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4	Manuscript accepted for publication in Development and Psychopathology
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6	The Intergenerational Transmission of Childhood Maltreatment: Non-specificity of Maltreatment
7	Type and Associations with Borderline Personality Pathology
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ABSTRACT

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One generation's experience of childhood maltreatment is associated with that of the next. However, whether this intergenerational transmission is specific to distinct forms of maltreatment and what factors may contribute to its continuity remains unclear. Borderline personality pathology is predicted by childhood maltreatment and characterized by features (e.g., dysregulated emotion, relationship instability, impulsivity, inconsistent appraisals of others) that may contribute to its propagation. Among 364 older adults and 573 of their adult children (total n=937), selfreported exposure to distinct forms of childhood maltreatment (i.e., emotional, physical and sexual abuse and emotional and physical neglect as assessed by the Childhood Trauma Questionnaire) showed homotypic and heterotypic associations across generations with little evidence that latent factors unique to specific forms of maltreatment show generational continuity. General nonspecific indices of childhood maltreatment showed evidence of intergenerational transmission after accounting for demographic factors and parent socioeconomic status (b = 0.126, p = 9.21×10^{-1} 4). This continuity was partially mediated by parental borderline personality pathology (assessed longitudinally through a variety of measures and sources, indirect effect: b = 0.031 [95% CI 0.003, 0.060]. The intergenerational continuity of childhood maltreatment may largely represent general risk for non-specific maltreatment that may, in part, be propagated by borderline personality pathology and/or shared risk factors.

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- Keywords: Borderline personality, childhood maltreatment, intergenerational transmission, stress,
- 37 abuse, neglect

INTRODUCTION

Maltreatment during childhood (i.e., abuse and/or neglect) is common and among the largest risk factors for adverse outcomes. By age 18, over 37% of children in the United States are investigated for maltreatment by child protective services (CPS); a third of these cases are substantiated (Kim, Wildeman, Jonson-Reid, & Drake, 2017). Consequences are severe and broad. Those who were abused/neglected have vastly increased odds and earlier onsets of later stress- and age-related disease and dysfunction (e.g., psychopathology, cardiovascular disease, obesity; Batten, Aslan, Maciewjewski, & Mazure, 2004; McLaughlin et al., 2012; Shields et al., 2016). Further, each case of nonfatal childhood maltreatment is estimated to cost society \$210,000 (in 2010 \$; Fang, Brown, Florence, & Mercy, 2012). Given the widespread prevalence and consequences of childhood maltreatment, it is critical to understand factors associated with its expression so that strategies and policies may be put in place to mitigate its burden.

Intergenerational Transmission of Childhood Maltreatment

Maltreatment during childhood tends to aggregate within families (see **Table 1** for a summary of research). Much of this work has focused on the intergenerational transmission of childhood maltreatment (ITCM) in vulnerable populations (e.g., young mothers, low SES, criminality). For instance, in the largest study of ITCM, adolescent first-time mothers (n=85,084) who were maltreated as children were more likely to be investigated for the perpetration of maltreatment by the time their children were 5 years old (substantiated HR: 3.19; unsubstantiated HR: 2.19; Putnam-Hornstein, Cederbaum, King, Eastman, & Trickett, 2015). Comparably less work has been conducted in community and general population samples, but findings are similar. For example, in a sample drawn from a birth registry of British twins, children were more likely to be physically

maltreated if their mother had experienced maltreatment (Jaffee et al., 2013). Indeed, parental history of childhood maltreatment is among the most potent risk factors for children to be maltreated, with evidence that its effect is even larger than other well-documented risk factors including poverty, criminality, and mental health conditions (Ben-David, Jonson-Reid, Drake, & Kohl, 2015). This robust evidence (**Table 1**) suggests that understanding childhood maltreatment requires an intergenerational family-based perspective. However, there has been limited work evaluating whether ITCM is general and/or specific to unique forms of maltreatment and what potential factors may contribute to its continuity.

ITCM: Maltreatment Specificity

Theoretical models pioneered by McLaughlin and colleagues (2014) speculate that distinct forms of childhood maltreatment may be associated with unique pathways to both shared and distinct outcomes. These models postulate that threat experiences (e.g., abuse) disrupt social and emotional processing, while deprivation (e.g., neglect) alters cognitive development to increase risk for psychopathology. However, whether ITCM represents continuity of non-specific childhood maltreatment or is associated with specific types of maltreatment remains uninvestigated.

Research on ITCM has evaluated broad dimensions of maltreatment (e.g., all abuse and all neglect), multiple specific subtypes (e.g., physical and emotional abuse and neglect and sexual abuse) or only one specific subtype (e.g., physical abuse) independently. Generally, this work provides evidence for the intergenerational transmission of homotypic (i.e., association of the same subtype of maltreatment across generations) and heterotypic (i.e., association of different subtypes of maltreatment across generations) childhood maltreatment (**Table 1**). For example, in the Add Health study, young parents (n=2,977) who were neglected and/or abused as children were more

likely to endorse being abusive and/or neglectful to their infants/toddlers (Kim, 2009). While some work has examined whether ITCM is characterized by non-specific and maltreatment type specificity in the context of a single study (e.g., Widom, Czaja, & DuMont, 2015), no studies, to our knowledge, have examined whether specific forms of childhood maltreatment exert unique patterns of association across generations above and beyond non-specific maltreatment. Indeed, we are aware of only one study that has tangentially tested this question; in that study, Newcombe and Locke (2001) found evidence that increased childhood maltreatment as assessed using a general latent factor was linked to poor parenting, but that maternal history of neglect was associated with poor parenting over and above the latent general maltreatment factor.

Borderline Personality Pathology: A Potential Mediator of Intergenerational Childhood

Maltreatment

Borderline personality pathology (BPP), which is characterized by pervasive emotion dysregulation, impulsivity, distress intolerance, inconsistent appraisals of others, feelings of isolation, and unstable interpersonal relationships, is a promising potential contributor to ITCM. Both retrospective and prospective data reveal that maltreatment during childhood is strongly associated with BPP and that this relationship remains even after accounting for other forms of psychopathology (e.g., depression) related to both early life stress and BPP (Gratz, Tull, Baruch, & Bornovalova, 2008; Hernandez, Arntz, Gaviria, Labad, & Gutiérrez-Zotes, 2012; Ibrahim, Cosgrave, & Woolgar, 2018; Joyce et al., 2003; Stone, 1981). For example, Zanarini and colleagues (1997) found that 90% of patients with borderline personality disorder were exposed to childhood maltreatment and that they were more likely to be exposed than patients with other personality disorder diagnoses.

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A comparatively small literature suggests that parents with BPP are more likely to be investigated for perpetrating childhood maltreatment by CPS, even after accounting for their own maltreatment (Perepletchikova, Ansell, & Axelrod, 2012; Bools, Neale, Meadow, 1994; Laporte, 2009). These findings are buttressed by evidence that BPP is associated with factors that mediate ITCM or have been associated with risk for maltreatment exposure in one's children (e.g., young parenthood, single parenthood, substance use disorder, depression, interpersonal stress and violence, stress exposure, familial instability, domestic violence; Chan, 2011; Conway, Boudreaux, & Oltmanns, 2018; Daley, Burge, & Hammen, 2000; De Genna, Feske, Larkby, Angiolieri, & Gold, 2012; Grant et al., 2008; Hopwood, Donnellan, & Zanarini, 2010; Liebke et al., 2017; Martín-Blanco et al., 2014; Radtke et al., 2015; Skodol et al., 1999; Smith et al., 2014; Weinstein, Gleason, & Oltmanns, 2012; Wilson, Stroud, & Durbin, 2017). For example, individuals with BPP are more likely to end long-term relationships and voluntarily or involuntarily lose custody of their children (Zanarini et al., 2014), and single parenthood is associated with heightened risk for childhood maltreatment (Gelles, 1989; Schneider, 2017). Moreover, BPP and childhood maltreatment exposure also share similar associations with other outcomes (e.g., impulsivity, emotional instability, relationship issues, substance use, epigenetic signatures within the glucocorticoid receptor gene NR3C1; Dammann et al., 2011; Geiger & Crick, 2001; Martín-Blanco et al., 2014; Rogosch & Cicchetti, 2005; Weaver et al., 2004). Collectively, these convergent data suggest that BPP may plausibly contribute to the propagation of childhood maltreatment across generations.

