Citation for published version (APA):
Developmental pathways to adolescent callous-unemotional traits: The role of environmental adversity, symptoms of borderline personality and post-traumatic disorder

Edward D. Barker, PhD and Alan J. Meehan, MSc

Institute of Psychiatry, Psychology & Neuroscience, King's College London, UK.

Corresponding Author: Edward D. Barker, PhD, Department of Psychology, Institute of Psychiatry, Psychology and Neuroscience, King’s College London, 16 De Crespigny Park, London, SE5 8AF. Phone: +44 (0) 207 848 0992. Email: ted.barker@kcl.ac.uk
**Introduction**

Psychopathy is a personality disorder that involves a constellation of interpersonal (e.g. superficial, grandiose, manipulative), affective (e.g. lack of empathy, remorse, or guilt, shallow emotional expression), and behavioral (e.g. impulsive, irresponsible, and/or antisocial) features (Cleckley, 1941, Hare and Neumann, 2006). This construct has been linked to high rates of community violence, violent and nonviolent criminal recidivism, institutional management difficulties and poor treatment outcomes (Yang et al., 2010, Salekin, 2008, Kiehl and Hoffman, 2011). With an estimated prevalence of 15-25% in forensic settings and 1% in the general population, the societal burden for psychopathic behavior is estimated to cost $460 billion per year in criminal social costs (Kiehl and Hoffman, 2011). Understanding the developmental precursors for psychopathy may therefore aid preventive efforts and lessen this societal burden.

It has been suggested that environmental adversity can influence the development of psychopathy. Based on clinical observations, Karpman (1941, 1948) believed that individuals may show a similar expression of psychopathy, but with different etiological origins. He theorised that primary (‘idiopathic’) psychopathy, characterized by a lack of anxiety, had no obvious environmental correlates, suggesting a more heritable, or biologically driven affective deficit. In contrast, the proposed secondary (‘symptomatic’ or ‘neurotic’) psychopath experienced negative emotions, particularly high levels of anxiety and emotional distress, in response to early environmental adversities, including parental abuse or maltreatment. Due to its distinct environmentally-based etiological underpinnings, it was suggested that the secondary subtype may be more responsive to treatment (i.e. sensitive to environmental input) than the primary subtype (Karpman, 1941, Karpman, 1948).
Historically, the role of biological correlates has received more attention than the environment, with psychopathy being viewed as a somewhat unitary construct, derived from a complex but relatively homogeneous pattern of heritable/congenital factors (see Poythress and Skeem, 2006, Porter, 1996). Nevertheless, in the last decade Karpman’s ideas have been revisited due to emerging research reporting that psychopathy can associate with both environment adversity (Dargis et al., 2016, Poythress et al., 2006) and stress-related disorders such as anxiety (Kubak and Salekin, 2009, Hicks et al., 2004) and depression (Price et al., 2013). These studies have largely been focused on adults and cross-sectional in design; hence, examining the early predictors that related to subsequent psychopathy has proven difficult.

One way to examine the etiological origins of psychopathy has been to examine its hypothesized developmental precursor in youth, callous-unemotional (CU) traits (i.e. lack of empathy/guilt, shallow affect), which represent the interpersonal-affective component of psychopathy. Research suggests that CU traits are not immutable in youth, and hence may be somewhat malleable to environmental influence (cf. Meehan et al., 2017). For example, developmental trajectories for psychopathic traits throughout childhood have identified a significant number of youth whose levels of psychopathic traits change (i.e. increase, decrease) over time (Byrd et al., 2016, Fontaine et al., 2010). In addition, general adversities, ranging from stressful life events to child maltreatment, have been found to associate with increased childhood CU and adolescent psychopathy (Farrington et al., 2010, Sharf et al., 2014, Barker et al., 2011b). In particular, negative life events can have strong effects on CU. For example, Barker and Salekin (2012) reported a direct link between victimization by peers (i.e. been hit, had things stolen, called names, had lies told about them) between ages 8-10 and subsequent CU traits at age 13.
Recent research has suggested that, in addition to anxiety and depression, psychopathy can associate with borderline personality disorder (BPD; Skeem et al., 2007) and post-traumatic stress disorder (PTSD; Porter, 1996), which are both psychopathological conditions associated with exposure to adverse/traumatic events. BPD is a serious mental illness associated with interpersonal dysfunction, affective dysregulation, self-harm and severe behavioral and emotional dysregulation (Leichsenring et al., 2011). BPD in youth is also associated with a range of environmental adversities, including prenatal risk factors (i.e. substance use, psychopathology; Winsper et al., 2015), harsh treatment in the family environment (Belsky et al., 2012) and being victimized by peers (Wolke et al., 2012).

