DIRECT AND INDIRECT EFFECTS FROM AGGRESSION TO INTERNALIZING SYMPTOMS: A GENETICALLY CONTROLLED STUDY

by

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This dissertation was prepared under the direction of the candidate’s dissertation advisor, Dr. Brett Laursen, Department of Psychology, and has been approved by all members of the supervisory committee. It was submitted to the faculty of the Charles E. Schmidt College of Science and was accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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ABSTRACT

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Behavioral difficulties in the early school years pose a risk to psychosocial functioning. The failure model suggests that peer rejection explains longitudinal associations between aggression and internalizing symptoms. The model postulates that aggression leads to increases in peer rejection, which, in turn, contributes to internalizing symptoms. This study tests pathways posited by the failure model, examining direct and indirect longitudinal effects. Direct effects models examined associations between reactive aggression and internalizing problems, reactive aggression and peer rejection, and peer rejection and internalizing symptoms. A mediation model examined the indirect effect of reactive aggression to internalizing symptoms, via peer rejection.

Because distinct components of the failure model are presumed to share genetic influences, removing potential genetic contributions is important when examining the environmental influences over developmental pathways posited by the model. To this end, longitudinal tests were conducted with traditional (non-genetically controlled) and
MZ twin difference (genetically controlled) designs. The latter disentangled nonshared environment effects from those for genetic factors from environmental factors.

Participants included 682 twin pairs (237 male dyads, 235 female dyads, 210 mixed-sex dyads). Data were collected when children were in Kindergarten ($M=6.04$ years, $SD=0.27$), Grade 1 ($M=7.07$ years, $SD = 0.27$), and Grade 4 ($M=10.00$ years, $SD= 0.28$). Teachers provided reports of reactive aggression and internalizing symptoms. Peer rejection was assayed with peer nominations.

The results identified a strong genetic component to individual variables and to previous findings that purportedly support the failure model. Univariate ACE models revealed genetic contributions to each study variable. Bivariate Cholesky models revealed that same genetic factors partially accounted for associations between the variables. Although conventional path analyses revealed direct and indirect longitudinal associations from reactive aggression to internalizing symptoms, via peer rejection, genetically-controlled analyses revealed that none of these associations could be attributed to nonshared environmental factors.

The findings indicate that while conventional analyses provide evidence supporting the causal pathways posited by the failure model, genetically-controlled analyses do not. The study calls into question the veracity of the failure model and suggests that its purported environmental effects, are a product of error, arising from the influence of unrecognized genetic factors.
DEDICATION

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INTRODUCTION

Behavioral difficulties in the early elementary school years can impact psychosocial functioning throughout the lifespan, giving rise to long-term socioemotional difficulties (Lereya et al., 2015). Early-onset aggression, in particular, is a risk for the development of a host of difficulties (Coie et al., 1995; Loth et al., 2014). While researchers typically focus on mechanisms that explain how early aggression contributes to later externalizing symptoms, the role of aggression in subsequent internalizing symptoms is less clear. The failure model suggests that peer difficulties partially explain the link between aggression and internalizing symptoms (Patterson & Capaldi, 1990). In this model, early aggression fosters interpersonal difficulties (Ostrov et al., 2013), which, in turn, contribute to the emergence of anxiety and depression (Mrug et al., 2012). However, few empirical studies have tested the model. Further, there is reason to suspect that at least some of the purported effects are a product of common genetic influences and gene-environment interplay.

The current study employs a genetically-controlled design to examine direct and indirect longitudinal associations from reactive aggression to internalizing symptoms. First, a bidirectional model will explore direct longitudinal pathways from reactive aggression to internalizing symptoms, as well as for each of the intervening steps in the failure model (i.e., from reactive aggression to peer rejection and from peer rejection to internalizing symptoms). Second, a longitudinal mediation model will explore the indirect path from reactive aggression to internalizing problems via peer rejection. Direct
and indirect effects obtained using traditional (i.e., nongenetically informed) models will be contrasted with results from genetically controlled models.

