JOINT TRAJECTORIES OF BULLYING VICTIMIZATION AND PERPETRATION: INVESTIGATING THE ROLE OF THE COMT GENE

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Abstract

Bullying research has grown tremendously throughout the years, and yet, there is a lack of research investigating the biological underpinnings of bullying victimization and perpetration. The single nucleotide polymorphism *catechol-O-methyltransferase* (*COMT*) *Val158Met* is an important candidate gene that has been demonstrated to interact with environmental factors and play an important role in emotion processing. However, it remains unknown whether *COMT Val158Met* influences youth and adolescents' involvement as both targets and perpetrators of bullying, considering bully-victims are found to struggle with emotion regulation. To address this knowledge gap, the role of *COMT Val158Met* on the joint trajectories of bullying victimization and perpetration was investigated in a longitudinal community sample. A latent class growth analysis (LCGA) was used to identify distinct patterns of bullying victimization and perpetration across the ages 10 to 18 years (n = 648). A three-class solution was chosen for bullying victimization where most participants were reflected in a trajectory of low decreasing bullying victimization (74%), followed by moderate stable trajectory of bullying victimization (23%) and a final group following a high stable trajectory of bullying victimization (3%). A two-class solution was chosen for bullying perpetration. As predicted, most participants were reflected in the low stable bullying perpetration group (83%) and a small group followed a moderate increasing/decreasing trajectory of bullying perpetration (16.4%). Dual trajectory models revealed distinct subgroup of individuals involved in bullying either as targets, perpetrators, or bully-victims. Conditional probabilities results suggest that highly victimized youth would in time perpetrate against others while remaining targets of high levels of perpetration (i.e., target to bully-victim), whereas youth moderately victimized were more likely to be uninvolved in bullying perpetration. There was no significant difference in allelic variations (i.e., any Met allele vs Val/Val) of *COMT Val158Met* between bully-victims and children uninvolved in bullying. Implications of these findings are discussed from a differential susceptibility model. Gaining an understanding of the mechanisms behind the impact of bullying victimization and perpetration on children and adolescent will help provide insight and support for school and clinical prevention and intervention efforts.

Keywords: Bullying victimization, Bullying perpetration, *COMT Val158Met*, Joint trajectories

Joint Trajectories of Bullying Victimization and Perpetration: Investigating the Role of the *COMT* Gene

Introduction

Bullying is a major public health issue (National Academies of Sciences, Engineering, and Medicine [NASEM], 2016), with one out of three adolescents experiencing bullying victimization in Canada (Craig et al., 2020). Bullying is defined as intentional repetitive negative behaviour that involves a power imbalance between the aggressor and the target (Olweus, 1993; Vaillancourt et al., 2008). Bullying victimization can take direct or indirect forms, such as verbal, physical, relational, and cyber (Vaillancourt et al., 2010a). A common past belief was that bullying victimization is a natural part of childhood and helps youth become resilient, but research on the impact of bullying on child and youth mental health has debunked this problematic myth. Indeed, several longitudinal studies have shown that bullying victimization and perpetration in childhood and adolescence are linked to severe and lasting negative mental health and behavioural outcomes (McDougall & Vaillancourt, 2015; Moore et al., 2017). For example, bullying victimization in childhood is associated with the development of internalizing problems (Arseneault et al., 2006; see meta-analyses by Moore et al., 2017; Reijntjes et al, 2010) and externalizing problems (Arseneault et al., 2008, Haltigan & Vaillancourt, 2014). Identifying risk factors for involvement in bullying is important to help identify the children who are vulnerable to these maladjustments and minimize the harmful effects of bullying. Although researchers have found that many individuals are involved in bullying at some point in childhood, longitudinal studies have highlighted three typical distinct groups of children who are involved in bullying (Pepler et al., 2008; Vaillancourt et al., 2010b): targets (i.e., victims), perpetrators (i.e., bullies), and target-perpetrators (i.e., bully-victims). Targets are children who are victims of bullying by their peers but do not bully others, perpetrators are children who bully their peers but are not bullied, and bully-victims are children who bully others and are bullied. Bully-victims have been shown to be at the highest risk for diverse negative maladjustment (Arseneault et al., 2010; Lereya et al., 2013).

Several longitudinal studies have identified potential risk factors that influence children's involvement in bullying (D'Urso & Symonds, 2021). One of these psychosocial risk factors is emotional dysregulation (Cooley & Fite, 2016; Fogleman et al., 2019; Gardner et al., 2017; Hanish et al., 2004; Kochenderfer-Ladd, 2004; Morelen et al., 2016; Rosen et al., 2012).

Emotion regulation is described as the change in an individual's activated emotional valence, intensity, and time of course to meet external demands (Cole et al. 2004; Thompson, 1994). Children who effectively regulate their emotions are more likely to develop and maintain healthier relationships with their peers (Kim & Cicchetti, 2010). This is because emotion regulation skills lead to increased communication and understanding of one's own and others' needs, allowing children to effectively navigate peer relationships. Alternatively, children with poor emotion regulation skills are at risk of adopting uncontrolled and overt displays of emotions leading to poor social functioning (Eisenberg, 2000; Eisenberg et al., 2010). Longitudinal studies investigating trajectories of bullying victimization and perpetration suggest that emotional dysregulation may influence the reciprocal cycle of bullying involvement and may increase the likelihood of targets transitioning into perpetrators (Barker et al., 2008; Haltigan & Vaillancourt, 2014; Kochenderfer-Ladd., 2004). This heterogeneous pattern of involvement highlights the importance of examining both bullying victimization and perpetration longitudinally rather than within time.

Although research on psychosocial risks like emotional dysregulation is important, there is also a call for more research investigating underlying biological risk factors for involvement in bullying (Vaillancourt, 2018). This expanded approach can help researchers and practitioners gain a better understanding of the diversity in outcomes seen in the literature. One potential genetic risk factor for youth involvement in bullying is the single nucleotide polymorphism catechol-O-methyltransferase *(COMT) Val158Met*. This common single nucleotide polymorphism is associated with modulation of dopamine levels in brain areas, of relevance to bullying involvement, that play an important role in emotion processing. Specifically, the allelic variation on *COMT Val158Met* is a risk factor for emotional dysregulation (see Mier et al., 2010 for a meta-analysis). Considering children who are involved in bullying as either a target, perpetrator, or bully-victim are often characterized as being emotionally dysregulated (Shields & Cicchetti, 2001), I used *COMT Val158Met* as a representation of emotion regulation in my thesis. Specifically, I examined *COMT Val158Met* as a genetic risk factor in the co-development of bullying victimization and perpetration across childhood and adolescents (Grade 5 to Grade 12).

Literature Review

Bullying and Emotion Regulation

Emotions play an essential role in how we communicate and relate to one another. Effective emotion regulation is essential in the management of interpersonal relationships, which we heavily rely on for support, a sense of belonging, and overall well-being (Denham et al., 2014). Youth struggling with regulating their emotions may be at risk for adopting uncontrolled and overt displays of emotions like physical aggression during conflicts with their peers (Cole et al., 2004). This may elicit unfavourable reactions from others, leading to poor social functioning, such as bullying (Shields & Cicchetti, 2001). Indeed, Rosen et al. (2012) proposed a model in which emotional dysregulation influences children's involvement in bullying as targets, perpetrators, and bully-victims. The model suggests that dysregulated emotional reactivity impairs children's ability to attend to socially threatening situations such as being bullied, resulting in adverse reactions. For example, emotionally dysregulated targets of bullying may aggress against their perpetrator in undesirable social manners. In effect, children demonstrating difficulties with regulating their emotions may be at risk for chronic and increased frequency of bullying victimization (Rosen et al., 2012). Rosen et al. (2012) demonstrated this pattern in 213 9–13-year-olds when investigating the association between dysregulated negative emotional reactivity and chronic peer victimization. In this study, emotional dysregulation was associated with concurrent victimization and increased rates of victimization over time.

A growing body of research demonstrates that children with poor emotion regulation have an increased risk of experiencing victimization and difficulties mitigating bullying (Cooley & Fite, 2016; Fogleman et al., 2019; Gardner et al., 2017; Hanish et al., 2004; Kochenderfer-Ladd, 2004; Morelen et al., 2016). In line with these findings, Palamarchuk and Vaillancourt (2022) proposed that the interpretation of a stressor (i.e., controllability and severity) is what influences psychological outcomes. That is, emotional dysregulation in children may influence poor cognitive appraisal of stressful situations leading to heightened reactivity (Palamarchuk & Vaillancourt, 2022). Considering that bullying victimization is often an uncontrollable and extreme stressor, emotionally dysregulated children may resort to maladaptive strategies that exacerbate and perpetuate bullying experience, instead of engaging in prosocial behaviour (Eisenberg et al., 2004; Eisenberg et al., 2010). Researchers have found that children who tend to struggle the most with emotion regulation and emotion processing are bully-victims (Garner et

al., 2010; Schwartz et al., 2001; O'Brennan et al., 2009; Palamarchuk & Vaillancourt, 2022; Toblin et al., 2005). In fact, heightened reactivity is thought to be a core mechanism explaining the dual role of victimization and perpetration (Palamarchuk & Vaillancourt, 2022). Children with heightened reactivity who are targets of bullying may engage in bullying behaviour to help cope with the stressor, as a form of retaliation or to help fill their need to belong (Underwood & Ehrenreich, 2014). This idea is supported by studies showing that targets of bullying are more likely to transition to perpetrators than the reverse (Barker et al., 2008; Haltigan & Vaillancourt, 2014), although a recent meta-analysis by Walters et al. (2021) suggests that the relation between bullying victimization and perpetration may in fact be bidirectional. A better understanding of the temporal ordering between bullying victimization and perpetration is needed to clarify these discrepancies. Moreover, given the complex course and development of involvement in bullying victimization and perpetration over time, joint trajectory studies are particularly needed over a large time frame to examine co-occurring involvement in bullying victimization and perpetration (bully-victims).

