dynamic risk factors (i.e., the potential care needs) informing clinical professionals on what should be targeted in treatment, so that the risk for victimization can be reduced. By determining which variables are true risk factors for CSA victimization, and what the predictive abilities of these factors are, the present review may help in providing an empirical foundation for developing (or improving) instruments for risk and needs assessment. Further, this study may offer grounds for improving and developing treatment services, so that all potential care needs of at-risk children (and their families) can be addressed, and the risk for CSA victimization is reduced (see also Van der Put, Assink, Gubbels, & Boekhout van Solinge, 2018).

### The Present Review

Until now, there was no quantitative overview of risk factors for child sexual abuse victimization. A meta-analysis of risk factors was necessary, as single primary studies cannot provide an accurate estimate of the true effect of risk factors, nor can they provide proper insight in how risk factor effects are influenced by study, sample, and/or risk factor characteristics. Accordingly, we conducted the present meta-analytic review to address two questions about risk factors for CSA victimization. The first question is what variables are true risk factors for CSA victimization and what are their effects? In answering this question, we first classified all variables of which the predictive value was examined in primary studies into one of a number of risk domains, which we defined as (broad) categories of risk factors that are similar in nature. Next, an overall mean effect was estimated for each of these risk domains

in a separate meta-analysis. The second research question is how are risk factor effects influenced by study, sample, and risk factor characteristics? We examined the potential impact of these characteristics on the predictive ability of risk factors by performing moderator analyses. In addressing these questions, the present meta-analytic review has the potential to provide more insight into risk factors for CSA victimization and to give rise to improvements of clinical practice.

## Method

### Defining Child Sexual Abuse

In defining child sexual abuse, we used the definition of the World Health Organization (1999) which described CSA as “the involvement of a child in sexual activity that he or she does not fully comprehend, is unable to give informed consent to, or for which the child is not developmentally prepared and cannot give consent, or that violate the laws or social taboos of society.” (p. 15). As for the concept “child,” there is no clear agreement on the absolute age ranges for childhood, adolescence, and adulthood in literature on (re-) victimization of CSA (see also Walker, Freud, Ellis, Fraine, & Wilson, 2017). Some researchers define experiences of CSA as victimization prior to 12 years of age (e.g., Miron & Orcutt, 2014), whereas other studies on CSA are directed on unwanted sexual activities up to the age of 18 years (e.g., Campbell, Greeson, Bybee, & Raja, 2008; Davis, DeMaio, & Fricker-Elhai, 2006). In the present study, we chose to examine (putative) risk factors for victimization of sexual abuse of children and adolescents up to the age of 18 years. In this way, we excluded risk factors related to sexual assault of (young) adults, which can be quite different in nature from risk factors for CSA victimization.

In the more rigorous studies in the CSA literature, sexual activities among peers are often ruled out by limiting CSA victimization of adolescents to specifically contact sexual abuse in the forms of unwanted attempted or completed rape by a perpetrator who is at least five years older than the victim (see also Stoltenborgh, Van IJzendoorn, Euser, & Bakermans-Kranenburg, 2011). According to this criterion, a 16-year-old who is flashed or mooned by a peer is not regarded as CSA victimization. On the other hand, if the sexual abuse perpetrator is an adult relative, experiences of any sexual activity (contact or noncontact) are often regarded as CSA victimization. This implies that a 12-year-old who is forced to watch pornography by an adult caretaker is regarded as CSA victimization. Unfortunately, in many primary studies that examine variables as potential risk factors for CSA victimization, no (sufficient) information is reported on the specific CSA type, the age difference between the perpetrator and the victim at the time of the sexual abuse episode(s), and/or the abuse context (intrafamilial or extrafamilial). Therefore, we decided not to further narrow our CSA definition based on these aspects. In sum, we focused on contact and noncontact CSA victimization of children and adolescents up to 18 years of age.

### Defining Risk Factors

As noted by Kraemer et al. (1997), the terms *risk*, *risk factors*, and especially the term *cause* have inconsistently and imprecisely been used in scientific literature, leading to incorrect scientific

claims and recommendations for research and policy not grounded in proper empirical research. To prevent this, a clarification of terms is necessary. According to Kraemer et al., a *correlate* is a factor that is associated with another factor. For instance, if children showing antisocial behavior tend to become victims of child sexual abuse, this may indicate that antisocial behavior of children is a correlate of CSA victimization. Because correlates can be associated with factors in different ways, specific study designs are needed to determine the nature of the association. A *risk factor* is a specific type of correlate that precedes the outcome of interest and can be used to classify a population into risk groups, such as low, medium, and high risk groups. Continuing the example, if children showing antisocial behavior (at Time 1) were more likely to become a victim of child sexual abuse (at Time 2), antisocial behavior of children would be a risk factor for CSA victimization. A *causal risk factor* is another type of correlate that can be identified when the manipulation of the correlate systematically changes the probability for the outcome of interest. Further continuing the example, if experimentally influencing the amount of antisocial behavior of children (by means of treatment) at Time 1 systematically changes the probability of CSA victimization at Time 2, antisocial behavior of children can be considered a causal risk factor. Distinguishing between these terms is essential, because causal risk factors are predictors and valuable treatment targets; (noncausal) risk factors are predictors, but may be less effective treatment targets; and correlates may be nothing more than poor predictors and ineffective treatment targets (Franklin et al., 2017). Franklin et al. note that improper use of these terms may have major negative impact on theory development, research, and clinical practice.

In scientific literature on risk factors for child sexual abuse, a retrospective cross-sectional design is far more used than a prospective longitudinal design. In the first, (adult) participants are often asked whether they ever were a victim of CSA, under what circumstances the victimization occurred, and what factors preceded the victimization. Following infants over time, keeping track of sexual abuse victimization, and assessing a variety of factors that may pose a risk for victimization at multiple points in time, are very costly research activities. Therefore, only a limited number of studies in the field of child sexual abuse is prospective and longitudinal, although this number has been increasing in recent years. Consequently, using the typology of Kraemer et al. (1997), it can be difficult to determine whether factors examined in retrospective cross-sectional studies are true risk factors, or merely correlates (see also the inclusion and exclusion criteria below). This especially holds for factors that are dynamic in nature, as many static factors—if recalled accurately by participants—precede the victimization because of their constant presence. In this study, we use the term *risk factor* for all variables that could be included in the present review given our inclusion and exclusion criteria.

### Inclusion and Exclusion Criteria

Because the aim of the present review was to provide a broad overview of risk factors for CSA victimization, all potentially eligible studies were examined for variables that were tested as risk factors. From this point onward, the terms “effect” and “effect size” refer to the magnitude of the association between a variable

and CSA victimization. The following criteria were used to select relevant studies reporting on risk factors and their effects.

**Language.** Only studies written in Dutch, English, French, or German, of which the full-text could be retrieved, were included.

**Published studies.** As a form of quality control, studies had to be published in peer-reviewed scientific journals or be (part of) a dissertation that was accessible to the authors of this review. We decided not to include unpublished studies, because these are far more difficult to locate, and because published studies have survived some form of a refereeing and editing process (Dunkin, 1996). Although often not peer-reviewed, we did decide to include dissertations, as these are subject to quality control in the form of supervising committees, and thus had an aspect of critical academic evaluation beyond the work of the authors (Merrell, Gueldner, Ross, & Isava, 2008).

**Outcome.** Studies had to examine CSA victimization, matching with our definition of CSA (see above).

**Risk factors.** As risk factors must precede the outcome of interest (Kraemer et al., 1997), only effect sizes of variables that preceded the CSA victimization were included. These variables could be either static or dynamic (i.e., changeable) in nature. More specific, primary studies had to report on at least one association between CSA victimization and a factor preceding the victimization, or a factor of which reasonably could be assumed to precede the victimization based on information described in the primary study. Primary studies only examining the association between gender and CSA victimization and/or ethnicity and CSA victimization (i.e., mere prevalence studies) were not included in the present review, because these studies were meta-analyzed by Stoltenborgh et al. (2011).

**Nontreatment studies.** Because our aim was not to perform a meta-analysis of the effects of treatment strategies for victims of child sexual abuse, and because treatment effects may influence risk factor effects, we did not extract effects of risk factors that are reported in studies examining treatment effects.

**Required statistical information.** Primary studies had to report on associations between (putative) risk factors and CSA victimization or on sufficient statistical information to calculate these associations. If measures of association (e.g., correlations or odds-ratios) were not directly reported in primary studies, the study had to discriminate between participants who were assessed as having a history of experiencing child sexual abuse (i.e., the abuse condition) and participants assessed as not having a history of experiencing child sexual abuse (i.e., the comparison condition). In this way, statistics such as means and standard deviations, and proportions, could be converted into correlations. As for the size of the two conditions, each had to consist of a minimum of five participants, because studies with small sample sizes are more susceptible to publication bias and tend to overestimate effect sizes (Sterne, Gavaghan, & Egger, 2000; Turner, Bird, & Higgins, 2013; Zhang, Xu, & Ni, 2013).

**Western studies.** Given that the prevalence of CSA victimization differs across countries (Stoltenborgh et al., 2011), and because risk factors may be very different in prevalence and nature across cultural settings, we decided to only include studies that were performed in Western countries (i.e., European countries, Australia, New Zealand, Canada, and the US).

## Literature Search

In our search for relevant primary studies, our purpose was to be as exhaustive as possible, and therefore we performed three complementary search strategies which were completed in June 2018. First, studies were identified through searching the electronic databases PsycINFO, ERIC, Sociological Abstracts, ScienceDirect, and Google Scholar. Searches included permutations of keywords related to “risk factors” and “child sexual abuse victimization.” These keywords were: “abuse,” “maltreatment,” “assault,” “violen\*,” “victim\*,” “child\*,” “youth,” “adolescen\*,” “juvenil\*,” “risk,” “risk factor\*,” “predict\*,” “sex\*,” and “correlate\*” (the asterisk represents one or more wildcard characters). Second, reference lists of relevant review articles (e.g., Bidarra, Lessard, & Dumont, 2016; Black, Heyman, & Smith Slep, 2001; Jones et al., 2012; Maniglio, 2015; McEachern, 2012; Meinck, Cluver, Boyes, & Mhlongo, 2015; Stith et al., 2009; Stoltenborgh et al., 2011) and the reference section of Bolan’s book (Bolan, 2001) on child sexual abuse were searched for missing references. Third, we searched the journals *Child Abuse & Neglect*, *Child Maltreatment*, and *Journal of Child Sexual Abuse* for missing references using each journal’s electronic search function. In each search, the keywords mentioned above were entered in different combinations. Finally, we screened the reference sections of all included primary studies. In determining whether identified studies in each search strategy met the inclusion and exclusion criteria, we read titles, abstracts, and, if necessary, full article texts. The final list of included studies was checked by all coauthors and is reported in Appendix A.

## Data Extraction and Coding

In coding each included primary study, we applied a coding system that was based on coding guidelines of Lipsey and Wilson (2000). This coding system was developed by the first author in consultation with the coauthors. Coding categories were developed prior to coding the studies, but were adapted in an iterative process when necessary. In coding studies for meta-analytic research, it is common practice to retrieve a large amount of information from primary studies (see for instance, Cooper, 2010; Lipsey & Wilson, 2000), after which the moderating effect of a variety of study, sample, and research design descriptors is tested. However, because the problem of multiple testing often dealt with in primary studies (see, for instance, Tabachnik & Fidell, 2013) is equally present in meta-analysis, we decided to code a specific set of variables that seemed most relevant in light of the aims of the present review.

**Type of child sexual abuse.** As there are different forms of CSA, we coded sexual abuse as (a) contact sexual abuse (i.e., involving physical contact), (b) noncontact sexual abuse (i.e., not involving physical contact), or (c) mixed (i.e., contact and noncontact sexual abuse).

**Child sexual abuse assessment.** Different approaches can be applied to assessing CSA and may influence the magnitude of risk factors. For instance, the results of MacMillan, Jamieson, and Walsh (2003) revealed that only a small percentage of respondents who reported to be a victim of CSA also reported having had contact with official authorities. In other words, official records only capture a small fraction of the actual number of CSA instances. Therefore, we coded type of CSA assessment using the

categories (a) self-report with a single-item assessment, (b) self-report with a multiitem assessment, (c) face-to-face interview, (d) telephone interview; (e) family report, or (f) official record (e.g., child protection services record). In case the self-report or interview method was used in primary studies, we also coded the number of screen/interview questions, as higher number of sexual victimization questions may result in higher reporting rates (Fricke, Smith, Davis, & Hanson, 2003), which in turn may influence estimates of risk factor effects.

**Age cut-off.** As there are differences in cut-off age in CSA definitions between primary studies, we coded the cut-off age as described in a study's definition.

**Publication year.** As the prevalence of both CSA and risk factors may fluctuate over time, we coded the year of publication for each published study.

**Risk domains.** Across all effect sizes that could be extracted from all included studies, there were too many risk factors to examine individually. For valid and intelligible analyses, we classified each individual risk factor into one of 35 risk domains, which can be defined as categories of risk factors that are (more or less) similar in nature. The risk domains are listed in Appendix B with examples of risk factors classified in each of the domains.

**Type of parent.** As effects of father-related risk factors may not be the same as effects of mother-related risk factors, we coded whether a (parental or family) factor was related to a father- or mother-figure, wherever possible.

**Sample age, gender, and ethnicity.** For each study, the mean sample age was coded and the sample's gender was coded as the percentage of males/boys. We also coded the percentage of Whites/Caucasians in samples.

**Statistical adjustment.** Combining and comparing differentially adjusted effect sizes may limit a robust estimation of the true effects of risk factors. Because scholars rarely use the exact same set of covariates in their studies, we aimed for including zero-order effects (i.e., unadjusted or bivariate effects) of each risk factor. Although unadjusted effects could be calculated for most risk factors reported in the primary studies, a number of studies only reported adjusted effect sizes. Not including these adjusted effects would limit the number of risk domains that could be examined in this review. Therefore, we decided to include both unadjusted and a relatively small number of adjusted effect sizes, and to code for each risk factor if the effect was adjusted or unadjusted. In this way, we could test statistical adjustment as a potential moderator of effects of risk domains.

Several additional data extraction and coding decisions were made. First, if primary studies reported on sufficient data for extracting effect sizes for subgroups of participants, we coded effects of risk factors for CSA in each subgroup. If coding within subgroups was not possible, we only coded risk factors for the total group of participants. Second, most of the reported factors in primary studies could reasonably be hypothesized to increase risk for future CSA victimization, but during the coding process there were complications to this general rule that required attention. Because the aim was to examine effects of risk factors (e.g., "the child has a physical disability"), we not only excluded true protective factors (e.g., "stable family relationships"), but also factors that merely indicated the absence of risk (e.g., "no episodes of CSA victimization in earlier generations of the child's family") or a rather low degree of risk (e.g., "low number of family conflicts

versus high number of family conflicts"). Third, we decided to disregard the age of children because there were not only differences between studies in how the child's age was measured (continuous vs. discrete), but also large differences in age categorizations across studies. Moreover, there is no clear linear pattern in the association between the child's age and CSA victimization (e.g., Finkelhor, 1993).

