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Child Sexual Abuse as a Unique Risk Factor for the Development of Psychopathology: The Compounded Convergence of Mechanisms

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Abstract

Meta-analytic, population cohort, prospective, and clinical studies provide systematic evidence that child sexual abuse accounts for unique variation in several deleterious outcomes. There is strong evidence for psychiatric disorders, including posttraumatic stress disorder and mood, anxiety, and substance use disorders, and mixed evidence for personality disorders. Evaluation of sex-specific outcomes shows strong evidence for teenage childbearing, sexual revictimization, and sexual dysfunction and mixed evidence for heightened sexual behaviors and sexual offending. This review further demonstrates not only that survivors suffer the noxious impact of traumatic sexualization but that additional transdiagnostic mechanisms, including the biological embedding of stress, emotion dysregulation, avoidance, and insecure attachment, converge to compound risk for deleterious outcomes. A road map to enhance the rigor of future research is outlined, and specific recommendations for evidence-based policy making to boost prevention efforts and increase access to treatment are discussed.

Keywords

sexual abuse; systematic review; traumatic sexualization; biological embedding

> Hoping they would exceed the distance to my bedroom, I would count his footsteps.
>
> When they so often did not, the faint squeak of my door opened into a shroud of secrecy that I prayed could contain the shame, my rage, . . . remnants of a sullied innocence.
>
> Footsteps and squeaky hinges echo still across the fortress of my shattered soul, the keys to which are held by no one.
>
> —Anonymous

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INTRODUCTION

The trauma of child sexual abuse (CSA) has long been assumed to be a potent risk factor for the development of psychopathology. This review critically evaluates the scientific evidence for such a link with careful attention to possible confounding effects of sociodemographic risk and other types of co-occurring trauma. This review considers the definition of CSA to be consistent with that of a recent multidisciplinary, international analysis (Mathews & Collin-Vézina 2017). By this definition, CSA includes sexual acts with a child under the age of 18 that involve direct physical contact and/or noncontact sexual acts, in which there is no or limited capacity to provide true consent. CSA occurs most often within the family, but it also occurs within institutions (e.g., schools, sports leagues, churches), among trusted individuals outside the family, at the hands of strangers, and in the form of Internet-initiated victimization through online exploitation, child pornography, and the luring of children offline for sexual encounters (Noll et al. 2013). The prevalence of CSA is disturbingly high; a recent meta-analysis across 24 countries reported pooled national prevalence estimates of 15% for girls and 8% for boys (Barth et al. 2013). The most recent US incidence report showed that over 63,000 children were victims of substantiated CSA in 2018 (Child. Bur. 2020)—up 6% from 2017 and representing the first increase in over 15 years (Finkelhor et al. 2020). Even if the rates are extrapolated across several decades, there is a marked discrepancy between these annual incidence rates compared with prevalence rates of adults retrospectively reporting CSA before age 18—that is, 1 in 5 women and 1 in 10 men (Pereda et al. 2009). Although it has been argued that retrospective reports of CSA might be inflated as a result of bias (Baldwin et al. 2019), this discrepancy suggests that there are likely substantial numbers of CSA cases that are never brought to the attention of authorities, never disclosed, and thus potentially untreated. When productivity losses as well as health care, education, child welfare, and criminal justice costs are considered, the lifetime economic burden of CSA is estimated to exceed $9.3 billion (Letourneau et al. 2018).

As this review demonstrates, several recent meta- and umbrella analyses clearly reinforce what the field has intuited for decades: CSA is associated with a host of psychopathology outcomes. However, because the list of deleterious outcomes is long and diverse, this review endeavors to discern whether the pathogenic influence of CSA stems mainly from its overall toxicity, which, other than perhaps because of its severity, is indistinguishable from that posed by any number of other noxious life events. Alternatively, given its sexually explicit nature, could CSA be highly unique in its conveyance of risk, such that it affects a litany of outcomes through this insidiousness? The distinction is important because it is one of equifinality versus multifinality (Cicchetti & Rogosch 1996). Equifinality would suggest that CSA is one among many pathways to psychopathology and that tools designed to abate associated symptoms are interchangeable across all types of trauma that produce similar symptoms. While the etiology may differ, the treatment for those symptoms does not, and thus there would be little need for tailored or novel, CSA-specific interventions. If, however, the pathogenic influences of CSA are distinguishable from those of other forms of trauma such that CSA constitutes a highly inimitable etiology and a distinct phenotype, then a multifinality explanation of psychopathology sequelae is warranted. This perspective would suggest that the injurious nature of CSA, in and of itself, disrupts healthy development

across multiple domains and that different intervention tools are likewise needed to address its uniqueness. It is, of course, possible that both are true—that CSA shares common components with other traumas and adversities (e.g., stress) but that some aspects are unique to CSA and not present in others (e.g., sexual exploitation). If such is the case, then a comprehensive model addressing both unique and shared mechanisms would best serve survivors.

To parse out whether CSA has unique effects on psychopathology, it is important to evaluate research on the basis of its ability to adequately disentangle the effects of CSA from the effects of other forms of trauma and adversity. Such disaggregation is increasingly difficult because of recent trends whereby CSA is regarded as one of the "adverse childhood experiences" (ACEs) and is thus included in widely reported aggregate ACEs scores (Anda et al. 2020) along with other ACEs including physical abuse, neglect, verbal abuse, family violence, parental mental illness, and divorce. Research on ACEs weights all ACEs equally and applies a dose effect whereby, regardless of type, having four ACEs is appreciably worse than having three or fewer. CSA is also a category of child maltreatment (CM), which, along with physical abuse and neglect (and, per recent trends, emotional and/or psychological abuse), is recognized by the child welfare system as worthy of intervention. Most research on ACEs and CM does not distinguish between types of adversity or trauma, and results are often reported in aggregate such that discerning the unique effects of any one particular type is nearly impossible. Moreover, ample research demonstrates that types of ACEs and CM rarely occur in isolation—for instance, physical abuse may also occur in a neglectful home—and it is difficult to disentangle the effects of one type without also considering the confounding impact of another (Vachon et al. 2015).

What follows is a careful review of research on the effects of CSA. Systematic reviews and meta-analytic approaches are prioritized with accompanying AMSTAR ratings (Shea et al. 2007), as are studies with appropriate designs, controls, or statistical methods that allow for the parsing out of variability that is uniquely attributable to CSA versus other types of trauma or adversity. The overall objective of this review is to provide a useful summary of findings that will serve the field in its deliberations regarding whether CSA is one type of adversity that lies on a continuum of toxic stress or whether there are discernible aspects of CSA that confer unique risk in the development of psychopathology. This review also considers research on the discovery of plausible transdiagnostic mechanisms that explain the relationship between CSA and psychopathology at multiple levels of analysis under the lens of developmental psychopathology (Toth & Cicchetti 2013). The review culminates in an integrative model of the compounded mechanisms that set the stage for CSA being a potent risk factor for psychopathology and suggests a road map to enhance the rigor of future research, as well as specific recommendations for boosting prevention efforts and increasing access to treatment.

CHILD SEXUAL ABUSE AND PSYCHOPATHOLOGY

Evidence for the association between CSA and psychopathology derives from three main sources: meta-analytic and umbrella analyses, population-based cohort studies, and well-controlled, prospective, longitudinal studies where CSA is confirmed via official child

welfare records and psychopathology is subsequently and rigorously tracked. Large clinical studies of diagnosed patients who retrospectively report having been sexually abused in childhood are also included in this review, as they offer valuable insight into the prevalence of CSA in treatment-seeking populations. Finally, the extent to which plausible confounds, including other types of trauma and adversity, are taken into account in analyses is highlighted whenever possible.

Mood Disorders, Anxiety Disorders, Posttraumatic Stress Disorder, and Substance Use

Several meta-analyses have offered a comprehensive picture of the large array of psychiatric outcomes associated with CSA. The most recent compilation is a 2019 umbrella review (Hailes et al. 2019) of meta-analyses that reported aggregate odds ratios (ORs) of 3.3 for conversion disorder, 2.9 for borderline personality disorder (BPD), 2.7 for both anxiety and depression, 2.3 for posttraumatic stress disorder (PTSD), 2.2 for eating disorders, 1.9 for somatoform disorders, 1.7 for substance misuse, and 1.4 for schizophrenia. However, on the basis of published heterogeneity estimates, publication bias including excess statistical significance and small study effects, prediction intervals, and AMSTAR ratings (Shea et al. 2007), the authors concluded that only two outcomes (PTSD and substance misuse) reached high-quality meta-analytic standards (and thus should be prioritized) and that little credence should be given to other disorders.

While an impressive undertaking, this umbrella analysis is far from definitive in terms of its contribution to the overall understanding of the effects of CSA on psychiatric outcomes. Quality ratings were based on aggregate assessments across all meta-analyses within diagnostic sets without regard for potential quality exemplars, and there were no distinctions between ORs that were adjusted versus unadjusted for potential confounds, making it difficult to discern unique effects of CSA for these outcomes. Most importantly, there were notable omissions of clearly relevant meta-analyses that did not meet exclusion criteria but were nonetheless left out of the umbrella analysis. For example, Teicher & Samson's (2013) meta-analysis of the effects of CSA on psychiatric outcomes, which has a high AMSTAR rating (10), presented aggregate effects by study type (prospective or retrospective) and was careful to distinguish between unadjusted ORs and those adjusted for potential confounds [i.e., adjusted ORs (AORs)], including other forms of maltreatment and adversities. Their criteria for CSA were well defined to include either CSA or a composite exposure measure, such as ACEs that included CSA. The meta-analysis contained 37 cross-sectional studies (total $N = 244,887$) and 8 prospective studies (total $N = 3,149,066$). Results indicated aggregate AORs of 2.2 for depressive disorders, 4.4 for PTSD, 2.4 for anxiety disorders, 2.0 for alcohol use disorders, and 2.3 for drug use disorders.

Although included in Teicher & Samson's (2013) meta-analysis, several population-based cohort studies deserve specific mention for the rigor imposed to parse out the potential unique contribution of CSA to psychopathology. In a large study of 1,411 female adult twins (Kendler et al. 2000), AORs for major depression, generalized anxiety disorders, panic disorder, bulimia nervosa, and alcohol and drug use disorders ranged from 2.5 to 6.5. Although analyses were controlled for a host of family factors and parental history of psychiatric disorders, it is unclear whether other forms of trauma were covaried. However,

this analysis demonstrated how increased CSA severity corresponded with linear increases in AORs. For example, AORs were modest for non-genital-contact CSA, were higher for genital contact, and were highest for intercourse, with most for the latter exceeding 3.0—a finding that was replicated across twins. In cases of discordant pairs, the exposed twin was consistently higher in risk for psychopathology. The National Comorbidity Survey (NCS) of 5,877 adults who received full diagnostic interviews (Molnar et al. 2001) exercised careful control over confounds including a host of alternative family factors, adverse experience, and parental maltreatment. This study also reported differences by gender. While the AOR for CSA with any disorder was 2.3 for both men and women, there were 14 disorders significant for women and only 5 for men. AORs were highest for PTSD (10.2 for women; 5.3 for men), followed by mania (9.1 for women; not reported for men) and drug dependence (2.0 for both women and men). In a subsequent analysis of NCS data that took into account comorbidities across diagnoses (Cougle et al. 2010), CSA was uniquely associated with social anxiety disorder, panic disorder, generalized anxiety disorder, and PTSD for females; for men, CSA and physical abuse were both associated with social anxiety disorder and PTSD.