Influential biosocial developmental models of BPP provide a framework for understanding how BPP might contribute to ITCM (Crowell et al., 2009; Linehan, 1993). These theoretical models postulate that genetically-driven vulnerability for dysregulated emotion and environmental

sensitivity interact with an invalidating environment characterized by maltreatment to promote the development of BPP. Accordingly, the moderate heritability of BPP (Reichborn-Kjennerud et al., 2013) may be passed from parents to offspring, leading to the expression of related prodromal temperamental traits in children (e.g., dysregulated emotion and stress sensitivity) that may evoke childhood maltreatment and inconsistent caregiver practices, especially among caregivers with BPP traits. This invalidating early environment may then potentiate BPP development in offspring already at risk by encouraging dysregulated and volatile emotion by inconsistently reinforcing and punishing emotional expression (Crowell et al., 2009; Gunderson & Lyons-Ruth, 2008; Linehan, 1993).

Consistent with this model, emotion dysregulation—a core component of BPP—among parents has been associated with their invalidation of adolescent emotion expression (a tenet of emotional abuse), which is further linked to BPP-related characteristics (e.g., poor emotion regulation; Buckholdt, Parra, & Jobe-Shields, 2014). Further, dysregulated emotion is associated with the propagation of maltreatment across generations (Smith et al., 2014) and indirectly links childhood emotional abuse to unhealthy relationship characteristics, such as intimate partner violence (Berzenski & Yates, 2010), which, in turn, indirectly links maltreatment across generations (Rodriguez, Silvia, Gonzalez, & Christl, 2018). Together, theoretical work supported by empirical data raise the intriguing possibility that BPP may indirectly link childhood maltreatment across generations. Such an indirect effect might reflect a behavioral mechanism underlying ITCM and/or common factors associated with exposure to childhood maltreatment (in both generations) as well as BPP expression.

The Current Study

Among a representative community sample (n=937) of older parents (n=364, age: 66.68±3.01) and their adult children (n=573, age: 38.67±7.39 years), we tested whether the intergenerational transmission of childhood maltreatment is general and/or specific to certain forms, and whether parental BPP mediates this continuity. Based on work suggesting general maltreatment continuity and heterotypic associations across distinct forms of child maltreatment, we hypothesized that non-specific childhood maltreatment would be transmitted across generations with little evidence that unique specific forms of maltreatment are. Further, because borderline personality pathology is associated with both exposure to childhood maltreatment and risk factors for its perpetuation, we expected that it would mediate childhood maltreatment continuity across generations.

[Insert Table 1 Here]

METHOD

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Participants and Procedure

Participants (n=937; **Table 2**) included 364 parents (G1; age 66.68±3.01 years, 59.44% female, 71.70% white) and 573 of their biological adult children (G2; age 38.67±7.39 years, 43.96% female, 73.73% white). These families were recruited from the St. Louis Personality and Aging Network (SPAN) study, which examines personality, health, biomarkers, and aging in later life (Oltmanns, Rodrigues, Weinstein, & Gleason, 2014). Of the original 659 SPAN participants (G1) who reported having biological children, 543 (82%) provided permission to contact their G2 offspring (465 provided contact information for all of their children, 78 provided contact information for some of their children). G2 participants were called or emailed up to 5 times inviting them to participate in our study. Of the potential 855 G2 participants, 610 (71%) completed questionnaires (through mail or online surveys) about themselves as well as their children (G3). Data from G1 participants were drawn from SPAN. In cases where G1 participants had insufficient or missing maltreatment reports, observations were excluded from analyses. Participants provided informed consent to a protocol approved by the Washington University in St. Louis Institutional Review Board. G2 participants were compensated \$30 for returning questionnaires. G1 participants received \$60 for completing each in-person SPAN session.

[Insert Table 2 Here]

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 $^{^{1}}$ G1 individuals who participated in our study and those with children who declined did not differ on summed CTQ subscale scores, BPP, or age, all ps>0.25). G1 participants had higher levels of education, higher household income, and were more likely to be female and white (all ps<0.05) relative to those who declined consent.

Measures

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Childhood Maltreatment

Self-reported childhood maltreatment experienced by G1 and G2 participants was assessed using the 28-item Childhood Trauma Questionnaire (CTQ) (Bernstein & Fink, 1998). It is composed of 5 subscales, which had good-excellent internal consistency across both generations in our sample [sexual abuse: $\alpha s = 0.92(G1)$, 0.94(G2); emotional neglect: $\alpha s = 0.91$, 0.88; emotional abuse: $\alpha s = 0.89$, 0.87; physical abuse: $\alpha s = 0.81$, 0.73; physical neglect: $\alpha s = 0.76$, 0.67).

Given evidence that CTQ subscales represent a general factor of childhood maltreatment (Spinhoven et al., 2014; Pezzoli et al., 2018), we used Mplus v. 7.3 software (Muthén & Muthén, 1998-2012) to conduct confirmatory bifactor analyses of G1 data using mean and varianceadjusted weighted least squares (WLSMV) and G2 data, nested by family, using maximum likelihood estimation with robust standard errors. Variances of the general and group specific factors were set to 1 and correlations between factors to 0, producing a general factor on which all items are permitted to load (representing shared variance among indicators) as well as second factors representing unique variance among sets of items with similar content. Thus, we modeled a single global factor of childhood maltreatment, as assessed by all CTQ items (n=25) contributing to the 5 subscales, as well as five group specific factors, as assessed by the five CTQ items composing each subscale. Model fit was acceptable-excellent in both G1 and G2 (RMSEA = 0.05 (G1), 0.05 (G2); CFI = 0.99, 0.94; TLI: 0.98, 0.85). However, one item (Item #14; family members saying hurtful or insulting things, part of the emotional abuse subscale) was removed from G1 data because it resulted in negative residual variance. Factor loadings on the general factor were moderate-large in both generations (G1: M = 0.70, range = 0.44 to 0.91; G2: M = 0.50, range = 0.22 to 0.78). Subscale specific factor loadings were generally lower (G1: Emotional Abuse M =

0.18, range = 0.047 - 0.33; Physical Abuse M = 0.51, range = 0.38 to 0.65; Sexual Abuse M = 0.510.75, range = 0.63 to 0.81; Emotional Neglect M = 0.43, range = 0.32 to 0.55; Physical Neglect M= 0.37, range = 0.22 to 0.59. G2: Emotional Abuse M = 0.34, range = 0.16 to 0.52; Physical Abuse M = 0.43, range = 0.35 to 0.57; Sexual Abuse M = 0.78, range = 0.61 to 0.87; Emotional Neglect M = 0.42, range = .24 to 0.62; Physical Neglect M = 0.34, range = 0.14 to 0.6). Following evidence that a general single latent factor represented these data well, we also constructed CTQ total scores (with and without log-transformation), consistent with prior work (MacDonald et al., 2016; Schmidt, Narayan, Atzl, Rivera, & Lieberman, 2018), so that our results would be more readily combined with and interpreted alongside other studies.

