Title: Associations between developmental trajectories of peer victimization, hair cortisol and depressive symptoms: a longitudinal study.

Running head: Peer victimization and hair cortisol

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ABSTRACT

Background: Peer victimization has been associated with long-lasting risks for mental health. Prior research suggests that stress-related systems underlying adaptation to changing environments may be at play. To date, inconsistent findings have been reported for the hypothalamic–pituitary–adrenal (HPA) axis, and its end-product cortisol. This study tested whether peer victimization was associated with hair cortisol concentrations (HCC), and if this association varied according to sex, timing and changes in exposure. We also examined whether peer victimization differentially predicted depressive symptoms according to HCC. **Methods**: The sample comprised 556 adolescents (42.0%; 231 males) who provided hair for cortisol measurement at 17 years of age. Peer victimization was reported at seven occasions between the ages of 6 and 15 years. **Results**: Peer victimization was non-linearly associated with HCC for boys only, whereas changes in peer victimization were related to HCC for boys and girls. Peer victimization predicted more depressive symptoms for all participants, except those with lower HCC. **Conclusion**: Our findings provide further support for persistent dysregulation of the HPA axis following exposure to chronic adversity, of which the expression may change according to sex and the severity of victimization.

Keywords: Peer victimization; HPA-axis; Hair cortisol; Depression; Non-linear models.

Abbreviations: HPA axis = hypothalamic–pituitary–adrenal; HCC = hair cortisol concentrations.

INTRODUCTION

Evidence shows that familial adversity increases risks of pediatric health problems (Oh et al., 2018). Adverse experiences occurring outside of the family environment, such as peer victimization, have also been associated with difficulties, from feeling unhappy to depression and suicidality (Arseneault, 2018; Geoffroy et al., 2018). Because peer victimization can be unpredictable, uncontrollable and threatening, it has been hypothesized that changes embedded in neurophysiological stress systems upholding adaptation to environments may be at play (Koss & Gunnar, 2018; Shonkoff, 2010). Mixed findings have, however, been reported between peer victimization and salivary diurnal secretion of cortisol (Grynderup et al., 2017; Knack, Jensen-Campbell, & Baum, 2011; Vaillancourt et al., 2008). One explanation may be that salivary cortisol does not capture long-lasting differences in cortisol and excludes nocturnal secretion. This is problematic because it is the chronic exposure to cortisol that is expected to increase neuronal toxicity in brain structures rich in mineralocorticoid and glucocorticoid receptors, such as the amygdala, hippocampus and frontal cortex (Koss & Gunnar, 2018). HCC may help to circumvent problems of protocol non-adherence and time-varying confounders affecting measures of salivary cortisol to a greater extent (Stalder & Kirschbaum, 2012). Despite these strengths, no study has yet investigated whether peer victimization is associated with HCC.

Prior studies investigating the association between child maltreatment and HCC have also yielded mixed findings, reporting lower (Hinkelmann et al., 2013; Steudte et al., 2013; White et al., 2017), higher (Ursache, Merz, Melvin, Meyer, & Noble, 2017; White et al., 2017), or non-significant findings (Fuchs et al., 2018; Gerber et al., 2017). This suggests that factors other than cortisol measurement may be involved. First, most studies used cross-sectional designs, which constrains our capacity to test whether chronicity, timing, or changes in exposure over time

affect HCC. Trickett *et al*. (2010), for example, have reported a shift from higher to lower morning cortisol secretion following sexual abuse. Similarly, brain structures regulating the HPA axis are expected to be more sensitive to earlier insults (Lupien, McEwen, Gunnar, & Heim, 2009). Second, the finding of lower *and* higher salivary diurnal and HCC in adverse contexts may also indicate that the effects of severity of adverse experiences on HCC are not adequately captured by linear functions. Essex and colleagues (2011) have reported that preschoolers who grew up with depressed mothers or in families displaying high levels of anger had *lower* daytime salivary cortisol levels at age 9 years, whereas those exposed to both adverse contexts had *higher* levels. Similarly, a shift in the direction of the association between SES and HCC was reported in adults from diverse ethnic backgrounds (O'Brien, Tronick, & Moore, 2013) and according to perceived stress (Wells et al., 2014). The idea that nonlinear associations may emerge in adverse contexts is not new (Doom, Cicchetti, & Rogosch, 2014; Miller, Chen, & Zhou, 2007), but evidence supporting this hypothesis remains scarce. Finally, heterogeneous findings may also pertain to sex differences emerging in adverse contexts (Del Giudice, Ellis, & Shirtcliff, 2011).

Determining whether peer victimization disrupts HCC matters because differences in stress response systems are thought to alter sensitivity to the environments (Boyce & Ellis, 2005; Del Giudice, Ellis, & Shirtcliff, 2011). Although there is emerging evidence for differential sensitivity to child maltreatment (Fuchs et al., 2018) and traumatic events (Steudte et al., 2013), no studies have yet tested if youth victimized by their peers fare better (or worse) according to their HCC. Depressive symptomatology is ideally suited to test this hypothesis as it is often reported by victims (Geoffroy et al., 2018; Sourander et al., 2016), and has been associated with disrupted HPA axis functioning (Stalder et al., 2017).

This study tested whether peer victimization, assessed from 6 to 15 years of age, predicted HCC measured two years later, at 17 years of age. We also tested whether this association was affected by sex, severity, timing and changes in peer victimization over time. Finally, we tested whether HCC modified the impact of peer victimization on depressive symptomatology.