BPD is also highly comorbid with both psychopathy and PTSD. BPD associates with constructs related to psychopathy, as well as psychopathy itself. For example, the National Epidemiologic Survey on Alcohol and Related Condition (Sanislow et al., 2002) reported that BPD associated with mood disorders, anxiety and, importantly, narcissistic personality. This finding deserves attention as, similarly, Paulhus and Williams (2002) identified a “Dark Triad” of psychopathic-associated personality styles that include Machiavellianism and narcissism. Following up on this “Dark Triad,” Miller et al. (2010) reported that BPD symptoms associated more with “vulnerable narcissism,” which reflects a defensive and fragile grandiosity that may serve to mask feelings of inadequacy, and is related to psychological distress and dysfunction. Moreover, in a non-clinical sample of French adolescents, Chabrol and Leichsenring (2006) reported that BPD symptoms associated with overall CU traits. Taken together, these results suggest BPD could index a profile that may be similar to the secondary psychopath.
High rates of BPD-PTSD comorbidity have been found in both clinical and community samples (Zanarini et al., 1998). This association is thought to be (partially) explained by shared cognitive biases for anger and threat due to traumatic and stressful experiences (Lobbestael and McNally, 2016). Indeed, a traumatic event is part of the symptomatology of PTSD in DSM-5, which include: persistent intrusions of the stressful event(s) (flashbacks, recurrent and distressing recollection for dreams), persistent avoidance of stimuli associated with the trauma (e.g. efforts to avoid external reminders), negative alterations in cognition or mood (numbing of general responsiveness), and persistent symptoms of increased arousal and reactivity (American Psychiatric Association, 2013).

Porter (1996) suggested that an association between PTSD symptoms and psychopathy might signpost individuals similar to the secondary psychopath, whose psychopathy develops as a result of having experienced adversity-related "de-activation" of basic affect and conscience. Notably, the association between PTSD symptoms and psychopathy may be higher in females than males. For example, in a general population sample of adults, Colins et al. (2017) reported that psychopathy associated with lower physical aggression but higher PTSD symptoms for females versus males. Likewise, Hicks et al. (2010) found that, in an adult female sample of incarcerated adults, PTSD symptoms associated with greater mental health problems, including psychopathy. However, similar results have been found with regard to CU traits within an adolescent sample of incarcerated males (Sharf et al., 2014) and in a mixed-gender community sample (Kahn et al., 2013). Hence, a consistent profile of sex differences in the association between PTSD and psychopathy is not firmly established.
The aforementioned body of research suggests that predictor domains for CU may inter-correlate, as in Figure 1, panel A. We hypothesize that the dynamic model (cf. Dodge et al., 2008) represented in Figure 1, panel B will describe this developmental process (but at the same time recognize that alternate cascade models could hold as well). This model begins with the child’s fetal development and birth into an adverse social context, characterized by stressful life events, parental psychopathology, poverty, family conflict and direct child victimization (via caregiver or peers). It is hypothesized that this adverse developmental context operates on adolescence CU primarily through affecting inter-relationships with peers. Children from high risk backgrounds, defined by both poverty and caregiver psychopathology, are at increased risk for chronic bullying and victimization experiences (Barker et al., 2008b). Victimization, in turn, may associate with the affective/emotional instability and interpersonal difficulties that are part of BPD (see Wolke et al., 2012). BPD symptoms in themselves may increase vulnerability for PTSD symptoms, via shared symptomology, including cognitive biases for anger and threat due to earlier traumatic and stressful experiences (Lobbestael and McNally, 2016), which, in this dynamic model, include both early adversity and victimization. Finally, PTSD symptoms, themselves associated with earlier adversity, victimization and BPD symptoms, are predicted to associate with higher CU traits via emotional numbing, due to these repetitive negative experiences. We also tested for sex differences in these pathways, given previous findings.