Borderline Personality Pathology

As described in our prior work (Conway, Boudreaux, & Oltmanns, 2018; Di Iorio et al., 2018) borderline pathology was assessed in G1 participants dimensionally from multiple perspectives (i.e., clinician, self, and informant ratings) across time. Because many people exhibit at least some personality pathology symptoms (Oltmanns et al., 2014) and subthreshold borderline personality disorder symptoms are associated with psychosocial impairment (Ellison, Rosenstein, Chelminksi, Dalrymple, & Zimmerman, 2016; Zimmerman, Chelminksi, Young, Dalrymple, & Martinez, 2013), scores were treated continuously to retain variation at subthreshold diagnostic levels. Borderline pathology was assessed using multiple sources and methods to improve our coverage of subthreshold presentations. Informant reports were included because they add unique information about an individual's personality that the participant may be unable or unwilling to report (Oltmanns & Turkheimer, 2006) and are predictive of health outcomes over and above self-report (Cruitt & Oltmanns, 2017). Interviewer ratings of borderline personality pathology were

acquired using the Structured Interview for DSM-IV Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997); self- and informant reports were collected using the Revised NEO Personality Inventory (NEO PI-R; Costa & McCrae, 1992) and the Multisource Assessment of Personality Pathology (MAPP; Oltmanns, Turkheimer, & Strauss, 1998). Interviewer ratings and self-reports were collected at three in-person sessions from participants: baseline (the initial session), and the first and second in-person follow-up session (IPFU1 and IPFU2, respectively). IPFU1 occurred 0.72-6.65 (M = 2.83) years following the baseline session; IPFU2 occurred 0.29-6.33 (M = 3.93) years following IPFU1. Informant reports were collected at these same times through mail or email.

Clinical Interview: The SIDP-IV is a semi-structured interview in which trained interviewers rate 80 items corresponding to criteria of the 10 PDs on a scale of 0 (no pathology present) to 3 (pathology strongly present). SIDP scores were treated continuously by summing responses across the 9 borderline personality disorder (BPD) criteria at each assessment (range: 0-4; n=67 (18.41%) met at least one criterion at one or more assessments; M±SD: baseline: 0.140±0.457; IPFU1: 0.123±0.448; IPFU2: 0.122±0.468). Trained full-time staff members, graduate students in clinical psychology, or undergraduate research assistants conducted all interviews. Inter-rater reliability ratings from a selected subsample of 265 video-recorded baseline interviews show excellent agreement (intraclass correlation coefficient: 0.77; Oltmanns et al., 2014).

Self and Informant Report: In addition to self-report, we also acquired informant-report of G1 personality. Most (95%) participants had an associated informant consent to the SPAN protocol and report on their (i.e., the participant's) personality at the baseline assessment (informant n = 344; age: range 25.86.83, M = 59.81, SD = 12.15; 65.41% female, 72.30%

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European-American, 24.78% African-American). Informants knew the participant for an average of 32.86 years (SD = 13.10) at the baseline SPAN assessment. Most were a spouse or romantic partner (54.65%, n = 188), other family member (29.07%, n = 100), or friend (14.53%, n = 50), with the remainder consisting of neighbors, coworkers, or other acquaintances (1.74%, n = 6). Informants completed questionnaires about their associated participant through the mail or online at baseline, IPFU1, and IPFU2 sessions and received \$30 remuneration.

The NEO PI-R (Form S for self; Form R for informant) consists of 240-items assessing the five domains of neuroticism, extraversion, openness, agreeableness, and conscientiousness, as well as six lower-order facets within each domain. NEO PI-R borderline scores were generated independently for self and informant report by summing anxiety, angry hostility, depression, impulsiveness, vulnerability, openness to feelings, openness to actions, compliance (reverse scored), and deliberation (reverse scored) facet scale scores within each assessment (self-report: range 57.80-225.00, baseline: 117.70±21.79; IPFU1: 115.80±21.41; IPFU2: 118.90±22.53; informant-report: range 44.00-252.00, baseline: 123.60±29.68, IPFU1: 123.70±30.03; IPFU2: 121.470±30.66) (Miller, Reynolds, & Pilkonis, 2004). The MAPP is an 80-item measure of personality pathology based on lay translations of DSM-IV PD diagnostic criteria. Self and informant MAPP BPD scores were calculated by summing responses across the 9 BPD items (self: range: 0-5; n=117 (32.14%) met at least one criterion at one or more assessments; baseline: 0.225 ± 0.559 ; IPFU1: 0.156 ± 0.461 ; IPFU2: 0.278 ± 0.619 ; informant: range: 0-8; n=161 (44.23%) met at least one criterion at one or more assessments; baseline: 0.417±0.850 IPFU1: 0.462±1.08; IPFU2: 0.438±0.966).

We performed an exploratory structural equation modeling (ESEM) analysis on borderline personality pathology scores derived from clinician-based interview (SIDP-IV) and self- and

informant-report (NEO PI-R and MAPP) data across all assessment times (baseline, IPFU1, IPFU2) using maximum likelihood estimation in Mplus 7.3 (Muthén & Muthén, 1998-2012). We hypothesized a one-factor model to account for the correlations among the 15 BPP measures (i.e., self and informant report and clinician ratings at baseline, IPFU1, and IPFU2 sessions), which fit the data well (RMSEA = .056, CFI = .976, TLI = .955) with factor loadings ranging from .45 to .70. Estimated factor scores were used to represent BPP in all subsequent analyses.

Other Relevant Covariates

Race, ethnicity, and gender were assessed via self-report. Participants were asked to choose from eight options to identify their race (e.g., "White, Caucasian," "Black, African American," "Other"), and were further asked to indicate whether they were Hispanic. Due to the low prevalence of races other than White and Black and endorsement of being Hispanic (**Table 2**), the present study formed one dichotomous variable indicating status as White/non-White and did not consider Hispanic/non-Hispanic. Gender was assessed via self-report. Socioeconomic status (SES) was formed as a composite measure of household annual income and level of education, each assessed via self-reported choice from nine and eight ranked options, respectively, and standardized prior to being summed.

Statistical Analyses

As the sample contains parents and one or more of their offspring, linear mixed effect models were used to nest data according to family (random factor), when examining G1-G2 associations, to account for the non-independence of measurement across generations using the lme4 R package (Bates, Maechler, Bolker, & Walker, 2015). The following fixed effect covariates were included

in models: G1 age, sex, race (white/non-white), SES (in all analyses); G2 age and sex (in all analyses involving G2). We first tested whether CTQ subscale summary scores (i.e., emotional abuse, physical abuse, sexual abuse, emotional neglect, and/or physical neglect) show evidence of intergenerational association before examining whether childhood maltreatment factors unique to these subscales have generational continuity. To correct for the 25 associations tested, each of these analyses was subjected to Benjamini and Hochberg (1995) false discovery rate (FDR) correction.

Given evidence of non-specific association across CTQ subscales and limited evidence that unique subscale-specific factors showed intergenerational continuity (see **Results**), we examined relationships between G1 childhood maltreatment (i.e., CTQ summed scores), G1 Borderline Personality Pathology (factor score), and G2 childhood maltreatment (i.e., CTQ summed scores) using FDR adjustment to account for these 3 comparisons. Subsequently, we evaluated our hypothesized mediational model by testing whether G1 borderline personality pathology indirectly links childhood maltreatment experienced by G1 to childhood maltreatment experienced by G2 in a 2-2-1 multilevel mediational model clustered by family using Mplus v. 7.3 software (Muthén & Muthén, 1998-2012). As with the linear mixed effects models, demographic variables were included as covariates on G1 BPP (m; G1 age, sex, race, SES) and G2 childhood maltreatment (y; G1 age, sex, race, SES, and G2 age and sex).