METHODS

Participants

Participants were members of the Quebec Longitudinal Study of Child Development (QLSCD), a population-based cohort of 2,120 children born in 1997-98 in Quebec, Canada. A sample of 1,150 youths (i.e., who still participated in the study, lived in the province of Quebec and could be contacted) were invited to collect hair for cortisol measurement at age 17. A total of 560 participated, but the weight of four (.7%) hair samples was insufficient for analysis, resulting in a final sample of 556 participants (42.0%; 231 males). Having hair less than 3cm long or avoiding reduced hair volume before high school prom were the main reasons offered not to participate. It would be hazardous to solely attribute this attrition to hair collection as a similar participation rate was noted for the concurrent collection of a single whole saliva sample for the measurement DNA methylation. Study fatigue may have also influenced this attrition. In comparison to the initial cohort (n=2,120), males (χ^2=17.56, p<.001), non-Caucasian (χ^2=6.35, p<.01) and youth from lower SES families (r_{pb}=-.09, p<.001) were less likely to have participated. Statistical analyses accounted for this selective attrition by including inverse-probability-of-attrition weights in all analyses.

We sent to the participants curved scissors, hair clamps and instructions explaining how to collect hair from the posterior vertex area of the scalp, as we had previously validated in a sample of similar age, male/female ratio and SES (Ouellet-Morin et al., 2016). Parents gave

informed consent and children assented. Ethical approval was granted by the ethical committees of the Institut de la Statistique du Québec and University of Montreal.

Measures

Peer Victimization

Information about perceived peer victimization was collected at seven occasions between ages 6 and 15 using a modified version of the Self-Report Victimization Scale (Ladd & Kochenderfer-Ladd, 2002), which measured the perceived occurrence of physical, verbal, relational and cyber peer victimization since the beginning of the school year. The questions were read out loud by a research assistant at younger ages and included seven items, such as "*Called you names or said bad/mean things to you*," "*Broke purposely something that is yours*," and "*Pushed, hit or kicked*", responded using a 3-point Likert scale "0" (never), "1" (once/twice) or "2" (often or very often). We summed and rescaled the scale to vary between 0 and 10. Cronbach alphas (α) ranged from .70 to .80.

Hair Cortisol

Wash and steroid extraction procedures was conducted at the Centre for Studies on Human Stress (Montreal, Canada), according to the protocol described by Kirschbaum and colleagues (2009), with the exception that the first 3 cm hair segment was washed as is (not ground/cut/ pulverized) into a 15 millilitre (ml) tube with 2.5 ml of isopropanol before mixing. After decanting, the wash cycle was repeated and left to dry overnight. Pure methanol (1.5 ml) was added before being rotated for 24 hours. The methanol was then spun down in a microcentrifuge and 1 ml was aliquoted. The methanol evaporated at 37°C under a constant stream of nitrogen. Finally, .4 ml of phosphate buffer was added to the tube before being vortexed for 15 seconds.

The reconstituted sample was measured in duplicate using a luminescence immunoassay (detection range: .005-4 µg/dl; intra-assay coefficient of variation=5.39%). The sample had an average of 16.19 pg/mg cortisol (*SD*=14.84; 3*SD*-winsorized), which was log10-transformed for the analyses.

Depressive Symptoms

At 17 years, youth reported whether they experienced eight DSM-based depression symptoms "1" (never), "2" (sometimes), or "3" (often) over the past 12 months using the validated Mental Health and Social Inadaptation Assessment for Adolescents (Cote et al., 2017). The scale included items such as "*I lost interest in things I usually like*" or "*Even small things were bothering me*". In this subsample, participants reported a mean score of 3.81 (*SD*=2.22), according to a scale varying from 0 to 10 (α=.91).

Potential confounders

A description of the hair-related, individual and familial measures considered as potential confounders is available online (Online Supplement 1).

Statistical analyses

From a wide range of hair and health factors, including medication, five were uniquely associated with HCC: smoking, month of hair collection, hair washed 24 hours before sampling, natural hair color and body mass index (BMI). We derived a standardized residual of HCC to control for these confounders. In the main analyses, we first estimated models including 1-to-4 groups to capture homogenous patterns of peer victimization from ages 6 to 15 (then specifically for childhood and adolescence) using growth mixture models. The best-fitting model was selected according to the Bayesian Information Criterion (BIC), LoMendell-Rubin likelihood

ratio test (LMR-LRT) and entropy estimate. Second, we tested whether youth belonging to distinct trajectories of peer victimization differed on potential confounders using analyses of variance (ANOVAs) and chi-squared tests. Third, we tested with an ANOVA whether HCC differed according to peer victimization, testing for sex interaction, while controlling for non-random attrition and confounders. Linear and quadratic contrasts were estimated. Fourth, we tested whether trajectories of childhood (6-10 years) and adolescence (12-15 years) peer victimization uniquely predicted HCC using the previously-described ANOVA. Fifth, we tested whether changes in peer victimization, from childhood to adolescence, was associated with HCC. Finally, we tested if HCC moderated the association between peer victimization and depressive symptoms using the SPSS macro Process (Hayes, 2012).