**Method**

**Sample**

The Avon Longitudinal Study of Parents and Children (ALSPAC) is an ongoing epidemiological study established in the UK to understand how genetic and
environmental characteristics influence health and development in parents and children. Pregnant women resident in the former Avon Health Authority with expected delivery dates between April 1, 1991 and December 31, 1992, were eligible for recruitment, resulting in a cohort of 14,541 pregnancies, of which 13,988 singletons/twins were alive at 12 months of age. ALSPAC has been found to be representative of the general UK population, based on 1991 National Census Data (Boyd et al., 2013). Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. The study website contains details of all the data that is available through a fully searchable data dictionary: http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/.

Measures

**Callous-unemotional traits** was assessed through a six-item questionnaire completed by mothers when their child was 13 years old (Moran et al., 2008). Items were rated on a three-point scale, from ‘not true’ to ‘certainly true’: (i) makes a good impression at first but people tend to see through him/her after they get to know him/her; (ii) shallow or fast-changing emotions; (iii) is usually genuinely sorry if s/he has hurt someone or acted badly (reverse coded); (iv) can seem cold-blooded or callous; (v) keeps promises (reverse coded); and (vi) is genuine in his/her expression of emotions (reverse coded). Items were selected based on factor analyses of scales measuring CU traits (Frick et al., 1994, Frick et al., 2000). The measure correlated highly ($r = .81$) with the CU scale of the Antisocial Process Screening Device (APSD) in 182 children aged 9-17 displaying antisocial behavior (Moran et al., 2009), and has previously shown acceptable internal reliability via confirmatory factor analysis (CFA; Barker et al., 2011).
Borderline personality disorder symptoms were assessed using the UK Childhood Interview for DSM-IV Borderline Personality Disorder (UK-CI-BPD; Zanarini et al. 2004), a face-to-face semi-structured interview based on the DSM-IV criteria for borderline personality disorder. The interview consists of nine sections: intense inappropriate anger; affective instability; emptiness; identity disturbance; paranoid ideation; abandonment; suicidal or self-mutilating behaviors; impulsivity; and intense unstable relationships. Interviewers made a judgement as to whether each symptom was ‘definitely present’, having occurred very frequently (i.e. daily or at least 25% of the time); ‘probably present’ if it had occurred repeatedly but did not meet the criterion for definitely present; or ‘not present’ if it had not occurred. The derived outcome variable summed index of all ‘definitely present’ symptoms. The reliability and validity of this measure has been established elsewhere (Zanarini et al., 2011).

Post-traumatic stress disorder symptoms were assessed using maternal reports on the well-validated Development and Well-Being Assessment interview (DAWBA; Goodman et al., 2011). The DAWBA was administered via a computer-based package of questionnaires, interviews, and rating techniques used to assess adolescent psychopathology based on DSM-IV criteria. Each question was introduced with the stem: ‘over the last 6 months, and as compared with other children the same age, has s/he often . . . .’ followed by the specific clause. Response categories were 0 = no, 1 = a little more than others, 2 = a lot more than others. We defined PTSD by the average of the xx symptoms: 1) relived stressful events with vivid memories, 2) repeated bad dreams of stressful event, 3) upset by reminders of stressful event, 4) avoided talking about stressful event, 5) avoided activities/places/people related to stressful event, 6) blocked out details of stressful event from memory, 7) reduced
interest in activities, 8) reduced range of feelings, problems sleeping, 9) seemed irritable/angry, 10) difficulty concentrating, and 11) alert for possible dangers. We created a summed index of these 11 PTSD symptoms.