Trajectories of Bullying Involvement

Trajectories of Bullying Victimization and Perpetration. Longitudinal studies investigating heterogeneous changes in bullying involvement across time have generally identified three to five bullying victimization and perpetration trajectories (e.g., Brendgen et al., 2016; Cho & Lee, 2021; Espelage et al., 2018; Geoffroy et al., 2018; Pabian & Vandebosch, 2016; Pepler et al., 2008; Reijntjes et al., 2013; Vaillancourt et al., 2023). Group-based bullying victimization trajectory studies have yielded trajectories consisting of a high chronic group, a low-stable, and a moderate-stable group (Biggs et al., 2010; Brendgen et al., 2016; Geoffroy et al., 2018; Goldbaum et al., 2003; Sumter et al., 2012), with some reporting decreasing and increasing groups over time (Biggs et al., 2010; Brendgen et al., 2016; Gauldbaum et al., 2003). Researchers examining bullying perpetration trajectories have found a low/stable and a high/chronic trajectory group, along with increasing or decreasing involvement over time groups (Cho & Lee, 2021; Espelage et al., 2018; Pabian & Vandebosch, 2016; Pepler et al., 2008; Reijntjes et al., 2013). For example, Pepler et al. (2008) identified four distinct bullying perpetration trajectories in youth aged 10 to 17 years comprising of a small stable high group (9.9%), a stable moderate group (35.15%), a moderate/declining group (13.4%), and a large stable/non-involvement in bullying group (41.6%). Although investigating trajectories of

bullying victimization and perpetration separately provide information on children involved in bullying as targets and perpetrators, it fails to account for the subgroup of children that are the most at risk for diverse maladjustment—bully-victims. To gain a better understanding of all the subgroups of children involved in bullying, joint trajectories should be used when investigating bullying victimization and perpetration.

Joint Trajectories of Bullying Victimization and Perpetration. When trajectories of bullying victimization and perpetration are examined together, three to six joint trajectories have been identified (Barker et al., 2008; Cho & Lee, 2020; Haltigan & Vaillancourt, 2014; Zhou et al., 2020). Barker et al. (2008) examined joint trajectories in a sample of British adolescents aged 13 to 16 years and found that a large group (75%) who reported little to no involvement in bullying as either targets or perpetrators and subgroups consisting of a high/decreasing bullying victimization and low/decreasing bullying perpetration (7%), a high/increasing bullying victimization and low/decreasing bullying perpetration (i.e., targets; 3%), a low/stable bullying victimization and high/increasing bullying perpetration (i.e., perpetrators; 11%), a high/decreasing bullying victimization and high/increasing bullying perpetration trajectory (i.e., those transitioning from targets to perpetrators; 3%), and a high/increasing bullying victimization and high/increasing bullying perpetration (i.e., bully-victims; 2%). In a sample of Canadian youth assessed from aged 10 to 14 years, Haltigan and Vaillancourt (2014) found a large group (73%) who had limited involvement in bullying and three additional subgroups with involvement. The first followed a low/stable bullying victimization and a moderate/increasing bullying perpetration trajectory (11%), another followed a moderate declining bullying victimization and low/stable bullying perpetration trajectory (10%), and the final group followed a moderate/declining bullying victimization and moderate/increasing bullying perpetration trajectory (6%; i.e., those transitioning from targets to perpetrators). Baker et al. (2008) and Haltigan and Vaillancourt (2014) results suggest that the temporal ordering was from target to perpetrator.

Cho and Lee (2020) found three joint trajectories of bullying victimization and perpetration among Korean youth in Grade 4 to Grade 8 using second order growth mixture modeling rather than Latent Class Growth Analysis (LCGA) as in Baker et al. (2008) and Haltigan and Vaillancourt (2014). Cho and Lee (2020) found a limited involvement/stable group (83.3%) and the other two subgroups were characterized by high/decreasing perpetration and

high/decreasing victimization (9.9%) and a moderate/stable bullying perpetration and moderate/increasing and late peak victimization trajectory (6.8%). In another study, Zhou et al. (2020) examined joint trajectories in Chinese youth from Grade 4, 5 and 6 to Grade 7, 8 and 9, respectively. Zhou et al. found four joint trajectories consisting of: (1) 73.1% following low stable perpetration and low stable victimization (i.e., non-involvement group), (2) 7.6% following increasing victimization and perpetration, (3) 6.1% following high/decreasing for both perpetration and victimization, and (4) 13.2% remaining at high levels of victimization and low levels of perpetration (i.e., targets). In this study, trends in specific forms of bullying were examined (i.e., verbal, relational, and physical bullying/victimization), and no specific differences were found, suggesting bullying victimization may be better defined by degree (i.e., severity) rather than by type (see also Haltigan & Vaillancourt, 2018; Nylund et al., 2007; Wang et al., 2010). Given these results, I did not examine different forms of bullying involvement, but rather, examined involvement in bullying as a composite score that combines general bullying with physical, verbal, social, and cyber bullying (Haltigan & Vaillancourt, 2018).

Taken together, researchers examining the trajectories of bullying victimization and perpetration have consistently found a large group of children with limited bullying involvement (73% to 83%) and a smaller portion of youth chronically involved in bullying as targets or perpetrators (17% to 27%; Barker et al., 2008; Cho & Lee, 2019; Haltigan & Vaillancourt, 2014; Zhou et al., 2020). However, there remains inconsistencies among the studies on the joint trajectories of bullying victimization and perpetration. Previous work has found high decreasing levels of both bullying victimization and perpetration (Zhou et al., 2020; Cho & Lee, 2020), whereas these trajectories were not found in the other studies (Baker et al., 2008; Haltigan & Vaillancourt, 2014). Moreover, Baker et al. (2008) and Zhou et al. (2020) found an increasing reciprocal pattern of victimization and bullying over time (i.e., bully-victims), whereas Baker et al. (2008) and Haltigan and Vaillancourt (2014) found a small group of youth initially high on bullying victimization experienced an increase in bullying perpetration over time, suggesting a transition from targets to perpetrators. Although previous research provides an overview of the developmental joint trajectories between bullying victimization and perpetration, they covered a relatively limited time frame (i.e., 3 to 6 years), limited age ranges (i.e., middle school), or were assessed biannually (Barker et al., 2008; Cho & Lee, 2020; Haltigan & Vaillancourt, 2014; Walters et al., 2021; Zhou et al., 2020). I expanded on these studies by assessing the joint

trajectories of bullying victimization and perpetration across 8 years of development, from childhood to late adolescence (i.e., Grade 5 to Grade 12).

Despite inconsistencies, there appears to be, for a small subgroup of children, a dynamic interplay between initial bullying victimization and the subsequent development of bullying behaviour and vice versa (Barker et al., 2008; Cho & Lee, 2020; Haltigan & Vaillancourt, 2014; Walters, 2021; Walters & Espelage, 2018; Zhou et al., 2020). This development pattern is represented in a small group of children. Why is this the case? Previous research within the area has largely focused on psychosocial risk and protective factors associated with the maladjustment of developmental patterns of bullying victimization and perpetration (Marini et al., 2008). For example, the association between emotion regulation and bullying involvement (Hanish et al., 2004; Rosen et al., 2012). Although studies investigating psychosocial risk and protective factors are useful, they fail to account for underlying biological processes that may influence bullying trajectories. Vaillancourt (2018) reviewed the complex interaction between bullying victimization as a chronic stressful experience, and the underlying biological mechanisms that lead to impairment. This review strongly emphasized the need for studies to parse these relations and increase the biopsychosocial comprehension of bullying victimization and perpetration, a gap my thesis addresses.

Emotion regulation is developed through complex interactions between multiple factors that influence a child's ability to thrive within their environment. That is, not all children who are bullied develop maladaptive forms of emotion regulation skills. Other factors such as the interaction between social and biological factors conferring risk for maladaptive patterns must be considered to gain clarity on understanding these heterogeneous outcomes. Indeed, emotional dysregulation is hypothesized to be a heritable characteristic risk factor for involvement in bullying as both a target and perpetrator (i.e., bully-victims; Ball et al., 2008). One possible mechanism that is shown to be associated with emotion processing difficulties in stressful situations that has not been explored longitudinally in the context of bullying and emotion regulation, is the single nucleotide polymorphism *COMT Val158Met* (Tunbridge et al., 2006). Considering that bullying is a highly stressful situation that evokes negative emotional responses, children with the at-risk allele of *COMT Val158Met* may have increased difficulties regulating their emotions when bullied. These children may retaliate in unfavorable manners further impacting their peer relationships leading to increased involvement in bullying. In other words, a

reactive effect could be seen where the biological risk for poor emotion regulation leads to increased involvement in bullying as targets and perpetrators, further impacting their emotion processing, creating a positive feedback loop (Harris, 1995; Rosen, 2014; Palamarchuk & Vaillancourt, 2022). In my thesis, I aimed to gain a better understanding on how the single nucleotide polymorphism *COMT Val158Met* influences the joint trajectories of bullying victimization and perpetration. Specifically, I investigated the influence of *COMT Val158Met* on the joint trajectories of bullying victimization and perpetration across an 8-year time span (Grade 5 to Grade 12) to gain a better understanding of this complex relation.