RESULTS

Youths who completed hair collection at 17 years were, at 5 months of age, generally living with their parents who were in their late-twenties to early-thirties (Table 1). Boys reported more peer victimization than girls at all ages, except at 15 years (TableS1). Perceived peer victimization remained stable from 6 to 10 years and declined afterward (repeated measures ANOVA: linear: $F(1,337)=235.91$, $p<.001$; quadratic: $F(1,337)=8.66$, $p=.003$). Moderate to strong individual stability was noted over time ($r_s=30.8\text{-}52.7$, $p_s<.001$). Growth mixture models with linear and quadratic time parameters were estimated according to the maximum available data ($n=1,751$), as it was done previously in this cohort (Geoffroy et al., 2018). Figure 1 shows the best-model trajectories, depicting participants exposed to "Lower", "Moderate" and "Higher" levels of peer victimization between ages 6 and 15.

[FIGURE 1 & TABLE 1]

Youth who were exposed to more peer victimization had lower family SES, experienced more coercive parenting, and were reported by their teachers as being more anxious, hyperactive and physically aggressive. We controlled for these factors in subsequent analyses. HCC did not vary according to peer victimization [$F(2,528)=.29$, $p=.75$]. However, a significant interaction emerged with sex [$F(2,525)=3.63$, $p=.03$]. Specifically, boys with distinct levels of peer victimization varied on HCC [$F(2,210)=4.91$, $p=.008$; girls: $F(2,310)=.55$, $p=.58$], whereby those who experienced moderate peer victimization had lower HCC at 17 years of age in comparison to those exposed to lower victimization [difference(*SE*)=-.39(.15), $p=.009$; Figure 2]. Conversely, boys exposed to higher victimization had *higher*, not lower, HCC than those exposed to moderate victimization [difference(*SE*)=-.38(.19), $p=.04$], hence the quadratic function [linear: difference(*SE*)=-.007(.15), $p=.96$; quadratic: difference (*SE*)=.32(.11), $p=.003$]. Boys exposed to lower and higher peer victimization did not differ on HCC [difference(*SE*)=-.01(.22), $p=.96$].

[FIGURE 2]

To test whether the timing of these experiences mattered, we re-estimated the growth mixture models according to childhood (6-10 years) and adolescence (12-15 years) peer victimization separately, which again led to 3-group models, hence "Lower", "Moderate" and "Higher" levels of childhood/adolescence peer victimization (Supplemental Figure S1-S2). Neither childhood nor adolescence peer victimization predicted HCC when tested concurrently [$F(2,210)=1.04$, $p=.36$; $F(2,310)=.17$, $p=.83$, respectively], separately [$F(2,522)=.99$, $p=.37$; $F(2,524)=.14$, $p=.87$] or in interaction with sex [$F(2,511)=.74$, $p=.48$; $F(2,511)=2.31$, $p=.10$]. Changes in peer victimization, derived by subtracting the childhood from the adolescence victimization levels, was non-linearly associated with HCC [linear: difference(*SE*)=-.22(.37), $p=.56$; quadratic: difference(*SE*)=-

.77(.32), *p*=.02]. Figure 3 shows that youth who reported larger decrease (or increase) in victimization in adolescence had a trend for (or significant) lower HCC in comparison to youth exposed to smaller changes over time [difference(*SE*)=-.68 (.40), *p*=.09;difference(*SE*)=.96 (.45), *p*=.03, respectively]. Conversely, youth with a moderate decrease in peer victimization showed a trend for higher HCC in comparison to those for whom victimization did not change [difference(*SE*)=.22 (.11), *p*=.06]. These findings should, however, be interpreted with caution because most youth experienced no (54.5%; *n*=303) or moderate (17.1%, *n*=95; 21.8%, *n*=121) changes in peer victimization from childhood to adolescence. Larger changes were rare (1.3%, *n*=7; 1.1%, *n*=6). These findings remained when controlling for childhood peer victimization.

[FIGURE 3]

We tested whether HCC modified the association between peer victimization and depressive symptoms according to childhood and adolescence peer victimization and increasing levels over time. While the interactions between peer victimization, HCC, and sex [β(*SE*)=.05(.30), *p*=.86] and between victimization and HCC did not predict depressive symptoms at 17 years of age [β(*SE*)=-.13(.14), *p*=.36], peer victimization was a significant predictor [β(*SE*.73(.15), *p*<.001]. Controlling for childhood peer victimization, no interaction was either detected between increasing peer victimization, HCC, and sex on depressive symptoms [β(*SE*)=.67(.42), *p*=.11], but a significant interaction emerged between peer victimization and HCC [β(*SE*)=.40(.20), *p*=.05]. Because HCC was continuously distributed, we probed the region of significance for the moderation using the J-N technique. With increasing levels of peer victimization, higher depressive symptoms was noted for all youth, except for those with HCC lower than the 15th percentile of the sample's distribution. Figure 4 shows the peer victimization-depressive symptoms association at lower (-1*SD*), moderate (sample's mean) and higher (+1*SD*) HCC,

which again depicted youth with lower HCC reporting similar levels of depressive symptoms according to increasing peer victimization. Nonetheless, it was only *in absence* of increasing peer victimization in adolescence that youth with lower, moderate and higher HCC significantly differed on depressive symptomatology. Specifically, youth with higher HCC reported less depressive symptoms in comparison to those with moderate and lower HCC [difference(*SE*)=-.79 (.36), *p*=.03; difference(*SE*)=.97 (.47), *p*=.04]. Both childhood and increasing levels of victimization in adolescence uniquely predicted depressive symptoms [β(*SE*)=1.21(.21), *p*<.0001; β(*SE*)=.93(.24), *p*=.0001].