**Peer Victimization** was measured using child reports, collected at ALSPAC’s Child in Focus Clinics (see Schreier et al., 2009). Children indicated how often (1 = never to 4 often) they had: 1) been hit; 2) had belongings stolen, 3) been called names, and 4) had lies told about them. These 4 items showed acceptable internal reliability at ages 8 and 10 via confirmatory factor analyses (Barker and Salekin, 2012)

**Early adversity** was assessed based on maternal reports. Risk items were organized into two developmental eras: (i) prenatal risks (18 weeks to 32 weeks) and (ii) early childhood risks (birth – age 7). For each developmental period, items were organized to create distinct but correlated risk domains: (i) *Life events* (e.g. death in family, accident, illness), (ii) *Contextual risks* (e.g. poor housing conditions, financial problems), (iii) *Parental risks* (e.g. parental psychopathology, criminal involvement and substance use), (iv) *Interpersonal risks* (e.g. intimate partner violence, family conflict), and (v) *Direct victimization* (e.g. child bullied by peers or physically hurt; available for birth to age 7 postnatal risk). These global risk scores showed high internal reliability via confirmatory factor analyses of the individual risk domains and also to extract one global risk score for each developmental era (Cecil et al., 2014).

**Selected sample of ALSPAC mothers and children**

Of the 13,988 mother-child pairs, 4039 (53% female) had information about BPD, PTSD and CU. These were the mothers and children included in the present study. Compared to those included, excluded mothers were lower in educational attainment (Odds Ratio [OR]=1.62, 95% Confidence Interval [95% CI] = 1.40, 1.88),
had early birth or pregnancy (OR=1.88, 95% CI=1.51, 2.34) and higher in poverty (OR=1.44, 95% CI=1.25, 1.67).

**Statistical Analysis**

For all analyses, we first examined the overall sample and then tested for sex differences. The analyses proceeded in two basic steps. In the first step, we conducted longitudinal path analysis. Here, we tested a developmental cascade where we hypothesized that predictor domains for CU may inter-correlate over time to influence CU. Specifically, the hypothesized cascade begins with cumulative adversity (prenatal to age 7), higher levels of which would associate with higher victimization by peer experiences, which in turn, would associate with higher BPD symptoms, which in turn, would associate with higher PTSD symptoms, which in turn, would then associate with higher CU. In addition, we also estimated a direct effect of each predictor domain on CU. Hence it was possible to examine smaller sets of cascading effects in addition to the total cascade; for example, if higher victimization associates with higher BPD symptoms, which in turn, associate with higher CU (above and beyond the other estimated parameters in the model). Sex differences were tested through multiple group models and nested model comparisons (i.e. chi-square difference tests).

The different cascades were described via indirect effects. The effects were defined by the product term of the pathways of interest (i.e., ‘early adversity to victimization’ BY ‘victimization to BDP’ BY ‘BPD to PTSD’ BY ‘PTSD to CU’). Because standard errors underlying indirect effects (i.e. product terms) are known to be skewed, we bootstrapped all indirect effects 10,000 times with bias corrected 95% confidence intervals. The indirect pathways reported below are based on the bootstrapped variability around the product of standardized path coefficient estimates.
We tested sex differences in indirect pathways (e.g. males vs females) by bootstrapping the difference of the respective indirect pathways. We tested differences in the unstandardized estimates.

The second step was reserved for following up any identified sex differences in the first step. For example, if we found that an indirect pathway involving PTSD or BPD symptoms was higher for males vs females, we would then break down the overall PTSD or BPD scores into their sub-domains and further explore these sex differences. Indirect pathways, as described above, were also examined in these follow-up analyses.

Model fit was determined through the Comparative Fit Index and Tucker-Lewis Index (CFI & TLI; acceptable fit => 0.90) (Bentler and Bonett, 1980) and root mean square error of approximation (RMSEA; acceptable fit <= 0.08) (Browne and Cudeck, 1993). Maximum likelihood estimation with robust standard errors was used to estimate the model parameters, and missing data were handled through full information maximum likelihood. All analyses were conducted using Mplus Version 7.2 for Windows (Muthén and Muthén, 1998-2016).

**Results**

Prior to discussing the results, we first describe the correlations and means of the variables. As can be seen in Table 1, for females (top) and males (bottom), CU significantly associated with victimization, BPD, PTSD and early adversity. For males, BPD and PTSD significantly associated with each other, and associated with victimization and early adversity. For females, BPD and PTSD did not significantly associate with each other, but as with males, they did associate with victimization and adversity. There were no significant sex differences in mean levels of the variables.