The data extraction and coding procedure was conducted using an SPSS sheet that was specifically designed for the present review. In the first coding round, the first, third, and fourth author independently coded 10 randomly selected studies that were eligible for inclusion. Next, the independent codings were compared, coding inconsistencies were discussed, and interrater agreements were determined by calculating an intraclass correlation ( $r$ ) for continuous variables and Fleiss' kappa ( $\kappa$ ) for categorical variables. For the continuous variables, the percentage of males in the sample, the percentage of Whites/Caucasians in the sample, mean age of the sample, study publication year, and the number of CSA screen/interview questions, the agreement was excellent ( $r = 1.000$ ). A slightly lower agreement was found for the calculated effect sizes ( $r = .952$ ). The agreement was also excellent for the categorical variables CSA assessment type, age cut-off, and statistical adjustment, with  $\kappa = 1.000$ . Somewhat less agreement was found for CSA type ( $\kappa = 0.810$ ), the risk domain in which a factor was classified ( $\kappa = 0.942$ ), and whether a factor was related to a mother- or father-figure ( $\kappa = 0.937$ ). When less than excellent agreement was reached for a variable, remaining inconsistencies were resolved until the three authors agreed on all final coding decisions. In case consensus on a final coding decision could not be reached, the second author was consulted and acted as an arbitrator. Prior to coding the remaining studies, the coding procedure and SPSS sheet were modified, where necessary. In the second coding round, the first author coded the remaining 62 studies.

## Calculating Effect Sizes

To quantify the effect of a (putative) risk factor for CSA victimization, we calculated the Pearson product-moment correlation coefficient as the common effect size for each risk factor reported in each primary study. Because reported associations between risk factors and CSA victimization were expressed in different forms across primary studies (e.g., correlations, means and standard deviations, and odds-ratios), it was often necessary to transform study-specific data into correlation coefficients. For these transformations, methods and formulas of Ferguson (1966), Lipsey and Wilson (2000), and Rosenthal (1994) were used. In addition, it was necessary that each correlation coefficient reflected the association between a (putative) risk factor and CSA victimization in the proper direction. Therefore, when higher levels of risk factors (e.g., higher levels of interparental problems, parental alcohol/drug use, or mental problems of children) were associated with higher levels of CSA victimization, a positive sign was given to correlation coefficients, whereas a negative sign was given when higher levels of factors were associated with lower levels of CSA victimization. For a total of 31 nonsignificant factors (reported in 10 studies), it was not possible to calculate a correlation coefficient, because the required statistical information was not provided in the primary studies. In these instances, we

assigned the value of zero to the coefficient, which is a conservative estimate of the true association (see also, Mullen, 1989). We preferred this method above excluding primary studies because of insufficient reporting on nonsignificant risk factors.

As recommended by several scholars (e.g., Cooper, 2010; Lipsey & Wilson, 2000), correlation coefficients should be transformed into Fisher's  $z$  scores in meta-analysis, because the latter have a normal sampling distribution. Therefore, in the final step of calculating effect sizes, all correlations were transformed into Fisher's  $z$  scores. After the statistical analyses were performed, the Fisher's  $z$  scores were converted back into correlations to facilitate interpretability.

## Analytic Strategy

**General strategy.** Because we were interested in effects of different risk factors for CSA victimization, we first estimated the overall strength of each risk domain. Additionally, we were interested in how the overall strength of individual risk domains is influenced by specific variables (e.g., CSA type, age cut-off, sample age, etc.). Therefore, we tested variables as potential moderators of the strength of individual risk domains in separate metaregressions. The complete dataset that we used in all analyses can be found in the [online supplemental material](#) for this review.

**Random effect models.** Because we considered the included primary studies to be a random sample of the population of studies, we used a random-effects approach in all our analyses (see, for instance, Raudenbusch, 2009; Van den Noortgate & Onghena, 2003). In fixed effects meta-analyses, it is assumed that there is one "true" effect (i.e., the population parameter) that is to be estimated, and that all effect sizes extracted from primary studies approximate this true effect. However, because there is often considerable variance between effect sizes as a result of methodological differences between primary studies, this assumption is rarely valid. As a result, inaccurate estimates of effect sizes are produced. In random effects meta-analyses, there is not one single "true" population parameter that is to be estimated, as it is assumed that real differences exist between primary studies in the strength of the effect. In terms of variance, fixed effects models only assume the presence of within-study sampling error, implying that all variation in effect sizes is caused by chance. On the other hand, random effects models assume the presence of both within-study sampling error and between-study variance. In most meta-analyses, random effects models are to be preferred over fixed effects models (Kelley & Kelley, 2012).

**Effect size dependency.** Because primary studies typically examined more than one variable as a (putative) risk factor for CSA victimization, more than one effect size could be extracted from each included study. Put differently, most included studies examined multiple—and qualitatively different—risk factors for CSA victimization, which were all relevant for the present review and had to be classified in different risk domains. Therefore, we were interested in extracting *all* relevant effect sizes from each included study. Besides examining multiple (putative) risk factors, other reasons primary researchers may have for reporting multiple effect sizes are the aim to examine risk factors for different aspects of CSA victimization (such as contact and noncontact CSA), and the use of different instruments to assess risk factors and/or CSA victimization.

An important assumption in meta-analytic research is independence of effect sizes (see, for instance, Cooper, 2010; Lipsey & Wilson, 2000). However, extracting multiple effect sizes from single studies violates this assumption, because these effect sizes are more alike than effect sizes extracted from different studies. After all, the former may be based on the same participants, instruments, and/or circumstances in which the research was conducted (Houben, Van den Noortgate, & Kuppens, 2015). In more traditional approaches to meta-analysis (i.e., fixed effect models or two-level random effect models), this assumption is often met by averaging or eliminating effect sizes in primary studies (e.g., Assink & Wibbelink, 2016). A drawback of these procedures is that important information is lost, leading to less statistical power in the analyses. Moreover, such procedures severely limit the ability to address specific research aims, such as estimating effects of multiple domains of (putative) risk factors for CSA victimization, which is central to the present review. Therefore, we extracted all relevant effect sizes from each included study, and dealt with effect size dependency by applying a three-level approach to meta-analysis (Assink & Wibbelink, 2016; Cheung, 2014; Hox, 2002; Van den Noortgate, López-López, Marin-Martinez, & Sánchez-Meca, 2013, 2014).

In a three-level meta-analytic model, three sources of variance are considered: Variance in effect sizes extracted from different studies (i.e., between study variance) at level 3 of the model, variance in effect sizes extracted from the same study (i.e., within study variance) at level 2 of the model, and sampling variance of the extracted effect sizes at level 1 of the model (Cheung, 2014; Hox, 2002; Van den Noortgate et al., 2013, 2014). This model allowed us to calculate an overall effect size and, if significant between study variance (at level 3) and/or within study variance (at level 2) was observed, to examine variables that may explain significant variance by extending the model with (possible) moderating variables. Note that the sampling variance of extracted effect sizes (level 1) is not estimated in this meta-analytic model, but is considered known. We used the formula of Cheung (2014, p. 215) to estimate the parameter that is associated with this source of variance. We refer the reader to the work of Assink and Wibbelink (2016) for an introduction to three-level meta-analysis.

**Overall strength of individual risk domains.** For obtaining an overall strength of each risk domain, we built a three-level meta-analytic model without predictors (i.e., intercept-only models) for each risk domain. In these models, the estimated intercept value represents the effect of one risk domain that was tested against the null hypothesis of no effect to determine its significance.

## Assessment of Bias

Although we tried to be as exhaustive as possible in our search for primary studies on risk factors for CSA victimization, it is possible that we missed relevant studies because of limitations in our search strategy or different forms of bias, such as publication bias or subjective reporting bias. In examining whether (a form of) bias was present in the effect sizes we analyzed, four analyses were conducted. First, we conducted the funnel-plot-based trim and fill method (Duval & Tweedie, 2000a, 2000b). In case of an asymmetrical distribution of effect sizes (i.e., an asymmetrical funnel plot), the trim-and fill method restores symmetry of the distribu-

tion by imputing effect size estimates from “missing” studies. Effect sizes can be imputed either in the left or right side of the funnel plot, depending on whether below average or above average effect sizes are underrepresented in the data. Second, we conducted a classical Egger’s test (Egger, Davey Smith, Schneider, & Minder, 1997), in which effect sizes were regressed on standard errors with effect size weights inversely proportional to the variance of effect sizes (Sterne et al., 2000). In this model, a significant slope is an indication of bias. Third, an adapted Egger’s test was conducted in which effect sizes were regressed on standard errors in a three-level meta-analytic model. Contrary to the classical Egger’s test, this adapted test accounted for effect size dependency. Once again, a significant slope is an indication of bias. Last, we estimated Begg and Mazumdar’s rank order correlation (Begg & Mazumdar, 1994) representing the rank association between standardized effect sizes and the weighting factors of each effect size, which are determined by the sample size on which the effect is based. A significant rank association implies the presence of bias in the data. These four analyses were performed in the R environment (Version 3.2.0; R Core Team, 2015) with the functions “trimfill,” “regtest,” “rma.mv,” and “ranktest” of the metafor package (Viechtbauer, 2010), respectively.

**Heterogeneity and moderator analyses.** Prior to testing variables as potential moderators of the strength of individual risk domains, we first determined whether heterogeneity in effect sizes was present within each risk domain. Specifically, we determined the significance of the variance at levels two (variance in effect sizes extracted from the same study) and three (variance between studies) of the meta-analytic model by performing two separate one-tailed log-likelihood ratio tests in which the deviance of the full model was compared with the deviance of the model excluding one of the variance parameters. In case of significant variance at level two and/or three, we proceeded with testing variables as potential moderators of the overall strength of an individual risk domain. In these moderator analyses, we only tested variables as moderators when (all categories of) these variables were based on at least five studies. To meet this lower bound, we sometimes combined categories of the originally coded discrete variables, which are described in the data extraction and coding section. In contrast, the moderating effect of statistical adjustment was tested for all risk domains in which both unadjusted and adjusted effect sizes were classified. Each potential moderator was examined in a separate three-level meta-analytic model in which the potential moderator was added as a covariate. Prior to conducting all moderator analyses, we centered each continuous variable around its mean and created dummy variables for each discrete variable.

**Statistical software and parameters.** All meta-analytic models were built in the R environment (Version 3.2.0; R Core Team, 2015) by using the function “rma.mv” of the metafor package (Viechtbauer, 2010). The R syntax was written so that the three-level structure as described by Cheung (2014) and Van den Noortgate et al. (2013, 2014) was applied to our meta-analytic models (see the tutorial of Assink & Wibbelink, 2016). The significance of model coefficients was tested two-tailed using the Knapp-Hartung adjustment (Knapp & Hartung, 2003), meaning that individual coefficients were tested using a *t*-distribution and that all model coefficients were tested using an *F*-distribution (i.e., omnibus test; excluding the intercept). In estimating the model parameters, the

restricted maximum likelihood estimation method was applied. In all analyses, a five percent significance level was used.

## Results

### Primary Studies

The search procedure resulted in 74 eligible articles (*k*) describing 72 studies (*l*), that were published between 1980 and 2017 (median publication year was 2003). Studies were conducted in U.S.A. (*l* = 45), Canada (*l* = 9), Australia or New Zealand (*l* = 9), and in Europe (*l* = 9). The mean age of the participants was 18.58 years (*SD* = 12.25). In total, 765 effect sizes could be retrieved from the primary studies, each reflecting the effect of a putative risk factor for CSA victimization. Of this total number of effect sizes, 687 effects were statistically unadjusted and 78 effects (extracted from six studies) were statistically adjusted. The mean number of extracted effect sizes per study was 10.63 (*SD* = 14.02). For more details on the included studies and several characteristics, see Appendix A.

### Overall Mean Effect of the Risk Domains and Heterogeneity in Effect Sizes

Of all 765 extracted (effects of) putative risk factors, we classified 762 factors in one of the 35 risk domains (as defined in the Method section). In Table 1, an overall mean effect for each of these 35 risk domains is presented in descending order, separately for child-, parent-, and family related risk domains. The overall mean effect of seven child-related risk domains, nine parent-related risk domains, and seven family-related risk domains was significant, and ranged from  $r = .101$  for the risk domain “Low family SES” to  $r = .360$  for the risk domain “Prior sexual abuse victimization of child or siblings.” According to the conventions described by Cohen (1988) for interpreting effect sizes ( $r = .1$ ,  $r = .3$ ,  $r = .5$  indicating small, medium, and large effects, respectively), the effect of one domain was large, the effects of four domains were medium, the effects of 25 domains were small, and the effects of five domains were very small in size. The effect of 12 domains did not significantly deviate from zero, meaning that the results did not produce evidence for a true association between these domains and CSA victimization. Table 1 also presents the effects of three single factors that could not be classified in any of the created risk domains, because of their unique nature. The effect of the factors “Child / Family moved six or more times” and “Prior victimization of a family member” were significant and small in size. The effect of “Child’s sibling(s) has a psychiatric condition” was not significant, implying that this variable was not identified as a risk factor for CSA victimization.

As for heterogeneity in effect sizes, we found significant within study (level 2) and/or between study (level 3) variance in 28 risk domains (see Table 1). In these domains, we could proceed with performing moderator analyses to examine the potential moderating effect of study, sample, and risk factor characteristics on the strength of individual risk domains. However, as (categories of) potential moderating variables had to be based on at least five studies (see Analytic Strategy paragraph in the Method section), we performed moderator analyses for only 18 risk domains.