COMT Val158Met and Emotion Processing

There are a variety of genetic factors associated with maladjusted developmental patterns (e.g., *5-HTTLPR, MAOA, DRD4*), but a common polymorphism on the *COMT* gene, found on chromosome 22q11.2, is growing in interest (Vaillancourt et al., 2018). The single nucleotide polymorphism results in an amino acid change from valine to methionine at codon 158 of the membrane known as *COMT Val158Met* (rs4680). The common single nucleotide polymorphism encodes the enzyme catechol-O-methyltransferase (COMT) involved in the catabolizing of catechol compounds including dopamine (Byers et al., 2020). The enzyme COMT has a role in the function of the dopamine system, playing an important part for the breakdown of dopamine in the prefrontal cortex (Tunbridge et al., 2006), the part of the brain associated with planning and decision making. The two alleles of interest (i.e., Val and Met) have differing levels of thermostability impacting enzyme activity (Lotta et al., 1995; Scanlon et al., 1979; Weinshilboum & Dunnette, 1981). Chen et al. (2004) found that adults who were homozygous for the Val allele had 38% higher COMT enzyme activity than adults who were homozygous for the Met allele. In other words, the COMT enzymes coded by the Val allele appear to have greater stability at physiological temperature compared to the COMT enzyme coded by the Met allele, resulting in greater functioning of the COMT enzyme in Val carriers (Lotta et al., 1995; Chen et al., 2004). Considering the COMT enzyme's influence on the dopamine system, Met carriers would have increased dopamine levels in the prefrontal cortex (PFC) compared to Val carriers (Meyer-Lindenberg et al., 2005; Tunbridge et al., 2006).

Dopamine modulates brain areas that play an important role in cognitive functioning and emotion processing (Gordon-Fennell & Stuber, 2021; Nieoullon & Coquerel, 2003). Therefore, the findings that *COMT Val158Met* influences emotional processing and cognitive functioning is

not surprising considering the polymorphism's association with the modulation of dopamine levels (see Mier et al., 2010 for a meta-analysis; Tunbridge et al., 2006). Neuroimaging studies have found increased brain activity in the corticolimbic circuit of emotional processing and arousal in Met carriers of *COMT Val158Met* compared to Val carriers (Drabant et al., 2006; Lonsdorf et al. 2011; Smolka et al., 2005). For example, Drabant et al. (2006) found increased hippocampus, amygdala, and ventrolateral prefrontal cortex activation in Met carriers (i.e., homozygous and heterozygous), compared to homozygous Val carriers when viewing faces displaying negative emotions among both men and women. Smolka et al. (2005) also found increased activation in the left hippocampus, right amygdala, and right dorsolateral prefrontal cortex in both men and women Met carriers when exposed to unpleasant stimuli such as unpleasant pictures. Similarly, Rasch et al. (2010) found homozygotes for the Met allele had increased activation in the right amygdala in response to processing unpleasant pictures compared to Val carriers. Moreover, Lonsdorf et al. (2011) reported that homozygotes for the Met allele showed higher amygdala reactivity to angry faces compared to carriers of at least one Val allele in men and women. However, these relations have not been explored in children and adolescents.

Met allele's impact on emotional processing is also supported by a meta-analysis by Mier et al. (2010) which reported a significant relation between *COMT Val158Met* genotype (i.e., Met/Met versus Val/Val) and prefrontal activation with a large effect size (d=0.73). In this meta-analysis, it was proposed that the Met allele may pose a risk for emotional dysregulation and the Val allele may confer a risk to executive cognitive functioning. Neuroimaging studies discussed above support this hypothesis, which is in line with the Warrior/Worrier model suggesting a trade-off between the Met and Val alleles (Goldman et al., 2005). The Warrior/Worrier model predicts that the Val allele increases emotional resilience at the expense of cognitive functioning, whereas the Met allele offers increased cognitive functioning at the expense of emotional resilience (Goldman et al., 2005).

In contrast, there are studies that have found Val carriers to have increased activity in brain areas involved in emotion processing when viewing an unpleasant stimulus (Domschke et al. 2012; Tunbridge et al., 2006; Weiss et al., 2007). For example, Domschke et al. (2012) found increased amygdala activation solely in women Val carriers when viewing angry/fearful facial stimuli. It is important to note that most of the studies linking Val carriers to increased activity in

brain areas involved in emotion processing (e.g., PFC and amygdala) were in clinical samples, hinting that the relation between *COMT Val158Met* and dopamine function may be more complex than simply the Met allele being associated with increased enzyme activity and the Val allele being associated with decreased enzyme activity (Tunbridge et al., 2006; Domschke et al., 2007; Lelli-Chiesa et al., 2011; Vai et al., 2017). An individual's environmental context, genetic background, and potential predisposition to dopaminergic abnormalities could play a large part on how *COMT Val158MET* exerts its effects, accounting for these inconsistencies across the literature (see Tunbridge et al., 2006 for a review). For example, Parkinson's disease is associated with dopaminergic abnormalities, which may influence an alternate effect of *COMT Val158Met* (Foltynie et al., 2004; Turnbridge et al., 2006). These findings are broadly consistent, suggesting Met carriers are at greater risk for emotional dysregulation compared to Val carriers.

Behavioural effects of *COMT Val158Met* have also been observed with similar findings to neural measures supporting the Warrior/Worrier model (Goldman et al., 2005). In a sample of boys and girls aged 9 to 15 years old, homozygous Met carriers were more likely to express greater anger during psychosocial tasks compared to homozygous Val carriers (Oppenheimer et al., 2013). In line with these findings are homozygous Met carriers, compared to homozygous Val carriers, showing greater negative bias toward perceiving neutral faces as expressions of anger, and attentional bias toward negative emotional expressions (Gohier et al., 2014; Gong et al., 2013). These findings lend support for the Met allele being a potential risk factor for emotional dysregulation, suggesting allelic variation on *COMT Val158Met* may influence youth involvement in bullying, such as bully-victims.

Previous findings have also associated the Met allele with higher stress responses (Armbruster et al., 2012; Jabbi et al., 2007). Jabbi et al. (2007) found higher stress response and higher endocrine response in homozygous Met carriers after completing a stress task compared to homozygous Val carriers. Additionally, homozygous carriers of the Met alleles have been associated with higher cortisol responses, especially in children who have experienced stressful life events (Armbruster et al., 2012). The potential increased vulnerability to adverse environments in carriers of the Met allele would make it an excellent candidate when examining bullying involvement given that bullying is an extremely stressful event (Palamarchuk & Vaillancourt, 2022). Although previous research has emphasized an association between the Met allele and emotional dysregulation, there remain inconsistencies. Amstadter et al. (2012) found

in 9 to 13 year olds, Val carriers were more likely to quit a stressful task compared to children without a Val allele, suggesting higher distress tolerance associated with carriers of at least one Met allele. Boettiger et al. (2007) also found decreased reward bias in Met carriers compared to those homozygous for the Val allele, suggesting increased emotion regulation. There have also been findings showing no overall effect of *COMT Val158Met* on emotion processing (Defrancesco et al., 2011; Kempton et al., 2009; Tamm et al., 2016).

To the best of my knowledge, there is only one study on the role of *COMT Val158Met* in the context of bullying victimization and perpetration. In a longitudinal study, Cao et al. (2017) investigated the moderating role of *COMT Val158Met* between bullying victimization and depressive symptoms. Participants consisted of a sample of 757 adolescent boys assessed at two time points (Grade 5 and Grade 6). Cao et al. found that *COMT Val158Met* and the *MAOA* gene moderated the relation between peer victimization and depressive symptomology. That is, adolescent carriers of both the Met allele and the *MAOA* G allele appeared more sensitive to the adverse experience of bullying victimization compared to other genotype combinations. Although useful, the study fails to capture the complex developmental processes involved between genes and environment. Future longitudinal studies assessing multiple time points over an extended period are needed to gain a comprehensive understanding of these developmental processes, considering that involvement in bullying also changes over time.

Current Study

My research objective was to examine the role of the single nucleotide polymorphism *COMTVal158Met* in relation to trajectories of bullying victimization and perpetration from childhood to adulthood. Specially, I examined the biological influence of the Met and Val allele of *COMT Val158Met* on the development of bullying involvement across eight years of development. Given prior research that bullying is best understood by degree/severity rather than type (Haltigan & Vaillancourt, 2018), I examined involvement using bullying as a composite score that combines general bullying with physical, verbal, social, and cyber bullying.
My primary research question was: What are the allelic differences (i.e., Met and Val allele) of *COMT Val158Met* polymorphism between the bully-victim and the non-involvement group?

Hypotheses

In line with previous research, I predicted that at least three joint trajectories of bullying victimization and perpetration will be found consisting of a low stable group (i.e., children

uninvolved in bullying as targets or perpetrators), and two bully-victim groups such as moderate/declining victimization and moderate/increasing bullying joint trajectory (i.e., those who transition from targets to perpetrators). Considering its association with emotional dysregulation, I predicted that carriers of the Met allele would be more likely to be involved in bullying as both targets and perpetrators (i.e., bully-victims), compared to homozygous carriers of the Val allele. Furthermore, carriers of the Met allele were predicted to follow a moderate/declining victimization and moderate/increasing bullying joint trajectory compared to homozygous Val carriers. That is, individuals carrying at least one Met allele who are bullied were expected to go on to bully others over time. Moreover, homozygous Val carriers were predicted to be overrepresented in the non-involvement group compared to homozygous and heterozygous Met carriers.