[FIGURE 4]

DISCUSSION

This study examined the association between peer victimization, HCC and depressive symptoms in youth for whom victimization was reported prospectively at seven occasions over a nine-year period. Our findings showed that long-lasting differences in peer victimization were associated with HCC at 17 years of age in boys, over and above the influence of many factors often confounded with peer victimization (e.g., family SES, youth's aggression). Specifically, boys exposed to moderate levels of peer victimization had lower HCC than those who experienced lower peer victimization. Yet, we uncovered a point of inflexion, a shift, whereby higher levels of victimization pointed to higher HCC. By and large, meta-analyses and systematic reviews investigating the association between early adversity and HCC have reported large heterogeneity estimates (Gray et al., 2018; Khoury, Bosquet-Enlow, Plamondon, & Lyons-Ruth, 2019). Our study extends this work by showing that this association is best described according to a non-linear function, suggesting that the gradient in the severity of exposure should be considered. Consistent with these findings, our team also reported a shift from moderate-to-lower-to-higher

levels of salivary cortisol responses to stress in adult males exposed to increasingly severe child maltreatment (Ouellet-Morin et al., 2018).

Our findings may thus offer a putative explanation as to why lower and higher HCC may be detected in participants exposed to adverse contexts. Nonlinear associations between early adversity and HPA axis functioning have been proposed before. For instance, the Adaptive calibration model of stress responsivity proposed four prototypical patterns of responsivity according to increasing levels of adversity (Del Giudice, Ellis, & Shirtcliff, 2011). Moderate-to-high responsivity is anticipated in safe environments, followed by a buffered pattern of responsivity in moderate stress. A point of inflexion toward higher responsivity is expected for individuals exposed to more dangerous and unpredictable environments, which remains high in severe and traumatic environments for females, but revert to unresponsive patterns of response for males. We also uncovered shifts in HCC according to changes in peer victimization from childhood to adolescence in boys and girls. If overlooked, these nonlinear associations could have been mistaken for non-significant findings. Depending on the range of severity captured in study samples, apparent findings denoting lower, higher, or non-significant differences may ensue.

Our findings are also in line with the Stress inoculation model (Parker, Buckmaster, Schatzberg, & Lyons, 2004). We speculate that youth confronted to moderate peer victimization may be more likely to perceive themselves as having the abilities and resources to cope with these experiences, leading to lower HCC over time. Conversely, severe victims may not show this buffered secretion because of the frequency of victimization experienced and the hypervigilance to detect future aggression that accompany these experiences. Relying on a cross-sectional study design, a similar (U-shaped) association was reported according to SES in

minority racial/ethnic background (O'Brien, Tronick, & Moore, 2013). Altogether, these findings suggest that nonlinear associations should be systematically tested in studies assessing biological markers associated with gradient of adversity.

We found that neither childhood nor adolescent peer victimization predicted HCC at 17 years of age. These findings contrast with the idea that brain structures regulating HPA axis activity are more malleable to earlier environmental influences (Lupien, McEwen, Gunnar, & Heim, 2009). One interpretation may be that the timing of peer victimization is less relevant to later HCC because these experiences typically start in childhood. While peer victimization can still be expected to affect the amygdala and frontal cortex, as these brain regions continue to develop at this time, the hippocampus is already mature and fully organized (Lupien, McEwen, Gunnar, & Heim, 2009). Similarly, our analyses statistically controlled for several individual and familial characteristics known to co-occur with peer victimization, such as low SES, coercive parental interactions and children's aggression, hyperactivity, and anxiety. Because these characteristics generally emerge at younger ages and are stress inductive, they may speed up the maturity of amygdala-medial prefrontal cortex network (Callaghan & Tottenham, 2016), thus lessening the sensitivity to more transient peer victimization taken place during childhood and adolescence. Therefore, it may be that only persistent experiences of peer victimization, from childhood to adolescence, are capable of exerting an influence on HCC at 17 years-old, as found in this cohort. Changes in peer victimization from childhood to adolescence (increasing and decreasing levels) were also shown to trigger further demands on the HPA axis, taking the forms of higher and lower HCC. We speculate that youth who experienced decreasing levels of peer victimization may remain vigilant to perceived threats. More research is needed to examine this possibility.

While sex differences in brain responses to stressors are expected (McEwen, Gray, & Nasca, 2015), they are not systematically tested (or reported). We found an association between peer victimization and HCC only for boys. These emulate, to some degree, those reporting higher and lower salivary diurnal cortisol levels in bullied boys and girls (Vaillancourt et al., 2008). The factors responsible for these differences are unknown, although a few lines of enquires are proposed. Our finding showed that boys were more severely victimized by their peers than girls from ages 6 to 13 years, which generally involved more physical assaults for boys (Arseneault, 2018). While these differential exposures could partly explain our sex-dimorphic findings, we cannot test this possibility because only a few items measured each type of victimization (e.g., physical versus verbal). In contrast, we reported that changes in peer victimization in adolescence similarly predicted HCC for boys and girls, a finding more aligned to a recent meta-analysis reporting no sex differences in the association between child maltreatment and HCC (Khoury, Bosquet-Enlow, Plamondon, & Lyons-Ruth, 2019). Furthermore, while the exposure to peer victimization was generally more severe for boys, HCC similarly modified the association between these experiences and depression for boys and girls. Considering the lack of systematic reports of sex differences between exposure to early adversity and HCC, caution is warranted in the interpretation of this finding.