Table 1  
 Mean Effect Sizes of the 35 Risk Domains and the Overall Risk Factor Strength With Corresponding Level 2 and Level 3 Variance Estimates

Risk domain	# Studies	# ES	Mean Fisher's Z (SE)	[95% CI]	Sig. mean Z (p)	Mean r	% Var. at Level 1	Level 2 variance	% Var. at Level 2	Level 3 variance	% Var. at Level 3
Child-related risk domains (#8)											
Child demonstrates gender nonconforming behavior	2	5 (0)	.575 (.470)	[-.730, 1.881]	.288	.519	.068	.010****	2.456	.414**	97.476
Child experienced a form of victimization other than (forms of) child abuse	4	9 (0)	.354 (.040)	[.263, .446]	<.001***	.340	8.978	.008***	80.498	.001	10.524
Low quality of child's relation with parents/parental attachment	2	7 (0)	.301 (.045)	[.190, .412]	<.001***	.292	8.600	.013****	91.399	.000	.000
Child is female	24	50 (0)	.299 (.041)	[.216, .382]	<.001***	.290	.456	.025****	57.120	.019	42.424
Shyness/low social skills of a child	2	3 (0)	.221 (.049)	[.009, .432]	.046*	.217	32.227	.000	.000	.003	67.773
Child has a physical or mental chronic condition	15	49 (2)	.195 (.040)	[.114, .275]	<.001***	.193	.786	.012****	42.891	.016**	56.323
Child frequently using the internet	1	4 (4)	.153 (.011)	[.117, .189]	<.001***	.152	100.000	.000	.000	.000	.000
Child ever used drugs/engaged in (violent) delinquency	1	8 (8)	.127 (.017)	[.087, .166]	<.001***	.126	23.078	.002	76.922	.000	.000
Parent-related risk domains (#15)											
High levels of parental/family stress	3	6 (0)	.317 (.237)	[-.292, .925]	.238	.307	1.229	.020****	12.200	.142	86.570
Parental history of child abuse victimization	13	37 (12)	.272 (.036)	[.199, .346]	<.001***	.265	17.994	.014****	57.152	.006*	24.854
Parental overprotection	3	12 (0)	.215 (.046)	[.113, .317]	<.001***	.212	5.645	.024****	94.355	.000	.000
Intimate partner violence between a child's caretakers	6	17 (0)	.190 (.064)	[.053, .327]	.010*	.188	5.643	.010****	38.028	.015*	56.329
Poor physical health of parents	2	4 (0)	.183 (.121)	[-.201, .568]	.226	.181	4.320	.056****	95.680	.000	.000
Parental relationship problems (excluding IPV)	7	12 (2)	.177 (.055)	[.061, .293]	.005**	.175	6.576	.009****	38.638	.013*	54.786
Parental substance (ab)use	16	56 (1)	.173 (.033)	[.106, .239]	<.001***	.171	1.443	.010****	46.843	.011****	51.714
Parental mental/psychiatric or physical problems	22	60 (4)	.171 (.046)	[.078, .263]	<.001***	.169	1.129	.023****	41.597	.031****	57.275
Low level of parental education	12	33 (0)	.152 (.038)	[.074, .229]	<.001***	.151	1.492	.012****	51.332	.011*	47.175
Low parental care/affection (excluding forms of child neglect)	8	31 (4)	.150 (.048)	[.052, .248]	.004**	.149	4.245	.020****	63.580	.010	32.157
Low (sense of) parenting competence	7	23 (8)	.137 (.034)	[.068, .207]	<.001***	.136	7.920	.021****	92.080	.000	.000
Young maternal age (20 or younger at child birth)	2	3 (1)	.126 (.054)	[-.105, .358]	.143	.125	6.703	.003**	48.056	.003	45.241
Conservative sexual/family values of parents	2	7 (0)	.108 (.061)	[-.041, .257]	.125	.108	10.617	.023****	89.383	.000	.000
Parental unemployment	4	5 (1)	.079 (.051)	[-.062, .219]	.194	.079	33.602	.005	38.505	.003	27.894
Dysfunctional maternal attitudes (pre- and postnatally)	1	12 (0)	.016 (.020)	[-.028, .060]	.430	.016	9.021	.004****	90.980	.000	.000
Family-related risk domains (#12) and 3 individual family-related risk factors											
Prior sexual abuse victimization of child or sibling	4	5 (1)	.377 (.096)	[.110, .643]	.017*	.360	1.838	.045****	98.162	.000	.000

(table continues)

Table 1 (continued)

Risk domain	# Studies	# ES	Mean Fisher's Z (SE)	[95% CI]	Sig. mean Z (p)	Mean <i>r</i>	% Var. at Level 1	Level 2 variance	% Var. at Level 2	Level 3 variance	% Var. at Level 3
Family history of antisocial/criminal behavior	5	10 (0)	.343 (.159)	[−.017, .703]	.059	.330	.467	.012***	9.040	.115**	90.493
Child/Family moved 6 or more times <sup>a</sup>	1	1 (0)	.303 (.038)	[.229, .377]	<.001***	.294	100.000	—	—	—	—
Prior victimization of a family member <sup>a</sup>	1	1 (0)	.291 (.023)	[.246, .336]	<.001***	.283	100.000	—	—	—	—
Prior or concurrent forms of (nonsexual) child abuse in the home environment	27	88 (10)	.274 (.023)	[.228, .320]	<.001***	.267	1.441	.005***	34.235	.010***	64.324
Problems in the family system functioning	10	39 (3)	.197 (.065)	[.064, .329]	.005**	.194	6.646	.004	9.169	.037***	84.185
Child or family members experience social isolation	9	19 (4)	.193 (.078)	[.029, .358]	.024*	.191	2.295	.009***	16.619	.046**	81.087
Child grew up in a non-nuclear family structure (excluding having a stepfather)	24	86 (5)	.166 (.020)	[.126, .207]	<.001***	.164	2.261	.010***	69.515	.004**	28.224
Sibling(s) experienced child abuse (other than sexual abuse)	1	2 (0)	.158 (.041)	[−.363, .680]	.162	.161	100.000	.000	.000	.000	.000
Child has a stepfather	8	12 (5)	.119 (.044)	[.022, .215]	.020*	.118	5.255	.009***	51.362	.006	43.383
Low family SES	21	33 (3)	.101 (.048)	[.003, .199]	.044*	.101	1.975	.033***	60.240	.021*	37.785
Large family size	3	4 (0)	.056 (.058)	[−.129, .241]	.403	.056	46.082	.000	.000	.005	53.918
Child's sibling(s) has a psychiatric condition <sup>a</sup>	1	1 (0)	−.010 (.058)	[−.124, .104]	.863	−.010	100.000	—	—	—	—
Strong religious affiliation of the child's family	5	7 (0)	−.047 (.101)	[−.294, .201]	.661	−.047	2.897	.042***	69.019	.017	28.084
Living in a violent community	3	4 (0)	−.061 (.170)	[−.603, .481]	.745	−.061	2.073	.000	.546	.084	97.381

Note. # Studies = number of studies; # ES = number of effect sizes with number of adjusted effect sizes in parentheses; Mean Fisher's Z = Mean effect size (Fisher's Z); SE = standard error; CI = confidence interval of each mean Fisher's Z; Sig = Significance; *r* = Mean effect size (Pearson's correlation); % Var = percentage of variance explained; Level 2 variance = variance between effect sizes extracted from the same study; Level 3 variance = variance between studies.

<sup>a</sup> One of three single risk factors that could not be classified into one of the 35 created risk domains.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

## Assessment of Bias

Table 2 presents the results of the four analyses that were conducted to assess bias in the estimated mean effects of each of the 35 risk domains. The results showed no indication of bias in 13 estimated risk domain effects (i.e., zero of four methods indicated bias), some indication of bias in 14 risk domain effects (i.e., one of four methods indicated bias), and moderate to strong indications of bias in eight risk domain effects (i.e., two or three of four methods indicated bias). In general, the results reflect indications of bias in most of the estimated risk domains. For brevity, the 35 funnel plots that were produced by the trim-and-fill analyses are not presented here, but are available upon request from the first author.

## Moderator Analyses

For 18 risk domains, moderator analyses were performed to find variables that can explain differences in observed effect sizes within and/or between studies. Put differently, we examined whether and how the overall strength of 18 individual risk domains was moderated by study, sample, and risk factor characteristics.

The results of these analyses are presented in Table 3, in which the potential moderators are classified into study, sample, and risk factor characteristics. In total, we identified five moderating variables of which three were study characteristics, one was a sample characteristic, and one was a risk factor characteristic. First, we found a moderating effect of sexual abuse type in three risk domains (see Table 3). In the risk domains "Low level of parental education" and "Low family SES," we found that effects of risk factors for the mixed category of sexual abuse (i.e., contact and/or noncontact sexual abuse) were significantly smaller—and even nonsignificant—than effects of risk factors for contact sexual abuse. In the risk domain "Child grew up in a non-nuclear family structure," we found that effects of risk factors for mixed sexual abuse and noncontact sexual abuse were significantly smaller than effects of contact sexual abuse.

Second, we found a moderating effect of the CSA cut-off age in the risk domain "Child grew up in a non-nuclear family structure" (see Table 3). The strength of this domain was significantly smaller when studies used a CSA cut-off age of 18 or 19 years than 17 years or younger. Third, publication year moderated the overall

Table 2  
*Results of Four Methods for the Assessment of Bias in the Estimated Mean Effects of the 35 Risk Domains*

Risk domain	<i>r</i>	Trim-and-fill analysis	Classical Egger's test <sup>a</sup>	Adapted Egger's test <sup>b</sup>	Rank correlation test	Number of methods indicating bias (of 4)
<b>Child-related risk domains (#8)</b>						
Child demonstrates gender nonconforming behavior	.519	Underestimation (1 ES missing)	$\beta_1 = 6.061, p = .408$	$\beta_1 = 4.791, p = .029^*$	$\tau = .447, p = .296$	2
Child experienced a form of victimization other than (forms of) child abuse	.340	Underestimation (2 ES missing)	$\beta_1 = -2.110, p = .502$	$\beta_1 = -2.622, p = .077$	$\tau = -.036, p = .900$	1
Low quality of child's relation with parents/parental attachment	.292	—	$\beta_1 = -.363, p = .943$	$\beta_1 = -1.844, p = .756$	$\tau = -.048, p = .999$	0
Child is female	.290	—	$\beta_1 = 2.708, p = .898$	$\beta_1 = -.367, p = .715$	$\tau = -.086, p = .401$	0
Shyness/low social skills of a child	.217	—	$\beta_1 = -9.901, p = .073$	$\beta_1 = -9.901, p = .392$	$\tau = -.817, p = .221$	0
Child has a physical or mental chronic condition	.193	Underestimation (12 ES missing)	$\beta_1 = -5.895, p = .061$	$\beta_1 = .806, p = .542$	$\tau = .452, p < .001^{***}$	2
Child frequently using the internet	.152	Overestimation (1 ES missing)	$\beta_1 = -8.502, p = .050^*$	$\beta_1 = -8.502, p = .269$	$\tau = -.667, p = .333$	2
Child ever used drugs/engaged in (violent) delinquency	.126	—	$\beta_1 = 2.069, p = .824$	$\beta_1 = 2.796, p = .763$	$\tau = .154, p = .610$	0
<b>Parent-related risk domains (#15)</b>						
High levels of parental/family stress	.307	—	$\beta_1 = 2.419, p = .309$	$\beta_1 = 2.128, p = .567$	$\tau = .298, p = .421$	0
Parental history of child abuse victimization	.265	Underestimation (1 ES missing)	$\beta_1 = .490, p = .714$	$\beta_1 = 1.493, p = .350$	$\tau = .227, p = .057$	1
Parental overprotection	.212	Underestimation (3 ES missing)	$\beta_1 = -.978, p = .852$	$\beta_1 = -.082, p = .975$	$\tau = -.031, p = .890$	1
Intimate partner violence between a child's caretakers	.188	—	$\beta_1 = -6.326, p = .067$	$\beta_1 = -5.003, p = .086$	$\tau = -.357, p = .091$	0
Poor physical health of parents	.181	—	$\beta_1 = 26.917, p = .649$	$\beta_1 = 26.917, p = .698$	$\tau = .408, p = .439$	0
Parental relationship problems (excluding IPV)	.175	Underestimation (1 ES missing)	$\beta_1 = -7.056, p = .031^*$	$\beta_1 = -4.690, p = .091$	$\tau = -.407, p = .035^*$	3
Parental substance (ab)use	.171	Underestimation (14 ES missing)	$\beta_1 = -2.247, p = .402$	$\beta_1 = -2.119, p = .096$	$\tau = .001, p = .994$	1
Parental mental/psychiatric or physical problems	.169	Underestimation (1 ES missing)	$\beta_1 = -5.065, p = .006^{**}$	$\beta_1 = -.641, p = .603$	$\tau = .401, p < .001^{***}$	3
Low level of parental education	.151	Underestimation (2 ES missing)	$\beta_1 = -1.000, p = .625$	$\beta_1 = 3.392, p = .025^*$	$\tau = .123, p = .331$	2
Low parental care/affection (excluding forms of child neglect)	.149	Overestimation (4 ES missing)	$\beta_1 = 3.924, p = .075$	$\beta_1 = 1.147, p = .555$	$\tau = .122, p = .346$	1
Low (sense of) parenting competence	.136	Overestimation (6 ES missing)	$\beta_1 = .485, p = .614$	$\beta_1 = 1.044, p = .276$	$\tau = .054, p = .727$	1
Young maternal age (20 or younger at child birth)	.125	—	$\beta_1 = 53.995, p = .186$	$\beta_1 = 59.201, p = .221$	$\tau = .999, p = .333$	0
Conservative sexual/family values of parents	.108	—	$\beta_1 = 7.792, p = .886$	$\beta_1 = 7.792, p = .906$	$\tau = -.211, p = .581$	0
Parental unemployment	.079	—	$\beta_1 = 5.069, p = .431$	$\beta_1 = 4.956, p = .490$	$\tau = .105, p = .801$	0
Dysfunctional maternal attitudes (pre- and postnatally)	.016	Overestimation (6 ES missing)	$\beta_1 = .485, p = .614$	$\beta_1 = 1.044, p = .276$	$\tau = .054, p = .727$	1
<b>Family-related risk domains (#15)</b>						
Prior sexual abuse victimization of child or sibling	.360	—	$\beta_1 = -5.408, p = .764$	$\beta_1 = -7.170, p = .563$	$\tau = -.316, p = .449$	0
Family history of antisocial/criminal behavior	.330	Underestimation (4 ES missing)	$\beta_1 = -13.959, p = .176$	$\beta_1 = -5.875, p = .190$	$\tau = .111, p = .728$	1
Prior or concurrent forms of child abuse in the home environment	.267	Underestimation (16 ES missing)	$\beta_1 = -.596, p = .689$	$\beta_1 = -.334, p = .612$	$\tau = .268, p < .001^{***}$	2
Problems in the family system functioning	.194	Overestimation (9 ES missing)	$\beta_1 = -3.303, p < .001^{***}$	$\beta_1 = .494, p = .659$	$\tau = .560, p < .001^{***}$	3
Child or family members experience social isolation	.191	—	$\beta_1 = 3.395, p = .092$	$\beta_1 = 1.566, p = .296$	$\tau = .295, p = .084$	0
Child grew up in a non-nuclear family structure (excluding having a stepfather)	.164	Overestimation (9 ES missing)	$\beta_1 = -1.675, p = .060$	$\beta_1 = .196, p = .752$	$\tau = -.081, p = .280$	1

(table continues)

Table 2 (continued)

Risk domain	<i>r</i>	Trim-and-fill analysis	Classical Egger's test <sup>a</sup>	Adapted Egger's test <sup>b</sup>	Rank correlation test	Number of methods indicating bias (of 4)
Sibling(s) experienced child abuse (other than sexual abuse)	.161	Overestimation (1 ES missing)	NA	NA	NA	1
Child has a stepfather	.118	—	$\beta_1 = 2.406, p = .298$	$\beta_1 = 1.195, p = .403$	$\tau = .152, p = .545$	0
Low family SES	.101	Overestimation (9 ES missing)	$\beta_1 = -1.624, p = .369$	$\beta_1 = 2.321, p = .153$	$\tau = .237, p = .053$	1
Large family size	.056	Underestimation (1 ES missing)	$\beta_1 = .215, p = .919$	$\beta_1 = -.095, p = .956$	$\tau = .600, p = .251$	1
Strong religious affiliation of the child's family	-.047	Underestimation (1 ES missing)	$\beta_1 = -2.540, p = .297$	$\beta_1 = -3.108, p = .341$	$\tau = -.195, p = .543$	1
Living in a violent community	-.061	Underestimation (1 ES missing)	$\beta_1 = -6.102, p = .231$	$\beta_1 = 10.062, p = .119$	$\tau = -1.000, p = .083$	1

*Note.* *r* = Mean effect size (Pearson's correlation; see also Table 1); Underestimation = Effect sizes were imputed to the right of the mean effect, implying that above average effect sizes were underrepresented and that the mean effect may be an underestimation of the true effect; Overestimation = Effect sizes were imputed to the left of the mean effect, implying that below average effect sizes were underrepresented and that the mean effect may be an overestimation of the true effect; NA = Not available, as only two effect sizes were classified in the corresponding risk domain. Dashes indicate that trimming and filling of effect sizes were not necessary according to the trim-and-fill algorithm.