Sex differences have been observed across studies suggesting COMT Val158Met may impact brain function differently for boys and girls. However, there are inconsistent results across the literature, thus sex differences should be addressed as a potential confound (Harrison & Turnbridge, 2008; Tunrbridge et al., 2006; Hawn et al., 2015). Furthermore, there are sex differences in bullying involvement with boys worldwide reporting more involvement in bullying as both targets, perpetrators, and bully-victims than girls (Cook et al., 2010). Yet, in Canada, girls tend to report more bullying victimization than boys (Inchley et al., 2020; Vaillancourt et al., 2021). Given these differences, the moderating role of sex was explored, and no directional predictions were made.

Theoretical Framework

Many existing studies have examined the genetic and environmental interactions concerning behavioural outcomes. From an evolutionary-biological basis, the diathesis-stress model states that behavioural, physiological, and genetic factors influence one's susceptibility to environmental situations, either optimizing or minimizing survival rate (Belsky & Pluess, 2009; Zuckerman, 1999). Moving beyond the diathesis-stress model, the differential susceptibility model, posited by Belsky and Pluess (2009) accounts for both adverse and beneficial environmental influences on individual attributes rather than solely negative environmental stressors. For example, some individuals may be at greater risk for following maladjusted trajectories in adverse environmental contexts such as bullying victimization (Belsky & Pluess, 2009; Haltigan & Vaillancourt, 2014). I propose the differential susceptibility model as the

theoretical framework for exploring the outcomes of the interactions between genes and the environment. Differences in alleles (i.e., Met and Val) on *COMT Val158Met* were explored in relation to susceptibility to bullying involvement within a school environment. According to the differential susceptibility hypothesis, children with genetic predispositions for increased emotional resilience (i.e., carriers of the Val allele) may fair better at mitigating bullying experiences compared to children with the Met allele which is associated with emotional dysregulation (Belsky & Pluess, 2009; Cooley & Fite, 2016; Mier et al., 2010). Considering the adverse long-term outcomes of bullying victimization, individuals who are carriers of the Met allele should benefit from not being subjected to bullying victimization and perpetration.

Methods

Participants and Procedures

Data from the McMaster Teen Study was used for this study. The McMaster Teen Study is an ongoing longitudinal cohort study that began in the spring of 2008 that was designed to examine mental health, bullying, and academic achievement using a multi-method, multi-informant approach. Ethics approval was received from the school board and the associated university every year. Participants were recruited from 51 randomly selected primary schools within a large southern Ontario public school board. Parental consent and student assent were also received each year for children's participation within the student survey and telephone interviews. In the first year of the study, youth completed the survey with a pen and paper at school. In recent years, the youth completed self-reports from home via paper and pen or an online format. Compensation for participation within the study consisted of gift cards worth $5 to $50, depending on the year of participation. Demographic information such as biological sex and age was collected from each participant and their parents. Household income was collected from the parents of participants. Participants were genotyped (i.e., for Val and Met) on the single nucleotide polymorphism *COMT Val158Met*, collected through saliva samples when they were between the ages of 18 and 20 years.

Data collected annually from Time 1 to Time 8 was used to model the joint trajectories of bullying victimization and perpetration. There was a total of 875 participants recruited to be a part of the longitudinal study at Time 1 and 80% (n = 703) contributed at least one follow up time point (from Grade 6 to Grade 12). To be included in the analytic sample, participants needed to have data on bullying victimization and perpetration on at least one time point between

Time 1 (Grade 5) to Time 8 (Grade 12). The final analytical sample consisted of 701 participants of which 53% of participants were girls. Most participants were self-reported European Canadian (White) = 76%, followed by African/West–Indian–Canadian = 4%; South–Asian–Canadian = 4%; Indigenous–Canadian = 2%; Asian–Canadian = 2%; Middle–Eastern–Canadian = 2%; South/Latin American–Canadian = 1%; Other = 2% and 8% of participants did no report their ethnicity. The median household income of participants was $70,000-80,000 and the median of parent completed education were college diploma or trades certificate. Participants were close to 11 years old at Time 1 (M_{age} = 10.93; *SD* = .33) and close to 18 years old at Time 8 (M_{age} = 18.00; *SD* = .35)

Hardy-Weinberg Equilibrium. The Hardy-Weinberg Equilibrium (HWE) is a principle used to explore whether or not allele frequencies for a population are expected to remain consistent over time under the assumption of a large population size, no natural selection, no mutation rate, no genetic drift and random mating (Hardy, 1908; Weinberg, 1908). The HWE was applied to test if there was significant deviations between the observed genotype frequencies and the expected genotype frequencies within the sample. There were 354 particpant that were genotyped (i.e., for Val and Met) on the single nucleotide polymorphism *COMT Val158Met* that were tested for HWE. The observed frequencies within the sample were $p = 0.49$ and $q = 0.51$ for the Val and Met alleles, respectively. The expected genotype frequencies were Val/Val = 0.24, Val/Met = 0.50 and Met/Met = 0.2612. Therefore, the expected count for each group was as follow: Val/Val = 84.53, Val/Met = 176.96 and Met/Met = 92.46. A chi-square test was used to test for HWE and revealed there was no significant deviation between the observed and expected frequencies of alleles ($\chi^2 = 0.009$, df = 2, $p = 0.995$), suggesting that the sample was in HWE.

Measures

Bullying Involvement

Involvement in bullying perpetration and victimization was assessed using five items adapted from the Olweus Bully/Victim Questionnaire (Olweus, 1994; Vaillancourt et al., 2010b). Participants were provided with a standard definition of bullying to ensure an accurate measure of the variable then asked about their general experience with bullying perpetration (i.e., “Since the start of the school year [September], how often have you taken part in bullying another student?”). The remaining four questions assessed experiences with physical (e.g., hitting,

shoving, kicking, spitting), verbal (e.g., name calling, mocking, hurtful teasing, or verbally threatening), social (e.g., excluding others from groups, gossiping, or spreading rumors about others), and cyber bullying (e.g., using computer or e-mail messages or pictures to hurt someone's feelings). Five similar questions were asked to assess for bullying victimization (i.e., "How often have you been bullied at school?"). Items were assessed on a five-point scale (0 = not at all to 4 = many times a week) and were averaged during each time point. Internal reliability for the bullying victimization items was good at each time point (α = .788 to .818). Similarly, internal reliability for the bullying perpetration items was good at each time point (α = .707 to .806).

COMT Val158Met

All participants were genotyped for *COMT Val158Met* to identify carriers of Met and Val using DNA from saliva samples collected at Time 8 (Time 9 to 10 if not completed prior) with the Oragene 500 DNA self-collection kit. The kit was mailed to participants with detailed instructions about how to collect and safely return the sample. All necessary mailing expenses and paperwork were provided to help ensure compliance. Participants were instructed to avoid eating, drinking, smoking, and chewing gum 30 minutes before providing their saliva sample, which required spitting into a funnel until the fill line was reached (~2 ml). Saliva samples were pre-labelled with participants' study ID code, and each participant recorded the time and date the sample was collected. Researchers performed DNA extraction and genotyping of *COMT Val158Met* using procedures based on the manufacturers' instructions and optimized in Dr. Paul Arnold's lab located in Calgary, Alberta, Canada. The lab has extracted over 20,000 samples from the Oragene 500 DNA self-collection kit with excellent results (mean concentration 160 ng/ul with 96% of samples having concentrations > 50ng/ul). All DNA samples were precisely quantified using PicoGreen (Molecular Probes). The region around the COMT Val158Met SNP was PCR-amplified and allelic discrimination was achieved with a Taqman assay run on an ABI-PRISM 7900HT real-time PCR system (Life Technologies).

Analysis

Analytic Plan

A latent class growth analysis (LCGA) was used to examine heterogeneity among the trajectories. LCGA allows for the identification of subgroups of individuals that share similar developmental patterns (Nylund-Gibson & Choi, 2018). The analysis included 8 years of

available participant data from Grade 5 (age 10) to Grade 12 (age 18) to identify the developmental progression of bullying victimization and perpetration in this sample. Bayesian information criterion (Jung & Wickrama, 2008; Nagin, 2005; Nylund et al., 2007), the Lo-Mendell-Rubin likelihood ratio test (LMR-LRT; Lo et al., 2001), and the bootstrapped likelihood ratio test (BLRT; McLachlan & Peel, 2000) were used to assess the best fitting model. To examine the probability of individuals belonging to a particular univariate trajectory group, posterior probabilities >.70 was used (Nagin, 2005). The final number of univariate trajectories was decided by considering theoretical soundness to ensure enough individuals were in each group (>10 participants), and mean levels and patterns were consistent with previous literature. The best fitting univariate trajectories were used to examine the joint trajectory models of bullying victimization and perpetration. Contrast codes were created to compare each of the two bully-victim groups, with the non-involvement group selected as the contrast. Logistic regression was used with the contrast codes to examine allelic differences (i.e., Any Met versus homozygous Val carriers) between the bully-victim groups and the non-involvement group. Sex differences were examined using a chi-square test.