A handful of carefully designed prospective studies also merit specific mention for their rigor in discerning the unique effects of CSA. In the prospective Female Growth and Development Study (FGDS) (Trickett et al. 2011), 84 females with substantiated CSA were referred by Child Protective Services agencies, and 102 nonabused control females, demographically matched on age, race, family constellation, ZIP code, and nonsexual traumas, were recruited via advertisements in the same neighborhoods. Participants were enrolled within 6 months of CSA disclosure at a mean age of 11 and then reassessed five additional times during childhood/early adolescence (Times 2 and 3; mean ages 12 and 13, respectively), mid- to late adolescence (Times 4 and 5; mean ages 18 and 19, respectively), and early adulthood (Time 6; mean age 24). Psychiatric interviews were conducted in young adulthood, with 88% of the sample retained. Results indicated that 54% of the CSA group (compared with 16% of the comparison group) had a psychiatric diagnosis, and there were significantly more cases of PTSD, dysthymia, major depression, panic disorder, and drug and alcohol dependence in the CSA group.

Widom and colleagues assembled a group of maltreated participants whose court records in the early 1970s showed documented CM (i.e., physical abuse, sexual abuse, or neglect) (Widom 1999). A comparison sample matched on demographic and neighborhood characteristics functioned as the counterfactual control group. Participants were first interviewed about their mental health in the early 1990s, approximately 20 years after the documented CM. In an overall analysis with 73% of participants retained (Horwitz et al. 2001), the team concluded, after controlling for alternative sources of stressful life events, that CM had little effect on adulthood mental health. Although the subsample of CSA (n = 96) was not disaggregated from other types of maltreatment, several subsequent papers reported effects of CSA for PTSD (Widom 1999), but associations with both BPD (Widom et al. 2009) and major depressive disorder (Widom et al. 2007) were nonsignificant.

In a prospective birth cohort study of 900 participants (Fergusson et al. 2013), self-reported CSA was assessed at ages 18 and 21, and then mental health outcomes were assessed again

at age 30. With 74% of participants retained, results showed that CSA was significantly associated with major depression, anxiety disorder, alcohol and drug dependence, and PTSD even after the authors controlled for demographic factors, family, and other types of CM. In another prospective study (LONGSCAN), 1,354 maltreated and at-risk children were followed longitudinally at 2-year intervals from age 4 to 16, with 72% retained; those with substantiated CSA exhibited higher rates of internalizing in adolescence (Lewis et al. 2016). Although this study did not ascertain psychiatric diagnosis per se, internalizing behavior problems are common adolescent precursors to adult mood disorders.

Personality Disorders

The empirical evidence for the association between CSA and personality disorders is less clear. Either the assessment of personality disorders is altogether lacking in the studies mentioned above or there is mixed evidence. For example, although the umbrella review (Hailes et al. 2019) reported an OR of 2.9 for BPD, this result was based on one meta-analysis conducted in 1999 (Fossati et al. 1999) and was judged by the original authors to be of poor quality. In fact, the original authors of the meta-analysis (Fossati et al. 1999) concluded that CSA is not a causal antecedent of BPD. Moreover, Widom and colleagues (2009) explicitly found that CSA is not independently associated with BPD. However, in a more recent systematic review of 40 studies (de Aquino Ferreira et al. 2018) of convenience samples and clinical studies, CSA was shown to be an important predictor of BPD: 40–71% of inpatients reported CSA and CSA severity associated with more severe clinical presentations and poorer prognosis of BPD (Lieb et al. 2004).

Because of the high comorbidity of PTSD and BPD, their overlapping symptoms, and the high representation of CSA as an etiology of each, the concept of complex PTSD (CPTSD) was proposed as a distinct syndrome to capture the wide array of symptoms observed in survivors of prolonged and repeated CSA (Herman 2012). Although several studies of CPTSD have demonstrated a clear link between CPTSD and CSA, and CPTSD is now listed in the *International Classification of Diseases for Mortality and Morbidity Statistics* (ICD), it is not its own diagnostic category in the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorder*s (DSM-5), and considerable debate persists regarding its distinctiveness from PTSD and BPD. A recent latent class analysis of PTSD, BPD, and CPTSD symptomology was conducted on a subsample of 956 sexual assault survivors drawn from the National Epidemiologic Survey on Alcohol and Related Conditions (Frost et al. 2020). Results showed a class compiled of CPTSD symptoms that was distinct from classes defining PTSD and BPD. However, CSA was equally represented in all classes, and thus it may not constitute a distinct etiology for CPTSD.

Psychosis, Suicidality, and Eating Disorders

In a national psychiatric survey of 7,353 adults, psychosis was strongly linked to a history of CSA (OR = 10.14); however, other forms of adversity and trauma were not adjusted (Bebbington et al. 2011). In a more recent meta-analysis of studies of adults with psychotic disorders, CSA was associated with psychosis but not significantly more so than other types of victimization in terms of effect sizes (de Vries et al. 2019). A recent meta-analysis of 47 studies (N = 151,476) reported a pooled OR of 1.89 for the link between CSA and suicide

attempts (Ng et al. 2018), but again, the authors concluded that robust controls of potential confounds were not systematic across studies, and thus no adjustment for other trauma or adversity was possible. In terms of eating disorders, a 2016 meta-analysis of 1,714 studies and more than 14,000 individuals reported a pooled OR for the unique effect for CSA equal to 2.73 for bulimia nervosa and 2.31 for binge eating disorder (Caslini et al. 2016). However, the OR for CSA and anorexia was nonsignificant after adjustment for publication bias. In the FGDS prospective cohort study, although subthreshold eating disorder symptoms were predictive of subsequent health problems, female CSA survivors did not exhibit diagnostic levels of either anorexia or bulimia in young adulthood (Li et al. 2018).

Sexual Dysfunction

Sexual dysfunction has not been consistently assessed in either population-based or cohort studies, perhaps because DSM-5 requires symptoms to be accompanied by "clinically significant distress," which may be difficult to discern in a nonclinical setting. Moreover, there has been debate over the distinction between sexual desire and subjective arousal, as well as over the quantification of sexual pain, which can affect operational definitions. However, in a recent systematic review, women with a history of CSA reported significantly higher rates of sexual dysfunction than their nonabused counterparts in well-controlled studies (Pulverman et al. 2018). Although symptoms such as flashbacks during sex, pain during sex, sexual aversion, and avoidance were reported, difficulties with arousal, desire, and orgasm were by far the most common sources of sexual dysfunction.

Summary

Overall, there is evidence that CSA is linked with psychiatric disorders, but the evidence for it being a unique etiology (i.e., the effect of CSA is maintained when other forms of trauma and adversity are controlled analytically) is stronger for some disorders than for others. There is strong evidence for CSA being a unique risk factor for mood disorders, anxiety disorders, PTSD, alcohol and substance use disorders, and sexual dysfunction. The evidence is less strong for personality disorders, and there is little systematic evidence for CSA being a unique predictor of psychosis and eating disorders. While there is certainly good evidence for an association, additional longitudinal research, in which confounds are systematically controlled, is needed before strong causal inferences can be drawn regarding the connection between CSA and psychiatric disorders.

MECHANISMS

This section is devoted to the plausible transdiagnostic mechanisms that explain why CSA might confer unique risk for the development of psychopathology. Several categories of mechanisms are evaluated: (*a*) those hypothesized to be associated with the distinctive features of CSA because of its explicitly sexual nature (i.e., traumatic sexualization); (*b*) products of the extreme and chronic stress (i.e., the biological embedding of stress) inherent in CSA but also observed in other forms of severe trauma and adversity; and (*c*) those that may be shared with other forms of trauma or adversity but that are otherwise highly prominent in the lives of CSA survivors, in part because of *a* and *b*. For mechanisms and their manifestations to be included in this section, two conditions were required; each had to

be theoretically and empirically associated with psychopathology, and, whenever discernibly possible, each had to be empirically evaluated as uniquely related to CSA with plausible confounds controlled.

Traumatic Sexualization

In their seminal writings, Judith Herman (1981) and Dianna Russell (1986) summarized, through clinical case studies and both qualitative and quantitative observational research, how and why CSA might differ from other forms of trauma and adversity. For example, the sexual violation of a child can fundamentally interfere with the shaping of sexual attitudes, beliefs, and norms in ways that can contribute to dysfunction and distortions such that the entire landscape of sexual development is disrupted. Such traumatic sexualization can contribute to misconceptions and inappropriate repertoires of sexual thoughts and behaviors, thus generating confusion about sexual health, efficacy, and agency as well as distorted emotional associations with sexual discourse, actions, and relationships. Since it is often shrouded in secrecy and perpetrated under the auspices of love and protection, CSA can result in profound feelings of shame and extreme violations of trust. When CSA is perpetrated in the absence of physical force or violence—which is often the case with prolonged grooming—survivors may have difficulty placing blame on the perpetrator and thereby engage in self-blame. When paired with violence or threats of violence, CSA can normalize involvement with exploitive or violent sex partners and can contribute to sexual harm and revictimization (Barnes et al. 2009).

Although this early work revolved mainly around father–daughter incest, Finkelhor and colleagues organized it, along with their observations from other forms of intra- and extrafamilial studies, into a useful conceptual framework of the unique features of CSA—namely, the Traumagenic Dynamics Model (Finkelhor & Browne 1985). This model, which has not been appreciably expanded upon since its introduction in 1985, explicates how the betrayal through sexual manipulation and breaking of trust bonds, the powerlessness of exploitation and body violations, and the stigmatization associated with sexual deviance and taboo all work to further compound the egregiousness of traumatic sexualization. The cognitive and affective distortions that develop from traumatic sexualization have been linked to sexual “self-schemas”—that is, attitudes about the self as a sexual being that affect the processing of sexually relevant cues and inform sexual behaviors (Meston et al. 2006). Damaged or impaired schemas can manifest in nonnormative sexual behaviors and outcomes that, although not necessarily considered psychopathology, can alter life circumstances, disrupt health trajectories, and increase the risk for victimization and dysfunction. Indeed, there is empirical evidence that CSA survivors subsequently experience manifestations of traumatic sexualization in terms of problematic sexual behaviors and cognitions—some in the form of heightened sexual behaviors or promiscuity, others in the form of sexual aversion or avoidance, and still others in the form of sexual ambivalence or the simultaneous pairing of sexual aversion with sexual preoccupation. What follows is a review of literature on outcome manifestations of traumatic sexualization that are not necessarily psychopathological in nature but are sex-specific outcomes of considerable public health concern (e.g., teen pregnancy, prostitution, sexual revictimization, and sexual

offending) and are associated with, or may have a bearing on the development of, mental health disorders.

Heightened sexual activity.—Several well-controlled studies have documented sex-specific outcomes associated with heightened sexual activity in CSA survivors. For example, intrusive sexual thoughts, the use of sexuality in relationships, and guilt associated with sex were found to be more potent mediators in the relationship between CSA and risky sexual behaviors above and beyond information, motivation, and behaviors among 481 females being treated for sexually transmitted infections (Senn et al. 2012). A recent meta-analysis of eight studies of men and women (N = 38,989) showed that substantiated CSA was associated with subsequent risky sexual behaviors in females (OR = 2.72) and in males (OR = 1.69) (Abajobir et al. 2017). In the FGDS study, CSA was associated with subsequent sexual preoccupation (e.g., in the form of pornography consumption, excessive masturbation, and an overactive sexual fantasy life) even when the investigators controlled for prior depression, anxiety, dissociation, and sexual behavior problems (Noll et al. 2003). Moreover, female CSA survivors were almost 2 years younger at their first voluntary intercourse than were nonabused females. Widom & Widom (2009) showed that CSA was associated with sexually transmitted infections (OR = 1.94). This team also reported a significant link between CSA and subsequent prostitution (OR = 2.38), but this relationship was fully mediated by early sexual contact (OR = 2.17) (Wilson & Widom 2008). Finally, CSA is a strong and important precursor to teenage pregnancy for females. A meta-analysis of 21 studies linking CSA to teenage pregnancy reported an aggregate AOR of 2.21 and found that 4.5 out of every 10 pregnant adolescents reported a history of CSA (Noll et al. 2009). In a more recent prospective cohort study of 514 females tracked longitudinally through adolescence, CSA was found to be uniquely associated with teenage motherhood after the authors controlled for a host of confounds including up to 18 alternative forms of maltreatment, trauma, and adversity (Noll et al. 2019).