<sup>a</sup> This test was performed in a weighted regression model with multiplicative dispersion in which the standard error was tested as a predictor of effect sizes (i.e., "a classical" Egger's test; Sterne, Gavaghan, & Egger, 2000). <sup>b</sup> This test was performed in a three-level meta-analytic model (see Methods section) in which the standard error was tested as a predictor of effect sizes. It is similar to the classical Egger's test, but in the current test, effect size dependency was accounted for.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

strength of this same risk domain. As studies were more recently published, the strength of the risk domain "Child grew up in a non-nuclear family structure" significantly decreased (see Table 3). Fourth, the percentage of males in study samples moderated the overall strength of the risk domain "Child has a stepfather." As the percentage of males increased, the overall strength of this domain significantly decreased (see Table 3).

The moderating effect of statistical adjustment was tested for 15 risk domains in which both unadjusted effects and at least one adjusted effect were classified (see Table 1 for the numbers of unadjusted and adjusted effect sizes that were classified in each risk domain). The results showed a significant moderating effect in the risk domains "Parental history of child abuse victimization,"  $F(1, 35) = 9.791, p = .004$ , and "Problems in the family system functioning,"  $F(1, 37) = 4.568, p = .039$ . In the former, statistical adjusted effects were significantly smaller (mean  $r = .131, p < .010$ ) than statistical unadjusted effects (mean  $r = .277, p < .001$ ). Likewise, in the latter, statistical adjusted effects were significantly smaller (mean  $r = -.042, p = .739$ ) than statistical unadjusted effects (mean  $r = .250, p < .001$ ).

## Discussion

Over the past decades, an increasing amount of research has been directed at identifying risk factors for CSA victimization, but a systematic quantitative review summarizing effects of (putative) risk factors for CSA victimization was not yet available. This review fills this gap in the literature and was aimed at estimating a mean effect of a number of different risk domains for CSA victimization. A risk domain was defined as a group of (more or less) similar risk factors. A second aim was to examine whether, and how, the overall strength of individual risk domains is moderated by study, sample, and risk factor characteristics. In total, 765 (putative) risk factors and their effects could be extracted from

72 studies, after which 762 factors were classified into 35 risk domains. The mean effect of each of these risk domains was estimated in a separate three-level meta-analysis. Three risk factors were evaluated individually instead of meta-analytically, as classifying these factors in one of the risk domains did not seem rational given the nature of these factors. The coded study, sample, and risk factor characteristics were tested as moderators of the overall strength of risk domains in which heterogeneity in effect sizes was found.

## Overall Mean Effect of Risk Domains and Bias Assessment

A significant mean effect was found for 23 of the 35 risk domains, ranging from  $r = .101$  for the risk domain "Low family SES" to  $r = .360$  for the risk domain "Prior sexual abuse victimization of child or siblings." We did not find a significant mean effect for the other 12 domains, implying that no sufficient evidence was obtained for designating these 12 domains as true risk domains. Given the pattern of significant domains and significant risk factors across domains, seven risk themes could be identified. First, the results showed that the largest effects were found for different risks that refer to (prior) victimization of not only the child, but also the child's family members. Specifically, substantial effects were found for prior CSA victimization of the child and/or its siblings ( $r = .360$ ), prior victimization of the child other than (forms of) child abuse ( $r = .340$ ), prior victimization of a family member ( $r = .283$ ), prior or concurrent forms of child abuse in the child's home environment ( $r = .267$ ), and a parental history of child abuse victimization ( $r = .265$ ).

Second, we found that multiple risk domains are related to different sorts of parental problems and difficulties. These risks were: intimate partner violence between the child's parents ( $r = .188$ ), other parental relationship problems ( $r = .175$ ), parental

Table 3  
*Results for Continuous and Categorical Variables Tested as Moderators (in Bivariate Models)*

Variables tested as moderators	# Studies	# ES	Intercept [95% CI]/ Mean Z [95% CI]	Mean <i>r</i>	$\beta$ [95% CI]	<i>F</i> ( <i>df</i> 1, <i>df</i> 2) <sup>a</sup>	<i>p</i> <sup>b</sup>	Level 2 variance	Level 3 variance
Risk domain: Child is female									
Study characteristics									
Type of sexual abuse examined						<i>F</i> (1, 48) = .085	.772	.026***	.019 <sup>+</sup>
Contact sexual abuse (RC)	16	28	.306 [.211, .401]***	.297					
Mixed (contact and noncontact)	13	22	.291 [.188, .393]***	.283	-.015 [-.121, .091]				
Type of assessment						<i>F</i> (1, 37) = .335	.566	.034***	.000
Self-report questionnaire—Single item or multiple items (RC)	11	32	.303 [.236, .369]***	.294					
Official records	6	7	.253 [.091, .414]**	.248	-.050 [-.225, .125]				
Age cut-off used in defining CSA (in years)						<i>F</i> (2, 47) = .625	.540	.025***	.020 <sup>+</sup>
17 or younger (RC)	11	34	.272 [.153, .392]***	.265					
18	8	8	.277 [.120, .434]***	.270	.005 [-.193, .202]				
19	5	8	.386 [.208, .563]***	.368	.113 [-.100, .327]				
Publication year	24	50	.283 [.201, .366]***	—	-.006 [-.014, .003]	<i>F</i> (1, 48) = 1.849	.180	.026***	.015
Number of questions used to elicit CSA episodes (in self-reports or interviews)	12	34	.269 [.198, .340]***	—	.004 [-.008, .016]	<i>F</i> (1, 32) = .363	.551	.027***	.001
Sample characteristics									
Mean age of the sample in years (at start of a study)	16	40	.298 [.210, .385]***	—	.007 [-.005, .202]	<i>F</i> (1, 38) = 1.390	.246	.026***	.009
Percentage of males in the sample	23	49	.308 [.222, .393]***	—	-.001 [-.010, .007]	<i>F</i> (1, 47) = .094	.760	.025	.019
Percentage of Whites/Caucasians in the sample	16	20	.330 [.202, .458]***	—	.002 [-.003, .007]	<i>F</i> (1, 18) = .596	.450	.010***	.045*
Risk domain: Child has a physical or mental chronic condition									
Study characteristics									
Type of assessment						<i>F</i> (1, 37) = .049	.826	.014***	.005 <sup>+</sup>
Self-report questionnaire—Single item or multiple items (RC)	6	26	.188 [.100, .276]***	.186					
Official records	5	13	.203 [.099, .307]***	.200	.015 [-.122, .151]				
Age cut-off used in defining CSA (in years)						<i>F</i> (1, 47) = .339	.563	.012***	.020**
18 or younger (RC)	9	35	.216 [.106, .327]***	.213					
19	6	14	.165 [.028, .303]*	.164	-.051 [-.227, .125]				
Publication year	15	49	.200 [.111, .290]***	—	.002 [-.009, .012]	<i>F</i> (1, 47) = .099	.754	.012***	.019**
Sample characteristics									
Mean age of the sample in years (at start of a study)	11	43	.199 [.078, .320]**	—	.004 [-.011, .019]	<i>F</i> (1, 41) = .302	.586	.008***	.033***
Percentage of males in the sample	13	46	.184 [.088, .279]***	—	.001 [-.000, .001]	<i>F</i> (1, 44) = 2.980	.091 <sup>+</sup>	.008***	.023***
Percentage of Whites/Caucasians in the sample	7	30	.139 [-.081, .359]	—	-.004 [-.012, .005]	<i>F</i> (1, 28) = .840	.367	.004***	.054***
Risk domain: Parental history of child abuse victimization									
Study characteristics									
Age cut-off used in defining CSA (in years)						<i>F</i> (1, 35) = 1.548	.222	.014***	.006 <sup>+</sup>
17 or younger (RC)	8	30	.243 [.157, .328]***	.238					
18–19	5	7	.339 [.207, .471]***	.327	.096 [-.061, .254]				
Publication year	13	37	.263 [.196, .330]***	—	-.006 [-.012, .001] <sup>+</sup>	<i>F</i> (1, 35) = 3.433	.072 <sup>+</sup>	.014***	.004
Sample characteristics									
Mean age of the sample in years (at start of a study)	10	32	.234 [.157, .310]***	—	-.003 [-.007, .001]	<i>F</i> (1, 30) = 2.104	.157	.015***	.003
Percentage of males in the sample	11	33	.245 [.175, .316]***	—	-.002 [-.006, .001]	<i>F</i> (1, 31) = 1.980	.169	.014***	.003
Percentage of Whites/Caucasians in the sample	8	29	.223 [.144, .301]***	—	.004 [-.003, .011]	<i>F</i> (1, 27) = 1.550	.224	.014***	.003
Risk domain: Intimate partner violence between a child's caretakers									
Study characteristics									
Publication year	7	17	.185 [.061, .309]**	—	.003 [-.008, .015]	<i>F</i> (1, 15) = .340	.569	.009***	.014 <sup>+</sup>
Sample characteristics									
Percentage of males in the sample	6	16	.232 [.065, .398]**	—	.002 [-.004, .009]	<i>F</i> (1, 14) = .737	.405	.009***	.015
Risk domain: Parental relationship problems (excluding IPV)									
Study characteristics									
Publication year	5	12	.207 [.039, .375]*	—	-.004 [-.029, .021]	<i>F</i> (1, 10) = .120	.736	.011***	.019
Risk domain: Parental substance (ab)use									
Study characteristics									
Type of sexual abuse examined						<i>F</i> (1, 54) = .915	.343	.010***	.013***
Contact sexual abuse (RC)	9	22	.192 [.111, .274]***	.190					
Mixed (contact and noncontact)	11	32	.155 [.077, .233]***	.154	-.038 [-.117, .041]				
Age cut-off used in defining CSA (in years)						<i>F</i> (1, 54) = 3.310	.074 <sup>+</sup>	.010***	.009***
17 or younger (RC)	9	35	.128 [.048, .208]**	.127					
18–19	7	21	.243 [.145, .340]***	.238	.115 [-.012, .241] <sup>+</sup>				

(table continues)

Table 3 (continued)

Variables tested as moderators	# Studies	# ES	Intercept [95% CI]/ Mean Z [95% CI]	Mean <i>r</i>	$\beta$ [95% CI]	<i>F</i> ( <i>df</i> 1, <i>df</i> 2) <sup>a</sup>	<i>p</i> <sup>b</sup>	Level 2 variance	Level 3 variance
Publication year	16	56	.172 [.103, .241]***	—	.001 [−.006, .008]	<i>F</i> (1, 54) = .058	.811	.010***	.012***
Number of questions used to elicit CSA episodes (in self- reports or interviews)	8	29	.210 [.082, .338]**	—	−.005 [−.028, .018]	<i>F</i> (1, 27) = .199	.659	.013***	.021***
Sample characteristics									
Mean age of the sample in years (at start of a study)	11	36	.138 [.090, .185]***	—	.003 [−.002, .007]	<i>F</i> (1, 34) = 1.654	.207	.005***	.003*
Percentage of males in the sample	15	54	.180 [.111, .250]***	—	.001 [−.001, .002]	<i>F</i> (1, 52) = .996	.323	.011***	.011***
Percentage of Whites/Caucasians in the sample	10	21	.171 [.120, .222]***	—	.002 [−.001, .005]	<i>F</i> (1, 19) = 1.489	.237	.005***	.002
Risk domain: Parental mental/psychiatric or physical problems									
Study characteristics									
Type of sexual abuse examined						<i>F</i> (1, 58) = .028	.868	.023***	.032***
Contact sexual abuse (RC)	14	43	.176 [.066, .285]**	.174					
Mixed (contact and noncontact)	10	17	.164 [.035, .292]*	.163	−.012 [−.156, .132]				
Type of assessment						<i>F</i> (1, 39) = .495	.486	.030***	.025**
Self-report questionnaire—Multiple items (RC)	9	15	.291 [.142, .440]***	.283					
Official records	6	26	.212 [.041, .383]*	.209	−.079 [−.305, .148]				
Age cut-off used in defining CSA (in years)						<i>F</i> (2, 57) = 1.167	.319	.022***	.034***
15 or younger (RC)	5	8	.054 [−.150, .257]	.054					
16–17	6	31	.150 [−.022, .321] <sup>+</sup>	.149	.096 [−.170, .362]				
18–19	11	21	.237 [.101, .374]***	.233	.183 [−.062, .428]				
Publication year	22	60	.161 [.063, .260]**	—	.004 [−.008, .016]	<i>F</i> (1, 58) = .441	.510	.022***	.033***
Number of questions used to elicit CSA episodes (in self- reports or interviews)	8	19	.216 [.123, .309]***	—	−.006 [−.036, .035]	<i>F</i> (1, 17) = .155	.699	.035***	.000
Sample characteristics									
Mean age of the sample in years (at start of a study)	16	45	.133 [.017, .249]*	.132	.008 [−.001, .016] <sup>+</sup>	<i>F</i> (1, 43) = 2.977	.092 <sup>+</sup>	.016***	.036***
Percentage of males in the sample	20	39	.150 [.058, .243]**	—	.002 [−.001, .006]	<i>F</i> (1, 37) = 2.261	.141	.024***	.024 <sup>+</sup>
Percentage of Whites/Caucasians in the sample	13	20	.175 [.002, .348]*	—	.001 [−.007, .010]	<i>F</i> (1, 18) = .111	.743	.001 <sup>+</sup>	.083***
Risk domain: Low level of parental education									
Study characteristics									
Type of sexual abuse examined						<i>F</i> (1, 31) = 19.090	<.001***	.005***	.015**
Contact sexual abuse (RC)	9	22	.201 [.116, .286]***	.198					
Mixed (contact and noncontact)	6	11	.049 [−.045, .143]	.049	−.153 [−.224, −.081]***				
Age cut-off used in defining CSA (in years)						<i>F</i> (1, 31) = .010	.920	.011***	.013*
16 or younger (RC)	6	17	.156 [.040, .271]	.155					
17–18	6	16	.147 [.027, .268]*	.146	−.008 [−.175, .159]				
Publication year	12	33	.142 [.058, .226]**	—	−.003 [−.012, .005]	<i>F</i> (1, 31) = .660	.423	.011***	.012*
Number of questions used to elicit CSA episodes (in self- reports or interviews)	5	17	.131 [.055, .207]**	—	.010 [−.004, .024]	<i>F</i> (1, 15) = 2.485	.136	.009***	.002
Sample characteristics									
Mean age of the sample in years (at start of a study)	9	28	.151 [.041, .260]**	—	−.005 [−.028, .018]	<i>F</i> (1, 26) = .236	.631	.011***	.018*
Percentage of males in the sample	10	29	.128 [.065, .190]***	—	−.001 [−.003, .002]	<i>F</i> (1, 27) = .171	.683	.012***	.003
Percentage of Whites/Caucasians in the sample	7	11	.113 [.034, .192]*	—	.002 [−.002, .006]	<i>F</i> (1, 9) = 1.627	.234	.012***	.000
Risk domain: Low parental care/affection (excluding forms of child neglect)									
Study characteristics									
Publication year	8	31	.152 [.057, .246]**	—	−.006 [−.016, .004]	<i>F</i> (1, 29) = 1.407	.245	.020***	.009
Risk factor characteristics									
Father- versus mother-related risk factors						<i>F</i> (1, 26) = .035	.853	.014***	.009 <sup>+</sup>
Mother-related risk factors (RC)	6	11	.188 [.086, .289]***	.186					
Father-related risk factors	5	10	.178 [.069, .288]***	.176	−.009 [−.111, .092]				
Sample characteristics									
Mean age of the sample in years (at start of a study)	6	22	.144 [.030, .258]*	—	−.030 [−.072, .013]	<i>F</i> (1, 20) = 2.137	.159	.017***	.010 <sup>+</sup>
Percentage of males in the sample	7	29	.129 [.035, .223]**	—	−.001 [−.003, .002]	<i>F</i> (1, 27) = .260	.614	.021***	.008
Risk domain: Low (sense of) parenting competence									
Study characteristics									
Publication year	7	23	.137 [.070, .204]***	—	.004 [−.001, .009]	<i>F</i> (1, 21) = 2.762	.111	.019***	.000
Sample characteristics									
Mean age of the sample in years (at start of a study)	6	21	.150 [.079, .221]***	—	−.019 [−.039, .001] <sup>+</sup>	<i>F</i> (1, 19) = 3.770	.067 <sup>+</sup>	.019***	.000
Percentage of males in the sample	6	17	.162 [.053, .271]**	—	−.001 [−.003, .001]	<i>F</i> (1, 15) = 1.219	.287	.008***	.009 <sup>+</sup>