RESULTS

Childhood Maltreatment: General and Specific Associations Across Generations

Associations between CTQ summed subscales and unique CTQ subscale factors are summarized in **Tables 3-4**. Broadly, with the exception of sexual abuse in G2, specific CTQ subscale total scores (i.e., emotional and physical abuse and neglect) showed evidence of homotypic and heterotypic association across generations (**Table 3**). However, unique subscale specific factor scores orthogonal to a general CTQ factor revealed little evidence of intergenerational association, with the only exceptions being heterotypic associations of G1 physical neglect and G2 sexual abuse ($p = 3.69 \times 10^{-3}$; $p_{\text{fdr}} = 0.0461$), and homotypic associations of childhood emotional neglect across generations ($p = 2.05 \times 10^{-4}$; $p_{\text{fdr}} = 5.13 \times 10^{-3}$; **Table 4**).² *Post hoc* analyses revealed no evidence that BPP mediated the association between the homotypic emotional neglect association across generations (indirect effects b = 0.013, 95% C.I. [-0.005, 0.032], p = 0.159) or the association between G1 physical neglect and G2 sexual abuse (indirect effects b = 0.024, 95% C.I. [-0.002, 0.029], p = 0.096). Unlike unique subscale factors, exposure to general childhood maltreatment, as measured using the general latent factor, was positively coupled across generations (b = 0.0233, SE = 0.00817, $p = 4.50 \times 10^{-3}$).

[Insert Tables 3 and 4 Here]

² Another way to address this question is to evaluate whether subscale scores show evidence of intergenerational continuity after accounting for summed scores of the other scales. Analyzing the data this way produces results analogous to our bifactor latent model approach, wherein only emotional neglect shows evidence of intergenerational homotypic continuity after controlling for demographic covariates. Emotional Abuse: b = 0.0800, SE = 0.0482, p = 0.0984, $p_{fdr} = 0.141$; Physical Abuse: b = 0.0823, SE = 0.0412, p = 0.0467, $p_{fdr} = 0.107$; Sexual Abuse: b = 0.0528, SE = 0.0485, p = 0.278, $p_{fdr} = 0.161$; Emotional Neglect: b = 0.185, SE = 0.0440, $p = 3.24 \times 10^{-5}$, $p_{fdr} = 1.22 \times 10^{-4}$; Physical Neglect: b = 0.0722, SE = 0.0371, p = 0.0522, $p_{fdr} = 0.107$).

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337 The experience of childhood maltreatment (i.e., total CTQ scores) was positively coupled across generations (without fixed effect covariates: b = 0.203, SE = 0.0368, $p = 8.34 \times 10^{-8}$, $p_{fdr} = 1.25 \times 10^{-8}$ 338 7; with fixed effect covariates: b = 0.126, SE = 0.0375, $p = 9.21 \times 10^{-4}$, $p_{fdr} = 9.21 \times 10^{-4}$). Further, 339 340 parent borderline personality pathology was positively associated with their own prior exposure to 341 maltreatment during childhood as well as their children's exposure (G1 without covariates: b = 0.0206, SE = 0.00235, $p < 2.22 \times 10^{-16}$, $p_{\text{fdr}} = 6.66 \times 10^{-16}$ with covariates; b = 0.0198, SE = 0.00320, 342 343 $p = 1.60 \times 10^{-9}$, $p_{fdr} = 4.80 \times 10^{-9}$; G2 without fixed effect covariates: b = 2.714 SE = 0.613, p = 0.613 1.28×10^{-5} , $p_{\text{fdr}} = 1.28 \times 10^{-5}$; with fixed effect covariates: b = 2.162, SE = 0.600, $p = 3.62 \times 10^{-4}$, $p_{\text{fdr}} = 0.600$ 344 $= 5.43 \times 10^{-4}$). 345 346 G1 borderline personality pathology indirectly linked non-specific childhood maltreatment 347 (i.e., CTQ total scores) experienced by G1 to non-specific childhood maltreatment experienced by 348 G2 (**Figure 1**; **Table 3**; b = 0.031 [95% CI 0.003, 0.060], p = 0.030). G1 childhood maltreatment 349 was positively coupled with G1 BPP (a pathway: b = 0.020 [95% CI 0.013, 0.027], p < 0.001)³ 350 which was associated with G2 childhood maltreatment (b pathway: b = 1.598 [95% CI 0.241, 351 2.955], p = 0.021) and indirectly linked G1 to G2 childhood maltreatment (c'pathway: b = 0.125352 [95% CI 0.013, 0.236], p = 0.021; c pathway: b = 0.142 [95% CI 0.055, 0.230], p = 0.001). Results 353 of the mediational model were consistent when using log transformed CTQ total scores (indirect 354 effect: b = 0.038 [95% CI 0.007, 0.069], p = 0.015; c' pathway: b = 0.116 [95% CI 0.028, 0.205], p = 0.010; c pathway: b = 0.153 [95% CI 0.059, 0.247], p = 0.001). 355

356 [Insert Figure 1 Here]

DISCUSSION

We evaluated whether the intergenerational transmission of childhood maltreatment (ITCM) is non-specific and whether parental borderline personality pathology mediates this continuity. Three primary findings emerged. First, we replicated a wealth of literature showing that childhood maltreatment is shared across generations (**Table 1**). Second, we found homotypic and heterotypic maltreatment type associations across generations (**Table 3**) with evidence that ITCM is largely shared across forms of maltreatment. Further, with the exception of a homotypic association of emotional neglect and a heterotypic association of G1 physical neglect and G2 sexual abuse, no specific forms of childhood maltreatment showed unique evidence of continuity across generations (**Table 4**). Third, parental borderline pathology partially mediated the association of global childhood maltreatment across generations (**Figure 1**; **Table 5**). Collectively, these data suggest that childhood maltreatment, broadly defined, is shared across generations and that parental borderline personality pathology may represent a shared risk factor associated with ITCM and/or a putative behavioral mechanism.

The Intergenerational Transmission of Broad Spectrum Childhood Maltreatment

The vast majority of childhood maltreatment research, including that on ITCM, has examined specific forms of maltreatment independently. While this approach is intuitively appealing, it does not account for common exposure to multiple forms of maltreatment and provides limited information with regard to the specificity of associations with distinct forms of childhood maltreatment.³ Most individuals maltreated during childhood are exposed to multiple forms of

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³ Studies showing that one form of maltreatment is significantly associated with an outcome while another is not may or may not be reflective of maltreatment type specificity. Forms of maltreatment that are rare and/or less severe may require relatively larger samples for the accurate estimation of their effects. Moreover, significant

abuse and neglect, and evidence indicates that a single general latent factor characterizes overall exposure well (Green et al., 2010; Kristjansson et al., 2016; Pezzoli, Antfolk, Hatoum, & Santtila, 2018; Spinhoven et al., 2014). Further, while limited studies have adopted such an approach, available evidence suggests that a general indicator of early life stress is associated with psychopathology, while unique aspects of specific forms of maltreatment are not (Green et al., 2010). Building on these findings, we find that broad exposure to childhood maltreatment is transmitted across generations with little evidence of unique distinct homotypic continuity. However, it may be that disentangling homotypic and heterotopic patterns of ITCM unique to specific forms of maltreatment requires much larger samples due to observed small effects.

Notably, unique aspects of emotional neglect unshared with the general childhood maltreatment factor showed evidence of homotypic continuity across generations (**Table 4**). While this finding was not hypothesized, the association survived correction for multiple testing and is broadly consistent with one prior study, in which maternal history of emotional neglect during childhood predicted poor parenting over and above a general maltreatment factor (Newcombe & Locke, 2001). Measurement and construct definitions of emotional neglect may contribute to this finding. With respect to measurement, emotional neglect is assessed on the CTQ entirely with reverse-scored items (e.g., "I felt loved, "My family was a source of strength and support"), which might constitute a relatively indirect measure of maltreatment that is conceivably more commonly endorsed outside of exposure to other forms (e.g., abuse) (Spinhoven et al., 2014). As a construct, some evidence suggests that neglect may be more specifically transmitted across generations than abuse; for example, Yang and colleagues (2018) found that parental history of neglect (broadly

associations with one form of childhood maltreatment may be attributable to shared variance with other forms as well as unique contributions.

conceived) was associated with neglect but not physical abuse in the next generation, while physical abuse was associated with both neglect and physical abuse. However, it is possible that differential endorsement rates between neglect and abuse could underlie these differential associations. Emotional neglect may be the most difficult form of maltreatment to define; unlike abuse, emotional neglect is often more chronic and less easily traced to specific instances, and there is a lack of consensus about what constitutes healthy emotional support (Stowman & Donohue, 2005).