Sexual avoidance or aversion.—In a recent review, the lack of positive emotions related to sex (rather than the presence of negative associations) was cited as the most common mechanism explaining the relationship between CSA and sexual dysfunction (Pulverman et al. 2018). In a prospective longitudinal study of 160 adolescents with confirmed CSA, negative associations with sexual activities stemming from the shame and self-blame associated with being abused predicted greater sexual difficulties 6 years later (Feiring et al. 2009). Women with a history of CSA have also been shown to experience sexual revictimization rates two to three times higher than those of their nonabused peers (Barnes et al. 2009, Roodman & Clum 2001); such findings suggest that negative associations with sex might be continually reinforced as a result of continued sexual revictimization experiences into late adolescence and early adulthood. In an imaging study, Heim and colleagues (2013) found a thinning of the genital somatosensory cortex representing the clitoris and surrounding genital area in women with a history of CSA. The authors hypothesized that this cortical thinning could explain how excessively adverse sensory experiences might lead to impaired neurodevelopment and explain why victims of CSA might avoid sexual contact in adulthood and develop sexual dysfunction. As this was a cross-sectional study, the authors conceded that the findings might also be explained by

overall decreases in the frequency of sexual behavior, which in turn may have resulted in cortical thinning.

Sexual ambivalence.—While heightened sexual behaviors and sexual avoidance or dysfunction likely explain the bulk of the multiple sexual development pathways experienced by CSA survivors who suffer traumatic sexualization, a third pathway has been posited. Noll and colleagues (2003) demonstrated that female CSA survivors in the FGDS study reported higher rates of "sexual ambivalence"—the simultaneous pairing of sexual preoccupation with marked attitudes of sexual avoidance and aversion. In this analysis, CSA characterized by early onset, penetration, and perpetration by a biological father without violence was most strongly associated with sexual ambivalence. This seemingly contradictory pairing of compulsions with aversions may develop when feelings of shame, guilt, betrayal, powerlessness, and exploitation are confused with sexual arousal, intimacy, and love. Such individuals may be at increased risk for continued emotional trauma and interpersonal difficulty, which can result in significant cognitive and affective sexual distortions including those consistent with compulsive sexual behavior disorder and documented in sexual offenders (Efrati et al. 2019).

Sexual offending.—Sexual offending is another hypothesized outcome of traumatic sexualization whereby victims display awareness, knowledge, and interest in sexual activity that are inappropriate for their age or that manifest in initiating sexual activity with unwilling peers or replicating their own victimization on younger children. The theory of sexual offending suggests that a complex system of interlocking neuropsychological functions, clinical symptoms, and social learning processes can produce sexually aggressive conduct (Ward & Beech 2006). Further, some compulsive sexual behaviors stemming from early sexual trauma can exacerbate this process and result in sexual aggression and dominance. Empirical evidence for this link is somewhat mixed. While an association between CSA and subsequent sexual offending was shown among 843 treatment-seeking adults, this result was mainly seen in men (30%) versus women (<1%) (Glasser et al. 2001). Widom & Massey (2015) found an AOR of 2.13 (1.94 for males and 2.50 for females), which did not reach significance. Studies of incarcerated populations have shown more consistent results, but again, mainly for men. For example, a study of 576 adult male sexual offenders (Reckdenwald et al. 2013) and data from 2,520 incarcerated male juvenile offenders (DeLisi et al. 2014) showed that CSA was associated with higher rates of sexual offending but not other types of offending such as homicides, property offending, and burglary. Most recently, a study of 865 active correctional clients used probation records and social service records to link CSA to official rape and sexual abuse charges and found that CSA had an incidence rate ratio of 2.91 after the authors controlled for a host of demographic variables and other adverse experiences including other forms of CM and out-of-home placements (Drury et al. 2019).

Sexual abuse severity and characteristics.—The severity of CSA seems to matter, as demonstrated by a population-based sample of twin females in which characteristics including attempted or completed intercourse, perpetration by a family member, the use or threat of force, and disbelief or lack of support at the time of disclosure were associated with

greater risk for deleterious psychiatric outcomes compared with less severe forms of CSA (Bulik et al. 2001). As to the specific or relative contributions of any one characteristic being more salient than others, the research is quite inconsistent. In more recent studies, adolescent (as opposed to childhood) onset and longer durations of CSA were potent predictors of anxiety and PTSD for females in a sample of 1,270 emerging adults (Adams et al. 2018), while younger age at CSA onset was inversely associated with rates of sexual offending in a study of correctional clients (Drury et al. 2019).

Results from a nuanced, person-centered approach showed differential outcomes based on profiles of abuse characteristics among three subgroups: abuse by multiple perpetrators (MPs), over relatively short periods of time, with accompanying violence; abuse by a single perpetrator (SP) who was not the biological father, for short durations, with no violence; and classic incest with abuse by the biological father (BF) over long durations, at young ages, with penetration, and with little violence (Trickett et al. 2001). In longitudinal analyses, the MP group showed initial deleterious outcomes in the short term followed by marked increases in functioning over time, the BF group showed persistent deleterious outcomes across development (including heightened sexual behaviors), and the SP group showed initial resilience followed by "sleeper effects" or decreased functioning over time (Noll et al. 2003). Although some would interpret MP abuse as "severe"and SP abuse as more "mild," these results demonstrate that severity may not necessarily function on a linear continuum but instead might be person-specific according to how characteristics cluster together and fluctuate over time.

The Biological Embedding of Stress

The past decade has seen a marked increase in the recognition that vulnerability to the development of psychopathology is mediated by biological processes. While stress is ultimately an adaptive process, overactivation of the stress-response system—specifically, the hypothalamic–pituitary–adrenal (HPA) axis—is assaultive over time and can lead to dysregulation and pathophysiology. Differential HPA patterns have been linked to internalizing and externalizing behaviors in maltreated children (Cicchetti & Rogosch 2001). Additionally, HPA dysregulation in the form of downregulation—that is, hypocortisolism (low overall diurnal patterns; blunted response to stressor paradigms)—has been implicated in the development of depression and PTSD in adults, potentially due to a failure in the connected neural circuitry implicated in emotional, neuroendocrine, and autonomic control (Heim et al. 2008). Because there have been few longitudinal studies of intraindividual changes in HPA functioning over time following CSA, it is not known whether CSA, specifically, confers risk for HPA dysfunction. However, the FGDS prospective study assessed cortisol levels over six time points across development and showed high circulating morning cortisol concentrations in childhood followed by a marked hyposecretion in adulthood approximately 15 years later, even after the authors controlled for other types of trauma, demographics, and time-varying anxiety and depression (Trickett et al. 2010).

This downregulation of the HPA is thought to be evidence of the biological embedding of stress, whereby chronic activation of the stress-response system becomes sufficiently embedded such that alterations in multiple biological and developmental processes

promote disease processes and illness (McEwen 2012). The stress associated with CSA, for example, can lead to acute increases of glucocorticoid secretion and activation of glucocorticoid receptors. In genetically vulnerable children, this activation can induce epigenetic modifications of stress-regulatory genes, including the glucocorticoid receptor gene, leading to subsequent glucocorticoid resistance. This glucocorticoid resistance can lead to disinhibition of further stress responses, resulting in increased HPA and autonomic reactivity, which promote activation of proinflammatory pathways. Increased glucocorticoid levels and inflammation can result in structural damage in the brain and functional changes on neural networks implicated in the regulation of stress and emotion, thus promoting further disinhibition of the stress responses and maladaptation. In this example, the fact that stress "gets under the skin" is the driving mechanism of pathology, and there is nothing inherently distinct about the experience of CSA—other than the notion that it might be exceedingly stressful—that would set it apart from other forms of chronic, early-life adversity in terms of conferring risk for subsequent maldevelopment.

DeBellis (2001) proposed the theory of Developmental Traumatology whereby the chronic and extreme stress of CM leads to alterations in the body's stress physiology (including HPA dysregulation) that interfere with optimal brain development. This brain maldevelopment contributes to cognitive, affective, and emotional health deficits that lead to behavioral problems, psychosocial impairments, and, ultimately, psychiatric disorders. Taking this concept further, Teicher & Samson (2013) hypothesized that because of its distinct clinical and neurobiological features—namely, physiological and neurohumoral changes that alter brain development trajectories—CM leads to a constellation of "ecophenotypes" that heighten the risk for the phenotypic expression of psychopathology. Owing in part to these advanced theoretical models of how CM confers risk for maldevelopment, the components of biological embedding and their consequences are now recognized as chief neurobiological mechanisms underlying a host of cognitive and emotional processes that contribute to psychiatric disorders (Nemeroff & Binder 2014). What follows is an accounting of the evidence for various aspects of biological embedding being associated with psychopathology and the extent to which there is theoretical and/or empirical justification that CSA poses unique risk in these pathways over and above it being a highly stressful type of early-life adversity.

Brain maldevelopment.—A comprehensive review of 108 imaging studies provided convincing evidence that CM is indeed associated with a host of structural, functional, and connectivity deficits that have direct implications for the development of psychopathology (Teicher et al. 2016). These include structural deficits in the hippocampus, corpus callosum, anterior cingulate, orbitofrontal cortex, and dorsolateral prefrontal cortex as well as functional deficits in the amygdala when detecting emotions and in the striatum with reward anticipation. These brain regions and functional deficits have been specifically implicated in the development of psychiatric disorders such as depression, PTSD, BPD, schizophrenia, antisocial personality disorders, and substance use disorders (e.g., De Bellis et al. 2015, Kumari et al. 2014, Wang et al. 2014). Many of these regions and deficits are also associated with executive functioning, emotion regulation, and social cognition—all of which have

been associated with the development of psychopathology (e.g., Amodio & Frith 2006, Kim & Cicchetti 2010, Minzenberg et al. 2008, Tottenham 2014).

Although there is not clear evidence that CSA confers unique risk for brain maldevelopment, several studies have provided some hypothesis-generating support. For example, Tomoda and colleagues (2009) showed that adults reporting CSA had reduced gray matter volume in the visual cortex and reduced thickness in right lingual, left fusiform, and left middle occipital gyri. Heim and colleagues (2013) showed a cortical thinning in the genital somatosensory cortex in women with self-reported prepubertal CSA, which they hypothesized might help explain the development of sexual dysfunction in some female CSA survivors. Although they did not examine effects for other types of CM or adversity, these studies either excluded females with other forms of trauma or statistically controlled for alternative forms of trauma, lending some credence to the uniqueness of CSA in these findings.