Theoretical models and accumulating evidence suggest that higher biological stress reactivity may not inherently indicate a risk for well-being, but rather represents a marker of sensitivity to the environments. We found that depressive symptoms were associated with increasing levels of peer victimization in adolescence, except for those who secreted lower HCC, for whom this association was not detected. Our findings are thus partly consistent with the Biological sensitivity to context model, whereby higher level of difficulties is anticipated to emerge in

individuals who are the most biologically sensitive to the environment when exposed to adversity, but otherwise relate to better functioning (Boyce & Ellis, 2005). Specifically, while we detected an association between victimization-depressive symptoms for youth with higher HCC, no mean differences were detected in the context of moderate and larger increases in victimization. It was in the absence of increasing peer victimization that youth with higher HCC had less depressive symptoms than those with moderate or lower HCC. We found only one study that had formally tested the interplay between early adversity and HCC in association with emotional and behavioral problems. While an interaction was reported between the children's HCC and their mothers' experiences of child maltreatment, only those exhibiting higher HCC manifested more behavior problems in *absence* of familial adversity (Fuchs et al., 2018). More research is needed to test whether higher HCC is health-undermining in high-risk contexts, but health-promoting in low-risk contexts, such as growing evidence suggests for salivary cortisol (Obradovic, 2012). Notably, we showed that the genetic and environmental associations between peer victimization and depression symptoms steadily increased with higher salivary morning cortisol levels in 14 year-old twins, also supporting the idea that peer victimization is more strongly correlated with internalizing difficulties in youth with heightened biological sensitivity (Brendgen et al., 2017).

This study is not, however, without limitations. First, our items inquiring about peer victimization were not preceded by an introductory statement specifying the context of power imbalance or intention necessary to refer more specifically to bullying victimization. Second, peer victimization were self-reported and, as such, should be viewed as perceptions of victimization. While distinct informants (e.g., child, parent) only modestly agree about bullying victimization, both provide reliable information that is similarly associated with children's

emotional and behavioral problems (Shakoor et al., 2011). Nonetheless, because depression symptoms were also self-reported, albeit years later, it raises a possibility of shared-method variance bias. Third, HCC was measured at only one point in time, after peer victimization. Future studies should ideally collect multiple measures of adversity and HCC to further ascertain the direction of this association. Finally, only a few items captured each type of victimization (e.g., physical versus relational), precluding a more refine analysis of the reported findings.

In conclusion, our findings offer evidence that persistent experiences of peer victimization predict HCC at the end of adolescence, with potential implication for depressive symptoms. By capturing systemic differences in chronic cortisol secretion in hair samples, this study suggests that preventive interventions helping victims to reduce the chronicity of their experiences may help to prevent the induction of long-lasting changes in HPA axis functioning.

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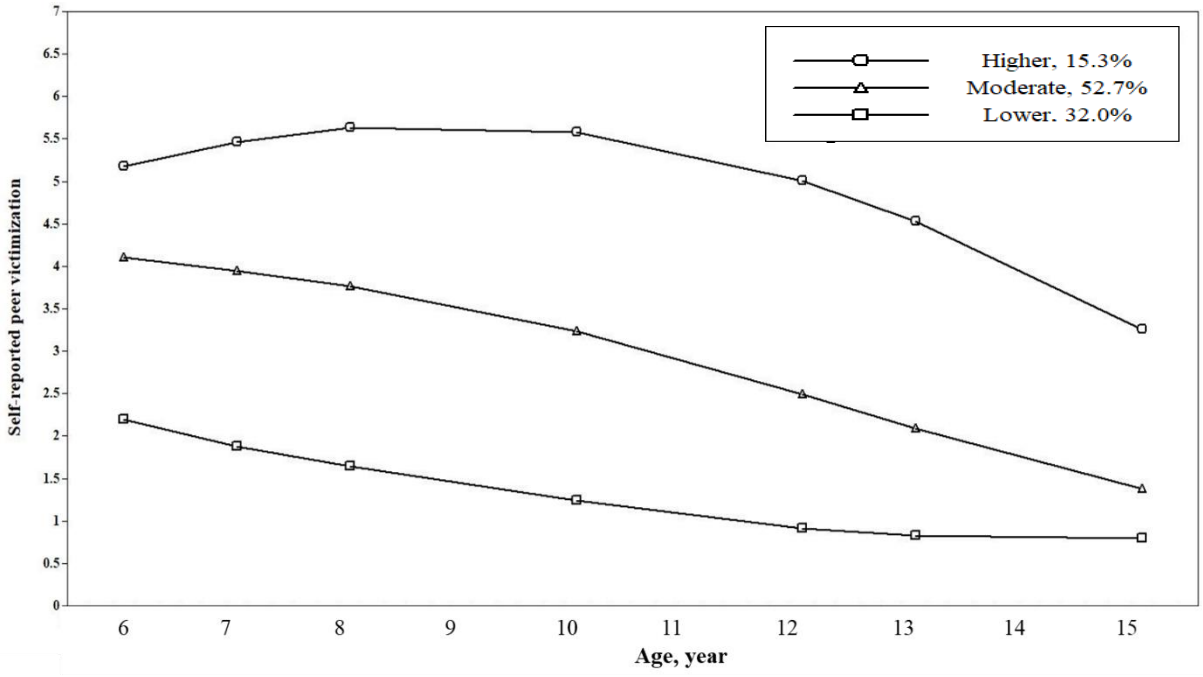
Table 1. Individual characteristics for the total sample and according to peer victimization.