**Step 1: Developmental cascade path analytic model**
The cascading model fit the data adequately: $\chi^2(37) = 233.19, p < .0001; \text{CFI} = .99, \text{TLI} = .94; \text{RMSEA} = .039 (90\% \text{CI} = .032 - .041)$. As hypothesized (see Figure 2, panel A), higher levels of early adversity prospectively associated with higher victimization ($b = 0.193$), which in turn, associated with higher BPD symptoms ($b = 0.371$), which in turn, associated with higher PTSD ($0.065$), which in turn associated with higher CU ($b = 0.099$). Significant individual direct effects on CU were as follows: early adversity ($b = 0.193$), victimization ($b = 0.077$) and BPD symptoms ($b = 0.109$). For the intermediate effects between the domain predictors, early adversity did not significantly associate with BPD symptoms, and victimization did not associate with PTSD symptoms; however, higher early adversity did significantly associate with higher PTSD symptoms ($b = 0.142$).

Indirect effects based on the significant associations described above are located in Table 1. The ‘grand’ cascade, from early adversity (18th week of gestation - age 7) to CU (age 13) via victimization (age 8-10), BPD symptoms (age 11) and PTSD symptoms (age 13) had a standardized effect size of 0.000. This developmental pathway was not supported by the data. Two indirect pathways (i.e. cascades) did show bootstrapped estimates with 95% confidence intervals that did not cross zero. These pathways included: (Effect 2) higher early adversity to higher CU via higher PTSD symptoms and (Effect 4) higher victimization to higher CU via higher BPD symptoms.

Next, we tested sex differences among the parameters involved in these indirect pathways (see Figure 2, panel B). For the association between BPD symptoms and CU, males had a higher estimate ($b = 0.163$) than females ($b = 0.060$). The difference between these estimates was on trend: $\Delta \chi^2 (df = 1) = 3.648, p = 0.056$. In addition, for the association between PTSD symptoms and CU, males had a lower
estimate \( (b = 0.060) \) than females \( (b = 0.129) \). The difference between these estimates was not on trend: \( \Delta \chi^2 (df = 1) = 2.338, p = 0.126 \).

**Step 2: Exploratory follow-up analyses based on observed sex differences**

Given the ‘on trend’ difference for males vs females in the relationship between BPD symptoms and CU, in a highly exploratory manner, we examined symptom domains for BPD that showed sufficient variability to further unpack potential differences. Here, self-harm was quite rare; hence, we did not follow up on these symptoms. We did, however, find sufficient variability in disturbed identity (i.e. frequent mood changes, felt empty, paranoid feelings), unstable interpersonal relationships (i.e. changed mind from love to hate, had stormy relationships, stopped talking/seeing people), and inappropriate intense anger, which will henceforth be referred to as ‘violence’ (i.e. threaten someone, shoved/slapped/punched/kicked someone, been in a fistfight, deliberately damaged property). Of note, these symptoms domains reflect a previously reported three factor solution of BPD symptoms (Sanislow et al., 2002).

We therefore estimated a new cascade model (see Figure 2) that focused on summed sub-scales of these symptom domains. This model showed adequate fit to the data: \( \chi^2(104)= 430.112, p < .0001; \) CFI = .94, TLI = .91; RMSEA = .040 (90% CI = .036 - .044). Early adversity did not significantly associate with the BPD symptom domains. Higher victimization, however, was significantly associated with higher identity disturbance and unstable relationships for females and males alike, but with higher violence for males \( (b = 0.262) \) but not females \( (b = 0.093) \) – a difference that was significant: \( \Delta \chi^2 (df = 1) = 7.053, p = 0.008 \). Higher violence also associated with higher CU for males \( (b = 0.205) \) but not females \( (b = 0.061) \); however, this difference was not significant: \( \Delta \chi^2 (df = 1) = 2.701, p = 0.100 \). Higher identity disturbance
associated with higher CU for males ($b = 0.092$) but not (significantly) for females ($b = 0.002$), a difference that was also non-significant: $\Delta \chi^2(df = 1) = 3.661, p = 0.056$.

Females showed higher estimates than males for two consecutive associations that could form an indirect pathway, but neither significantly differed from the estimates for males: higher violence to higher PTSD ($b_{\text{male}} = 0.043; b_{\text{female}} = 0.085; \Delta \chi^2[df = 1] = 1.336, p = 0.247$) and higher PTSD to higher CU ($b_{\text{male}} = 0.060; b_{\text{female}} = 0.130; \Delta \chi^2[df = 1] = 2.112, p = 0.146$).