Results

Missing Data Analyses

Little's MCAR test was used to examine whether data was missing at random for bullying victimization and perpetration. Little's MCAR test was significant ($\chi_2(1033) = 1259.46$, $p<.001$). After applying the Benjamini-Hochberg correction, the false discovery rate was .002 and two *t*-tests remained. The *t*-test revealed youth with data in Grade 9 reported higher mean bullying victimization ($t(196) = 3.1$, $p = .002$) rates in Grade 7 compared to those missing in Grade 9. The t-test also revealed youth with data in Grade 6 reported higher mean bullying perpetration ($t(62.6) = 3.3$, $p = .002$) rates in Grade 10 compared to those missing data in Grade 6. The results suggest children involved in bullying were more likely to remain in the study. Considering these were the only differences, missingness related to the outcomes was treated as ignorable.

Descriptive Statistics

Means and standard deviations and the number of participants who completed bullying victimization and perpetration items across elementary and secondary school are presented in Table 1. Correlations between bullying victimization and perpetration from Grade 5 to Grade 12

are presented in Table 2. Bullying victimization and perpetration were significantly positively correlated at all time points except for Grade 5 victimization with Grade 10 to 12 perpetration, Grade 6 victimization with Grade 11 perpetration, and Grade 5 perpetration with Grade 8 to 12 victimization.

Table 1

Descriptive and Frequency Statistics for all Study Variables

		Sample Range			
	n	Min	Max	*M*	*SD*
Bullying victimization					
Grade 5	648	0.00	3.80	0.84	0.76
Grade 6	599	0.00	3.60	0.66	0.69
Grade 7	548	0.00	3.40	0.59	0.65
Grade 8	506	0.00	3.60	0.58	0.64
Grade 9	489	0.00	2.80	0.41	0.49
Grade 10	452	0.00	3.20	0.40	0.53
Grade 11	436	0.00	3.60	0.30	0.44
Grade 12	448	0.00	3.60	0.30	0.44
Bullying Perpetration					
Grade 5	644	0.00	3.20	0.22	0.34
Grade 6	598	0.00	2.40	0.21	0.31
Grade 7	546	0.00	2.20	0.23	0.33
Grade 8	509	0.00	2.40	0.28	0.39
Grade 9	488	0.00	3.20	0.22	0.37
Grade 10	453	0.00	2.40	0.18	0.31
Grade 11	437	0.00	2.40	0.17	0.31
Grade 12	447	0.00	3.40	0.15	0.31
COMT					
Val/Val	85	-	-	-	-
Met/Met	93	-	-	-	-
Val/Met	176	-	-	-	-

Table 2

Correlations Among all Study Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. VIC GR5	-	.52**	.40**	.32**	.32**	.38**	.31**	.24**	.22**	.19**	.16**	.15**	.10*	.09	.09	.07
2. VIC GR6		-	.57**	.40**	.29**	.29**	.24**	.21**	.14**	.37**	.29**	.21**	.19**	.15**	.06	.18**
3. VIC GR7			-	.56**	.36**	.37**	.35**	.25**	.12**	.31**	.40**	.20**	.19**	.23**	.14**	.18**
4. VIC GR8				-	.46**	.49**	.44**	.27**	.03	.22**	.27**	.38**	.23**	.23**	.18**	.19**
5. VIC GR9					-	.53*	.54**	.34**	.09	.22**	.20**	.24**	.45**	.27**	.28**	.24**
6. VIC GR10						-	.58**	.49**	.05	.17**	.25*	.25**	.30**	.44**	.29**	.27**
7. VIC GR11							-	.52**	.04	.10*	.17**	.18**	.26**	.28**	.47**	.29**
8. VIC GR12								-	.06	.21**	.21**	.13**	.16**	.39**	.35**	.45**
9. PERP GR5									-	.40**	.44**	.32**	.26**	.19**	.14**	.22**
10. PERP GR6										-	.57**	.44**	.44**	.40**	.25**	.38**
11. PERP GR7											-	.55**	.45**	.45**	.33**	.34**
12. PERP GR8												-	.58**	.47**	.36**	.27**
13. PERP GR9													-	.57**	.51**	.36**
14. PERP GR10														-	.59**	.52**
15. PERP GR11															-	.44**
16. PERP GR12																-

Note: *$p < .05$. **$p < .01$; VIC=bullying victimization; PERP=bullying perpetration; GR=Grade

Developmental Trajectories

Bullying Victimization. Four possible classes of bullying victimization and perpetration were tested. A three-group solution was chosen as the final model based on model fit, parsimony, and theoretical soundness. There was an improvement of the BIC with each additional class

added to the model and BLRT values was significant for each of the models. Entropy scores were high and approximately equal across groups and LMR-LRT values were all non-significant. The three-class was favoured over the four-class solution because the latter resulted in clusters each totaling less than 2% of the sample. Fit indices are present in Table 3. Trajectory group names were chosen based on the most prominent features of the trajectory (i.e., mean levels, shapes, and slopes) relative to one another (see the final solution groups presented in figure 1). Most participants reflected a low initial level of victimization in Grade 5 with a curvilinear decline until the end of secondary school (low decreasing group; 74%, n = 516; see Table 4 for parameter estimates). The second group followed medium initial level of victimization remaining moderately stable overtime until the end of secondary school (moderate stable group; 23%, n = 163). The final group followed high initial levels of victimization at Grade 5 remaining stable overtime until the end of secondary school (high stable group; 3%, n = 22). Participants were well identified within their trajectory group as the posterior probabilities were .971 for the low decreasing group, .818 for the moderate stable group, and .820 for the high stable group.

Table 3

Fit Indices for Latent Class Growth Analyses for Bullying Victimization and Bullying Perpetration

No. of Groups	BIC	LMR-LRT *p*	BLRT *p*	Entropy
Bullying victimization				
1 Class	7339.82	-	-	-
2 Class	6448.39	0.007	< .001	0.833
3 Class	6233.97	0.443	< .001	0.841
4 Class	6042.47	0.680	< .001	0.833
Bullying perpetration				
1 Class	2704.11	-	-	-
2 Class	1626.61	0.153	< .001	0.868
3 Class	1416.62	0.258	< .001	0.842
4 Class	1269.38	0.853	< .001	0.847

Note. BIC = Bayesian information criterion; LMR-LRT = Lo-Mendell-Rubin likelihood ratio test; BLRT = bootstrapped likelihood ratio test.

Figure 1

Developmental trajectories of bullying victimization across childhood to late adolescence (ages 10 to 18).

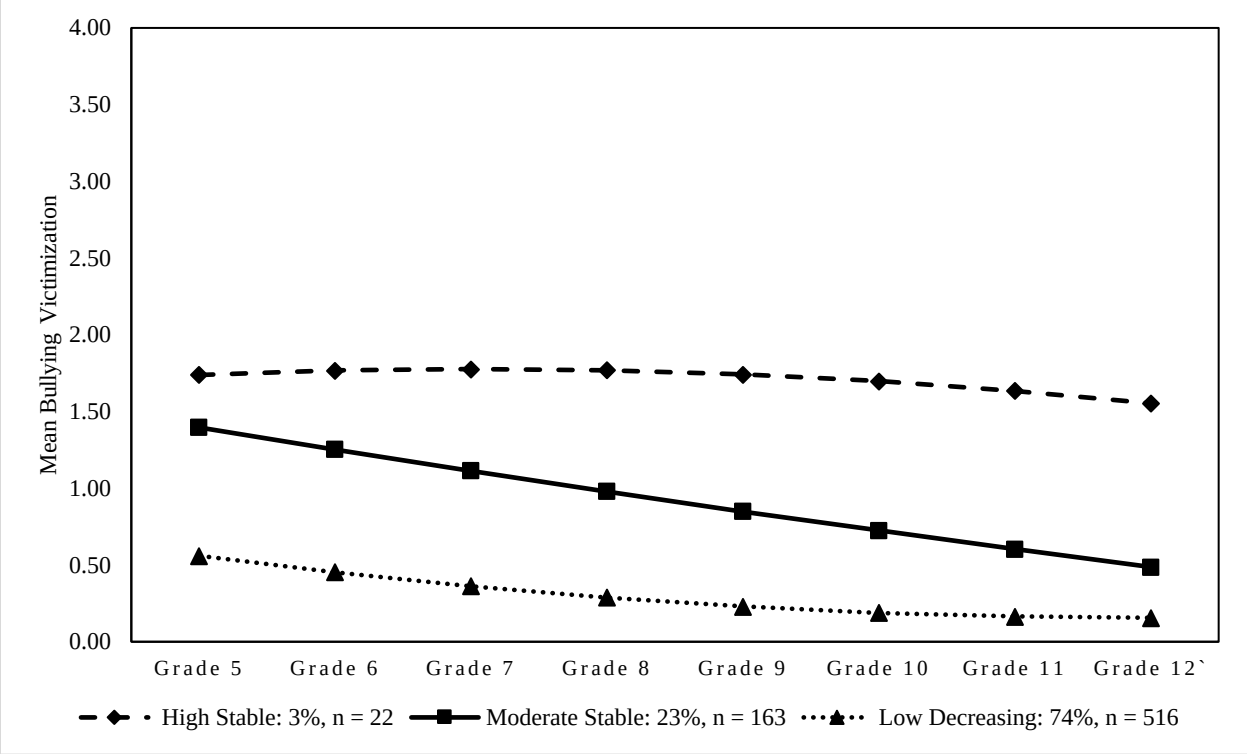


Table 4

Parameter estimates for bullying victimization and perpetration curves.