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Table 3 (continued)

Variables tested as moderators	# Studies	# ES	Intercept [95% CI]/ Mean Z [95% CI]	Mean <i>r</i>	$\beta$ [95% CI]	<i>F</i> ( <i>df</i> 1, <i>df</i> 2) <sup>a</sup>	<i>p</i> <sup>b</sup>	Level 2 variance	Level 3 variance
Risk domain: Family history of antisocial/criminal behavior									
Study characteristics									
Publication year	5	10	.319 [−.083, .720]	—	.018 [−.084, .119]	<i>F</i> (1, 8) = .163	.697	.013***	.121*
Sample characteristics									
Percentage of males in the sample	5	10	.331 [−.037, .006] <sup>+</sup>	—	.002 [−.002, .006]	<i>F</i> (1, 8) = 1.422	.267	.011***	.116**
Risk domain: Prior or concurrent forms of child abuse in the home environment									
Study characteristics									
Type of sexual abuse examined									
Contact sexual abuse (RC)	14	45	.291 [.237, .345]***	.283	—	<i>F</i> (1, 82) = 2.290	.134	.005***	.010***
Mixed (contact and noncontact)	16	39	.254 [.202, .307]***	.249	−.037 [−.085, .012]	—	—	—	—
Type of assessment									
Self-report									
questionnaire—Multiple items (RC)	13	60	.277 [.209, .345]***	.270	—	<i>F</i> (1, 78) = .219	.641	.005***	.012***
Interview (face-to-face or telephone)	7	20	.305 [.208, .401]***	.296	.028 [−.090, .146]	—	—	—	—
Age cut-off used in defining CSA (in years)									
13 or younger (RC)	5	13	.295 [.186, .404]***	.287	—	<i>F</i> (3, 84) = .728	.538	.005***	.011***
16–17	6	29	.218 [.116, .321]***	.215	−.076 [−.226, .074]	—	—	—	—
18	9	31	.308 [.230, .387]***	.299	.014 [−.121, .148]	—	—	—	—
19	7	15	.256 [.159, .353]***	.251	−.039 [−.185, .107]	—	—	—	—
Publication year	27	88	.277 [.230, .325]***	—	.002 [−.003, .008]	<i>F</i> (1, 86) = .616	.435	.005***	.010***
Number of questions used to elicit CSA episodes (in self-reports or interviews)									
	14	65	.280 [.215, .344]***	—	−.001 [−.010, .009]	<i>F</i> (1, 63) = .011	.917	.004***	.012***
Sample characteristics									
Mean age of the sample in years (at start of a study)	20	74	.257 [.219, .295]***	—	.002 [−.001, .005]	<i>F</i> (1, 72) = 2.449	.122	.005**	.004***
Percentage of males in the sample	25	86	.276 [.228, .325]***	—	.000 [−.002, .002]	<i>F</i> (1, 84) = .001	.979	.005***	.010***
Percentage of Whites/Caucasians in the sample	16	44	.275 [.208, .341]***	—	−.000 [−.003, .003]	<i>F</i> (1, 42) = .025	.874	.008**	.011***
Risk domain: Problems in the family system functioning									
Study characteristics									
Publication year	10	39	.184 [.035, .334]*	—	.004 [−.013, .021]	<i>F</i> (1, 37) = .205	.653	.004 <sup>+</sup>	.042***
Sample characteristics									
Mean age of the sample in years (at start of a study)	8	36	.141 [−.000, .283] <sup>+</sup>	—	−.003 [−.022, .016]	<i>F</i> (1, 34) = .102	.751	.005 <sup>+</sup>	.032***
Percentage of males in the sample	9	37	.238 [.122, .355]***	—	−.000 [−.002, .002]	<i>F</i> (1, 35) = .010	.921	.006 <sup>+</sup>	.023*
Percentage of Whites/Caucasians in the sample	6	31	.295 [.128, .461]**	—	.002 [−.005, .009]	<i>F</i> (1, 29) = .210	.651	.009*	.028*
Risk domain: Child or family members experience social isolation									
Study characteristics									
Type of sexual abuse examined									
Contact sexual abuse (RC)	5	10	.241 [.060, .423]*	.236	—	<i>F</i> (1, 17) = 1.137	.301	.011***	.038*
Mixed (contact and noncontact)	5	9	.146 [−.032, .325]	.145	−.095 [−.284, .093]	—	—	—	—
Publication year	9	19	.193 [.019, .366]*	—	−.006 [−.027, .015]	<i>F</i> (1, 17) = .362	.556	.009***	.052**
Sample characteristics									
Mean age of the sample in years (at start of a study)	6	13	.124 [−.031, .279]	—	−.013 [−.052, .025]	<i>F</i> (1, 11) = .581	.462	.014***	.017
Percentage of males in the sample	9	19	.192 [.024, .361]*	—	−.000 [−.003, .003]	<i>F</i> (1, 17) = .013	.910	.011***	.047*
Percentage of Whites/Caucasians in the sample	6	13	.243 [−.035, .521] <sup>+</sup>	—	.003 [−.012, .017]	<i>F</i> (1, 11) = .175	.684	.015***	.078*
Risk domain: Child grew up in a non-nuclear family structure (excluding having a stepfather)									
Study characteristics									
Type of sexual abuse examined									
Contact sexual abuse (RC)	13	41	.214 [.166, .262]***	.211	—	<i>F</i> (2, 83) = 5.976	.004**	.009***	.003**
Noncontact sexual abuse	5	12	.119 [.048, .189]**	.118	−.096 [−.164, −.027]**	—	—	—	—
Mixed (contact and noncontact)	12	33	.130 [.080, .181]***	.129	−.084 [−.146, −.021]**	—	—	—	—
Type of assessment									
Self-report questionnaire—Single item or multiple items (RC)									
Face-to-face or telephone interview	5	29	.118 [.050, .185]***	.117	−.059 [−.143, .025]	<i>F</i> (1, 70) = 1.958	.166	.010	.003
Age cut-off used in defining CSA (in years)									
17 or younger (RC)	11	54	.205 [.159, .250]***	.202	—	<i>F</i> (1, 84) = 6.856	.011*	.011***	.002
18–19	13	32	.116 [.066, .166]***	.115	−.089 [−.157, −.021]*	—	—	—	—
Publication year	24	86	.167 [.128, .206]***	—	−.004 [−.008, −.000]*	<i>F</i> (1, 84) = 4.496	.037*	.010***	.004***
Number of questions used to elicit CSA episodes (in self-reports or interviews)									
	13	53	.150 [.108, .192]***	—	−.005 [−.011, .000] <sup>+</sup>	<i>F</i> (1, 51) = 3.704	.060 <sup>+</sup>	.004***	.003***
Risk factor characteristics									

(table continues)

Table 3 (continued)

Variables tested as moderators	# Studies	# ES	Intercept [95% CI]/ Mean Z [95% CI]	Mean <i>r</i>	$\beta$ [95% CI]	<i>F</i> ( <i>df</i> 1, <i>df</i> 2) <sup>a</sup>	<i>p</i> <sup>b</sup>	Level 2 variance	Level 3 variance
Father- versus mother-related risk factors									
Mother-related risk factors (RC)	7	24	.146 [.069, .222]***	.145		<i>F</i> (1, 37) = .843	.365	.005***	.006***
Father-related risk factors	5	15	.170 [.091, .249]***	.168	.025 [−.030, .079]				
Sample characteristics									
Mean age of the sample in years (at start of a study)	16	62	.165 [.116, .213]***	—	.002 [−.002, .007]	<i>F</i> (1, 60) = .944	.335	.011***	.003 <sup>+</sup>
Percentage of males in the sample	23	85	.162 [.123, .201]***	—	−.000 [−.001, .000]	<i>F</i> (1, 83) = 1.472	.228	.010***	.004**
Percentage of Whites/Caucasians in the sample	15	32	.124 [.069, .180]***	—	.000 [−.004, .004]	<i>F</i> (1, 30) = .001	.982	.017***	.001
Risk domain: Child has a stepfather									
Study characteristics									
Publication year	8	12	.108 [.007, .209]*	—	−.005 [−.014, .004]	<i>F</i> (1, 10) = 1.506	.248	.008***	.007
Sample characteristics									
Mean age of the sample in years (at start of a study)	6	9	.149 [−.008, .305] <sup>+</sup>	—	−.002 [−.018, .015]	<i>F</i> (1, 7) = .048	.834	.004***	.017 <sup>+</sup>
Percentage of males in the sample	8	12	.100 [.009, .192]*	—	−.0019 [−.0034, −.0003]*	<i>F</i> (1, 10) = 7.133	.024*	.004***	.007
Percentage of Whites/Caucasians in the sample	5	8	.077 [.012, .141]*	—	−.003 [−.007, .001]	<i>F</i> (1, 6) = 2.903	.139	.003***	.000
Risk domain: Low family SES									
Study characteristics									
Type of sexual abuse examined						<i>F</i> (1, 31) = 8.527	.007**	.018***	.040**
Contact sexual abuse (RC)	13	20	.194 [.069, .319]**	.192					
Mixed (contact and noncontact)	10	13	−.029 [−.168, .111]	−.029	−.222 [−.377, −.067]**				
Age cut-off used in defining CSA (in years)						<i>F</i> (2, 30) = .170	.844	.034***	.024*
13 or younger (RC)	5	6	.152 [−.067, .371]	.151					
15–16	6	15	.101 [−.073, .274]	.101	−.051 [−.331, .228]				
17–19	10	12	.076 [−.079, .230]	.076	−.077 [−.344, .191]				
Publication year	21	33	.102 [.002, .202]*	—	−.003 [−.014, .008]	<i>F</i> (1, 31) = .251	.620	.034***	.023*
Number of questions used to elicit CSA episodes (in self-reports or interviews)	9	11	.100 [.012, .187]*	—	−.008 [−.025, .009]	<i>F</i> (1, 9) = 1.157	.310	.004***	.008
Sample characteristics									
Mean age of the sample in years (at start of a study)	16	28	.108 [.010, .206]*	—	−.006 [−.017, .006]	<i>F</i> (1, 16) = 1.066	.311	.030***	.014*
Percentage of males in the sample	19	29	.066 [−.032, .164]	—	−.001 [−.004, .003]	<i>F</i> (1, 27) = .169	.684	.040***	.012
Percentage of Whites/Caucasians in the sample	14	18	.026 [−.101, .153]	—	−.000 [−.006, .005]	<i>F</i> (1, 16) = .010	.921	.053***	.006
Risk domain: Strong religious affiliation of the child's family									
Study characteristics									
Publication year	5	7	−.050 [−.329, .229]	—	.006 [−.021, .032]	<i>F</i> (1, 5) = .293	.612	.047***	.021
Sample characteristics									
Percentage of males in the sample	5	7	−.048 [−.236, .140]	—	.008 [−.001, .016] <sup>+</sup>	<i>F</i> (1, 5) = 5.160	.072 <sup>+</sup>	.033***	.000

Note. # Studies = number of studies; # ES = number of effect sizes; Mean Z = Mean effect size (Fisher's Z); CI = confidence interval; Mean *r* = Mean effect size (Pearson's correlation);  $\beta$  = estimated regression coefficient; Level 2 variance = residual variance between effect sizes from the same study; Level 3 variance = residual variance between studies.

<sup>a</sup> Omnibus test of all regression coefficients in the model. <sup>b</sup> *p* value of the omnibus test.

<sup>+</sup> *p* < .10. \* *p* < .05. \*\* *p* < .01. \*\*\* *p* < .001.

substance (ab)use (*r* = .171), psychiatric/mental/or physical problems of parents (*r* = .169), and a low level of parental education (*r* = .149). Naturally, a parental history of child abuse victimization can also be regarded as a parental difficulty, because it may be indicative for unresolved trauma or attachment-related problems (Assink et al., 2018). Related to this risk theme are different *parenting problems and difficulties* that parents may experience. We found significant effects for a low quality of the parent–child relation including low parental attachment (*r* = .292), parental overprotection (*r* = .212), low levels of parental care/affection (*r* = .149), and a low (sense or levels of) parenting competence (*r* = .136).

A *non-nuclear family structure* may also pose a risk for CSA victimization. We found that a child growing up in a non-nuclear family (*r* = .191), and a child having a stepfather (*r* = .118), are both

significant risks. Other *family (system) problems* could also be designated as significant risks. The results showed that problems in the functioning of the family system (*r* = .194), social isolation of the family or the child (*r* = .191), and a low family SES (*r* = .101), were significant risk domains. An additional family characteristic that proved to be a risk factor was a substantial times (six or more) of moves/resettlements of the child and its family. Finally, there were several *child problems* and other *child characteristics* that could be identified as risk factors. As for child problems, we found that a physical and/or mental chronic condition (*r* = .193) may put children at risk for CSA victimization as well as using drugs or engaging in (violent) delinquent behavior (*r* = .126). Child characteristics that increase the risk for CSA victimization are a low level of social skills of the child (including shyness, *r* = .217), and a child's frequent use of the Internet (*r* = .152).