Based on these results, we examined potential sex differences in the indirect pathways that involved victimization, violence, and CU. As presented in Table 2, the 95% bias corrected confidence for males (not females) did not cross zero for the indirect pathway from higher adversity to higher CU (via higher victimization and higher identity disturbance). The 95% bias corrected confidence intervals for difference in the indirect pathways for males and females, however, did cross zero: $b_{\text{diff-unstandardized}} = 0.002, 95\% \text{ CIs: } -0.002, 0.005$.

**Discussion**

This study reports three major findings that, together, support a cascading model showing how CU in adolescence develops from pregnancy to through adolescence. The major contribution of this study is an improved understanding how predictor domains inter-correlate over time to associate directly and indirectly with CU traits.

First, each of the four predictor domains associated with CU above and beyond each other. Hence, each made a unique/additive contribution to adolescence CU and these prospective patterns were generalized across both male and female groups. The current study also provides a highly detailed account of how these temporally adjacent predictor domains inter-correlated and related to CU. All of the temporally adjacent risk domains were significantly related to each other; however,
adversity did not associate with BPD (above and beyond victimization) and victimization did not associate with PTSD (above and beyond BPD). In addition, the overall indirect effect of adversity leading to CU via victimization, BPD and PTSD, was not supported by the data. Instead, smaller chains of indirect effects were identified: for example, early adversity associated with CU via PTSD and victimization by peers associated with CU via BPD.

The second major finding of this study is interpreting why these ‘smaller’ cascading patterns might emerge. The relationship between early (pre- and postnatal) adversity and PTSD symptoms at age 13, which in turn, associated with higher CU, may support the “latent vulnerability” hypothesis (McCrory et al., 2017). McCrory et al. (2017) suggest that early trauma/adversity can form a latent vulnerability, which is defined as a complex phenotype that functions as a “maladaptive calibration” in neural systems important for socioemotional and affective functioning. However, they also state that the emergence of the psychiatric difficulties may only manifest under conditions of stress – in the present case, perhaps, the onset of adolescence. Indeed, there is a large body of literature that has examined how prenatal and postnatal adversity may affect neural systems underlying neurocognitive function and brain development (Lupien et al., 2009, Jensen et al., 2015, Blair and Raver, 2016). Of interest, brain areas that are affected by adversity/trauma are also (largely) implicated in PTSD symptoms – and hence potentially secondary psychopathy. These areas include the amygdala (threat hypervigilance), the striatum (reward processing, depressive symptomatology) and anterior cingular cortex (emotion regulation) (see McCrory et al., 2017, McCrory and Viding, 2015). We therefore suggest that a latent vulnerability may underlie the relationship between early adversity, PTSD symptoms and CU. However, additional research is needed to identify the conditions of stress (in
adolescence) that may have potentiated this latent vulnerability and the manifestation of an association between PTSD symptoms and CU traits.

The other indirect effect was peer victimization associating with BPD symptoms, which in turn, associated with CU. Of note, the association between BPD symptoms and CU was the only effect with a significant sex difference – for males not females. We therefore unpacked BPD into the symptom domains of disturbed identity (i.e. frequent mood changes, felt empty, paranoid feelings), unstable interpersonal relationships (i.e. changed mind from love to hate, had stormy relationships, stopped talking/seeing people), and violence (inappropriate intense anger, violent behavior). Of interest, for males and females alike, peer victimization associated with higher disturbed identity and instability in relationships, suggesting the ubiquitous and damaging long-term effects of chronic victimization (Takizawa et al., 2014, Barker et al., 2008a)

For males alone, however, early adversity associated with peer victimization, which in turn associated with BPD-disturbed identity and thereafter CU – the overall developmental cascade. Bateman and Fonagy (2008) suggest that individuals showing symptoms of trauma-related BPD may have rigid schematic representations of the mental states of self and others (i.e. mentalization), particularly in their explanations of others’ behavior. Here, interpersonal stress may result in the aforementioned “vulnerable narcissism” (Miller et al., 2010) that can create an atmosphere of distress and fear (perhaps related to previous adversity and victimization), which, in turn, could lessen the concern for the welfare of others (i.e. enhance callous-unemotional traits). To the extent that emotional distress may characterize certain youth showing CU traits, BPD-disturbed identity may be a construct of interest to future research – it
has been reported that long-term effects of chronic victimization by peers includes both social paranoia and negative affect (see Singham et al., 2017).