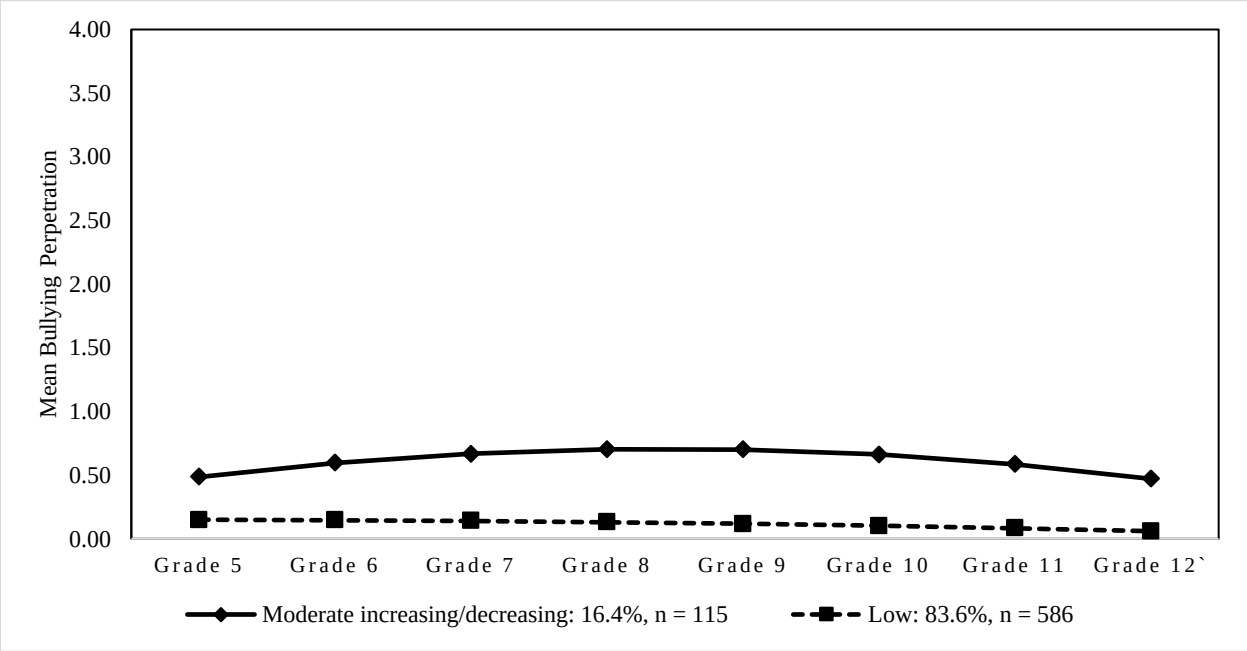
	n/% of sample	*unstandardized*	*p*
Bullying victimization			
Trajectory 1 – low decreasing	516/74%		
Intercept		0.56	<.001
Slope		-0.116	<.001
Quadratic		0.008	<.001
Trajectory 2 – moderate stable	163/23%		
Intercept		1.396	<.001
Slope		-0.146	0.06
Quadratic		0.002	0.767
Trajectory 3 – high stable	22/3%		
Intercept		1.739	0.02
Slope		0.037	0.925
Quadratic		-0.009	0.847
Bullying Perpetration			
Trajectory 1 – low stable	586/83.6%		
Intercept		0.15	0
Slope		-0.002	0.828
Quadratic		-0.002	0.072
Trajectory 2 - moderate increasing/decreasing	115/16.4%		
Intercept		0.487	<.001
Slope		0.128	<.001
Quadratic		-0.019	<.001

Bullying Perpetration. Similar patterns of results were found within the models of bullying perpetration. There was a steep drop in BIC from class 1 to class 2, and a steady decline thereafter and all LMR-LRT values were non-significant. BLRT was significant for 2-, 3-, and 4-class model and there was approximately equal high entropy across the groups. As a result, a two-group solution was chosen as the final model based on parsimony and theoretical soundness (see the final solution groups presented in figure 2). Most participants followed low stable levels of perpetration from Grade 5 to Grade 12 (low stable group; 83.6%, n = 586). The second group reflected a moderate initial level of bullying perpetration, increasing from Grade 5 to Grade 9, and decreasing from Grade 9 to Grade 12 (moderate increasing/decreasing group; 16.4%, n =

115). Participants were well identified within their trajectory group as the posterior probabilities were .986 for the moderate increasing/decreasing group and .986 for the low stable group.

Figure 2

Developmental trajectories of bullying perpetration childhood to late adolescence (ages 10 to 18).



Joint trajectories of bullying victimization and perpetration. There were six possible joint trajectory groups (3 X 2) with distinct developmental patterns of bullying victimization and perpetration. The top section of Table 5 reflects the proportion of participants in each group. Most of the participants reflected a joint trajectory pattern of low decreasing bullying victimization and low stable bullying perpetration (66.33%, *n* = 465). These youth had little involvement in bullying as both targets and perpetrators across the middle school to secondary school years (i.e., limited involvement group). The targets of bullying were represented in two groups. One group reflected a joint trajectory pattern of moderate stable victimization and low stable perpetration (16.26%, *n* = 114) and the other reflected a high stable victimization and low stable perpetration (1.00%, *n* = 7). The perpetrators were represented in one joint trajectory reflecting a low decreasing victimization and moderate increasing/decreasing perpetration (7.28%, *n* = 51). The final two joint trajectories consisted of the participants that engage in both

bullying victimization and perpetration over time (i.e., bully-victims). Group one was characterized by joint trajectories of moderate stable victimization and moderate increasing/decreasing perpetration (6.99%, *n* = 49). Group two was characterized by high stable victimization and moderate increasing/decreasing perpetration (2.14%, *n* = 15). Participants were well-identified within their trajectory group as the posterior probabilities for all joint trajectory groups were > .82.

Table 5

Joint and Conditional Probabilities of Peer victimization and perpetration

	Perpetration	
	Low Stable	Moderate Increasing/Decreasing
Victimization	Probabilities of Joint Trajectory Membership[a]	
Low Decreasing	0.922 (*n* = 465; 66.33%)	0.848 (n = 51; 7.28%)
Moderate Stable	0.829 (n = 114; 16.26%)	0.884 (n = 49; 6.99%)
High Stable	0.858 (n = 7; 1.00%)	0.823 (n = 15; 2.14%)
	Probabilities of Victimization Conditional on Perpetration[b]	
	Low Stable	Moderate Increasing/Decreasing
Low Decreasing	0.802	0.314
Moderate Stable	0.187	0.519
High Stable	0.011	0.167
	Probabilities of Perpetration Conditional on Victimization[c]	
	Low Stable	Moderate Increasing/Decreasing
Low Decreasing	0.916	0.084
Moderate Stable	0.605	0.395
High Stable	0.212	0.788

[a]Cells total 1.
[b]Columns total 1.
[c]Rows total 1.

Conditional probabilities. The bottom section of Table 5 shows the conditional probabilities of the trajectories of bullying victimization trajectories as a function of the bullying perpetration trajectories, and the conditional probabilities of the trajectories of perpetration as a function of bullying victimization trajectories. These results suggest that low stable bullying perpetration was a better indicator of low decreasing bullying victimization (.802) and moderate

increasing/decreasing bullying perpetration was a better predictor of moderate stable bullying victimization (.519). In contrast, low decreasing (.916) and moderate stable (.605) bullying victimization trajectory was a better indicator of low stable bullying perpetration trajectory. Moreover, high stable (.788) bullying victimization trajectory was a better indicator of moderate increasing/decreasing bullying perpetration trajectory.

Predictors of Trajectory Group Membership

Sex differences in proportion of sample in joint trajectories. Sex differences in the joint trajectory groups were examined by comparing the proportions of boys and girls in each joint trajectory groups (vs. all others). There were differences in the proportion of boys and girls across joint trajectory group $\chi_2(5) = 16.490$, $p = .006$. There were significantly more boys in the low decreasing victimization and moderate increasing/decreasing perpetration group than girls (9.4% boys vs. 5.4% girls) based on the adjusted standardized residuals of -2.0.

Sex differences in proportion of sample for COMT Val158Met. There were no differences in the proportion of boys and girls of COMT Val158Met Val/Val carriers compared to any Met carriers $\chi_2(1) = .258$, $p = .612$.

***COMT Val158Met* and Bully-Victims.**

COMT Val158Met did not significantly differentiate the joint moderate stable victimization and moderate increasing/decreasing perpetration from the joint low decreasing bullying victimization and low stable bullying perpetration (OR = 1.748, 95% CI [.766, 3.988], $p = .185$). *COMT Val158Met* also did not significantly differentiate the joint high stable victimization and moderate increasing/decreasing perpetration from the joint low decreasing bullying victimization and low stable bullying perpetration (OR = 4.428, 95% CI [.961, 20.409], $p = .056$).