Epigenetic modifications.—In a study of 1,411 adult twin females, Kendler and colleagues (2000) found that CSA was equally predictive of a wide array of psychiatric disorders; on the basis of these findings, the authors posited that CSA may not have any diagnostic specificity per se but instead may act to lower the threshold for expression of psychopathology when there is already a genetic predisposition. Such an assertion implies that CSA is an environmental signal that modifies the state of the genome, leading to changes in gene expression. As shown in animal models and, more recently, in humans (for a review, see O'Donnell & Meaney 2020), variations in the early environment are associated with changes in gene expression and biological functioning that persist into adulthood well beyond the duration of the initial event. Such alterations occur with the addition of a methyl group onto cytosines of the DNA (i.e., DNA methylation), which then becomes an additional layer of information contained in the genome. Epigenetics—the study of genetic modification brought on by a posteriori influences of the environment—suggests that variable methylated sites within regions of the genome that are associated with early-life stress (e.g., glucocorticoid receptor sites) are present in adults who report early adversity and, furthermore, that certain polymorphisms actually moderate the development of psychiatric conditions including PTSD and depression (Binder et al. 2008, Bradley et al. 2008). While some work has expressly focused on CSA (Beach et al. 2010, Perroud et al. 2011), there is currently no hypothesis that CSA results in epigenetic alterations other than those that can be accounted for by the stress of early-life trauma. In fact, one notable paper showed that polymorphisms in the corticotropin-releasing hormone receptor 1 (*CRHR1*) gene explained neuroticism in maltreated children, but the authors did not find this gene–environment interaction to be present for the subset of the sample who had experienced CSA (DeYoung et al. 2011).

Pubertal timing.—Another important aspect of biological embedding is the interaction between the HPA and hypothalamic–pituitary–gonadal (HPG) axes. Circulating gonadal steroids associated with pubertal development, especially estrogen, are important modulators of the HPA axis and may provide a potential route for estrogen-mediated increases in glucocorticoid levels that can influence the rate of growth and timing of sexual

maturation (Young 1995). Using the gold standard of direct observational Tanner staging, one longitudinal study of maltreated females showed that attenuated HPA functioning was predictive of accelerated pubertal development (Negriff et al. 2015); however, no specific associations or hypotheses were generated that would suggest a unique effect of CSA on pubertal timing. Herman-Giddens and colleagues (1988) were the first to suggest that CSA patients enter puberty earlier than other females; their hypotheses were based on clinical observations of pubic hair and breast development before the age of 8 years. Theories began to proliferate about potential pathways, including the following:

1. Environmental instability, such as the absence of a biological father (which is the case with many CSA survivors postdisclosure), signals accelerated maturational development as an adaptive response to maximize reproduction opportunities (Belsky et al. 1991).
2. Obesity, which has also been shown to be related to early adversities (Danese & Tan 2014), is implicated in early pubertal onset due to adiposity-related endocrine mechanisms that stimulate enzymes necessary for the production of adrenal androgens (Kaplowitz 2008).
3. Early maturation could signal sexual maturity, thus increasing vulnerability for being targeted for CSA (James et al. 2012).

Perhaps the most definitive analysis of pubertal timing in CSA survivors is the FGDS study, in which Tanner staging was continually assessed across adolescence (Noll et al. 2017). Results showed that CSA was associated with earlier pubertal onset: 8 months earlier for breasts (AOR = 3.06) and 12 months earlier for pubic hair (AOR = 3.49). Because pre-, peri-, and postpubertal onset of CSA was modeled in these analyses, and because obesity status and biological father absence were dynamically controlled in cumulative logit models, the authors concluded that these alternative explanations were less plausible than that of the HPA–HPG interaction. Hence, the stress associated with CSA was shown to be the most plausible explanation for early pubertal timing in this analysis.

Early pubertal timing has been implicated in the development of psychopathology owing to the risk conferred on adolescents whose physical appearance outpaces their psychological, cognitive, and social maturity. These adolescents are more likely to engage in age-inappropriate risk-taking, affiliate with delinquent peers, and become targets of bullying and exploitation (Ullsperger & Nikolas 2017). Early puberty is also associated with HPA dysregulation (Natsuaki et al. 2009) and altered neural responses to emotional stimuli (Whittle et al. 2015), which can set the stage for psychopathology. Although there appear to be sensitive periods of neurobiological development (including puberty) whereby exposure to hormones interacts with brain development that might influence environmental sensitivity and risk for mental health problems (Byrne et al. 2017), the specific mechanisms by which HPG axis activity, or the psychosocial sequelae of early pubertal timing, increases the risk of psychopathology are largely speculative. However, one notable analysis of 4,937 adolescent females in a nationally representative cohort showed that earlier age at menarche mediated the association between early-life threat and postmenarchal fear, distress, and externalizing behavior problems (Colich et al. 2020). The authors of that study put forth early menarche as a potent transdiagnostic mechanism in the development of psychopathology.

Inflammation.—Another key aspect of biological embedding is the interaction between the neuroendocrine and autonomic nervous systems with the immune system. Noradrenergic input stimulates inflammatory responses (Raison et al. 2006), and several inflammatory markers—including proinflammatory cytokines, such as interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6), and acute-phase proteins, such as haptoglobin, fibrinogen, and C-reactive protein (CRP)—are multifunctional signaling molecules of the immune system that act as key mediators in both central and peripheral inflammation. Neuroendocrine findings (i.e., heightened autonomic stress reactivity coupled with glucocorticoid receptor resistance) are evidence of proinflammatory states that accelerate pathophysiological processes in individuals experiencing early-life stress (Baumeister et al. 2016). In addition to a host of physical health consequences, these inflammatory processes have been linked to psychopathology as shown in meta-analyses of depression (Howren et al. 2009), BPD (Dargél et al. 2015), psychosis (Miller et al. 2011), and PTSD (Passos et al. 2015).

As summarized in a recent review, stress-associated glucocorticoid resistance, together with autonomic stimulation, can promote activation of inflammatory pathways, which ultimately leads to structural damage in the brain and functional changes in connected neural networks implicated in the further regulation of stress and emotion (Danese & Baldwin 2017). However, the authors were careful to point out that there is only indirect evidence for the connection between childhood trauma and psychopathology through immune system pathways due to plausible bidirectional associations—for instance, depressive symptoms such as distress could elevate the stress-response system, thereby increasing inflammation, and vice versa. Moreover, there are shared genetic vulnerabilities to both inflammation and psychiatric diagnoses, and early expressions of these conditions, including emotion dysregulation and externalizing behavior problems, may predispose risk for CM. While longitudinal studies are clearly needed, a meta-analysis of 25 studies of over 16,000 individuals clearly established a link between maltreatment in childhood and CRP, IL-6, and TNF-α in adulthood (Baumeister et al. 2016). Again, it should be noted that there are currently no hypotheses that CSA confers unique risk for inflammation over and above the notion that it may be inherently more stressful than other forms of maltreatment. In fact, CSA-specific findings were scant in this meta-analysis, with only TNF-α being significantly associated with CSA, a trend toward significance with IL-6, and no results for CRP.

Emotion Dysregulation

Disruptions in emotion regulation, including emotional reactivity, maladaptive cognitive and behavioral responses to distress, rumination, and impulsive behaviors, have been implicated in the development of psychopathology for maltreated children (McLaughlin et al. 2015). Although the pathways are not altogether clear, neuroimaging studies have linked CM to heightened emotional reactivity, amygdala responses to negative emotional stimuli, disengagement from regulatory processes via atypical prefrontal-amygdala circuitry, and antisocial behaviors associated with cortical thickness in the left parahippocampal gyrus (e.g., Busso et al. 2017, Gee et al. 2013, Peverill et al. 2019). Moreover, at-risk environments can be conducive to maltreated children adopting ruminative coping strategies and/or the modeling of ineffective emotional responses by caregivers (e.g., impulsiveness), leading

to depression, externalizing behaviors, substance use, eating disorders, conduct problems, and anxiety disorders (e.g., McLaughlin et al. 2014). Longitudinal modeling has been instrumental in understanding emotion regulation as a transdiagnostic mechanism for the development of psychopathology for maltreated children. For example, in a sample of 439 adolescents in a community-based cohort study followed prospectively for 5 years, emotional reactivity and maladaptive responses to distress mediated the association between CM and both internalizing and externalizing psychopathology (Heleniak et al. 2016). However, those with CSA scored only slightly higher than those with other forms of CM on measures of emotional reactivity but did not differ in their cognitive or behavioral responses to distress; this finding suggests that CSA does not pose appreciably greater risk in these mediational processes.

Avoidance

An empirical review of 39 studies of adults coping with CSA showed that survivors employ increased use of avoidant coping strategies both in the immediate aftermath and over time (Walsh et al. 2010). As confirmed in a recent meta-analysis of 212 studies (80,850 participants), avoidant coping strategies such as disengagement, emotional suppression, and denial were associated with concurrent and subsequent psychopathology (Compas et al. 2017). Experiential avoidance—or efforts to suppress and avoid painful or aversive stimuli such as thoughts, emotions, memories, and physiological arousal associated with trauma (Hayes et al. 1996)—is a maladaptive coping strategy associated with the development and severity of PTSD (Cameron et al. 2010) and BPD (Neacsiu et al. 2014). When tested in a multiple-mediator model along with autonomic nervous system and HPA dysregulation, experiential avoidance was the more potent predictor of subsequent PTSD symptoms in maltreated children and adolescents (Shenk et al. 2014). Even as maladaptive coping has been shown to be an important predictor of subsequent psychological distress in CSA survivors (Rosenthal et al. 2005), there is no specific working hypothesis that CSA confers unique risk over and above what would be expected for other forms of CM or other traumas.

Insecure Attachment

The danger, violation of trust, and betrayal associated with CSA can be especially disruptive to internal working models of attachment characterized by insecurity, and this disruption can enhance the use of self-protection strategies and induce subsequent interpersonal and relational difficulties (Alexander 1992). Indeed, insecure attachment is prominent in children with CSA (Ensink et al. 2020) and has been shown to moderate the association between CSA and behavior problems in preschoolers (Charest et al. 2019), trauma symptoms in adolescent psychiatric patients (Jardin et al. 2017), and psychiatric symptoms in adulthood (Lind et al. 2018). Meta-analyses have shown that, in general, children with insecure and disorganized attachment have greater risk for concurrent and later internalizing and externalizing difficulties (Fearon et al. 2010, Groh et al. 2012) and psychiatric disorders in adulthood (Raby et al. 2018). Disruptions in internal working models of attachment can extend into the next generation such that mothers with a CSA history generalize attachment insecurity to relationships formed with their own children (Kwako et al. 2010).

The mechanisms from insecure attachment to the development of psychopathology are not well understood; however, pathways through emotion dysregulation (Alink et al. 2009) and social cognition (Venta et al. 2017) have been shown in maltreated children and adolescents. Although such experiences could be present with other forms of maltreatment and adversity (e.g., child abandonment), the violation, victimization, and betrayal by a caregiver, particularly when coupled with a lack of responsiveness and support from others at the time of CSA disclosure, are thought to sufficiently erode trust, compound the assault on working models of attachment, and thus exacerbate psychopathology outcomes (McTavish et al. 2019b).

DISCUSSION

The Compounded Convergence of Mechanisms Model of Child Sexual Abuse

Meta-analytic studies, well-controlled population cohort studies, and prospective, longitudinal studies have provided strong evidence, after accounting for confounds and other types of trauma and adversity, that CSA accounts for unique variation of several forms of psychopathology (mood, anxiety, substance use disorders, and PTSD) as well as additional sex-specific sequelae (teenage childbearing, sexual revictimization, and sexual dysfunction). Such findings support the notion that CSA is a potent and unique risk factor in the development of these problematic outcomes. The Compounded Convergence of Mechanisms (CCM) model depicted in Figure 1 organizes this evidence by depicting how and why CSA should be considered unique. First, there are some mechanisms, such as traumatic sexualization, that are more likely than others to be uniquely attributable to CSA given the sexually explicit nature of the trauma; the betrayal, powerlessness, and stigma associated with sexual violations; and the disrupted sexual self-schemas and sexual ambivalence that can ensue. Second, CSA survivors have a high likelihood of experiencing additional risk factors, including insecure attachment, avoidance, emotion dysregulation, and the biological embedding of stress, which have been shown to be mechanistic of psychopathology in CSA survivors as well as in those who suffer from other forms of CM, traumatic experiences, and early-life adversity. Third, the model depicts how the presence of any single mechanism confers relatively low risk for problematic outcomes, whereas risk increases with their co-occurrence and overlap. The convergence of all mechanisms compounds risk to explain why CSA is a common etiological precursor for such a large array of both sex-specific and psychiatric outcomes.