Lastly, we observed a heterotypic association between unique aspects of physical neglect in G1 and unique aspects of sexual abuse in G2 that was negative (**Table 4**). This diverges from prior evidence documenting that neglect in one generation is positively correlated with sexual abuse in the subsequent one within documented cases of maltreatment (Widom et al., 2015). While our unanticipated finding survived multiple testing correction and had a similar directional pattern of association in subscale tests (i.e., subscales not orthogonal to general childhood maltreatment; **Table 3**), it should be noted that physical neglect and sexual abuse were the least highly endorsed subscales in this sample and further that physical neglect had the lowest internal consistency (see Methods), which may contribute to imprecise estimates of association.

Borderline Personality Pathology: A Putative Mediator of Childhood Maltreatment Generational Continuity

Clinical and subclinical forms of borderline personality pathology are associated with exposure to abuse and neglect in childhood and the perpetration of offspring maltreatment in parents (e.g., Ibrahim et al., 2018; Perepletchikova et al., 2012). Our study critically extends this literature by

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showing that parental BPP mediates the intergenerational transmission of childhood maltreatment.

Multiple, non-mutually exclusive pathways may contribute to this observation.

Consistent with theoretical models (e.g., Linehan, 1993) hypothesizing that childhood maltreatment plays a causal role in the development of BPP and associated evidence relating stressful family environments and predispositions for emotion dysregulation to the development of BPP, our findings show that maltreatment is associated with BPP development. In turn, the expression of BPP is associated with a heighten likelihood that one's children are exposed to abuse or neglect. A series of observations arising from epigenetic studies are consistent with theoretical models suggesting that childhood maltreatment may play a causal role in the development of BPP, which may, in turn, contribute to maltreatment in the subsequent generation. Studies in rodents have shown that maternal care affects later adult parenting behavior through epigenetic regulation of the glucocorticoid receptor gene (NR3C1) to influence stress-related cortisol function (Weaver et al., 2004). Specifically, cross fostering studies in rats show that pups raised by mothers who provide less maternal care (i.e., less licking and grooming and arched back nursing) are characterized by epigenetic changes resulting in an impaired ability to return to homeostasis following stress exposure. These changes persist throughout the rat's lifespan and promote stresssensitive behavior and reduced maternal care of the subsequent generation; hence less caring mothers beget relatively stress-sensitive pups that become less caring mothers through experience dependent mechanisms. However, this generational continuity can be disrupted; a pup born to a less caring mother who is subsequently raised by a more caring mother is characterized by epigenetic signatures associated with stress-resilience and increased maternal care in a subsequent generation. A human post mortem study suggests that this mechanism may be conserved across species (McGowan et al., 2009). Intriguingly, differential NR3C1 methylation patterns in

peripheral blood have been linked to both BPP and childhood maltreatment, suggesting that maltreatment-induced epigenetic signatures in stress responsive systems may contribute to the expression of BPP and the continuity of maltreatment (Dammann et al., 2011; Martín-Blanco et al., 2014; Perroud et al., 2011; Radtke et al., 2015; Steiger, Labonté, Groleau, Turecki, & Israel, 2013; Teschler et al., 2013).

Attachment theory offers further insight into the relationship between BPP and childhood maltreatment. For instance, evidence suggests that parents with BPP may be more likely to engage in inconsistent parenting styles that unpredictably oscillate between sensitivity and care to punishment (Stepp, Whalens, Pilkonis, Hipwell, & Levine, 2011). Theoretical models postulate that mothers with BPP are more likely to misperceive and invalidate their children's emotions, interfering with healthy emotional development and contributing to emotional dysregulation (Stepp et al., 2011). Such parenting styles are associated with the development of insecure, and more specifically disorganized, attachment, which may impair children's capacity for distress regulation (Crowell et al., 2009). Together with dysregulated emotion, insecure attachment, which is common among individuals with BPP and a history of childhood maltreatment (Fonagy et al., 1995), indirectly links parental exposure of childhood maltreatment to parental potential for abuse (Finzi-Dottan & Harel, 2014).

Other non-mutually exclusive pathways challenging conventional wisdom that childhood maltreatment contributes to the development of BPP may also explain our observed mediation. For instance, it is plausible that the early expression of BPP-like characteristics in children evokes childhood maltreatment from parents and others. In this sense the moderate heritability of BPP (Reichborn-Kjennerud et al., 2013) may be transmitted from parent to offspring, with resulting child behavior evoking maltreatment from those in the environment. As such, childhood

maltreatment may travel alongside BPP, while not contributing to its expression. It is also possible that childhood maltreatment evoked by prodromal BPP-like traits may further potentiate the development of BPP (Gunderson & Lyons-Ruth, 2008). Lastly, childhood maltreatment may not mechanistically contribute to BPP and BPP-like behavioral expressions in children may not evoke maltreatment. Instead, unmeasured familial factors may correlate with both childhood maltreatment and BPP. Twin research shows that BPP and related characteristics, as well as the perpetration of violence and likelihood of being exposed to maltreatment, have a moderate genetic component (Reichborn-Kjennerud et al., 2013; Pezzoli et al., 2018) with a recent GWAS identifying variation within genes linked to borderline personality disorder (Witt et al., 2017). While the genetic correlation between BPP and exposure to childhood maltreatment has not been estimated, to our knowledge, it is possible that common genetic influences on child maltreatment (both exposure for oneself and one's child) and BPP may account for their co-occurrence.

Strengths and Limitations

Our study is relatively large (n=937; **Table 1**) for a parent-child study and is characterized by multiple design recommendations for the study of ITCM (e.g., representative sample, independent reports of childhood maltreatment in both generations; see Thornberry et al., 2012) and a thorough assessment of borderline personality pathology (e.g., repeated evaluation over time, use of dimensional measurement and multiple sources of information). Further, unlike many ITCM studies, which focus on younger parents, our sample of older parents from the broader community allowed us to assess childhood maltreatment dimensionally throughout the entirety of childhood in both parents and their adult children, as opposed to only severe forms experienced during young childhood. However, our study has limitations to consider when interpreting results.

First, much like the majority of ITCM and parent-child studies, parental exposure to childhood maltreatment and borderline personality pathology were assessed with regard to one parent only, which prevented us from evaluating additive and interactive effects across parents/caregivers or other possible moderating factors (e.g., social support from other sources). Relatedly, our broad assessment of childhood maltreatment did not include details of who perpetrated the abuse/neglect. Given that borderline personality pathology is associated with aggression as well as familial instability, it is possible that child maltreatment may be perpetrated by parents with BPP and/or may arise from other caregivers or non-relatives. However, even if such measures were available, our sample size likely does not provide sufficient power to investigate these potentially complex interactive or specific effects.

Second, the CTQ is a retrospective and subjective measure of childhood trauma that may be susceptible to memory errors and reporting biases. For example, despite evidence that the CTQ shows convergence with clinician-rated childhood maltreatment interviews as well as strong test-retest reliability (Bernstein et al., 1994; Bernstein et al., 2003), a recent study found that retrospective self-reports of maltreatment on the CTQ have slight-fair agreement with prospective informant (i.e., caregiver, researchers, clinicians) reports and that the CTQ was associated with a relative underreporting of maltreatment and increased correspondence to psychopathology (Newbury et al., 2018). Thus, it is possible that G1 with higher levels of BPP were more likely to retrospectively report more childhood maltreatment; however, other evidence suggests that current psychopathology does not inflate the association between such retrospective reports and psychopathology (Fergusson, Horwood, & Boden, 2011). Relying on documented reports of childhood maltreatment also introduces problems, particularly for studies of ITCM, due to potential surveillance or detection bias. More specifically, among participants who self-report

childhood maltreatment, those whose parents have a documented history of exposure to childhood abuse and neglect themselves are more than twice as likely to have a CPS report than maltreated individuals whose parents have no documented history (Widom et al., 2015). As such, relying on documented cases of maltreatment may lead to an overestimation of intergenerational continuity, even while official records tend to underestimate exposure to maltreatment (Widom et al., 2015), and in particular exposure to multiple different forms (Kim et al., 2017). While there is clearly no current gold standard for the assessment of childhood maltreatment, our study would have benefited from a multisource assessment of childhood maltreatment (e.g., Widom et al., 2015) similar to our approach for characterizing borderline personality pathology.