Discussion

The aim of my thesis was to investigate the influence of the single nucleotide polymorphism *COMT Val158Met* on the joint trajectories of bullying victimization and perpetration. Bullying impacts over 30% of youth (Biswas et al., 2020) and is linked to a host of long-term mental health problems (McDougall & Vaillancourt, 2015; Vidourek et al., 2016; Wolke et al., 2013). Although bullying can have a detrimental impact on children's development, few studies have investigated the underlying biological risk factors for involvement in bullying (Vaillancourt, 2018). Understanding the gene by environmental interactive effect on

developmental trends in bullying victimization and perpetration is important to gain a better picture of the dynamic process of bullying in children and adolescents. This more comprehensive understanding can be used to appropriately implement effective and targeted prevention and intervention programs (Zych et al., 2020). The present thesis contributes to the existing literature in a few notable ways. Despite the growing literature on bullying, this is the first study to investigate *COMT Val158Met* in the context of bullying victimization and perpetration joint growth, thus increasing the biopsychosocial comprehension of bullying involvement. Furthermore, the examination of the co-occurring involvement of bullying victimization and perpetration is over a larger time frame (i.e., 8 years) than what has been previously studied (see Walter et al., 2021 for a meta-analysis). Investigating the joint trajectories of bullying victimization and perpetration across 8 years of development enabled us to better assess the dynamic changes in bullying involvement, from childhood to late adolescence, and to capture the subgroups of children most at risk for diverse maladjustment (i.e., bully-victims).

Univariate trajectories of bullying victimization and perpetration

When examining the univariate trajectories of bullying victimization alone, three trajectory groups consisting of a low decreasing group (74%), moderate stable group (23%) and a high stable group (3%) were found. Two trajectories were found when examining the univariate trajectories of bullying perpetration alone. The trajectories consisted of a low stable group (84%) and moderate increasing/decreasing group (16%). These developmental trends are consistent with previous research examining the univariate trajectories of bullying victimization and perpetration (Brendgen et al., 2016; Espelage et al., 2018; Geoffroy et al., 2018; Pepler et al., 2008; Reijntjes et al., 2013; Vaillancourt et al., 2023).

Joint Trajectories of bullying victimization and perpetration

The joint trajectories model identified six subgroups of children involved in bullying either as targets, perpetrators, or bully-victims. Taking a joint trajectory approach to examining bullying victimization and perpetration across elementary to secondary school allowed for (1) a more in-depth investigation of the complex development and co-occurrence of bullying victimization and perpetration and (2) to clarify discrepancies within the literature concerning the temporal ordering of bullying victimization and perpetration. As predicted, most participants had little involvement in bullying as both target and perpetrators across elementary school to secondary school (66%). These results are consistent with previous studies (e.g., Craig et al.,

2020) highlighting that most youth are not involved in bullying. Nevertheless, as seen in population-based studies, about 30-35% of youth are involved in bullying victimization and perpetration (Biswas et al., 2020). This leaves one out of three youth at risk for severe and lasting negative mental health and behavioural outcomes (see McDougall & Vaillancourt, 2015, for a review).

Targets and Perpetrators of Bullying. The targets of bullying were represented within two joint trajectories. There was a small group of participants who experience high consistent victimization across elementary to secondary school (1%). Although this group represents only 7 youth, their experience with peer abuse was extreme and deserves attention and intervention. There was also a group of participants who experienced moderate stable levels of victimization over time (16%). Both groups were relatively uninvolved in bullying perpetration. The perpetrators of bullying were represented in one joint trajectory reflecting moderate initial levels of perpetration increasing until the end of middle school and then declining until the end of secondary school and low decreasing levels of victimization (7%).

Contrary to previous findings, we did not identify decreasing or increasing trajectories of bullying victimization. Previous research in the area has suggested that bullying victimization is higher during childhood and declines as children mature (Smith et al., 1999; Smith et al., 2001). Although the victimization trends found within my study may be at odds with previous research, it may be that investigating a large time frame from elementary school to the end of secondary school is in fact a truer account of victimization patterns over time. Previous research that has identified decreasing trends in victimization assessed children within a shorter time frame across elementary school and middle school (i.e., when bullying is highest; Baker et al., 2008; Biggs et al., 2010; Brendgen et al., 2016; Goldbaum et al., 2003; Haltigan & Vaillancourt, 2014; Sumter et al., 2012; Zhou et al., 2020). Assessing bullying involvement within a shorter time frame and at a younger age where bullying victimization and perpetration is highest, may capture more subtle changes in bullying involvement as children learn to navigate social relationships. The bullying perpetration trends found are generally consistent with previous findings suggesting there are some youth who engage in moderate levels of perpetration (Espelage et al., 2008; Haltigan & Vaillancourt, 2014; Pepler et al., 2008). Although they represent a small portion of children, they pose a substantial risk to the wellbeing of their targets (McDougall & Vaillancourt, 2015; Moore et al., 2017; Reijntjes et al., 2010) and are at risk of poorer outcomes as well

(Bender & Lösel, 2011). Children involved in bullying perpetration are at significant risk for future violent behaviour (Copeland et al., 2013; Lösel & Bender, 2011; Ttofi et al., 2012), and thus, effective intervention and prevention programs should be implemented in elementary school (i.e., when bullying victimization and perpetration is highest; Vaillancourt et al., 2010; Vaillancourt et al., 2023). Implementing intervention and prevention programs when bullying involvement is highest can help decrease the risk of future involvement in bullying (Gaffney et al., 2019), and in turn, minimize the long-term adverse effects of bullying victimization and perpetration.

Bully-victims. Out of the six possible joint trajectories, particular interest was in the joint trajectories representing bully-victims given their predicted links to *COMT Val158Met.* There were two bully-victim trajectories identified in the current study. The first group followed moderate consistent levels of bullying victimization and moderate increasing/decreasing levels of perpetration (7%) and the second group followed high consistent levels of bullying victimization and moderate increasing/decreasing levels of bullying perpetration (2%). The current findings help clarify some of the inconsistent findings concerning the temporal ordering of bullying (Barker et al., 2008; Haltigan & Vaillancourt, 2014; Walters et al., 2021). Previous research has suggested victimization may increase the likelihood of future perpetration (Barker et al., 2008; Haltigan & Vaillancourt, 2014; Walters & Espelage, 2018), but it remains unclear if this sequence follows a transactional pattern (e.g., target to perpetrator to target). In the present study, youth who were highly victimized did in time perpetrate against others but remained targets of high levels of perpetration (i.e., target to bully-victim), whereas youth who were moderately victimized were more likely to be uninvolved in bullying perpetration. The difference in the progression of these two groups could represent the ways children and adolescents cope with the trauma of being a target of bullying. As children mature, some targets of bullying may learn to cope with their bullying experiences by avoiding, ignoring, or relying on external resources. Conversely, children that are bullied at high levels may be at risk of developing maladaptive coping strategies. Children who perceive and interpret their stressors (i.e., bullying victimization) as severe and uncontrollable, may also have a heightened stress response leading to poor emotion processing (Palamarchuk & Vaillancourt, 2022). Bullying victimization is associated with poor emotion processing (Rudolph et al., 2016; Telzer et al., 2018, 2020; Will et al., 2016) and bully-victims are found to have poor emotion regulation (Garner et al., 2010; O'Brennan et al., 2009;

Schwartz et al., 2001). These children struggling with emotion regulation may cope with their anger and anxiety from being bullied by acting out or bullying others. This can in turn perpetuate and escalate bullying interactions leading to consistent involvement in bullying (Mahady et al., 2000). These children can also develop unhealthy cognitive schemas from their poor interpersonal relationships which promotes social learning — that is children learn to bully their peers from being bullied (Bandura, 1986; Walter & Espelage, 2018). Emotionally dysregulated bully-victims are consistently being exposed to the long-term adverse effect of being both a target and perpetrator of bullying. This may lead youth involved in bullying as both a target and a perpetrator to become even more dysregulated in adulthood as they continue to adopt these maladaptive behavioural patterns in their everyday life (Wolke & Lereya, 2015).

COMT Val158Met and Bully-Victims

The current study is the first to investigate *COMT Val158Met* in the context of bullying victimization and perpetration joint growth trends. There has been a growing interest in studying the biological underpinning of bullying involvement to provide insight into the mechanisms associated with heterogeneity outcomes of bullying victimization and perpetration. Gaining a better understanding of these mechanisms help explain why not all children who are targets or perpetrators of bullying go on to have long term maladaptive outcomes in adulthood such as internalizing and externalizing problems (McDougall & Vaillancourt, 2015). There is also a lack of knowledge concerning the association between the timing, duration, and severity of bullying victimization and perpetration, genetic risks, and adverse health outcomes. Understanding these knowledge gaps is pertinent when it comes to making evidence-based policy recommendations and implementing bullying prevention and intervention work.

Allelic variation of *COMT Val158Met* has been linked to different mental health problems and emotional dysregulation (Cao et al., 2017; Domschke et al., 2012; Shifman et al., 2004; Stein et al., 2005; Taylor, 2017). However, results are inconsistent (Domschke et al. 2012; Drabant et al., 2006; ; Lonsdorf et al. 2011; Smolka et al., 2005; Tunbridge et al., 2006; Weiss et al., 2007). Considering the Met allele of *COMT Val158Met* has previously been hypothesized to be associated with poor emotion processing (Drabant et al., 2006; Lonsdorf et al. 2011; Mier et al., 2010; Smolka et al., 2005), it was hypothesized bully-victims would be more likely to be carriers of the Met allele because bully-victims are found to be emotionally dysregulated (Garner et al., 2010; O'Brennan et al., 2009; Schwartz et al., 2001). However, in the present study, the

results were non-significant — there were no differences in allelic variations of *COMT Val158Met* between bully-victims and children uninvolved in bullying.