The third major finding of this study lies in the other pathways identified in the cascading model of CU. Here we comment on one. The association between BPD and PTSD symptoms was low in effect size. However, when we unpacked BPD symptoms, for females, the association between BPD-related violence increased in effect size, but not by much. On the one hand, the association may support the idea that BPD and PTSD share cognitive biases for anger and threat due to traumatic and stressful experiences (Lobbestael and McNally, 2016). On the other hand, the small effect size may suggest that we are not accounting for other potential features by which these two disorders associate. For example, research suggests PTSD may be a better predictor of BPD than the converse: fear and sadness, a core feature of PTSD, may create vulnerability for negative affect, which is a core feature of BPD (Scheiderer et al., 2016). Hence, the effect size of the association between BPD and PTSD may increase with the inclusion of a wider range of common features, and with PTSD predicting BPD.

In summary, in this study we tested an idealized dynamic cascading model of CU. Although the overall cascade was not supported by the data, we did find that early adversity associated with CU via PTSD symptoms, whereas victimization by peers associated with CU via BPD symptoms. Moreover, when we unpacked the symptom domains of BPD, the data indicated that BPD-related identity disturbance leading to CU was an important pathway for males, whereas BPD-related violence leading to PTSD symptoms may be an important pathway for females.

Several limitations should be borne in mind when interpreting the results of this study. First, only one measure each was available for CU (at age 13) and BPD (at
age 11), precluding analysis of stability and change across childhood. Moreover, although PTSD was available at multiple time points, we included only one assessment, as per our hypothetical model. Second, although we referred to Karpman’s taxonomy in building a case for the cascade model, we did not actually test this sub-typing. Rather, we assumed we would find adversity-related associations based on the hypothesized existence of heterogeneity underlying the CU score. Future research may want to test if the developmental cascades identified here apply more to the secondary than the primary subtype of psychopathy. Third, although ALSPAC represents a broad, representative spectrum of socioeconomic backgrounds, the cohort features relatively low rates of ethnic minorities, necessitating replication with more ethnically-diverse samples. Fourth, like most large longitudinal cohorts, ALSPAC has experienced attrition over time, with children of younger and more socially disadvantaged mothers more likely to be lost in follow-up. However, previous studies of ALSPAC found that, while attrition affected prevalence rates of externalizing and internalizing disorders, associations between risks and outcomes remained intact, though conservative of the likely true effects (Wolke et al., 2009). Fifth, CU, BPD and PTSD all associate with genetic, epigenetic and neurocognitive mechanisms (Herpertz et al., 2001, McCrory et al., 2017, Viding et al., 2005, Cecil et al., 2014). An interdisciplinary approach may inform the nature of the results reported here, with regard to certain youth who may be more or less vulnerable for certain psychopathological outcomes following the experience of adversity.
Table 1. Descriptive statistics or the study variables by females (top, n = 2131) and males (bottom, n = 1908).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. CU (age 13)</td>
<td>--</td>
<td>0.136*</td>
<td>0.11*</td>
<td>0.162*</td>
<td>0.253*</td>
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<tr>
<td>2. Victimization (age 8-10)</td>
<td>0.181*</td>
<td>--</td>
<td>0.334*</td>
<td>0.003</td>
<td>0.215*</td>
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<tr>
<td>3. BPD Symptoms (age 11)</td>
<td>0.219*</td>
<td>0.407*</td>
<td>--</td>
<td>0.052*</td>
<td>0.094*</td>
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<tr>
<td>4. PTSD Symptoms (age 13)</td>
<td>0.106*</td>
<td>0.054*</td>
<td>0.093*</td>
<td>--</td>
<td>0.14*</td>
</tr>
<tr>
<td>5. Early Adversity (18wks gestation – age 7)</td>
<td>0.216*</td>
<td>0.171*</td>
<td>0.097*</td>
<td>0.148*</td>
<td>--</td>
</tr>
</tbody>
</table>

Male: Mean (StdDev)  
10.70 (3.22)  0.67 (0.73)  0.38 (0.87)  0.38 (1.61)  0.06 (1.39)  
Female: Mean (StdDev)  
10.76 (3.19)  0.53 (0.63)  0.33 (0.64)  0.51 (2.00)  0.05 (1.39)  

Note. * = p < 0.05; † = saved factor score.
Table 2. Indirect effects for overall dynamic cascade model.