Third, our assessment of borderline personality pathology among parents was conducted during later life and was temporally disconnected from the time these individuals were parenting their children. While borderline personality pathology is relatively stable (Bornovalova, Hicks, Iacono, & McGue, 2009), there is evidence that it "matures out" with age (Shea et al., 2009; Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012). Thus, it is plausible that associations between G1 BPP and G2 childhood maltreatment may be imprecise, as some parents may have expressed BPP while their children were young, but do not express it or express it with less severity in later life. More broadly, the lack of our ability to establish temporality with regard to relationships between borderline personality pathology and both G1 and G2 childhood maltreatment limits the inferences that can be generated from our mediational model (Maxwell, Cole, & Mitchell, 2011).

Fourth, though our study shows evidence that BPP indirectly links childhood maltreatment across generations, BPP is a heterogeneous construct. It is unclear what aspects or correlates of BPP may contribute to its association with ITCM. Given evidence that negative affectivity and

emotion dysregulation mediate the relationship between parent and child experiences of maltreatment (Smith et al., 2014), it is entirely possible that these facets of BPP are responsible for the current finding. Consistent with this speculation, maltreated children who score highly on measures of borderline symptomatology exhibit more intense emotional lability/negativity and are further rated by peers as being more disruptive, aggressive, and disliked, relative to children who did not experience maltreatment (Cicchetti et al., 2014). This evidence indicates that that affective instability, inappropriate anger and impulsivity, and unstable relationships may be the most important features of BPP in the context of ITCM. However, disarticulating these specific aspects of BPP would require a large clinical sample; the levels of BPP in the present sample do not have sufficient variability (see descriptive statistics provided in **Methods**).

Fifth, a substantial portion of individuals exposed to maltreatment during childhood experience multiple different forms of abuse/neglect (Kim, Mennen, & Trickett, 2017). This represents a major challenge to research attempting to evaluate associations of specific forms of childhood maltreatment. As such, it is possible that there are unique specific aspects of childhood maltreatment that show generational continuity, but that we are unable to detect them due to our assessment and/or the common occurrence of multiple different types of abuse/neglect that would require larger samples to detect.

Lastly, we evaluated childhood maltreatment across only two generations. Despite recent calls advocating for studies across three generations (Cheng Johnson, & Goodman, 2016), only a handful of studies to date have done so (e.g., Bailey, Hill, Oesterle, & Hawkins, 2009; Doumas, Margolin, & Jon, 1994; Lev-Wiesel, 2007). In order to identify the most enduring factors that contribute to ITCM, it will be important to extend this work to more than two generations.

Conclusions

Limitations notwithstanding, our study adds to a large literature documenting ITCM by replicating this relationship among a sample of older adults and their adult children and showing that childhood maltreatment is non-specifically transmitted across generations. The intergenerational transmission of non-specific maltreatment may be partially attributable to parental borderline personality pathology, which may plausibly reflect a behavioral mechanism underlying ITCM and/or emerge due to common risk contributing to exposure to childhood maltreatment, the expression of borderline personality pathology, and the likelihood that one's child is exposed to maltreatment. Providing treatment for borderline personality pathology in the context of parenting and development may help interrupt the continuity of childhood maltreatment and related health problems across generations.

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Figure 1. Parental Borderline Personality Pathology Indirectly Links Childhood Maltreatment Across Generations.

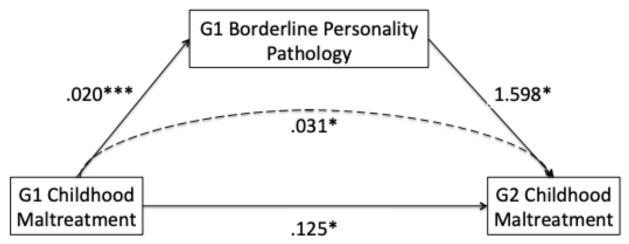


Figure 1. Parental Borderline Personality Pathology Indirectly Links Childhood Maltreatment Across Generations. G1 childhood maltreatment is associated with G1 Borderline Personality Pathology (path a), which is associated with G2 childhood maltreatment (path b). The association between G1 and G2 childhood maltreatment (path c: b=0.142, *p*<0.001) is attenuated by the inclusion of G1 Borderline Personality Pathology as a mediator (path c'). G1 and G2 childhood maltreatment represent latent general factors of childhood maltreatment generated from all CTQ items. G1 BPP represents a latent Borderline Personality Pathology factor gleaned from Borderline Personality Scores on the SIDP, participant- and informant-rated MAPP, and NEO-PI-R Personality Disorder Count Scores. The 95% CI represents a BCA simple bootstrapped confidence interval. This mediation model includes the following covariates: G1 and G2 Age, Gender, Race/Ethnicity (White, Black, Hispanic), and G1 Socioeconomic Status (a summed score of standardized education level and annual household income).

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

Table 1. Prior studies of intergenerational transmission of childhood maltreatment

Study Reference: Sample; Maltreatment Measure

Altemeier et al. (1986): Mothers (n = 927, 10.2% of whom experienced ch mal, A=20.8) and their infants (21-48 months); official reports of infant mal (protective agencies, Juvenile Court and DHS records), self-report mat exp to ch mal.

Appleyard et al. (2011): Mothers (n=499, A=27.3) and infants (n=499, A=0-26 months); PC-CTS, county records

Bartlett et al. (2012): Mothers (n=92, A=16.0) and children (A=7.9); PC-CTS, CPS reports.

Bartlett et al. (2017): Mothers (n=417, A=18.8) and children (A=4.8); DCF records. **Ben-David et al. (2015):** Low-income children (n=6,935) followed from age 1.5-11 and 18-26; Investigated report of maltreatment (statewide child welfare data).

Berlin et al. (2011): Mothers (n=499, *A*=27.3) and infants (*A*=0-26 months); PC-CTS, country records.

Bert et al. (2009): Mothers (n=681, *A*=19.8) and children; CTQ, KIDI, CAPI, PSEQ **Choi et al. (2018):** Mothers (n=1,016, *A*=28.0) and their same-sex 12-year-old twins (n=2,032, 51% female); CTQ, interview with mothers.

Conger et al. (2013): Parents (G2; n=290, A=25.6, 58.6% female), their children (G3; n=290, A=2.3, 48.9% female), and their mothers (G1); observation of G1 parenting during adolescence, observation of G2 parenting.

Cort et al. (2011): Mothers (n=104; A=31.3) and children (A=10-12); CTQ, CPS and DHS records

Dixon et al. (2005): Newborns (n=4,351; 3.1% of whom had caregivers who reported ch mal); Interview self-report (by mother or partner) and CPS referral.

Dixon et al. (2008): Newborns (n=4351; 3.1% of whom had caregivers who reported ch mal); Interview self-report (by mother or partner) and CPS referrals for infant mal.

Egeland et al. (1988): Mothers (n=267, *A*=20.5 at time of birth) and their children, from lower SES backgrounds; Interview, home observations (validated by official records)

Egeland et al. (1996): Mothers (n=24, A=20.5 at birth); Interview.

Finzi-Dottan et al. (2014): Parents (n=213, A=20-60) and children (A≤6); CAPI, CTQ.

Haapasalo et al. (1999): Mothers (n=50, A=40.0) and children (A=12.0) with (50%) and without CPS involvement; Structured interview, CPS records.