Methodological Considerations for Future Research

Care has been taken in this review to interpret literature in terms of the extent to which the research methodologies adequately isolated the unique effects of CSA through exacting control over variability in outcomes attributable to other forms of maltreatment, trauma, and adversity. Such methodologies were not uniform, varied widely in terms of rigor, or were completely absent. As such, there are likely additional analyses (or reanalyses) that could further inform these findings, the mechanisms included in the CCM model, and the evidence base for outcomes. Failing to disaggregate effects by type—in studies of ACEs and CM, for example—will further stymie any scientific advancement in this area of inquiry.

Of course, types of trauma and adversity can co-occur within the same family or individual, and parsing the unique effect of one versus another is exceedingly difficult. However, there have been some new and noble attempts to make sense of this overlap. A set of elegant analyses examined the synergistic effects of CSA in the study of ACEs (F.W. Putnam et al. 2020, K.T. Putnam et al. 2013). Synergy refers to a quantitative interaction such that the additive proportion (AP) of variance accounted for by the combined effect is significantly greater than the sum (or the product) of the individual effects. Based on a subsample from the National Comorbidity Survey-Replication (NCS-R; $n = 5{,}692$), results showed that, when predicting multiple psychiatric diagnoses, CSA was the most common synergistic ACE, especially for women with APs of 1.72 when in combination with domestic violence, 1.69 with the absence of a parent, 2.30 with being a victim of a crime, and 1.62 with economic hardship (Putnam et al. 2013). A similar analysis predicted clinical levels of internalizing and externalizing behaviors in a sample of 10,355 clinic-referred youth in the National Child Traumatic Stress Network data set. Results showed that CSA was synergistic with 16 ACEs, and the top four APs were 1.61 with physical abuse, 1.53 with domestic violence, 1.51 with emotional abuse, and 1.31 with neglect (Putnam et al. 2020). Taken together, these results demonstrated that CSA has an interactive and additive effect in the prediction of psychopathology over and above other ACEs. This set of analyses also has underscored how the use of synergy variables—or the inclusion of CSA within specified aggregate combinations, as well as variables such as “any CSA”—will advance the field in ways that ACE dose-effect analyses and studies that fail to disaggregate the effects of CSA will not.

If nonstrategic, aggregate analyses continue, the ability to distinguish between equifinality and multifinality will be further hampered, and the field runs the risk of developing (or perpetuating the use of) prevention and treatment tools that, although sharp enough to chisel away at commonalities, may be too blunt to affect any one outcome or transdiagnostic mechanism with precision. The momentum behind aggregate and dose-effect analyses has resulted in mountains of studies that simply place all adversities into a single bucket labeled “trauma,” “adversity,” “maltreatment,” or “toxic stress.” The perpetuation of this type of aggregate research is nothing short of concept creep—the notion that concepts lose their meaning when definitions are greatly expanded to encompass a broad variety of phenomena that are qualitatively different from one another and span a wide range of severity (Haslam 2016). Overly expanded and too-inclusive definitions, although perhaps well intentioned, can hamper the development of novel interventions designed to target mechanisms that act uniquely on specific phenomena.

Any disaggregation or sufficient control of confounds necessarily requires adequate, reliable assessments of trauma and adversity. The chief methods for doing so include objective verification through official records or subjective (usually retrospective) self-reports. There are problems with both approaches—objective methods will omit subthreshold or unreported CSA; subjective reports are vulnerable to both under- and overreporting—and outcomes can vary accordingly. In Widom and colleagues’ study in which both court records and subjective reports were obtained, the agreement between the two was quite low for CSA ($\kappa = 0.17$) (Widom 1999). Moreover, subjective methods yielded much higher rates of psychopathology compared with sole reliance on objective records (Danese & Widom

2020). The agreement level was substantially lower than that reported in the FGDS prospective study, in which the false-negative rate was only 10.11% and intraclass kappa coefficients were remarkably high for agreement with case records obtained 15 years prior: κ= 0.70 for severity (penetration versus genital contact); κ= 0.85 for age at onset; κ= 0.87 for age at offset; and κ = 1.0 for perpetrator identity (Barnes et al. 2009). Reasons for discrepancies across studies concerning the accuracy of CSA self-reports are unknown; however, the assessment methods used (intensive semistructured interviews versus questionnaires) and the frequency of face-to-face assessment points (more assessment points can build trust and familiarity) might play a role. What is clear is that both methods should be accomplished in any well-designed study, as demonstrated in a prospective study of 514 adolescents where a multimethod approach, which included both child welfare and self-report methods, increased the AOR from 1.66 to 2.21 for teenage births and from 1.28 to 2.95 for subsequent major depression (Shenk et al. 2016). This paper was also useful in illustrating how "contamination"—the occurrence of CSA in counterfactual conditions—can substantially truncate effect sizes. Because this type of contamination is likely present in most studies of CM, the effects of CSA most assuredly have been underestimated in the myriad of studies where contamination was not assessed, underdetected because of the exclusion of either the subjective or objective assessments, or uncontrolled in analyses. Finally, although there have been notable works that examined gender effects (e.g., Cougle et al. 2010, Romano & De Luca 2001), there is a dearth of information regarding differences in outcomes or mechanisms for males versus females.

CONCLUSIONS: PREVENTION, TREATMENT, AND POLICY

This review has highlighted the ways in which CSA does and does not account for unique variation in problematic outcomes. Strong evidence has been found for PTSD, as well as mood, anxiety, and substance use disorders, with mixed evidence for personality disorders. A number of additional sex-specific outcomes also have been evaluated, with strong evidence for teenage childbearing, sexual revictimization, and sexual dysfunction and mixed evidence for risky sexual behaviors and sexual offending. CSA has a noxious impact in terms of traumatic sexualization due to its sexually explicit nature, and survivors are also at risk for a host of conditions that have been shown to be mechanistic of psychopathology in studies of other traumas, CM, and early-life adversity. These additional mechanisms include insecure attachment, emotion dysregulation, avoidance, and the biological embedding of stress. Traumatic sexualization, coupled with the convergence of all of these mechanisms, is compounded for CSA survivors, who are thus at inordinate risk for these deleterious outcomes.

Unlike interventions for other forms of CM such as physical abuse and neglect, in which parenting skills are imparted to those at risk for perpetration, the primary prevention of CSA requires psychoeducation and inoculation efforts—including raising awareness, challenging social norms, teaching self-protection skills, and increasing knowledge about how to recognize signs and report suspected CSA—to be delivered across several levels and modalities. For example, these principles are included in age-appropriate delivery to school-aged children (Pulido et al. 2015), delivered to adults in general community settings (Rheingold et al. 2015), and, more recently, developed as indicated prevention efforts

targeted to at-risk households where parents have been referred for services within the child welfare system (Guastaferro et al. 2020). Other promising strategies involve intervention with adolescents or adults who have an attraction to young children (Beier 2018, Letourneau et al. 2017).

By far the most widely researched and lauded (McTavish et al. 2019a) treatment for CSA is trauma-focused cognitive behavioral therapy (TF-CBT) (Cohen et al. 2005). Originally developed to target posttraumatic stress symptoms, TF-CBT involves psychoeducation, coping skills, gradual exposure, cognitive restructuring, and safety skills training. There are many additional evidence-based treatment models that reduce trauma symptoms associated with CSA and/or operate on one or more of the transdiagnostic mechanisms linking CSA with psychopathology, including dialectical behavior therapy (Neacsiu et al. 2014), cognitive processing therapy (Rosner et al. 2019), prolonged exposure (Foa et al. 2013), attachment and biobehavioral catch-up (Zajac et al. 2020), interpersonal psychotherapy–trauma (Duberstein et al. 2018), acceptance and commitment therapy (Hayes 2004), and CBT-based sexual risk-reduction therapy focused on reducing traumatic sexualization (Senn et al. 2017).

Despite the wide array of options and the advanced state of the evidence base, surprisingly few CSA survivors receive and complete treatment. One study of 1,357 CSA patients in an urban hospital setting reported that only 36% were referred for evidence-based treatments, 19% were successfully linked, and 7% completed treatment (McPherson et al. 2012). Common barriers to treatment and completion include limited treatment availability, access obstacles, cost, the demands of the treatment, perceived relevance, and therapeutic alliance (de Haan et al. 2013). To improve access and response rates, it will be increasingly important to maximize the pool of trained providers and to ensure that evidence-based treatments are optimized for efficiency and cost-effectiveness (Brown et al. 2017).

The child advocacy center model, which includes the formation and use of multidisciplinary investigative teams on which child advocates, social workers, and law enforcement personnel work closely with mental health providers to ensure evidence-based treatment referrals, has been shown to result in better outcomes for survivors (Nwogu et al. 2016). The federal Victims of Crime Act established the Crime Victims Fund—primarily comprising federal criminal fines and forfeiture of profits from criminal activity—to assist and compensate survivors and to provide grants to states that fund victim services agencies, compensate providers of evidence-based trauma treatments, and remunerate crime-related losses (including counseling costs) for survivors. The Violence Against Women Act (S.2843), the Trauma-Informed Care for Children and Families Act of 2017 (S.774/H.R.1757), and the SUPPORT for Patients and Communities Act of 2018 (Pub. L. 115–271) are additional federal policies designed to bolster resources and services for CSA survivors, the continued support and reauthorization of which will substantially increase access for, and break down barriers to, evidence-based mental health services.

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LITERATURE CITED

Abajobir AA, Kisely S, Maravilla JC, Williams G, Najman JM. 2017. Gender differences in the association between childhood sexual abuse and risky sexual behaviours: a systematic review and meta-analysis. Child Abuse Negl. 63:249–60 [PubMed: 27908449]

Adams J, Mrug S, Knight DC. 2018. Characteristics of child physical and sexual abuse as predictors of psychopathology. Child Abuse Negl. 86:167–77 [PubMed: 30308347]

Alexander PC. 1992. Application of attachment theory to the study of sexual abuse. J. Consult. Clin. Psychol 60:185–95 [PubMed: 1592947]

Alink LR, Cicchetti D, Kim J, Rogosch FA. 2009. Mediating and moderating processes in the relation between maltreatment and psychopathology: mother-child relationship quality and emotion regulation. J. Abnorm. Child Psychol 37:831–43 [PubMed: 19301118]

Amodio DM, Frith CD. 2006. Meeting of minds: the medial frontal cortex and social cognition. Nat. Rev. Neurosci 7:268–77 [PubMed: 16552413]

Anda RF, Porter LE, Brown DW. 2020. Inside the Adverse Childhood Experience score: strengths, limitations, and misapplications. Am. J. Prev. Med 59:293–95 [PubMed: 32222260]

Baldwin JR, Reuben A, Newbury JB, Danese A. 2019. Agreement between prospective and retrospective measures of childhood maltreatment: a systematic review and meta-analysis. JAMA Psychiatry 76:584–93 [PubMed: 30892562]

Barnes JE, Noll JG, Putnam FW, Trickett PK. 2009. Sexual and physical revictimization among victims of severe childhood sexual abuse. Child Abuse Negl 33:412–20 [PubMed: 19596434]

Barth J, Bermetz L, Heim E, Trelle S, Tonia T. 2013. The current prevalence of child sexual abuse worldwide: a systematic review and meta-analysis. Int. J. Public Health 58:469–83 [PubMed: 23178922]