		Peer victimization trajectories % or Mean (*SD*)			
	Total sample % or Mean (*SD*)	No (fewer) (32.0%)	Moderate (52.7%)	Higher (15.3%)	*F* or χ^2
Mothers' age	29.90 (5.06)	29.93 (4.91)	30.03 (5.07)	29.21 (5.45)	.73
Fathers' age	32.59 (5.44)	32.56 (5.23)	32.61 (5.54)	32.56 (5.62)	.004
Family SES	.21 (1.06)	.24 (1.06)	.28 (1.03)	-.15 (1.15)	4.51**
Coercive parenting					
Lower	32.9	41.9	50.5	7.6	
Moderate	54.9	31.0	56.5	12.5	24.77***
Higher	12.2	14.5	60.9	24.6	
Anxiety					
Lower	26.4	38.7	56.4	4.9	29.03***
Moderate	69.3	32.7	53.6	13.7	
Higher	4.3	13.0	43.5	43.5	
Hyperactivity					
Lower	48.8	46.0	49.4	4.6	67.38***
Moderate	40.4	24.3	59.2	16.5	
Higher	10.8	10.3	55.2	34.5	
Aggression					
Lower	59.4	41.9	51.2	6.9	47.90***
Moderate	34.3	23.8	57.8	18.4	
Higher	6.3	5.9	58.8	35.3	

Note. Data were compiled from the final master file of the QLSCD (1998-2015), Institut de la Statistique du Québec. *SD*=Standard deviation; SES = Socioeconomic status; BMI=Body mass index. Based on maximum available *n* value. *** $p < .001$; * $p < .05$.

Figure 1. Developmental trajectories of peer victimization from childhood to adolescence.



Notes. The 3-group model offered the best fit indices [1-group: BIC=40126.45, entropy=not applicable, LMR-LRT=not applicable; 2-groups: BIC=38906.28, entropy=.61, LMR-LRT=1209.56, p<0.001; 3-groups: BIC=38678.33, entropy=.61, LMR-LRT=249.47, p<0.001; 4-groups: BIC=38564.27, entropy=.53, LMR-LRT=139.26, p=.15].

Figure 2. Association between peer victimization and HCC according to sex.

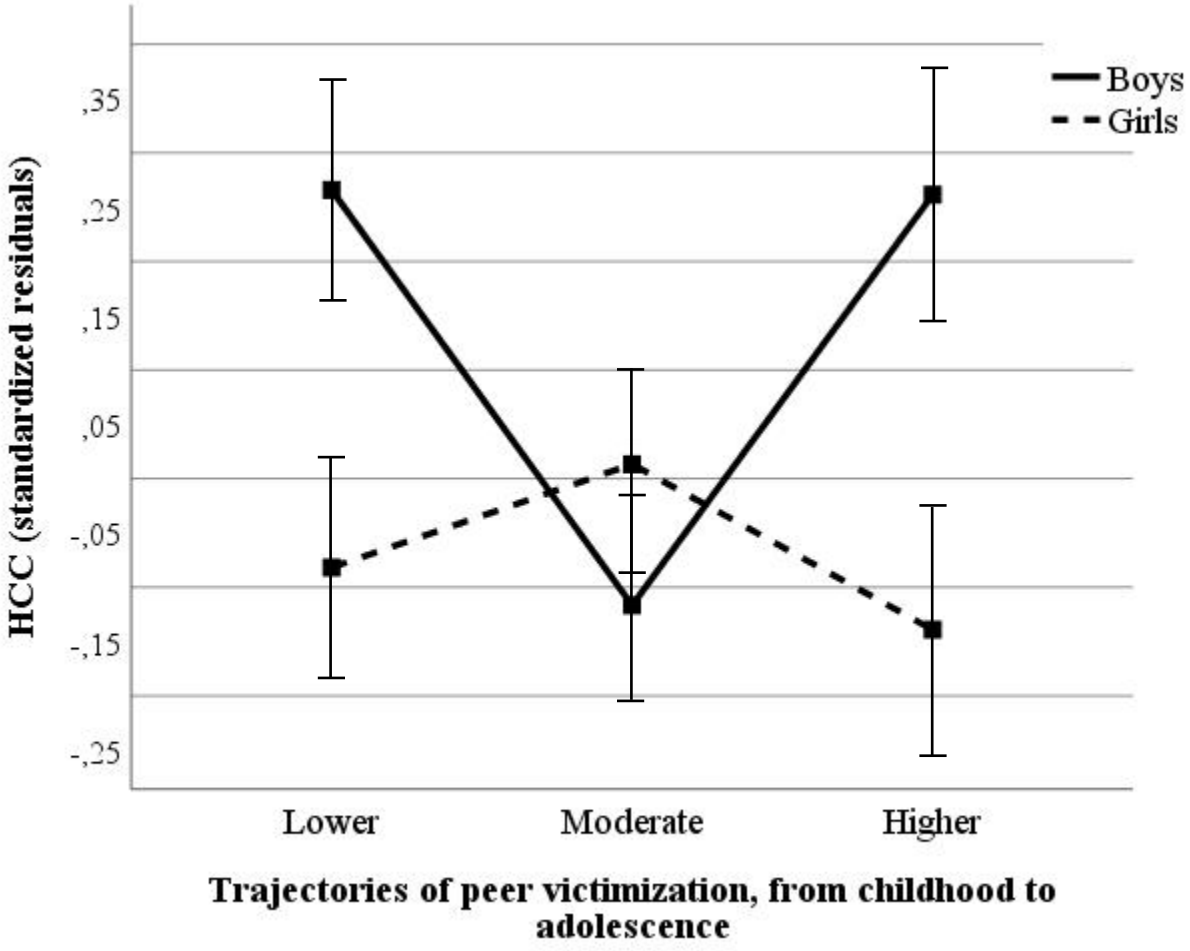


Figure 3. Association between temporal changes in peer victimization and HCC.