On a more general note, the results revealed that multiple child-related, parent-related, and family related risk factors contribute to the risk for CSA victimization. This observation is in line with Stith et al. (2009) who meta-analytically reviewed the literature on effects of risk factors for child physical abuse and child neglect. They concluded that child abuse should be examined from a multifactorial perspective, because risk factors at different levels contribute to child abuse. Given our results, this conclusion also holds for CSA victimization. Our results are also in line with Finkelhor and Asdigian (1996), who stated that both child characteristics and environmental factors put a child at risk for sexual abuse victimization, and with Belsky's (1980, 1993) developmental-ecological perspective on the etiology of physical child abuse and neglect. Belsky argued that child abuse is determined by different risk factors operating at multiple "levels of analysis," which is reflected by our results in the sense that different types of risk factors contribute to the risk of CSA victimization. On the other hand, our results do not enable us to make inferences on transactions between child characteristics and environmental factors, as for instance described by Cicchetti and Rizley (1981), nor on the dynamic nature of risk levels as described by Bolan (2001). Thoroughly testing these theoretical notions in the context of CSA victimization should be done in future research.

In comparing the present results to effects of risk factors for child physical abuse and neglect as analyzed by Stith et al. (2009), differences can be observed. Stith et al. found that "the parent's perception of the child as a problem" as well as parental "anger/hyper-reactivity" are among the strongest risk factors for physical abuse and neglect. Other strong risk factors for neglect (i.e., "high parental stress," "low parental self-esteem," and "poor-parent-child relationship") and physical abuse (i.e., "high family conflict," "low family cohesion," and "parental anxiety") are parent- or family related, or concern the parent-child relationship (Stith et al., 2009). Our results confirm that different risk domains related to parents, the family, or the parent-child relationship, are among the strongest and most important risk factors for child sexual abuse. However, we also found that a number of child-related risk domains is relatively strongly associated with CSA victimization (e.g., "Child experienced a form of victimization other than (forms of) child abuse," "Child is female," "Low social skills of a child," and "Child has a physical or mental chronic condition"). Further, there are differences in observed effects between the risk domains that we examined and the factors that Stith et al. examined. For instance, we found a significant positive effect for "Child has a stepfather" ( $r = .118$ ), whereas Stith et al. found a much smaller effect for the child physical abuse factor "Non-biological parent in the home" ( $r = -.03$ ), which was even insignificant. Similarly, we found a significant positive effect for growing up in a family with a non-nuclear structure ( $r = .164$ ), whereas Stith et al. reported a smaller effect of single parenthood for both child physical abuse ( $r = .12$ ) and child neglect ( $r = .08$ ). Although our risk domains conceptually differ from the risk factors examined by Stith et al., it is clear that there are differences in the risk factors that are most strongly related to child sexual abuse, physical abuse, and neglect.

When inspecting individual risk domains and their effects, several issues are noteworthy. First, our mean effect of the risk domain "Child being female" ( $r = .290$ ) was only slightly higher than the effect found by Stoltenborgh et al. ( $r = .250$ ;  $\Delta r = .040$ ), who meta-analyzed worldwide prevalence of CSA victimization

and included 331 studies in their review. Despite our more strict inclusion criteria and consequently, a smaller number of studies and effect sizes included in our gender domain, both effects are quite comparable to each other. Second, the risk domain "Low quality of a child's relation with parents/parental attachment" was classified as a child-related risk domain in this review, but naturally, parents play a crucial role in developing and maintaining positive parent-child relationships. Although the effect of this risk domain was relatively strong, the importance of child factors should not be overstated, and parent/family factors should not be understated. After all, parents, guardians, and/or caretakers are primarily responsible for fostering and maintaining a child rearing environment in which children are safe from harm.

Keeping a focus on the parent- and family factors, the results produced nonsignificant mean effects for 12 domains. However, we cannot be sure that these domains do not truly contribute to the risk for CSA victimization. Given the relatively low number of studies and effect sizes as well as the wide confidence intervals of most of these domains, the nonsignificant effects particularly indicate that more research is needed to get a better estimate of the true effects of these (putative) risk domains. Moreover, it is important to keep in mind that the parent and family domains were examined as (putative) risk domains for CSA victimization, and not for *perpetration* of CSA by parents, though parental substance abuse, a parental history of experiencing child sexual abuse, and psychiatric problems of parents may be examples of factors contributing to the risk for perpetrating CSA by parents. In fact, most experiences of CSA take place outside the family (e.g., Russell, 1983). Therefore, the presence of (an accumulation of) risk domains for CSA victimization endangers the child safety, and makes a child an easier target for CSA for a perpetrator who is inside or outside the family.

According to the bias assessment techniques that we applied to our data, there were no indications of bias in estimated mean effects of 13 risk domains, whereas the results showed some or strong indications of bias in the estimated mean effects of 22 risk domains. Interestingly, this bias may not be ascribed to publication bias alone. The trim and fill analyses showed that *above* average effect sizes had to be imputed to restore symmetry in the funnel plot of 14 risk domains. In other words, the estimated mean effects of these 14 risk domains may be *underestimations* of the true effects. Taken together, we should definitely acknowledge that bias may be a problem in a substantial number of estimated risk domain effects, which hampers drawing firm conclusions about the true effects of the risk domains that were examined in this review. Perhaps what the bias assessment results show most is that more methodologically strong research is needed to get a better grasp of the true effects of risk domains and/or risk factors. As most risk domains were based on a rather small number of effect sizes and/or studies, the risk for error and bias in effect size estimates was substantial. Therefore, the present review represents a call for future high quality studies on determining risk factors for CSA victimization, in which valid and reliable risk factor effects can be obtained.

### Mechanisms Linking Risk Domains to CSA Victimization

An important question is why the risk factors/domains with significant positive effects pose a risk for CSA victimization. Therefore, we discuss some explanations for the link between a

number of key risk factors/domains and CSA victimization. First, the risks posed by low levels of parental care and a child's social isolation may be explained by the routine activities theory (Cohen & Felson, 1979) and the work of Finkelhor and Asdigian (1996), in which capable "guardianship" plays a crucial role. If children spend much time alone or away from the eye of parents or other caretaking figures, the possibility of parental supervision or guardianship is reduced, which is needed to protect the child from CSA victimization. A low level of parental care can also mean a lack of Internet supervision, which may lead to dangerous situations for children. Many children are sexually solicited on the Internet, and in an increasing number of cases, this has led to victimization (Finkelhor, Mitchell, & Wolak, 2000). It is therefore not surprising that a frequent use of the Internet by a child was identified as a risk factor. According to Finkelhor (2008), family social isolation can also be linked to the guardianship component of the routine activities theory. But, in socially isolated families, the guardians may not refer to the caretakers themselves, but to members of a related social network whose supervision might inhibit the abuse (p. 58).

Guardianship or supervision may also be impaired when caretakers are addicted to substances (Onigu-Otite & Belcher, 2012), fight with each other (Finkelhor & Asdigian, 1996), and/or suffer from mental health problems (Fleming, Mullen, & Bammer, 1997), which all came out as significant risk domains. Parents with such issues may be too occupied with their own problems to adequately supervise and protect their children, which increases a child's vulnerability to CSA victimization. This may also hold for parents with a history of child abuse victimization, who may have trauma-related issues that interfere with adequate parenting (Assink et al., 2018) and supervision. On the other hand, too much parental involvement in the form of parental overprotection was also found as a significant risk domain. In scientific literature, there is evidence that an overprotective parenting style encourages children to behave passively or dependently (Ladd & Ladd, 1998), which limits a child's ability to defend itself against potential offenders. Perry, Hodges, and Egan (2001) suggested that when parents are overprotecting, children may develop a "victim schema" in which they view their parents as controlling and themselves as weak and helpless. Children with such conceptions may become too dependent on adults and behave in ways so that they might become an easy target for a potential offender.

The results showed that several child-related domains were significant predictors of CSA victimization, which can also be explained by the routine activities framework (Cohen & Felson, 1979), and the elaborating work of Finkelhor and Asdigian (1996). For instance, for most sex offenders, the female gender of a child represents a characteristic that makes the child a congruent target for sexual abuse, as in general, sexual assault is mostly committed by heterosexual males with a preference for girls (Finkelhor, 2008). So, a child's female gender can be regarded as an operationalization of target suitability (Cohen & Felson, 1979) and target gratifiability (Finkelhor & Asdigian, 1996). Significant effects were also found for low social skills of children (including shyness) and physical or mental chronic conditions of children. Drawing on the concept of target vulnerability, these child characteristics may decrease children's ability to resist or deter victimization, making them more vulnerable to CSA victimization than other children. For instance, children with different types of problems, such as cognitive, physical, or communicative disabilities,

can be less capable in avoiding dangerous situations and seeking help. Such children can also be more isolated from peer groups and social networks, in which help-seeking is facilitated and risk disclosure may occur (Brownlie, Jabbar, Beitchman, Vida, & Atkinson, 2007; Reiter, Bryen, & Shachar, 2007). Mobility disabilities may diminish a child's capacity to deter victimization, simply because children with such disabilities cannot physically escape from high-risk situations. Finally, delinquent or drug using youths may actively seek out high-risk situations, in which supervision is lacking and violence and delinquency occur. As such, antisocial youth may put themselves at risk for CSA victimization, which is central to the routine activities framework of Cohen and Felson (1979).

Our results showed that non-nuclear family structures, such as families with a stepfather and single parent families, pose a risk for CSA victimization. Finkelhor (2008) noted that it is not the family structure itself that increases this risk, but features that accompany specific family structures. Although much needs to be done in examining and identifying those features, Finkelhor offers a number of explanations. For instance, he states that children in non-conventional families are exposed to more unrelated and perhaps predatory or aggressive people, because there is a larger social network, the parent is dating frequently, or there are more frequent moves of the family. Notably, our results confirmed that frequently moving is a substantial risk factor for CSA victimization. A different explanation is that children who experienced family disruptions may have developed dysfunctional interpersonal patterns, because they have been exposed to conflict, aggression, and violence (Finkelhor, 2008). These children may have learned conflict-escalation rather than conflict-reduction skills, and may find themselves in more conflict situations with higher risks for victimization. A third explanation is that children in disrupted families have less control over their environment, and are therefore less able to avoid high-risk situations and victimization. For a more thorough discussion of these and additional explanations, we refer to the work of Finkelhor (2008; pp. 51–52).

Finally, prior victimization of the child—either in the form of child abuse or in any different form—or victimization of close family members, such as siblings, pose substantial risks for CSA victimization. These results underline what Finkelhor (2008) referred to as *transitivity* among victimization risks: the risk for victimization is substantially higher for a child who has previously been victimized. There may be different pathways leading to multiple repeated victimization of children, or poly victimization (Finkelhor, 2008, pp. 55–56). First, children may grow up in a victimization-filled home environment, in which domestic violence occurs and in which children may be maltreated in different ways. Such negative developmental experiences may pave the way for subsequent victimization outside a child's family through a child's emotional deficits and negative cognitive mindset. A second pathway may lead to victimization through family disruption and adversity, such as problems in the family system functioning, poverty, and low parenting competence, for which we found significant effects. Mechanisms like poor supervision, emotional deprivation, and exposure to multiple potential offenders may increase the risk for CSA and other forms of victimization. Third, there may be characteristics of the child itself that contribute to the risk for poly victimization. Specific temperaments, incapacities, and disabilities may be burdensome for caregivers or elicit irrita-

tion, anger, or frustration of caregivers or others. Children who look or behave differently, for instance because of a mental or physical disability, may also elicit feelings of dislike or resentment (i.e., target antagonism), which increases the risk for victimization.

### Moderating Variables of the Overall Strength of Risk Domains

Several study, sample, and risk factor characteristics were examined as potential moderators of the overall strength of risk domains in which heterogeneity in effect sizes was identified. In total, we found five moderating variables, but the moderating effects were rather modest.

**Study characteristics.** Across primary studies, CSA is not equally defined and different types of CSA are assessed. We found that the risk domains “Low level of parental education” and “Low family SES” were predictive of contact CSA, but not of mixed CSA (i.e., contact and/or noncontact CSA). Further, we found that the risk domain “Child grew up in a non-nuclear family structure” was less predictive of both mixed CSA and noncontact CSA relative to contact CSA. These results show that contact CSA can—at least to some extent—be better predicted than noncontact CSA. This may be explained by the fact that noncontact CSA has less severe and long-lasting consequences than contact CSA, and that therefore the threshold for perpetrating noncontact CSA may be lower relative to contact CSA. Based on this, it can be reasoned that CSA perpetrators may rather engage in noncontact than in contact CSA, making noncontact CSA a less specific phenomenon that is more difficult to predict. On the other hand, [Pereda, Guilera, Forn, and Gómez-Benito \(2009\)](#) did not find a difference in worldwide prevalence between noncontact and contact CSA. This may imply that it is not so much about a difference in thresholds, but rather about a qualitative difference between noncontact and contact CSA perpetrators. Although there is overlap in risk factors between noncontact and contact CSA perpetration, there may be additional risk factors relevant for either of the CSA types. Because noncontact CSA was examined in much fewer primary studies than contact CSA, there may be less knowledge about variables that form (strong) risk factors for noncontact CSA.

Next, we found that the cut-off age that is used in defining CSA influenced the overall strength of the risk domain “Child grew up in a non-nuclear family structure.” This domain is more predictive of CSA occurring prior to 18 years of age relative to CSA occurring at 18 or 19 year of age. This may not be a surprising finding as developmental theory suggests that family influences decrease as juveniles become older and develop autonomy from their parents ([Buhrmester & Furman, 1987](#); [Eccles, 1999](#)). Publication year also moderated the overall strength of this same risk domain, in the sense that more recently published studies reported smaller effects of risk factors that could be classified in this risk domain. Put differently, published risk factor effects decline in strength over time, and these results may support the presence of a “decline effect” ([Schooler, 2011](#)), which was referred to as the “law of initial results” by [Ioannidis \(2005\)](#). This tendency of risk factor effects becoming smaller over time may be explained by the fact that more rigorous studies with larger samples have been conducted over time leading to regression to the mean. As such, a moderating effect of publication year can also be interpreted as an indication of bias in estimated overall effects in review studies.