Baumeister D, Akhtar R, Ciufolini S, Pariante CM, Mondelli V. 2016. Childhood trauma and adulthood inflammation: a meta-analysis of peripheral C-reactive protein, interleukin-6 and tumour necrosis factor-alpha. Mol. Psychiatry 21:642–49 [PubMed: 26033244]

Beach SRH, Brody GH, Todorov AA, Gunter TD, Philibert RA. 2010. Methylation at *SLC6A4* is linked to family history of child abuse: an examination of the Iowa Adoptee sample. Am. J. Med. Genet. B Neuropsychiatr. Genet 153B:710–13 [PubMed: 19739105]

Bebbington P, Jonas S, Kuipers E, King M, Cooper C, et al. 2011. Childhood sexual abuse and psychosis: data from a cross-sectional national psychiatric survey in England. Br. J. Psychiatry 199:29–37 [PubMed: 21508437]

Beier KM. 2018. Preventing child sexual abuse—the Prevention Project Dunkelfeld. J. Sex. Med 15:1065–66 [PubMed: 29861360]

Belsky J, Steinberg L, Draper P. 1991. Childhood experience, interpersonal development, and reproductive strategy: an evolutionary theory of socialization. Child Dev 62:647–70 [PubMed: 1935336]

Binder EB, Bradley RG, Liu W, Epstein MP, Deveau TC, et al. 2008. Association of *FKBP5* polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. JAMA 299:1291–305 [PubMed: 18349090]

Bradley RG, Binder EB, Epstein MP, Tang Y, Nair HP, et al. 2008. Influence of child abuse on adult depression: moderation by the corticotropin-releasing hormone receptor gene. Arch. Gen. Psychiatry 65:190–200 [PubMed: 18250257]

Brown CH, Curran G, Palinkas LA, Aarons GA, Wells KB, et al. . 2017. An overview of research and evaluation designs for dissemination and implementation. Annu. Rev. Public Health 38:1–22 [PubMed: 28384085]

Bulik CM, Prescott CA, Kendler KS. 2001. Features of childhood sexual abuse and the development of psychiatric and substance use disorders. Br. J. Psychiatry 179:444–49 [PubMed: 11689403]

Busso DS, McLaughlin KA, Brueck S, Peverill M, Gold AL, Sheridan MA. 2017. Child abuse, neural structure, and adolescent psychopathology: a longitudinal study. J. Am. Acad. Child Adolesc. Psychiatry 56:321–28.e1 [PubMed: 28335876]

Byrne ML, Whittle S, Vijayakumar N, Dennison M, Simmons JG, Allen NB. 2017. A systematic review of adrenarche as a sensitive period in neurobiological development and mental health. Dev. Cogn. Neurosci 25:12–28 [PubMed: 28077245]

Cameron A, Palm K, Follette V. 2010. Reaction to stressful life events: What predicts symptom severity? J. Anxiety Disord 24:645–49 [PubMed: 20466515]

Caslini M, Bartoli F, Crocamo C, Dakanalis A, Clerici M, Carrà G. 2016. Disentangling the association between child abuse and eating disorders: a systematic review and meta-analysis. Psychosom. Med 78:79–90 [PubMed: 26461853]

Charest F, Hebert M, Bernier A, Langevin R, Miljkovitch R. 2019. Behavior problems in sexually abused preschoolers over a 1-year period: the mediating role of attachment representations. Dev. Psychopathol 31:471–81 [PubMed: 29681251]

Child. Bur. 2020. Child Maltreatment: 2018 Washington, DC: US Dep. Health Hum. Serv., Admin. Child. Youth Fam.

Cicchetti D, Rogosch FA. 1996. Equifinality and multifinality in developmental psychopathology. Dev. Psychopathol 8:597–600

Cicchetti D, Rogosch FA. 2001. The impact of child maltreatment and psychopathology on neuroendocrine functioning. Dev. Psychopathol 13:783–804 [PubMed: 11771908]

Cohen JA, Mannarino AP, Knudsen K. 2005. Treating sexually abused children: 1 year follow-up of a randomized controlled trial. Child Abuse Negl 29:135–45 [PubMed: 15734179]

Colich NL, Platt JM, Keyes KM, Sumner JA, Allen NB, McLaughlin KA. 2020. Earlier age at menarche as a transdiagnostic mechanism linking childhood trauma with multiple forms of psychopathology in adolescent girls. Psychol. Med 50:1090–98 [PubMed: 31020943]

Compas BE, Jaser SS, Bettis AH, Watson KH, Gruhn MA, et al. 2017. Coping, emotion regulation, and psychopathology in childhood and adolescence: a meta-analysis and narrative review. Psychol. Bull 143:939–91 [PubMed: 28616996]

Cougle JR, Timpano KR, Sachs-Ericsson N, Keough ME, Riccardi CJ. 2010. Examining the unique relationships between anxiety disorders and childhood physical and sexual abuse in the National Comorbidity Survey-Replication. Psychiatry Res 177:150–55 [PubMed: 20381878]

Danese A, Baldwin JR. 2017. Hidden wounds? Inflammatory links between childhood trauma and psychopathology. Annu. Rev. Psychol 68:517–44 [PubMed: 27575032]

Danese A, Tan M. 2014. Childhood maltreatment and obesity: systematic review and meta-analysis. Mol. Psychiatry 19:544–54 [PubMed: 23689533]

Danese A, Widom CS. 2020. Objective and subjective experiences of child maltreatment and their relationships with psychopathology. Nat. Hum. Behav 4:811–18 [PubMed: 32424258]

Dargél AA, Godin O, Kapczinski F, Kupfer DJ, Leboyer M. 2015. C-reactive protein alterations in bipolar disorder: a meta-analysis. J. Clin. Psychiatry 76:142–50 [PubMed: 25742201]

de Aquino Ferreira LF, Queiroz Pereira FH, Neri Benevides AML, Aguiar Melo MC. 2018. Borderline personality disorder and sexual abuse: a systematic review. Psychiatry Res 262:70–77 [PubMed: 29407572]

De Bellis MD, Hooper SR, Chen SD, Provenzale JM, Boyd BD, et al. 2015. Posterior structural brain volumes differ in maltreated youth with and without chronic posttraumatic stress disorder. Dev. Psychopathol 27:1555–76 [PubMed: 26535944]

de Haan AM, Boon AE, de Jong JT, Hoeve M, Vermeiren RR. 2013. A meta-analytic review on treatment dropout in child and adolescent outpatient mental health care. Clin. Psychol. Rev 33:698–711 [PubMed: 23742782]

de Vries B, van Busschbach JT, van der Stouwe ECD, Aleman A, van Dijk JJM, et al. 2019. Prevalence rate and risk factors of victimization in adult patients with a psychotic disorder: a systematic review and meta-analysis. Schizophr. Bull 45:114–26 [PubMed: 29547958]

DeBellis MD. 2001. Developmental traumatology: the psychobiological development of maltreated children and its implications for research, treatment, and policy. Dev. Psychopathol 13:539–64 [PubMed: 11523847]

DeLisi M, Kosloski AE, Vaughn MG, Caudill JW, Trulson CR. 2014. Does childhood sexual abuse victimization translate into juvenile sexual offending? New evidence. Violence Vict 29:620–35 [PubMed: 25199390]

DeYoung CG, Cicchetti D, Rogosch FA. 2011. Moderation of the association between childhood maltreatment and neuroticism by the corticotropin-releasing hormone receptor 1 gene. J. Child Psychol. Psychiatry 52:898–906 [PubMed: 21438878]

Drury AJ, Elbert MJ, DeLisi M. 2019. Childhood sexual abuse is significantly associated with subsequent sexual offending: new evidence among federal correctional clients. Child Abuse Negl 95:104035 [PubMed: 31252352]

Duberstein PR, Ward EA, Chaudron LH, He H, Toth SL, et al. 2018. Effectiveness of interpersonal psychotherapy—trauma for depressed women with childhood abuse histories. J. Consult. Clin. Psychol 86:868–78 [PubMed: 30265045]

Efrati Y, Shukron O, Epstein R. 2019. Compulsive sexual behavior and sexual offending: differences in cognitive schemas, sensation seeking, and impulsivity. J. Behav. Addict 8:432–41 [PubMed: 31394911]

Ensink K, Borelli JL, Normandin L, Target M, Fonagy P. 2020. Childhood sexual abuse and attachment insecurity: associations with child psychological difficulties. Am. J. Orthopsychiatry 90:115–24 [PubMed: 30829518]

Fearon RP, Bakermans-Kranenburg MJ, van Ijzendoorn MH, Lapsley AM, Roisman GI. 2010. The significance of insecure attachment and disorganization in the development of children's externalizing behavior: a meta-analytic study. Child Dev 81:435–56 [PubMed: 20438450]

Feiring C, Simon VA, Cleland CM. 2009. Childhood sexual abuse, stigmatization, internalizing symptoms, and the development of sexual difficulties and dating aggression. J. Consult. Clin. Psychol 77:127–37 [PubMed: 19170459]

Fergusson DM, McLeod GF, Horwood LJ. 2013. Childhood sexual abuse and adult developmental outcomes: findings from a 30-year longitudinal study in New Zealand. Child Abuse Negl 37:664–74 [PubMed: 23623446]

Finkelhor D, Browne A. 1985. The traumatic impact of child sexual abuse: a conceptualization. Am. J. Orthopsychiatry 55:530–41 [PubMed: 4073225]

Finkelhor D, Saito K, Jones L. 2020. Updated trends in child maltreatment, 2018 Rep., Crimes Against Child. Res. Cent., Univ. N.H., Durham

Foa EB, McLean CP, Capaldi S, Rosenfield D. 2013. Prolonged exposure versus supportive counseling for sexual abuse-related PTSD in adolescent girls: a randomized clinical trial. JAMA 310:2650–57 [PubMed: 24368465]

Fossati A, Madeddu F, Maffei C. 1999. Borderline personality disorder and childhood sexual abuse: a meta-analytic study. J. Pers. Disord 13:268–80 [PubMed: 10498039]

Frost R, Hyland P, Shevline M, Murphy J. 2020. Distinguishing complex PTSD from borderline personality disorder among individuals with a history of sexual trauma; a latent class analysis. Eur. J. Trauma Dissociation 4:100080

Gee DG, Gabard-Durnam LJ, Flannery J, Goff B, Humphreys KL, et al. . 2013. Early developmental emergence of human amygdala-prefrontal connectivity after maternal deprivation. PNAS 110:15638–43 [PubMed: 24019460]

Glasser M, Kolvin I, Campbell D, Glasser A, Leitch I, Farrelly S. 2001. Cycle of child sexual abuse: links between being a victim and becoming a perpetrator. Br. J. Psychiatry 179:482–94 [PubMed: 11731348]

Groh AM, Roisman GI, van Ijzendoorn MH, Bakermans-Kranenburg MJ, Fearon RP. 2012. The significance of insecure and disorganized attachment for children's internalizing symptoms: a meta-analytic study. Child Dev 83:591–610 [PubMed: 22235928]

Guastaferro K, Felt JM, Font SA, Connell CM, Miyamoto S, et al. 2020. Parent-focused sexual abuse prevention: results from a cluster randomized controlled trial. Child Maltreat 10.1177/1077559520963870

Hailes HP, Yu R, Danese A, Fazel S. 2019. Long-term outcomes of childhood sexual abuse: an umbrella review. Lancet Psychiatry 6:830–39 [PubMed: 31519507]

Haslam N2016. Concept creep: psychology's expanding concepts of harm and pathology. Psychol. Inq 27:1–17

Hayes SC. 2004. Acceptance and commitment therapy, relational frame theory, and the third wave of behavioral and cognitive therapies. Behav. Ther 35:639–65