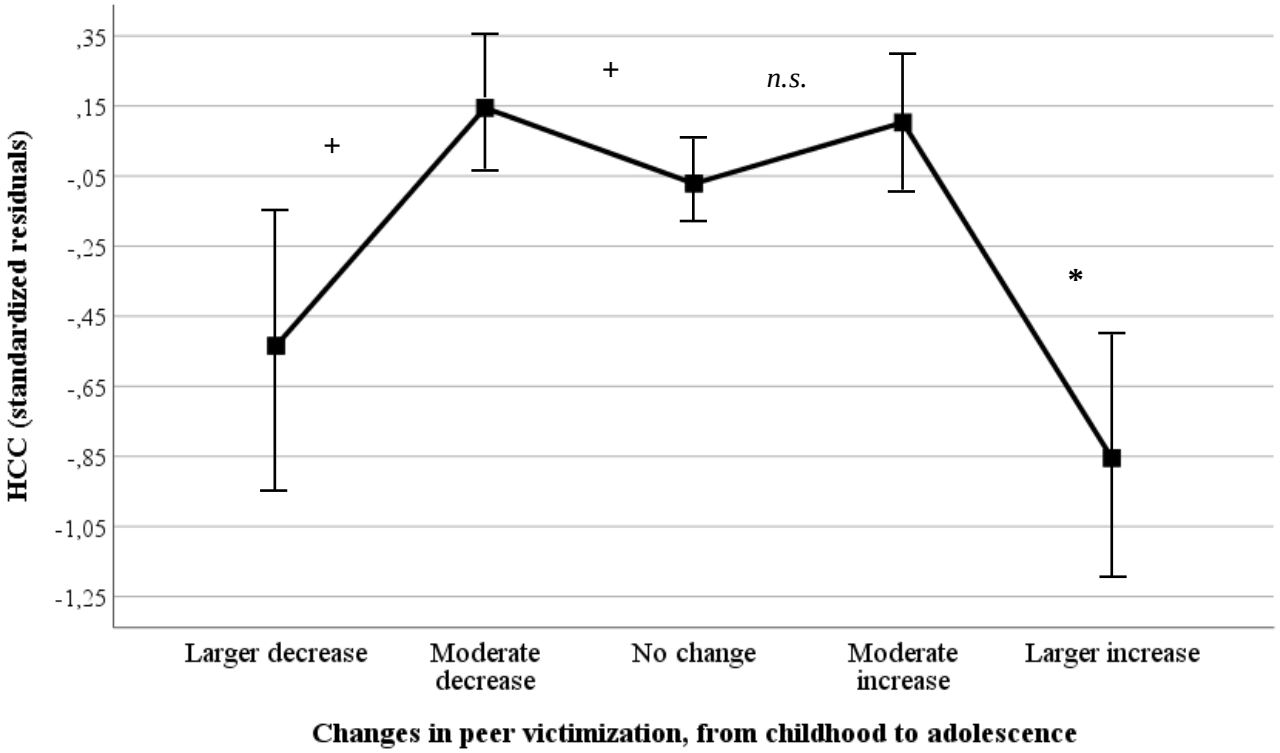
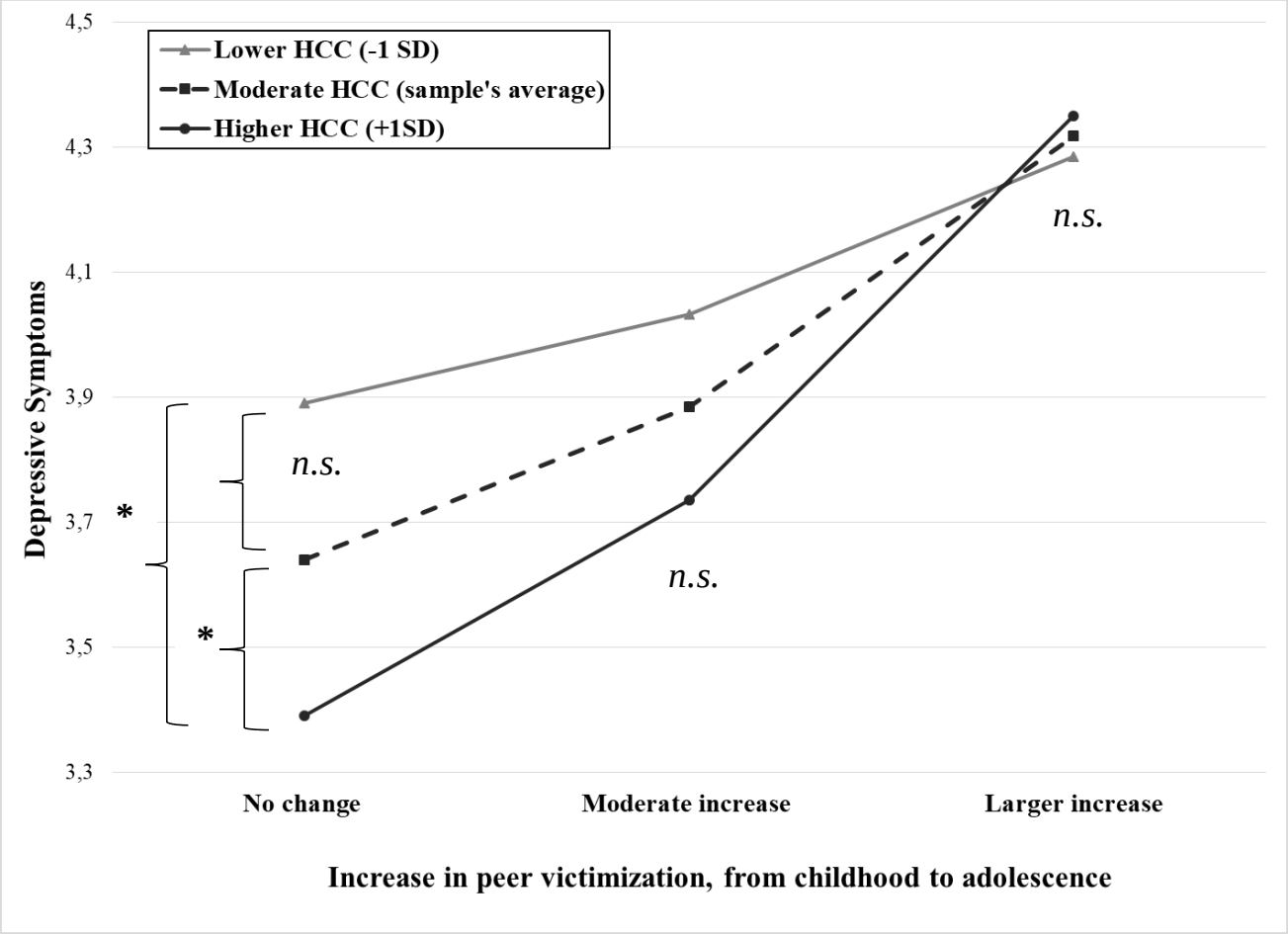