The genetic underpinnings of complex traits such as emotion processing are difficult to investigate. There is a complicated interplay between genetic and environmental factors which can make it difficult for researchers to explore. For instance, there are continuous interactions between genes and environment (e.g., epigenetic processes) which make it hard to capture gene by environmental interactions. Further, the use of a single gene variant rather than considering multiple gene variants may have hindered the investigation of trait outcomes. There is growing literature supporting that most human behavioural and disease outcomes are highly polygenic (Belsky & Domingue, 2023; Visscher et al., 2008). For example, a genome wide meta-analysis examining the association between independent genetic variations and depression using 807,553 individuals identified 102 independent genetic variants that were associated with depression (Howard et al., 2019). Therefore, although an individual may carry an allele that confers risk for maladjusted traits, they may also cary many protective alleles that could act as protection from the effects of a risk allele. Moreover, research examining single nucleotide polymorphism as a moderator are often underpowered leading to replicability failures within the literature (Burton et al., 2009; Munafò & Gage, 2013). The bully-victim groups I examined were also relatively small (i.e., 15 and 49 participants) leading to power issues. Therefore, future research should consider multiple gene variants and larger sample sizes when examining outcomes as it may represent the new benchmark when investigating candidate genes (Border & Keller, 2017).

In all, these fundamental problems encountered when examining candidate genes by environment interaction may help explain why we were unable to identify the influence of candidate genes on bully-victims' trends within my thesis. Future research should consider these challenges in examining candidate genes and evolve analytic procedures to gain a more comprehensive understanding how candidate genes interacted with the environment and their effect on trait outcomes. The results of my thesis highlight and add to the existing literature concerning joint trajectories of bullying victimization and perpetration, as well as the growing areas of research on candidate genes. Although there was no significant effect of *COMT Val158Met* on children that engage in both victimization and perpetration (i.e., bully-victims), my thesis highlights the need to further investigate the impact of genetics to help gain a comprehensive understanding of the biological underpinning of bullying. The lack in

understanding of the mechanisms behind bullying involvement significantly impacts the abilities for mental health professionals and school boards to implement evidence-based policy changes. Without having well-tailored approaches to reducing bullying based in science, there will be limited effectiveness in addressing this issue. There are youth that are suffering every day from the impact of bullying victimization and perpetration. This is why it is important that researchers and mental health professionals continue to investigate underlying mechanisms to help reduce bullying and its negative sequelae.

Limitations and Future Research Directions

The current study had several strengths such as the inclusion of a large sample of participants and consistent measures within an extended (i.e., 8 years) longitudinal design. Joint trajectories of bullying victimization and perpetration were also investigated rather than univariate trajectories as recommended to examine the overlap between the two roles of bullying (Marsh et al., 2022). Nevertheless, there remains some limitations to the current study. First, although the present sample was large, it was smaller than previous research that has investigated trajectories of bullying victimization and perpetration (see Walters et al., 2021 meta-analysis). A smaller sample can inhibit the ability to discover alternate trajectories from childhood to late adolescence and limits the cell size within each of the joint trajectories. Future research should use larger sample sizes when investigating predictors (i.e., candidate genes) of joint trajectories to increase power.Second, a noteworthy limitation pertained to the absence of the *COMT Val158Met* genotype for some of the participants within the data because genotyping of *COMT Val158Met* occurred at Time 9 within the longitudinal study. Third, even though joint trajectories allowed us to examine the dynamic interplay between bullying victimization and perpetration overtime, the analysis does not allow for causal predictions (Reijntjes, et al., 2013). Fourth, given that the genetic etiology of psychopathologies is likely polygenic, the use of a single nucleotide polymorphism approach rather than a polygenic approach may make it difficult to replicate in future studies and to draw conclusions from the findings (Belsky & Domingue, 2023; Howard et al., 2019; Visscher et al., 2008). Fifth, a drawback of the sample was that 76% of the participants were White, potentially resulting in population stratification. Population heterogeneity can be attributed to variation in allele frequencies across ancestral subpopulations thus large, diverse samples are ideal (Cardon & Palmer, 2003).

Keeping these potential shortcomings in mind, future studies should aim to have larger samples to ensure the statistical analysis is not underpowered (Burton et al., 2009; Moore & Thoemmes, 2016; Munafò & Gage, 2013) and investigate the interaction of multiple polymorphisms to capture a more refined understanding of the influence of candidate genes on trajectories of victimization and perpetration. Seeing that children involved in bullying follow different developmental trends and develop different mental health outcomes, understanding the mechanisms behind heterogeneity will help researchers and mental health professionals implement theoretically sound prevention programs to effectively minimize future involvement in bullying.

Educational and Clinical Implication

The findings of my thesis highlight the extent that youth engage in bullying victimization and perpetration across childhood to adolescence, while also exploring a possible biological factor associated with bullying involvement. A major implication of the present research is that bully-victims may be engaging in perpetration as a means to cope with the trauma of being a target. This highlights the need for comprehensive intervention and prevention programs to address the range of difficulties faced by bully-victims (McDougall & Vaillancourt, 2015; Lereya et al., 2015). Prevention and intervention programs for bullying are currently implemented through the educational system (Beeson & Vaillancourt, 2016). However, there is a great need for practitioners to screen and assess for bullying given that bullying is a major public health issue (NASEM, 2016), and children often avoid discussing bullying with other adults (Mishna et al., 2005; Trach et al., 2010). However, children and adolescents report feeling more comfortable discussing their bullying issues with health care practitioners (Scott et al., 2016; Vaillancourt et al., 2016). As such, counselling psychology professionals may be uniquely positioned to help targets of bullying. Moreover, keeping in mind that bully-victims are at risk for an array of long term maladjusted and mental health outcomes, practitioners should be vigilant in assessing the wide scope of issues faced by this subgroup of children. For example, bully-victims are often found to have emotion processing difficulties (e.g., Habashy, 2013; Swearer et al., 2001), which may contribute to the cycle of violence of bullying, (i.e., coping with victimization by perpetrating). Thus, counselling practitioners can advocate for policy changes within schools and help children involved in bullying access relevant resources to help

cope with the stress of being bullied which may in turn minimize their involvement as future perpetrators.

There are many therapeutic approaches that may be helpful in reducing the cycle of violence in bullying seen among bully-victims. Cognitive Behavioural Therapy (CBT; Beck & Beck 1995), which focuses on changing negative thought patterns and behaviour, can help bully-victims develop more efficient strategies to cope with unhelpful behaviour and manage difficult emotions that arise when involved in bullying. Considering bully-victims may develop a negative bias due to poor emotion processing, CBT may be helpful for children by developing proper evaluation of other children's behaviour (e.g., cognitive reappraisal; Palamarchuk & Vaillancourt, 2022). For example, reappraisal of stressful events to help children develop prosocial reactions to stressors that does not escalate or perpetuate future victimization. Other examples of therapeutic orientations that may be beneficial for bully-victims is mindfulness-based stress reduction (Kabat-Zinn, 2003; Roemer et al., 2015) which can help bully-victims developed coping strategies to manage difficult and negative emotions to help them establish healthier peer support networks. Moreover, given that bullying victimization can be considered a traumatic event (Idsoe et al., 2021), trauma informed approaches may be helpful in processing and healing individuals stressful experiences with bullying victimization (Nixon & Linkie, 2023).

The findings from my thesis also contribute to the growing literature on candidate gene influences on bullying involvement and maladjustment outcomes. Gaining insight into the biological underpinning to bullying involvement will help support researchers to develop strategies to reduce bullying victimization and perpetration. Investigating candidate genes in terms of bullying involvement also highlight the differences seen among individuals involved in bullying. That is, bullying is a complex behaviour that is influenced by multiple factors and gaining an understanding of the biological factors that contribute to bullying victimization and perpetrations will help shift the stigma away from the individuals to focus on working toward minimizing the harmful and stressful behaviour of bullying. Bullying victimization and perpetration can have a detrimental impact on development and with a growing body of research continuing to support the findings of the long-lasting negative outcomes of bullying, there is a great need to support and implement well-informed prevention and intervention efforts.

Conclusion

I investigated the role of *COMTVal158Met* in joint trajectories of bullying victimization and perpetration across childhood to late adolescence. This work helped fill a gap within the literature by gaining a better understanding of the influence of candidate gene polymorphisms on the involvement in bullying victimization and perpetration, as well as increasing the knowledge of the dynamic processes of bullying victimization and perpetration capturing the subgroup of children most at risk of diverse maladaptive outcomes, bully-victims (Zych et al., 2020; Lereya et al., 2015). Results from my thesis demonstrate that bully-victims engage in consistent levels of bullying victimization and perpetration. This pattern of behaviour is worrisome because children often maintain maladaptive behavioural patterns into adulthood which can have long term impacts on their overall health (McDougall & Vaillancourt, 2015). Although results did not lend support for allelic variation of *COMTVal158Met* influencing bully-victim developmental trends, it does not negate the importance of continuing to explore the impact of biological factors on bullying involvement to increase the biopsychosocial comprehension of bullying victimization and perpetration. The mechanisms behind bullying involvement and the reason why not all children develop significant mental and physical health problems are still relatively understudied, leaving clinicians at a disadvantage when offering recommendations for prevention and intervention work (Vaillancourt et al., 2017). Continuing to research the biological underpinning of bullying (e.g., candidate gene, epigenetic mechanisms, and neuroendocrinology) will help support policy recommendations and well-tailored, evidence-based prevention and intervention programs to adequately minimize bullying victimization with early interventions. Bullying victimization and perpetration, although common experiences, should not be a natural part of childhood. By continuing to investigate all these key gaps in evidence, clinicians and researchers will be able to effectively work together to minimize this stressful event.

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