Hayes SC, Wilson KG, Gifford EV, Follette VM, Strosahl K. 1996. Experimental avoidance and behavioral disorders: a functional dimensional approach to diagnosis and treatment. J. Consult. Clin. Psychol 64:1152–68 [PubMed: 8991302]

Heim CM, Mayberg HS, Mletzko T, Nemeroff CB, Pruessner JC. 2013. Decreased cortical representation of genital somatosensory field after childhood sexual abuse. Am. J. Psychiatry 170:616–23 [PubMed: 23732967]

Heim CM, Newport DJ, Mletzko T, Miller AH, Nemeroff CB. 2008. The link between childhood trauma and depression: insights from HPA axis studies in humans. Psychoneuroendocrinology 33:693–710 [PubMed: 18602762]

Heleniak C, Jenness JL, Stoep AV, McCauley E, McLaughlin KA. 2016. Childhood maltreatment exposure and disruptions in emotion regulation: a transdiagnostic pathway to adolescent internalizing and externalizing psychopathology. Cogn. Ther. Res 40:394–415

Herman J 1981. Father-Daughter Incest Cambridge, MA: Harvard Univ. Press

Herman J 2012. CPTSD is a distinct entity: comment on Resick et al. (2012). J. Trauma Stress 25:256–57 [PubMed: 22729977]

Herman-Giddens ME, Sandler AD, Friedman NE. 1988. Sexual precocity in girls: an association with sexual abuse? Am. J. Dis. Child 142:431–33 [PubMed: 3348186]

Horwitz AV, Widom CS, McLaughlin J, White HR. 2001. The impact of childhood abuse and neglect on adult mental health: a prospective study. J. Health Soc. Behav 42:184–201 [PubMed: 11467252]

Howren MB, Lamkin DM, Suls J. 2009. Associations of depression with C-reactive protein, IL-1, and IL-6: a meta-analysis. Psychosom. Med 71:171–86 [PubMed: 19188531]

James J, Ellis BJ, Schlomer GL, Garber J. 2012. Sex-specific pathways to early puberty, sexual debut, and sexual risk taking: tests of an integrated evolutionary-developmental model. Dev. Psychol 48:687–702 [PubMed: 22268605]

Jardin C, Venta A, Newlin E, Ibarra S, Sharp C. 2017. Secure attachment moderates the relation of sexual trauma with trauma symptoms among adolescents from an inpatient psychiatric facility. J. Interpers. Violence 32:1565–85 [PubMed: 26058980]

Kaplowitz PB. 2008. Link between body fat and the timing of puberty. Pediatrics 121(Suppl. 3):S208–17 [PubMed: 18245513]

Kendler KS, Bulik CM, Silberg J, Hettema JM, Myers J, Prescott CA. 2000. Childhood sexual abuse and adult psychiatric and substance use disorders in women: an epidemiological and cotwin control analysis. Arch. Gen. Psychiatry 57:953–59 [PubMed: 11015813]

Kim J, Cicchetti D. 2010. Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology. J. Child Psychol. Psychiatry 51:706–16 [PubMed: 20050965]

Kumari V, Uddin S, Premkumar P, Young S, Gudjonsson GH, et al. 2014. Lower anterior cingulate volume in seriously violent men with antisocial personality disorder or schizophrenia and a history of childhood abuse. Aust. N.Z. J. Psychiatry 48:153–61 [PubMed: 24234836]

Kwako LE, Noll JG, Putnam FW, Trickett PK. 2010. Childhood sexual abuse and attachment: an intergenerational perspective. Clin. Child Psychol. Psychiatry 15:407–22 [PubMed: 20603427]

Letourneau EJ, Brown DS, Fang X, Hassan A, Mercy JA. 2018. The economic burden of child sexual abuse in the United States. Child Abuse Negl 79:413–22 [PubMed: 29533869]

Letourneau EJ, Schaeffer CM, Bradshaw CP, Feder KA. 2017. Preventing the onset of child sexual abuse by targeting young adolescents with universal prevention programming. Child Maltreat 22:100–11 [PubMed: 28413921]

Lewis T, McElroy E, Harlaar N, Runyan D. 2016. Does the impact of child sexual abuse differ from maltreated but non-sexually abused children? A prospective examination of the impact of child sexual abuse on internalizing and externalizing behavior problems. Child Abuse Negl 51:31–40 [PubMed: 26712421]

Li J, Noll JG, Bensman HE, Putnam FW. 2018. Childhood sexual abuse increases risks for eating disorder symptoms and eating disorder-related health problems in females. In Child Maltreatment Research, Policy, and Practice: Contributions of Penelope Trickett K, ed. Negriff S, pp. 11–26. Cham, Switz.: Springer

Lieb K, Zanarini MC, Schmahl C, Linehan MM, Bohus M. 2004. Borderline personality disorder. Lancet 364:453–61 [PubMed: 15288745]

Lind MJ, Brown RC, Sheerin CM, York TP, Myers JM, et al. 2018. Does parenting influence the enduring impact of severe childhood sexual abuse on psychiatric resilience in adulthood? Child Psychiatry Hum. Dev 49:33–41 [PubMed: 28488144]

Mathews B, Collin-Vézina D. 2017. Child sexual abuse: toward a conceptual model and definition. Trauma Violence Abuse 20:131–48 [PubMed: 29333990]

McEwen BS. 2012. Brain on stress: how the social environment gets under the skin. PNAS 109(Suppl. 2):17180–85 [PubMed: 23045648]

McLaughlin KA, Aldao A, Wisco BE, Hilt LM. 2014. Rumination as a transdiagnostic factor underlying transitions between internalizing symptoms and aggressive behavior in early adolescents. J. Abnorm. Psychol 123:13–23 [PubMed: 24661155]

McLaughlin KA, Peverill M, Gold AL, Alves S, Sheridan MA. 2015. Child maltreatment and neural systems underlying emotion regulation. J. Am. Acad. Child Adolesc. Psychiatry 54:753–62 [PubMed: 26299297]

McPherson P, Scribano P, Stevens J. 2012. Barriers to successful treatment completion in child sexual abuse survivors. J. Interpers. Violence 27:23–39 [PubMed: 21859762]

McTavish JR, Santesso N, Amin A, Reijnders M, Ali MU, et al. 2019a. Psychosocial interventions for responding to child sexual abuse: a systematic review. Child Abuse Negl In press. 10.1016/j.chiabu.2019.104203

McTavish JR, Sverdlichenko I, MacMillan HL, Wekerle C. 2019b. Child sexual abuse, disclosure and PTSD: a systematic and critical review. Child Abuse Negl 92:196–208 [PubMed: 30999168]

Meston CM, Rellini AH, Heiman JR. 2006. Women's history of sexual abuse, their sexuality, and sexual self-schemas. J. Consult. Clin. Psychol 74:229–36 [PubMed: 16649867]

Miller BJ, Buckley P, Seabolt W, Mellor A, Kirkpatrick B. 2011. Meta-analysis of cytokine alterations in schizophrenia: clinical status and antipsychotic effects. Biol. Psychiatry 70:663–71 [PubMed: 21641581]

Minzenberg MJ, Poole JH, Vinogradov S. 2008. A neurocognitive model of borderline personality disorder: effects of childhood sexual abuse and relationship to adult social attachment disturbance. Dev. Psychopathol 20:341–68 [PubMed: 18211741]

Molnar BE, Buka SL, Kessler RC. 2001. Child sexual abuse and subsequent psychopathology: results from the National Comorbidity Survey. Am. J. Public Health 91:753–60 [PubMed: 11344883]

Natsuaki MN, Klimes-Dougan B, Ge X, Shirtcliff EA, Hastings PD, Zahn-Waxler C. 2009. Early pubertal maturation and internalizing problems in adolescence: sex differences in the role of cortisol reactivity to interpersonal stress. J. Clin. Child Adolesc. Psychol 38:513–24 [PubMed: 20183638]

Neacsiu AD, Lungu A, Harned MS, Rizvi SL, Linehan MM. 2014. Impact of dialectical behavior therapy versus community treatment by experts on emotional experience, expression, and acceptance in borderline personality disorder. Behav. Res. Ther 53:47–54 [PubMed: 24418652]

Negriff S, Blankson AN, Trickett PK. 2015. Pubertal timing and tempo: associations with childhood maltreatment. J. Res. Adolesc 25:201–13 [PubMed: 26146470]

Nemeroff CB, Binder E. 2014. The preeminent role of childhood abuse and neglect in vulnerability to major psychiatric disorders: toward elucidating the underlying neurobiological mechanisms. J. Am. Acad. Child Adolesc. Psychiatry 53:395–97 [PubMed: 24655648]

Ng QX, Yong BZJ, Ho CYX, Lim DY, Yeo WS. 2018. Early life sexual abuse is associated with increased suicide attempts: an update meta-analysis. J. Psychiatr. Res 99:129–41 [PubMed: 29454220]

Noll JG, Guastaferro K, Beal SJ, Schreier HMC, Barnes J, et al. 2019. Is sexual abuse a unique predictor of sexual risk behaviors, pregnancy, and motherhood in adolescence? J. Res. Adolesc 29:967–83 [PubMed: 30019514]

Noll JG, Shenk CE, Barnes JE, Haralson KJ. 2013. Association of maltreatment with high-risk Internet behaviors and offline encounters. Pediatrics 131:e510–17 [PubMed: 23319522]

Noll JG, Shenk CE, Putnam KT. 2009. Childhood sexual abuse and adolescent pregnancy: a meta-analytic update. J. Pediatr. Psychol 34:366–78 [PubMed: 18794188]

Noll JG, Trickett PK, Long JD, Negriff S, Susman EJ, et al. 2017. Childhood sexual abuse and early timing of puberty. J. Adolesc. Health 60:65–71 [PubMed: 27836531]

Noll JG, Trickett PK, Putnam FW. 2003. A prospective investigation of the impact of childhood sexual abuse on the development of sexuality. J. Consult. Clin. Psychol 71:575–86 [PubMed: 12795580]

Nwogu NN, Agrawal L, Chambers S, Buagas AB, Daniele RM, Singleton JK. 2016. Effectiveness of Child Advocacy Centers and the multidisciplinary team approach on prosecution rates of alleged sex offenders and satisfaction of non-offending caregivers with allegations of child sexual abuse: a systematic review. JBI Database Syst. Rev. Implement. Rep 13:93–129

O'Donnell KJ, Meaney MJ. 2020. Epigenetics, development, and psychopathology. Annu. Rev. Clin. Psychol 16:327–50 [PubMed: 32084320]

Passos IC, Vasconcelos-Moreno MP, Costa LG, Kunz M, Brietzke E, et al. 2015. Inflammatory markers in post-traumatic stress disorder: a systematic review, meta-analysis, and meta-regression. Lancet Psychiatry 2:1002–12 [PubMed: 26544749]

Pereda N, Guilera G, Forns M, Gomez-Benito J. 2009. The prevalence of child sexual abuse in community and student samples: a meta-analysis. Clin. Psychol. Rev 29:328–38 [PubMed: 19371992]

Perroud N, Paoloni-Giacobino A, Prada P, Olie E, Salzmann A, et al. 2011. Increased methylation of glucocorticoid receptor gene (NR3C1) in adults with a history of childhood maltreatment: a link with the severity and type of trauma. Transl. Psychiatry 1:e59 [PubMed: 22832351]

Peverill M, Sheridan MA, Busso DS, McLaughlin KA. 2019. Atypical prefrontal-amygdala circuitry following childhood exposure to abuse: links with adolescent psychopathology. Child Maltreat 24:411–23 [PubMed: 31146576]