Herrenkohl et al. (2013): Children (G2; n=268, 48.5% female) followed into adulthood; composite G1 harsh phy discipline (par self-report and severity ratings from specialists), G2 reports of harsh phy discipline toward children

Heyman et al. (2002): Parents (n=2,704; 55% mothers) of ch under 18 years old; CTS and exposure to violence in parents during childhood.

Findings

No significant difference in frequency of reports to protective services between mothers who were and were not exp to ch mal.

Mat substance use mediates relationship between mat. exp to ch sexual and phy abu and infant os victimization.

Mat. exp to ch phy abu: greater neg of os.

Mat ch mal exp: increased perpetration of os mal (OR=2.52).

Ch mal exp: increased young adult perpetration of ch mal.

Mat exp to ch phy abu: increased os victimization, mediated by mat social isolation and aggression.

Mat exp to emo and phy abu associated with increased potential for child abu.

Mat postpartum depression symptoms mediated relationship between mat exp to ch mal and subsequent child exp to harm.

Romantic partner warmth and nurturing behaviors interrupted intergenerational continuity in harsh parenting

Mat exp to ch mal predicted os mal; no indirect effects through romantic attachment, intimate partner violence, or psychological distress.

History of mal associated with 4x greater risk of perpetration, increased to 17x when par were young, psychopathology history, and residing with a violent adult.

ITCM: increased caregiver isolation and serious financial difficulty.

Mothers who broke cycle of abu more likely to have emo support, therapy, and supportive partner; those who continued cycle has more life stress and were more anxious, dependent, immature, and depressed.

ITCM: idealization, inconsistency, and escapism, higher on dissociation in parents.

Serial mediation: history of mal. \rightarrow insecure attachment \rightarrow emo control \rightarrow cognitive appraisals of stress and viewing parenthood as threat \rightarrow potential of ch mal.

Mothers with CPS contact had history of more ch psychological abu. No differences in terms of self-reported abu toward one's child.

G1 harsh phy discipline predicted G2 harsh phy discipline of similar type. No mediating or moderating effects of safe, stable, and nurturing relationships.

Mat exp to 2 types of mal: increased family violence as adults. Frequency of exp to ch family violence predicted future child abu.

Jaffee et al. (2013): Mothers (n=1,116) with same-sex 5-year-old twins; CTQ, interview with mothers.

Kim (2009): Young parents (n=2,977, A=22.5; ch A≤2.60); Parent self-report of parenting behavior and experience of ch mal.

Lamela et al. (2013): Parents (n=924, A=37.12); CHQ, CAPI.

Leve et al. (2015): Women (n=166, A=15.3) followed for 10 years; CWS records and self-reported CWS contact, CTS.

Li et al. (2011): Children (n=405, 49.9% female) assessed at ages 4 and 8 and their mothers, from mostly high-poverty areas; mat CHLV, ch CPS records

Milaniak et al. (2014): Children assessed again in adulthood (n=1,196, A=29.2, 49% female); Official records of mal. (physical and sexual abu and neg.), perpetration of mal. assessed via arrest records and self-report.

Milner et al. (2010): Navy recruits (n=5,394, A=19.7) and college students (n=716, A=19.2); CAPI, CTS-PC, ch history of sexual abu survey.

Newcomb et al. (2001): Parents (n=383, *A*=35); CTQ, PARQ

Noll et al. (2008): Women (n=164; 50% sexually abu). (G2; A=11.1 at T1 and A=24.9 at T5); os (G3; n=123, 56.1% female); mothers' caregivers (G1; n=128); G2 CTI, G3 CPS involvement, G1 history of ch sexual abu (retrospective self-report developmental questionnaire).

Oliveria et al. (2012): Caregivers (n=10, A=42.8) with exp to ch mal who maltreated children; Semi-structured interview, clinical files of abu children.

Pears et al. (2001): Male children (n=109, A=10.0) and their par (n=179, A=36.2, 59.2% female) form high crime area; AE-III (par abu by parents), modified AE-III for children.

Plant et al. (2013): Mothers (n=125, A=26.3 at birth) and their children at age 16; Mat ch mal (retrospective reports; 2+ types of mal), CAPA

Putnam-Hornstein et al. (2015): Children (n=85,084) of first-time mothers (ages 15-19); CPS reports for both generations

Renner et al. (2006): Mothers (n=1,005, A=33.4) and their children; CPS reports, self-report of mat exp to ch mal.

Robboy et al. (2011): Adolescent girls evaluated for sexual abu (n=139, A=14.5); Self-, par-, medical examiner-, and forensic interviewer-reports of current and past mal; mat history of sexual abu.

Relative to families in which mothers but not children experienced mal those in which both generations experienced mal had more supportive intimate relationships, more maternal warmth, and less partner violence.

Par exp to ch neg more likely to report neg (2.6x) and phy abu (2x) of their children. Par exp to ch phy abu more likely to report phy abu (5x) and neg (1.4x).

Par psychosomatic symptoms partially mediate relationship between ch phy victimization and current mal risk toward children.

30-42% rate of intergenerational continuity, depending on measurement. Intervention reduced self-reported mal chronicity.

Mat history of ch mal predicted CPS reports of mal (OR=2.26).

History of ch abu and/or neg associated with perpetration of violence (relative risk = 1.26; criminal violence, child abu, intimate partner violence)

History of physical abu associated with greater odds (2-3x) of perpetrating physical abu,trauma symptoms mediated this relationship.

General factor of child mal associated with general factor of poor parenting in both mothers and fathers.

Children of mothers who were exp to ch sexual abu were more likely to be exp to ch sexual abu (G2) and be involved with CPS (G3).

Evidence of ITCM, but no difference in form of mal.

23% rate of intergenerational transmission. Parents of children who reported abu 2x as likely to have been abu. Parental depression, PTSD and consistency of parental discipline predicted child abu but did not mediate intergenerational relationship Mothers with history of ch mal and antenatal depression had os with higher levels of mal; only for os exp to mat depression in utero did mat ch mal contribute significant variance in predicting os mal.

Increased substantiated mal in os of mothers with history of CPS involvement between age 10 and conception; history of substantiated or unsubstantiated mal. strongest predictor of reported and substantiated os mal by age 5.

Some evidence for ITCM of neg, no evidence for phy abu

Adolescents whose mothers exp to ch sexual abu were more likely to experience poly-victimization than those whose mothers did not.

Rodriguez et al. (2018): Mothers (n=180-201 depending on time point, *A*=26.04 at T1) and fathers (n=140-146, *A*=28.87 at T1); Par mal history (CTS-PC, CES, KDA) and par-child aggression (PCA) risk (CAPI, AAPI-2, ReACCT, CTS-PC).

Sidebotham et al. (2001): Children (n=14,138) and their par; Par self-report of exp to ch mal, Social Services child protection registers

Smith et al. (2014): Primarily African American and low-income mothers (n=83, A=34.8) of children aged 6-13; CTQ, TEI, CAPI.

Thompson (2006): Mothers (n=220, A=26.2) and children; CPS records, mat history of victimization (as a child and as an adult, MHLH).

Thornberry et al. (2013): Female adolescents (n=748, *A*=13.6) followed into adulthood; CPS records for history and perpetration of mal

Tracy et al. (2018): Children (n=11,384; 48.4% female) and mothers (A=28.4 at birth); Mat self-report of ch mal, report of os mal (by mother or partner) and timing of mal, os self-report of phy violence in adulthood

Valentino et al. (2012): Mothers (n=70, *A*=17.4 at birth) and children (n=70) followed 18 years; CTQ (mothers and os abu subscales only).