The moderator analyses showed that the overall strength of risk domains was moderated by study characteristics to only a limited extent. This was somewhat surprising, because the method that is used to assess CSA, the cut-off age used in definitions of CSA, and the number of questions used in eliciting CSA victimization may influence CSA prevalence estimates, and thus estimates of risk factor effects. For instance, in studies using self-report methods for assessing CSA victimization, only participants willing to disclose their history of experiencing CSA can be identified. In addition, it may be difficult for participants to recall (details of) CSA experiences, because of the traumatic nature of CSA or simply because of the time elapsed between the moment of abuse and the self-report of it. The number of questions asked to participants in self-report methods may also influence prevalence estimates. [Stoltenborgh et al. \(2011\)](#) state that multiple questions can describe more specific information of a CSA definition than a single question. They also mention that multiple questions can cover more CSA aspects than a single question, which may lead to higher prevalence rates. On the other hand, using records of official authorities for assessing CSA victimization may also be problematic, because a substantial proportion of child maltreatment instances is not reported to child protection authorities ([Finkelhor, Ormrod, Turner, & Hamby, 2005](#)). This implies that using official records may lead to underestimations of true CSA prevalences. Also, it may be expected that the cut-off age used in CSA definitions influence CSA prevalence rates, and thus risk factor effects. As the cut-off age increases, the period in which participants may have had experienced CSA becomes longer, which may increase prevalence estimates. In general, the results show that the overall strength of risk domains is not influenced by assessment method and the number of questions that are asked to participants in eliciting CSA episodes.

In line with this, the year in which primary studies were published moderated the overall strength of only a single risk domain. This result reveals not only that effects of risk factors in most risk domains are in general consistent over time, but also that there has not been much progression in the CSA literature when it comes to identifying specific variables that are strong risk factors for CSA victimization. Given that CSA is a multifactorial and not a single factorial determined phenomenon, it is perhaps difficult to identify new risk factors that on their own are strong predictors of CSA victimization. Risks for CSA victimization particularly increase when multiple risk factors are present in children and their (rearing) environment. As accumulation of—and interactions between—risk factors have been scarcely examined, future research should address this, so that more knowledge is gained about what combinations of risk factors are especially predictive for CSA victimization.

**Sample characteristics.** The percentage of males in samples moderated the overall strength of the risk domain “Child has a stepfather,” in the sense that the strength of this domain decreased as the percentage of males increased. This result indicates that the presence of a stepfather is in particular a risk factor for CSA victimization in girls, which may be explained by the fact that CSA perpetrated by stepfathers is far more often inflicted upon (step-)daughters than (step)sons ([Finkelhor, Hotaling, Lewis, & Smith, 1990](#)). The percentage of males in samples did not moderate the overall strength of other risk domains, and the percentage of Whites/Caucasians in samples did not moderate the overall

strength of any risk domain. These results indicate that most risk domains are equally predictive of CSA victimization in males, females, and different ethnic groups. However, it must be noted that risk factors for CSA victimization have been more examined in female than in male samples, as the CSA prevalence is higher among females than males. This is reflected in the significant and positive mean effect of  $r = .290$  that we found for the risk domain “Child is female.” Further, only Western studies were included in the present review, and moreover, the cultural diversity of these Western samples was limited. Therefore, our results do not permit drawing firm conclusions on the influence of gender and cultural background on the effects of risk domain. Determining differences in (effects of) risk factors between males and females, and between different ethnic groups is an important challenge for future research.

Also, no moderating effect was found for the mean age of samples on the effect of any risk domain, which may be attributable to a restriction of range. More than 80% of the risk factor effects were retrieved from studies with a sample mean age of 21 years or younger. This means that risk factor effects obtained in adult samples could not be properly compared with risk factor effects obtained in child or adolescent samples. However, comparing this is important, because studies on CSA victimization mostly use self-report methods. This means that for adults, more time has passed since experiences of CSA victimization occurred than for adolescents and children, which may negatively affect recall precision, and thus risk factor effects. The fact that no moderating effect of the mean sample age was found in the present review does not mean that this effect is absent in reality.

**Risk factor characteristics.** The results did not reveal a difference in effect between father- and mother-related risk factors, which was tested for the risk domains “Low parental care/affection” and “Child grew up in a non-nuclear family structure.” However, it should be noted that an important methodological limitation of the existing literature is that fathers and father-related risk factors are understudied. In particular mothers are available or willing to participate in research, and therefore, there is limited knowledge of how father-related risk factors contribute to the risk of CSA victimization. At this point, we cannot infer that there is a statistical significant and clinically relevant difference in effect between father- and mother-related risk factors. What we can state is that both fathers and mothers are equally responsible for fostering and maintaining caring environments in which children can achieve their full developmental potential without being harmed by any form of abuse or neglect.

In extracting effects of (putative) risk factors from included studies, we aimed for obtaining unadjusted effects. For most effects (687; 89.80%) this was possible, but a relatively small number of adjusted effects (78; 10.20%) were included in case unadjusted effects could not be extracted from primary studies. The results showed that, in the two risk domains “Parental history of child abuse victimization” and “Problems in the family system functioning,” the mean effect of adjusted effect sizes was significantly lower than the mean effect of unadjusted effect sizes. This moderating effect is not surprising, as in general, analyses controlling for different variables (that may be risk factors for CSA victimization on their own) produce smaller risk factor effects. This result implies that the estimated mean effect of at least two risk domains may be an underestimation to some extent. Of note is

that statistical adjustment could not be tested as a moderator in the risk domains “Child frequently using the internet” and “Child ever used drugs/engaged in (violent) delinquency,” as these domains were entirely based on adjusted effect sizes. For these domains, larger overall effects could have been obtained if unadjusted effect sizes were classified in these risk domains. However, as most of the examined risk domains are based on a relatively small number of adjusted effect sizes (see Table 1), the underestimation of mean effects due to statistical adjustment of risk factor effects seems limited.

## Study Limitations

The results of the present review must be interpreted in light of several limitations. The most important limitation is that we cannot be sure that our sample of included studies is representative of all studies on (putative) risk factors for CSA victimization. In our search strategy we tried to be exhaustive, but as already noted by Stith et al. (2009), a huge amount of literature on CSA has been generated and the number of published studies is continuously growing. A related issue specifically problematic in the current review was that a substantial number of relevant primary studies has not been indexed with keywords derived from the “risk factor” concept, making it more difficult to identify these studies. To deal with this problem, we used three complementary strategies in our search for primary studies, but still, we may have missed studies. Further, as a form of quality control, we restricted study inclusion to published studies and dissertations, which may have increased the risk of overestimating effects due to publication bias. The possibility that our results are biased was strengthened by the results of the bias assessment, indicating that we may have missed effect sizes in more than half of the examined risk domains. However, the trim-and-fill analyses yielded underestimated rather than overestimated mean effects of risk domains, indicating that specifically *publication* bias may not have been very likely.

Second, in our analytic strategy, we decided a priori to only examine study, sample, and risk factor characteristics as potential moderators of risk domain effects if (categories of) these characteristics were based on at least five studies. This decision was made as performing a large number of moderator analyses (27 risk domains with heterogeneity in effect sizes  $\times$  8 variables to examine as potential moderator) is not only impractical, but also statistically unwise, because insufficient data and capitalization on chance pose important problems. Still, the significant results produced by the moderator analyses may be attributable to spuriousness, for instance because potential moderators were examined in bivariate models without control variables. Specifically, the age cut-off used in CSA definitions, publication year of studies, and the percentage of males in samples were significant moderators of single risk domains, but were tested as a moderator in nine, 18, and 17 risk domains, respectively. Type of sexual abuse appears to be a somewhat more robust moderator, because significant moderating effects of this variable were found in three (of eight examined) risk domains. In general, the moderating effects that we found were more modest than substantial. On the other hand, it should be acknowledged that the effect size heterogeneity in 28 risk domains may imply that the overall effects of these 28 domains is not equally strong across different study designs and for children with different background characteristics (e.g., boys and girls). There-

fore, more focused and rigorously designed research aimed at examining effects of specific risk factors in different groups and under different circumstances, is needed.

Third, all methods currently available for assessing bias in meta-analysis have their own limitations and drawbacks. A general drawback is that all available methods were originally designed for meta-analyses in which independence of effect sizes is assumed. This poses a particular problem in the present review, as multiple effect sizes were extracted from most included studies. The assumption of effect size independency was therefore clearly violated in the bias assessment tests that we conducted. Further, the performance of the trim-and-fill method is limited when effect sizes prove to be heterogeneous (Peters, Sutton, Jones, Abrams, & Rushton, 2007; Terrin, Schmid, Lau, & Olkin, 2003). Egger, Davey-Smith, and Altman (2001) noted that applying the trim and fill method could mean adding and adjusting for nonexistent effect sizes in response to funnel plots that are asymmetrical simply because of random variation. As for the Egger's test and Begg and Mazumdar's rank test, the statistical power is very poor in small meta-analyses and in the case of the presence of small bias in data (Nik Idris, 2012). More recently developed methods for bias assessment have also limitations: PET only performs well when heterogeneity in effect sizes is low and a true effect cannot be assumed (Stanley, 2017), PEESE has a poor coverage of true effects (Inzlicht, Gervais, & Berkman, 2015), and *p* curve analysis assumes the absence of nonsignificant effect sizes in the data to be analyzed (McShane, Böckenholt, & Hansen, 2016). All these methods have in common that they have not been tested and evaluated yet in three-level meta-analysis, in which effect size dependency is modeled. The considerable and ongoing debate in scientific literature about the appropriateness of different methods for detecting and handling missing data reflects the importance of interpreting the results of our bias assessment with caution.

Several other issues should be addressed that affect the inferences that can be drawn from our results. The present review does not permit drawing conclusions about (the direction of) causality, because of the nonexperimental nature of this review. Further, in extracting effects of (putative) risk factors from primary studies, we focused on antecedents of CSA victimization (see also the in- and exclusion criteria in the Method section), but because many included studies were retrospective in nature, we cannot be certain that all factors classified into risk domains were true antecedents rather than outcomes. Further, our aim was to estimate mean effects of individual risk domains, but it is to be expected that specific combinations of (or interactions between) risk domains are particularly predictive of CSA victimization, as accumulation of risk factors plays an important role in multiple theories on the etiology of child abuse. Examining interactions between multiple risk (and protective) factors is therefore an important challenge for future research. A last issue involves the nature of comparison or control conditions used in primary studies (comprising the participants assessed as not having a history of CSA victimization). As already noted by Mulder, Kuiper, Van der Put, Stams, and Assink (2018), it is difficult to rule out experiences of child abuse and neglect in control participants, as underreporting is a problem in essentially all available methods for assessing child abuse and neglect (e.g., Fergusson, Horwood, & Woodward, 2000; Finkelhor, 2008). Additionally, different forms of child abuse and neglect often co-occur (Arata, Langhinrichsen-Rohling, Bow-

ers, & O'Farrill-Swails, 2005). Consequently, our estimates of risk domains may have been affected by the fact that (levels of) risk factors were not compared with *true* control participants, but rather with participants who in reality did have experiences of CSA, another form of child abuse or neglect, or a combination of abuse types. In particular this last limitation reflects how difficult it is to get a grasp on true prevalences of child sexual abuse, and in turn, true effects of risk factors for CSA victimization.

## Clinical Implications

One of the advantages of synthesizing primary studies on risk factors for CSA victimization is that it provides suggestions for improving clinical practice. The results of this review show that the risk for CSA victimization can best be assessed from a multifactorial perspective, meaning that child-, parent-, and family related risks deserve attention in instruments for risk assessment. In such instruments, specific attention needs to be given to the assessment of both static and dynamic risks that are related to prior CSA and non-CSA victimization of the child and its family members, because these risk factors have relatively strong effects, and therefore, important predictive value. However, other risk factors should also be assessed, because substantial effects were found for a variety of factors related to parents (e.g., intimate partner violence between a child's caretakers, parental overprotection, and parental mental and physical health problems), the family (e.g., problems in the family system functioning, social isolation of the family, low family SES), and the child (e.g., child is female, and low social skills of a child). The quality of the parent-child relation and the parental attachment is also a risk that should be assessed. In risk assessment strategies, it is important to assess a variety of risk factors that can be present at different levels or social systems influencing the child. After all, from scientific literature we know that it is the accumulation of risks, rather than single risk factors, that increases a child's risk for child abuse victimization (e.g., Belsky, 1980, 1993; Cicchetti, Toth, & Maughan, 2000; MacKenzie, Kotch, & Lee, 2011).

A similar approach is necessary in assessing the care needs of children and their family members, which is also referred to as needs assessment. Contrary to risk assessment, needs assessment should be directed at measuring only those risk factors for CSA victimization that are dynamic, and thus modifiable in (preventive) interventions. Also in needs assessment, multiple child, parent, and family risk factors should be assessed, as this review showed that many of these risks for CSA victimization are dynamic in nature. Examples of dynamic factors with relatively strong effects are a low quality of the parent-child relation/low parental attachment, parental overprotection, problems in the family system functioning, and current episodes of (non-CSA) forms of child abuse in the child's home environment. As for victimization, prior occurrences of (CSA and non-CSA) victimization of the child or its family members is static in nature, and should therefore be assessed in instruments for *risk* assessment. However, in *needs* assessment it is essential to pay attention to the potential consequences of any prior victimization of children and family members. For instance, parents with a history of child abuse victimization—which proved to be a risk domain with a substantial effect for CSA victimization—may suffer from unresolved trauma that requires attention in treatment (Assink et al., 2018).

Drawing on the risk and need principles of the RNR-model (Andrews & Bonta, 2010; Andrews et al., 1990; Bonta & Andrews, 2007, 2010), proper risk and needs assessment is essential in answering (a) *which children* are at risk for CSA victimization, and thus require one or more (preventive; family) interventions to reduce this risk, (b) with *what intensity* these children and their families should be treated to effectively reduce the risk, and (c) *what care needs* of children and their families must be addressed in these interventions. The knowledge that this review produced on risk factors for CSA victimization and their effects is very important in developing and improving psychometric valid instruments that support clinical practitioners in answering these three questions. Of note is that in the clinical practice of child welfare, safety assessment is the first essential step in the diagnostic process, which is aimed at assessing (and resolving) the immediate child safety (Vial, Assink, Stams, & Van der Put, 2018). The current review has only limited value for improving or developing instruments for safety assessment.

The results of the moderator analyses generally suggested that effects of risk domains for CSA victimization are not substantially different between boys and girls. However, there is an exception, as the effect of having a stepfather was stronger for girls than for boys. This implies that the risk of having a stepfather should receive more weight in risk (and perhaps needs) assessment in girls than in boys. Similarly, no moderating effect was found for the percentage of Whites/Caucasians on the effect of risk domains, suggesting that effects of risk domains are the same for children of different ethnicity and cultural background. These results may imply that for different groups of children (and their families), a rather general assessment strategy works equally well, but caution must be exercised in drawing such conclusion. First, we could not test moderating effects of gender and ethnicity on the effect of all risk domains, and second, multiple scholars have stressed the importance of validating assessment instruments in different populations, so that in each population, risk factors are properly weighed to obtain the optimal assessment outcomes (e.g., Assink et al., 2015; Van der Put et al., 2011).

Following the broad and multifactorial perspective that is needed in risk and needs assessment, (preventive) interventions should also be based on the notion that CSA victimization results from the presence of multiple determinants at different ecological (social) systems surrounding a child. In practice, this means that care needs of the child, the parent(s) or caretaker(s), and other family members must be addressed to effectively reduce the risk for CSA victimization. A multifactorial perspective in clinical practice probably stimulates a systemic-oriented approach to (preventive) interventions, reducing a misleading focus on individual risk factors, or an individual child or caretaker. It may also minimize blame on not only the child, but also individual caretakers. This is important, because blaming nonoffending mothers for CSA victimization of their child has serious consequences for both the mother (Fong & Walsh-Bowers, 1998; Jackson & Mannix, 2004) and the child (Lovett, 2004).