Pulido ML, Dauber S, Tully BA, Hamilton P, Smith MJ, Freeman K. 2015. Knowledge gains following a child sexual abuse prevention program among urban students: a cluster-randomized evaluation. Am. J. Public Health 105:1344–50 [PubMed: 25973809]

Pulverman CS, Kilimnik CD, Meston CM. 2018. The impact of childhood sexual abuse on women's sexual health: a comprehensive review. Sex. Med. Rev 6:188–200 [PubMed: 29371141]

Putnam FW, Amaya-Jackson L, Putnam KT, Briggs EC. 2020. Synergistic adversities and behavioral problems in traumatized children and adolescents. Child Abuse Negl 106:104492 [PubMed: 32447141]

Putnam KT, Harris WW, Putnam FW. 2013. Synergistic childhood adversities and complex adult psychopathology. J. Trauma Stress 26:435–42 [PubMed: 23893545]

Raby KL, Labella MH, Martin J, Carlson EA, Roisman GI. 2018. Childhood abuse and neglect and insecure attachment states of mind in adulthood: prospective, longitudinal evidence from a high-risk sample—Corrigendum. Dev. Psychopathol 30:367–70 [PubMed: 28583213]

Raison CL, Capuron L, Miller AH. 2006. Cytokines sing the blues: inflammation and the pathogenesis of depression. Trends Immunol 27:24–31 [PubMed: 16316783]

Reckdenwald A, Mancini C, Beauregard E. 2013. The cycle of violence: examining the impact of maltreatment early in life on adult offending. Violence Vict 28:466–82 [PubMed: 23862310]

Rheingold AA, Zajac K, Chapman JE, Patton M, de Arellano M, et al. 2015. Child sexual abuse prevention training for childcare professionals: an independent multi-site randomized controlled trial of Stewards of Children. Prev. Sci 16:374–85 [PubMed: 25015782]

Romano E, De Luca RV. 2001. Male sexual abuse: a review of effects, abuse characteristics, and links with later psychological functioning. Aggress. Violent Behav 6:55–78

Roodman AA, Clum GA. 2001. Revictimization rates and method variance: a meta-analysis. Clin. Psychol. Rev 21:183–204 [PubMed: 11293365]

Rosenthal MZ, Rasmussen Hall ML, Palm KM, Batten SV, Follette V. 2005. Chronic avoidance helps explain the relationship between severity of childhood sexual abuse and psychological distress in adulthood. J. Child Sex. Abuse 14:25–41

Rosner R, Rimane E, Frick U, Gutermann J, Hagl M, et al. 2019. Effect of developmentally adapted cognitive processing therapy for youth with symptoms of posttraumatic stress disorder after childhood sexual and physical abuse: a randomized clinical trial. JAMA Psychiatry 76:484–91 [PubMed: 30969342]

Russell DEH. 1986. The Secret Trauma: Incest in the Lives of Girls and Women New York: Basic Books

Senn TE, Braksmajer A, Hutchins H, Carey MP. 2017. Development and refinement of a targeted sexual risk reduction intervention for women with a history of childhood sexual abuse. Cogn. Behav. Pract 24:496–507 [PubMed: 29062220]

Senn TE, Carey MP, Coury-Doniger P. 2012. Mediators of the relation between childhood sexual abuse and women's sexual risk behavior: a comparison of two theoretical frameworks. Arch. Sex. Behav 41:1363–77 [PubMed: 22282323]

Shea BJ, Grimshaw JM, Wells GA, Boers M, Andersson N, et al. 2007. Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews. BMC Med. Res. Methodol 7:10 [PubMed: 17302989]

Shenk CE, Noll JG, Peugh JL, Griffin AM, Bensman HE. 2016. Contamination in the prospective study of child maltreatment and female adolescent health. J. Pediatr. Psychol 41:37–45 [PubMed: 25797944]

Shenk CE, Putnam FW, Rausch JR, Peugh JL, Noll JG. 2014. A longitudinal study of several potential mediators of the relationship between child maltreatment and posttraumatic stress disorder symptoms. Dev. Psychopathol 26:81–91 [PubMed: 24444173]

Teicher MH, Samson JA. 2013. Childhood maltreatment and psychopathology: a case for ecophenotypic variants as clinically and neurobiologically distinct subtypes. Am. J. Psychiatry 170:1114–33 [PubMed: 23982148]

Teicher MH, Samson JA, Anderson CM, Ohashi K. 2016. The effects of childhood maltreatment on brain structure, function and connectivity. Nat. Rev. Neurosci 17:652–66 [PubMed: 27640984]

Tomoda A, Navalta CP, Polcari A, Sadato N, Teicher MH. 2009. Childhood sexual abuse is associated with reduced gray matter volume in visual cortex of young women. Biol. Psychiatry 66:642–48 [PubMed: 19560122]

Toth SL, Cicchetti D. 2013. A developmental psychopathology perspective on child maltreatment. Child Maltreat 18:135–39 [PubMed: 23886641]

Tottenham N 2014. The importance of early experiences for neuro-affective development. Curr. Top. Behav. Neurosci 16:109–29 [PubMed: 24264369]

Trickett PK, Noll JG, Putnam FW. 2011. The impact of sexual abuse on female development: lessons from a multigenerational, longitudinal research study 23:453–76

Trickett PK, Noll JG, Reiffman A, Putnam FW. 2001. Variants of intrafamilial sexual abuse experience: implications for short- and long-term development. Dev. Psychopathol 13:1001–19 [PubMed: 11771904]

Trickett PK, Noll JG, Susman EJ, Shenk CE, Putnam FW. 2010. Attenuation of cortisol across development for victims of sexual abuse. Dev. Psychopathol 22:165–75 [PubMed: 20102654]

Ullsperger JM, Nikolas MA. 2017. A meta-analytic review of the association between pubertal timing and psychopathology in adolescence: Are there sex differences in risk? Psychol. Bull 143:903–38 [PubMed: 28530427]

Vachon DD, Krueger RF, Rogosch FA, Cicchetti D. 2015. Assessment of the harmful psychiatric and behavioral effects of different forms of child maltreatment. JAMA Psychiatry 72:1135–42 [PubMed: 26465073]

Venta A, Hatkevich C, Mellick W, Vanwoerden S, Sharp C. 2017. Social cognition mediates the relation between attachment schemas and posttraumatic stress disorder. Psychol. Trauma 9:88–95 [PubMed: 27336218]

Walsh K, Fortier MA, Dilillo D. 2010. Adult coping with childhood sexual abuse: a theoretical and empirical review. Aggress. Violent Behav 15:1–13 [PubMed: 20161502]

Wang L, Dai Z, Peng H, Tan L, Ding Y, et al. 2014. Overlapping and segregated resting-state functional connectivity in patients with major depressive disorder with and without childhood neglect. Hum. Brain Mapp 35:1154–66 [PubMed: 23408420]

Ward T, Beech A. 2006. An integrated theory of sexual offending. Aggress. Violent Behav 11:44–63

Whittle S, Simmons JG, Byrne ML, Strikwerda-Brown C, Kerestes R, et al. 2015. Associations between early adrenarche, affective brain function and mental health in children. Soc. Cogn. Affect. Neurosci 10:1282–90 [PubMed: 25678548]

Widom CS. 1999. Posttraumatic stress disorder in abused and neglected children grown up. Am. J. Psychiatry 156:1223–29 [PubMed: 10450264]

Widom CS, Czaja SJ, Paris J. 2009. A prospective investigation of borderline personality disorder in abused and neglected children followed up into adulthood. J. Pers. Disord 23:433–46 [PubMed: 19817626]

Widom CS, DuMont K, Czaja SJ. 2007. A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. Arch. Gen. Psychiatry 64:49–56 [PubMed: 17199054]

Widom CS, Massey C. 2015. A prospective examination of whether childhood sexual abuse predicts subsequent sexual offending. JAMA Pediatr 169:e143357 [PubMed: 25561042]

Wilson HW, Widom CS. 2008. An examination of risky sexual behavior and HIV in victims of child abuse and neglect: a 30-year follow-up. Health Psychol 27:149–58 [PubMed: 18377133]

Wilson HW, Widom CS. 2009. Sexually transmitted diseases among adults who had been abused and neglected as children: a 30-year prospective study. Am. J. Public Health 99(Suppl. 1):S197–203 [PubMed: 19218173]

Young EA. 1995. The role of gonadal steroids in hypothalamic-pituitary-adrenal axis regulation. Crit. Rev. Neurobiol 9:371–81 [PubMed: 8829851]

Zajac L, Raby KL, Dozier M. 2020. Sustained effects on attachment security in middle childhood: results from a randomized clinical trial of the Attachment and Biobehavioral Catch-up (ABC) intervention. J. Child Psychol. Psychiatry 61:417–24 [PubMed: 31677152]

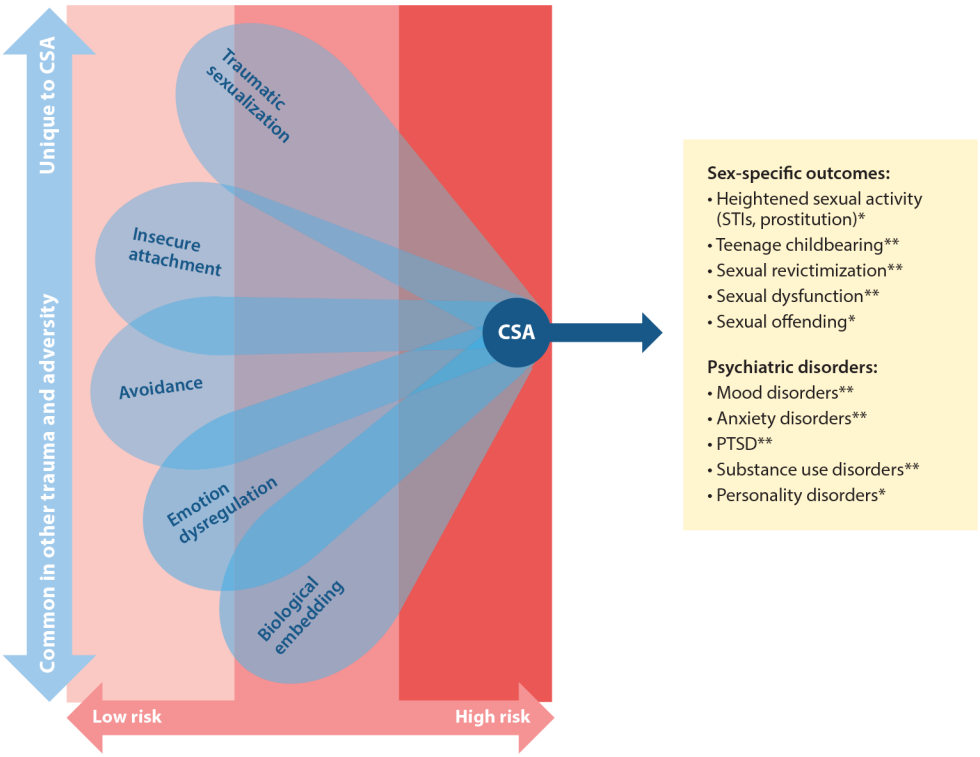

Figure 1.

The CCM model of the risk that CSA confers on psychiatric disorders and sex-specific outcomes. Lower risk is associated with singular or independently occurring mechanisms; higher risk is associated with multiple or co-occurring mechanisms. Single asterisks indicate mixed or moderate evidence for CSA elevating risk; double asterisks indicate strong evidence for CSA elevating risk. Abbreviations: CCM, Compounded Convergence of Mechanisms; CSA, childhood sexual abuse; PTSD, posttraumatic stress disorder; STI, sexually transmitted infection.