Widom et al. (2015): Children with documented cases of mal followed longitudinally (n=649, A=47.0 at assessment) and matched comparisons (n=497) and their children (n=697, A=22.8); CPS records for history of mal and reports of mal their children, number of filed reports against participants and chronicity of reports, self-reports by par or other caregivers; self-report measures to asses their children's histories of mal. **Yang et al. (2018):** Mothers (n=1200, A=30.7) and children; CPS records of mat neg and phy abu; mat self-report of exp to ch neg and phy abu.

History of physical and psychological aggression predicted PCA risk prenatally and during toddlerhood, for both mothers and fathers. Different mediators and moderators of mal continuity among mothers and fathers.

Only mat exp to ch sexual abu significantly predicted child mal

Mat negative affect and emotional dysregulation mediated relationship between mat exp to ch abu and abu potential.

Association between mat history of phy victimization and risk of mal their children disappeared when marital status, depressive symptoms, and adult experiences of victimization were added to model

Adolescent, but not ch-limited mal, increases odds of later perpetration of mal

Among mothers with and without history of mal but not currently experiencing intimate partner violence, social support reduced mal risk.

Exp to community violence and lower authoritarian parenting attitudes increased risk for continuity of abu across generations. Authoritarian attitudes were only protective among African American families

Strongest evidence for intergenerational transmission of neg and sexual abu; little evidence for phy abu. Par with documented mal more likely to have a CPS report with os compared to matched controls.

Mat neg history increased risk of neg their children, while their phy abu history increased risk of both neg and physically abu their children. Mat depressive symptoms mediated transmission of mat neg to child phy abu but not mat phy abu to child neg.

Note. AAPI-2 = Adult Adolescent Parenting Inventory-2 (Bavolek & Keene, 2001); AE-III = Assessing Environments-III questionnaire (Berger, Knutson, Mehm, & Perkins, 1988); CAPI = Child Abuse Potential Inventory (Milner, 2004); CES = Compliance Expectations Scale (Rodriguez, Smith, & Silvia, 2016); CHLV = Caregiver's History of Loss and Victimization (Hunter & Everson, 1991); CHQ = Childhood History Questionnaire (Milner, Robertson, & Rogers, 1990); CIDI-SF = Composite International Diagnostic Interview-Short Form (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998); CPS = Child Protective Services; CTQ = Childhood Trauma Questionnaire (Bernstein & Fink, 1998); CTS = Conflict Tactics Scale (PC = Primary Caregiver; Straus, 1979); CWS = Child Welfare Services; DCF = Department of Children and Families; KDA = Knowledge of Discipline Alternatives (Rodriguez et al., 2016); KIDI = Knowledge of Infant Development Inventory (MacPhee, 1981); MHLH = Mother's History of Loss and Harm (Hunter & Everson, 1991); PARQ = Parental Acceptance-Rejection Questionnaire (Rohner, 1991); PSEQ = Parenting Style Expectations Questionnaire (Bavolek, 1984); ReACCT = Response Analog to Child Compliance Task (Rodriguez, 2016). A= Average Age. Mat = Maternal. Exp = Exposure. Abu = Abuse. Neg = Neglect. Ch = childhood. Mal = maltreatment. OS = offspring. Phy = physical. Emo = emotional. Par. = parent. A = mean reported age, if not available, range is reported. Reported in years unless otherwise noted.

Table 2. Demographic and study characteristics of G1 and G2 generations

Variable	G1%(n)/M(SD)	G2%(n)/M(SD)
Age	66.7 (3.01)	38.7 (7.39)
Race		
White	71.7 (261)	73.7 (421)
Black	24.7 (90)	22.9 (131)
Gender		
Female	59.3 (216)	44.0 (251)
Male	40.7 (148)	56.0 (320)
Education		
High school or less	12.7 (46)	6.3 (36)
Some college, vocational or associates	29.3 (106)	25.9 (148)
College degree	27.4 (99)	35.9 (205)
Graduate degree	30.7 (111)	31.9 (182)
Annual household income (baseline)		
Under \$20,000	9.7 (34)	9.0 (51)
\$20,000 - \$40,000	12.0 (42)	12.4 (70)
\$40,000 - \$60,000	20.6 (72)	15.9 (90)
\$60,000 - \$80,000	15.7 (55)	10.8 (61)
\$80,000 - \$100,000	8.0 (28)	10.8 (61)
\$100,000 - \$120,000	9.4 (33)	9.2 (52)
\$120,000 - \$140,000	7.1 (25)	9.4 (53)
\$140,000 or more	17.4 (61)	22.6 (128)
Childhood Trauma		
Emotional Abuse	7.85 (4.22)	7.88 (3.93)
Physical Abuse	7.06 (3.16)	6.48 (2.49)
Sexual Abuse	6.40 (3.51)	6.33 (3.62)
Emotional Neglect	8.80 (4.42)	8.37 (3.77)
Physical Neglect	6.40 (2.57)	6.11 (2.06)
Borderline Personality Pathology*		
SIDP	0.70 (1.15)	
PMAPP	3.40 (2.76)	
IMAPP	4.64 (4.11)	
PNEO	117.47 (20.68)	
INEO	122.99 (28.62)	

Note. G1 = parents; G2 = children; SIDP = Structured Interview for DSM-IV Personality; PMAPP = Participant-rated Multisource Assessment of Personality Pathology; IMAPP = Informant-rated Multisource Assessment of Personality Pathology; PNEO = Participant-rated Revised NEO Personality Inventory; INEO = Informant-rated Revised NEO Personality Inventory

^{*} Total scores, averaged across the three time points.

Table 3. CTQ Subscale Summary Score Associations Across Generations

G2 Childhood Abuse and Neglect						
		Abuse			Neglect	
G1 CTQ	Emotional	Physical	Sexual	Emotional	Physical	
Emotional Abuse	0.17*	0.12*	0.01	0.17*	0.07	
Physical Abuse	0.14*	0.15*	0.001	0.11*	0.06	
Sexual Abuse	0.16*	0.15*	0.07	0.12*	0.12*	
Emotional Neglect	0.11*	0.07	-0.05	0.19*	0.06	
Physical Neglect	0.09	0.10	-0.10	0.12*	0.08	

Note. N = 568; k = 362, where k = number of families. * = pfdr < 0.05. G1 and G2 age, sex, and race, and G1 SES, were included as covariates. All estimates reflect standardized values.

Table 4. Unique CTQ Subscale Factor Score Associations Across Generations

	G2 Childhood Abuse and Neglect					
		Abuse			Neglect	
G1 CTQ	Emotional	Physical	Sexual	Emotional	Physical	
Emotional Abuse	0.05	0.0003	0.06	-0.02	01	
Physical Abuse	0.01	0.02	-0.05	-0.08	-0.05	
Sexual Abuse	0.02	0.04	0.01	-0.04	0.02	
Emotional Neglect	-0.04	-0.01	-0.07	0.16*	0.02	
Physical Neglect	-0.003	0.08	-0.13*	0.01	0.08	

Note. N = 569; k = 362, where k = number of families. *= pfdr < 0.05. G1 and G2 age, sex, and race, and G1 SES, were included as covariates. All estimates reflect standardized values.

Table 5. Mediation Model Statistics: G1 Borderline Personality Pathology Indirectly Links Childhood Maltreatment Across Generations

	Borderline Pathology (a pathway)		G2 Child Malt (b and c' path	
	b	р	b	р
G1 Gender	-0.060	0.496	-3.175	0.511
G1 White	0.420	< 0.000	-3.454	0.071
G1 Age	-0.030	0.052	0.031	0.951
G1 SES	-0.094	0.001	0.096	0.954
G2 Gender			-51.704	0.329
G2 Age			0.192	0.646
G1 Child Maltx	0.020	<0.000	0.125	0.028
G1 Borderline PP			1.598	0.021

Note. b = unstandardized beta regression coefficient. P = p-value of association.

PP = Personality pathology. SES = Socioeconomic status. C'pathway: b = 0.125 [95% CI 0.013, 0.236], p = 0.021; C pathway: b = 0.142 [95% CI 0.055, 0.230], p = 0.001