<table>
<thead>
<tr>
<th>Effect</th>
<th>Age and measure</th>
<th>Indirect Effects</th>
<th>Estimate</th>
<th>95% CIs</th>
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<td></td>
<td></td>
<td></td>
<td>LL</td>
<td>UL</td>
</tr>
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<td>(1) Early Adversity</td>
<td>Victimization</td>
<td>BPD symptoms</td>
<td>PTSD symptoms</td>
<td>CU</td>
</tr>
<tr>
<td>(2) Early Adversity</td>
<td></td>
<td></td>
<td>PTSD symptoms</td>
<td>CU</td>
</tr>
<tr>
<td>(3) Victimization</td>
<td>BPD symptoms</td>
<td></td>
<td>PTSD symptoms</td>
<td>CU</td>
</tr>
<tr>
<td>(4) Victimization</td>
<td>BPD symptoms</td>
<td></td>
<td></td>
<td>CU</td>
</tr>
<tr>
<td>(5) BPD symptoms</td>
<td>PTSD symptoms</td>
<td></td>
<td></td>
<td>CU</td>
</tr>
</tbody>
</table>
### Table 3. Indirect effects for Exploratory model focused on sex difference in BPD symptoms.

<table>
<thead>
<tr>
<th>Effect</th>
<th>Prenatal – Age 7</th>
<th>Age 8 - 10</th>
<th>Age 11</th>
<th>Age 13</th>
<th>Estimate</th>
<th>95% CIs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1 – male)</td>
<td>Cumulative Risk</td>
<td>Victimization</td>
<td>Identity disturbance</td>
<td>CU</td>
<td>0.002</td>
<td>0.000 – 0.005</td>
</tr>
<tr>
<td>(1 – female)</td>
<td>Cumulative Risk</td>
<td>Victimization</td>
<td>Identity disturbance</td>
<td>CU</td>
<td>0.000</td>
<td>-0.002 – 0.003</td>
</tr>
<tr>
<td>(2 - male)</td>
<td>Victimization</td>
<td>Identity disturbance</td>
<td>CU</td>
<td>0.050</td>
<td>-0.003 – 0.116</td>
<td></td>
</tr>
<tr>
<td>(2 – female)</td>
<td>Victimization</td>
<td>Identity disturbance</td>
<td>CU</td>
<td>0.002</td>
<td>-0.058 – 0.065</td>
<td></td>
</tr>
</tbody>
</table>
Figure 1. Hypothesized correlations among domains in the development of CU (panel A) and hypothesized dynamic cascade model of the development of CU (panel B)

Panel A

Panel B

Note. BPD = borderline personality disorder symptoms; PTSD = post-traumatic stress disorder symptoms; CU = callous-unemotional traits.
Figure 2. Dynamic cascade model (panel A) and testing sex differences (panel B)

Panel A

Panel B

Note. Dotted lines = $p > 0.05$; Solid lines = $p < 0.05$; Circles = latent variables.

Rectangles = observed variables. Estimates = males / females; ns = not significant;

BPD = borderline personality disorder symptoms; PTSD = post-traumatic stress disorder symptoms; CU = callous unemotional traits.
Figure 2. Exploratory dynamic cascade model – sex differences in BPD symptoms

Note. Dotted lines = $p > 0.05$; Solid lines = $p < 0.05$; Circles = latent variables.

Rectangles = observed variables. Estimates = males / females; superscript ns = not significant; ID disturb = BPD symptoms of identity disturbance; Relations = BPD symptoms of disturbed relationships; Violence = BPD symptoms of aggressive and antisocial behavior; PTSD = post-traumatic stress disorder symptoms; CU = callous unemotional traits
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