As a final remark on improving clinical practice, we underline that this review synthesized studies in which primarily child-, parent-, family-, and only occasionally, community-related factors were examined. In other words, particularly factors present in children and in rather proximal ecological systems surrounding children have been studied throughout the years. Naturally, such

primary research produces valuable knowledge for clinical practitioners, who constantly make important decisions on *who* is in need of (preventive) care, and *what* care is needed. However, it should not be ignored that more distal ecological systems also influence the risk for CSA victimization. Factors operating at the macrosystem (Belsky, 1980, 1993) influence cultural values and beliefs about how children should be treated, and how child sexual abuse is perceived by different societies. Such values and beliefs can, for instance, be influenced by public messages communicated through (mass) media campaigns and require governmental leadership (e.g., Wurtele, 2009). So, although the results of this review are not informative on the effects of societal risk factors for CSA victimization, this does not undermine the value of attention to the macrosystem in the perspective on CSA victimization. A key challenge in future research is to expand the knowledge on societal and cultural risk factors for CSA victimization.

## Conclusion

To our knowledge, this is the first meta-analytic review of primary research on effects of risk factors for child sexual abuse victimization. Our results revealed that 25 of 35 examined risk domains—defined as groups of risk factors that are more or less similar in nature—are associated with child sexual abuse victimization, indicating that a substantial number of risk domains contribute to the risk of child sexual abuse. Significant and substantial effects were found for risk domains that refer to: prior (CSA and non-CSA) victimization of the child and/or its family members, different types of parental problems (e.g., mental health problems and intimate partner violence), parenting problems and difficulties (e.g., a low quality of the parent–child relation and low parenting competence), a non-nuclear family structure (e.g., presence of a stepfather and growing up in a family with a non-nuclear structure), family problems (e.g., a family being socially isolated), child problems (e.g., having a physical or mental chronic condition and low social skills), and other child characteristics (e.g., child being female). These results imply that a multifactorial approach is needed in risk and needs assessment, and in interventions aimed at reducing the risk for child sexual abuse victimization. The results also showed that the effect of two risk domains was stronger for contact sexual abuse victimization than for noncontact sexual abuse victimization, indicating that the former may be somewhat better predicted than the latter. We did not find significant differences in effects between father- and mother-related risk factors, meaning that risk factors related to both fathers and mothers deserve attention in clinical practice. The results of this review provide valuable insights for the development and improvement of both assessment and (preventive) intervention strategies.

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## Appendix A

### Characteristics of Included Studies

Reference	Year	% Males in sample	% Whites in sample	Mean age of sample	Age cut-off	# Extracted risk factors
Agyapong et al.	2017	38.60	73.90	35.67	18	2
Alriksson-Schmidt et al.	2010	0	63.20	—	19	1
Amodeo et al.	2006	0	54.50	37.35	17	3
Avery et al.	2002	48.60	55.30	7.90	17	1
Baril & Tourigny	2016	—	—	—	18	11
Bergner et al.	1994	0	84.40	19.30	17	8
Boney-McCoy & Finkelhor	1995	52.10	80.00	12.87	17	12
Briscoe-Smith & Hinshaw	2006	0	53.00	9.58	13	1
Brunnberg et al.	2012	0	—	17.50	19	4
Buckle et al.	2005	49.38	—	16.02	18	6
Cohen et al.	2001	48.95	91.87	—	18	3
Cuevas et al.	2009	50.30	75.70	9.50	18	1
DiLillo et al.	2000	0	61.50	26.15	18	1
Dong et al.	2004	46.00	74.00	56.00	19	9
Edmond et al.	2003	0	33.00	16.33	19	5
Ensink et al.	2017	38.96	98.00	6.17	13	6
Estes & Tidwell	2002	50.00	50.00	5.80	12	2
*Fergusson, Horwood, et al.	1997	0	—	18.00	16	39
*Fergusson, Lynskey, et al.	1996	49.36	86.16	18.00	16	45
Finkelhor	1980	0	—	17.00	17	36
Finkelhor, Hotaling, et al.	1990	0–100	—	—	19	22
Finkelhor, Moore, et al.	1997	51.00	82.16	—	18	11
Finkelhor, Ormrod, et al.	2007	—	—	—	19	8
Fleming et al.	1997	0	—	—	16	24
Gagné et al.	2005	0	—	16.30	12	6
Hamby et al.	2010	50.00	53.00	—	18	4
Higgins & McGabe	2000	26.30	—	31.46	13	4
Howes et al.	2000	67.70	52.30	4.00	6	9
Hussey et al.	2006	—	—	—	13	4

(Appendices continue)

## Appendix A (continued)

Reference	Year	% Males in sample	% Whites in sample	Mean age of sample	Age cut-off	# Extracted risk factors
Kanamüller et al.	2014	0	98.20	—	18	14
Kenny & McEachern	2000	0	23.00	37.00	18	1
Kim et al.	2010	0	—	11.10	17	6
Kvam	2004	0	—	—	17	2
Laaksonen et al.	2011	100	—	29.31	16	45
Lacelle et al.	2012	0	—	21.20	18	5
Langeland et al.	2015	48.90	—	—	18	3
Lanktree	1991	51.60	6.30	12.10	17	3
Lynch & Cicchetti	1998	62.70	23.70	8.79	13	1
MacMillan et al.	2013	48.81	—	27.10	15	9
Maikovich-Fong & Jaffee	2010	23.00	49.00	11.14	16	2
Maker et al.	1999	0	70.00	22.20	16	5
Manion et al.	1996	—	—	10.21	16	35
Martin et al.	2011	47.07	—	21.00	16	38
Martinez	—	48.00	46.00	7.44	10	9
McClellan et al.	1995	62.93	80.76	13.50	19	11
McCloskey & Bailey	2000	0	53.00	9.20	13	12
Mohler-Kuo et al.	2014	52.32	—	15.50	17	32
Mueller-Johnson et al.	2014	0–100	95.28	15.41	18	56
Mulder et al.	1998	—	—	—	19	1
Mullen et al.	1996	0	—	—	15	14
Ney	1987	46.10	41.80	47.00	13	13
Noll et al.	2009	0	49.00	11.11	17	1
Nuttall & Jackson	1994	44.25	—	45.90	19	1
Oates et al.	1998	26.19	—	9.33	16	7
Paradise et al.	1994	76.81	24.15	7.30	13	11
Perez-Fuentes et al.	2013	47.79	70.99	—	18	4
Plaza et al.	2014	0	100	32.60	18	5
Ray et al.	1991	0	91.50	18.90	17	16
Roberts et al.	2012	0–100	—	—	18	4
Sansonnet-Hayden et al.	1987	46.30	—	14.60	19	8
Shipman et al.	2003	0	95.00	8.96	13	6
Sobsey et al.	1997	0–100	—	2.90–14.00	18	6
Spencer et al.	2005	—	—	—	19	2
Stout-Miller et al.	1997	40.05	91.18	—	18	9
Suris et al.	1996	0–100	87.60	15.00	19	6
Turner, Finkelhor et al.	2017	51.20	56.80	8.60	18	1
Turner, Vanderminden et al.	2011	51.10	59.60	9.60	18	12
<sup>b</sup> Walsh et al.	2003	0–100	—	—	19	14
<sup>b</sup> Walsh et al.	2002	45.40	—	—	19	5
Widom et al.	2015	46.10	41.80	47.00	13	15
Williamson et al.	1991	18.00	84.00	14.18	18	16
Yama et al.	1996	0	92.00	20.20	17	1
Zolotor et al.	2007	51.40	84.70	—	18	9
Zuravin et al.	1996	—	—	—	19	1

*Note.* Year = publication year; % Male = percentage of males in a sample; % Whites = percentage of Whites/Caucasians in a sample; Age cut-off = The age cut-off (in years) that was used in a primary study's definition of child sexual abuse; # Extracted risk factors = number of risk factors extracted from a study. Studies sharing the same superscript (<sup>a</sup> or <sup>b</sup>) were assigned the same study id in the dataset, because the same sample of participants was used in these studies. Dashes indicate that a statistic could not be retrieved from a study. Ranges indicate that extracted effect sizes were based on at least two (sub)groups of participants for which the statistic varies between the reported lower and upper limit.

(Appendices continue)

## Appendix B

## Overview of Risk Domains With Examples of Risk Factors Classified in Each Domain

Risk domain	Examples of risk factors
<b>Child-related risk domains (#8)</b>	
Child demonstrates gender non-conforming behavior	Male child has history of cross-dressing; Child is gender non-conform to a substantial degree; Child scores in the highest decile of gender non-conformity
Child experienced a form of victimization other than (forms of) child abuse	Child experienced parental death; Child was a victim of a crime; Child was a victim of peer/sibling non-sexual assault; Child was indirect victimized or a witness of non-sexual assault in the last year; Child experienced other kind of non-sexual trauma
Low quality of child's relation with parents/parental attachment	Child has poor relation with parents; Child scores in lowest quartile/quintile of parental attachment score
Child is female	Child is female
Shyness/Low social skills of a child	Child is not doing well socially at school; Child is shy
Child has a physical or mental chronic condition	Child is diagnosed with ADHD; Child has a physical disability; Child has learning difficulties; Child has a speech and/or language disorder; Child has a low IQ; Child has a visible or nonvisible physical chronic condition; Child is deaf; Child has a psychiatric disorder; Child has a functional limitation; Child has a history of multiple hospitalizations
Child frequently using the internet	Child is frequently using the internet
Child ever used drugs/engaged in (violent) delinquency	Child ever used drugs; Child is/was a (violent) delinquent
<b>Parent-related risk domains (#15)</b>	
High levels of parental/family stress	Mother experiencing stress; High level of family stress; High number of family stressors
Parental history of child abuse victimization	Parental history of physical/sexual abuse; Parent was neglected in own childhood; Non-offending parent was sexually abused in own childhood; Mother experienced psychological violence in own childhood; Mother or mother's partner experienced incest in own childhood
Parental overprotection	In highest quartile/quintile of maternal or paternal protection score; Overprotecting and/or overcontrolling mother; Overprotecting and/or overcontrolling father; Parental overprotection
Intimate partner violence between a child's caretakers	Father is violent toward mother; Mother is violent toward father; Violence between parents; Mother experienced physical domestic violence during her life
Poor physical health of parents	Father/Mother is often ill; Father/mother has poor physical health
Parental relationship problems (excluding IPV)	Parents had unhappy marriage; Parents showed little mutual affection; Extreme conflict between parents; Low couple satisfaction; Parents score in highest quartile of marital conflict score; Frequent parental conflict
Parental substance (ab)use	Maternal/Paternal substance abuse; Mother is a heavy drinker; Mother is drug dependent; A parent is too drunk or high to care for the child; There is a history of substance abuse in the family
Parental mental/psychiatric or physical problems	Parental history of depression/mania/schizophrenia/depression/anxiety; Parental mental/psychiatric illness; Parental suicide attempts; Mother has a poor mental health; Mother/father has a psychological disorder; Family has a history of mood/psychotic disorders
Low level of parental education (high school or less)	Parental education of high school or less; Father/mother did not complete high school; Father/mother has no educational qualifications
Low parental care/affection (excluding forms of child neglect)	Child is not emotionally close to mother; Child is not close to father; Lack of physical affection from father; Child has no male/female adult who is caring; Father/mother is cold and rejecting; Low parental monitoring; Parental hostility; Parents score in the lowest quartile of parental care scores
Low (sense of) parenting competence	Father/mother has low sense of parenting competence; Mother is unstructured in parenting (e.g., inconsistent, unfair, and/or irritable); Mother has low level of reflective functioning/mentalizing abilities; Father/mother has feelings of personal inadequacy and inferiority in comparison with others; Father/mother is harsh in parenting; Parents have low expectations of a child's development; Sex education of father/mother is inadequate; Father/mother gives no sex education
Young maternal age (20 or younger at child birth)	Child has a young mother (i.e., 13–19 years); Young maternal age at birth of first child (20 or younger)
Conservative sexual/family values of parents	Father/mother is sexually punitive (i.e., punishing a child's normal sexual behavior); Father/mother has conservative family/sexual values
Parental unemployment	Mother/father is unemployed
Dysfunctional maternal attitudes (pre- and postnatally)	Maternal pre-pregnancy attitude toward child is unpositive; Mother does not want to have contact with the newborn infant; Mother does not engage in breastfeeding; Maternal contact with the baby is ambiguous (wanted & unwanted)

(Appendices continue)

## Appendix B (continued)

Risk domain	Examples of risk factors
Family-related risk domains (#12)	
Prior sexual abuse victimization of child or sibling	Prior sexual abuse; Prior genital violence; Sibling (brother/sister) has been sexually abused; Child was a victim of sexual victimization in the last year
Family history of antisocial/criminal behavior	Father/mother has a history of criminal offending/antisocial behavior; Presence of a criminal household member; High parental sociopathy; Father/mother has a record of criminal offending
Prior or concurrent forms of (non-sexual) child abuse in the home environment	Child was exposed to emotional/physical abuse; Child was exposed to emotional/physical neglect; Child experienced psychological abuse; Child witnessed psychological or physical violence between parents; Parental use of physical punishment was too frequent or too severe; Child us under child welfare placement
Problems in the family system functioning	High levels of anger/sadness in the family; High family chaos; Low role clarity of family members; Unhealthy family functioning; Low family cohesion; High levels of conflict in the family; Low family adaptability; Unhappy family members; Low independence of family members; Low number of active-recreational activities of family members; Low emotional expressiveness of family members; High levels of control in the family
Child or family members experience social isolation	Child has two or fewer/only few good friends; Child is dissatisfied with social life; Mother reports low social support satisfaction; Mother has a low perception of social support; Child is raised in a family with a high degree of social isolation
Child grew up in a non-nuclear family structure (excluding having a stepfather)	Child lives with only one parent; Child is separated from father/mother; Parental separation/divorce; Child has a stepmother; Child lives in a foster home/institution; Mother is a single parent; Presence of a stepmother; absence of biological father/mother; Child lives with grandparents/foster parents; Child is growing up separate from parents; Child had a stepparent before age 15
Sibling(s) experienced child abuse (other than sexual abuse)	Sibling(s) experienced physical abuse; Sibling(s) witnessed domestic violence
Child has a stepfather	Child has a stepfather; Mother's partner is biologically unrelated to the child
Low family SES	Family is of unskilled or semiskilled SES; Low family SES; Low annual family income (<\$25,000); Child is living in poverty; Low monthly family income; Low parental socio-economic status
Large family size (i.e., larger family size relative to families of nonabused children)	Larger family size; More crowded home
Strong religious affiliation of the child's family	Family has a religious background; High moral-religious emphasis of family members; Highly religious family; Religious upbringing of a child; Child is involved in religious activities to a high degree
Living in a violent community	Child lives in a high violent community; Child lives in a relatively dangerous community

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