Figure 4. Association between increasing levels of peer victimization and depressive symptoms.



Note: SD=Standard deviation.

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Online Supplement 1

Hair-related potential confounders

Information about the natural state of the hair (e.g., color, curvature), usual care (e.g., frequency of washing, treatments), and health (e.g., medications, sleeping habits, body mass index (BMI), tabacco, drug and alcohol consumption) were reported by the participants. Specifically, the participants reported their use of medication, including those potentially affecting the HPA axis, with a reference timeframe of three months before hair collection. Each medication category was examined in association with HCC. Those with a significant bivariate association with HCC were then re-examined concurrently with all potential confounders. None of the medication-related variables had unique contributions with HCC, including the glucocorticoid medication reported to have been taken at least once in the preceding three months by 73 participants (13.2% of the sample). Because the unique contribution of glucocorticoid medication to HCC was not significant (B=.04, t=1.01, p=.31), these participants remained included in the study sample.

Individual and familial potential confounders

To test whether peer victimization co-occurred with known risk factors for HCC and depressive symptoms, we used an aggregated index of SES, including information about family annual income, single parenthood, mothers' and fathers' education and occupational prestige collected on many occasions between 5 months and 15 years. When the participating child was 5 months of age (first wave of data collection), 87% and 83.5% of mothers and fathers, respectively, had completed their high school diploma, 89.6% of fathers were being remunerated for their work and most families (83.0%) reported having sufficient income for their needs. Coercive parenting referred to power-assertive behaviors (8 items, e.g., *use of physical punishment*) were reported by the mothers at 10 occasions between 2.5 to 15 years (Boivin et al., 2005). The information

across the time points was summarized using group-based mixture models, of which the 3-group was the best fitting model (« Lower » 33.8%, « Moderate» 51.8%, and « Higher» 14.4% coercion in this sample). Behaviors indicative of hyperactivity-impulsivity (3 items; e.g., *could not sit still*), physical aggression (3 items; e.g., *got into a fight*), and anxiety (4 items; e.g., *appears fearful/nervous*) were rated on a 3-point scale by school teachers at ages 6, 7, 8, 10 and 12 years using items derived from the Canadian National Longitudinal Study of Children and Youth (Statistics Canada, 1996), which incorporates items from the Child Behaviour Checklist (Achenbach, 1991), the Ontario Child Health Study scales (Offord, Boyle, & Racine, 1989) and the Preschool Behaviour Questionnaire (Tremblay, Desmarais-Gervais, Gagnon, & Charlebois, 1987). The data related to each behavior were summarized using growth mixture models, as done previously in this cohort (Vergunst et al., 2018), for which the 3-group models also fitted best the data (hyperactivity-impulsivity: « Lower » 48.8%, « Moderate» 40.4%, and « Higher» 10.8%; physical aggression: « Lower » 59.4%, « Moderate » 34.3%, and « Higher » 6.3%; and anxiety: « Lower » 26.4%, « Moderate » 69.2%, and « Higher » 4.3%, in this sample).

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