**An Overview of Childhood Aggression**

Aggression is defined as a behavior intended to harm others (Krahé, 2013). Aggression can be distinguished by its different forms and functions. Forms describe the different types of aggression and the ways they are used (e.g., physically, relationally; Tremblay, 2000). Functions describe the motives behind displays of aggression, why it is used. There are two primary functions of aggression: reactive and proactive. Reactive aggression is triggered by anger or frustration. It is an immediate and largely impulsive response to a provocation, frustration, or threat, with the primary goal of hurting the source (Vitaro & Brendgen, 2005). Reactive aggression is often described as “hot-blooded” or “defensive” and is viewed as an unconditioned temperament-based reaction (Berkowitz, 1989). Theoretically, it is best described by the frustration-aggression model, which posits that arousal from frustration biases how a person interprets their environment. When frustrated or aroused by the environment, children may reactively aggress, assuming negative intent on the part of the target (Berkowitz, 1993). Proactive aggression, in contrast, is motivated by personal gain, such as securing goods or dominating others. It is best described by social learning theory, which posits that aggression is a learned behavior that allows an individual to obtain a desired goal (Bandura, 1973). Proactive aggression is often described as “cold-blooded” or “offensive” (Dodge et al., 1997) and is associated with bullying and remorselessness (Orobio de Castro et al., 2010). Although the two functions of aggression are distinct, conceptually, and empirically, they tend to co-occur, with a majority of aggressive
children displaying both types of aggression (Hubbard et al., 2009). Because they tend to co-occur, it is important to control for one when examining the other, so as to isolate the variance unique to the specific function of aggression.

**Pathways from Aggression to Internalizing Symptoms: Theoretical Models**

The aggressive behavior of early starters persists well beyond childhood. They are responsible for most adolescent and adult criminal activity (Malti & Rubin, 2018). Mental health problems are also prevalent among early starters (Coie et al., 1992). My dissertation will focus on how early aggressive behavior can give rise to internalizing symptoms, inwardly experienced maladjustment symptoms typically encompasses depressive and anxiety-related behaviors (Sloman et al., 2003). Several conceptual models have been advanced to explain how aggressive behavior leads to internalizing symptoms. Direct and indirect pathways are discussed.

**Direct Effects Models.**

Although there are no clear theoretical models that posit direct associations from general aggressive behaviors to internalizing symptoms, there are theories that suggest reactive aggression and internalizing symptoms may be associated through shared underlying emotional deficits. Reactive aggression initially stems from the frustration-aggression model, which postulates that aggression is driven by anger and a desire to retaliate. More recently, Berkowitz & Harmon-Jones (2004) have proposed that reactive aggression stems from heightened emotionality more broadly. Children who are temperamentally prone to over-react may be vulnerable to reacting aggressively to perceived threats or provocations (Vitaro et al., 2002). At the same time, emotional overreactivity may also make children vulnerable to experiencing depressive symptoms.
or anxiety if faced with loss or uncertainty. It is therefore likely that reactive aggression is associated with internalizing symptoms. Other theorists propose alternative explanation. Children who are reactively aggressive may experience shame and remorse upon committing aggressive acts, reflecting increased levels of internalizing problems (Moore et al., 2019).

**Indirect Effects Models**

**The Failure Model.** Patterson and Capaldi’s failure model (1990) proposes an indirect pathway from externalizing symptoms -including aggression- to internalizing symptoms, through psychosocial failures. The model posits that aggressive children encounter difficulties in accomplishing typical developmental tasks, often experiencing failure in a series of domains. The wear and tear of such nonsuccesses ultimately gives rise to internalizing symptoms (Masten et al., 2006).

Peer rejection is an important failure in the social domain, and thus a proposed mediator between aggression and internalizing symptoms (Patterson & Capaldi, 1990). Upon entering school, children spend a significant amount of time alongside peers (Coie & Cillessen, 2003). The degree to which children are accepted or rejected by the peer group becomes increasingly important. Peer rejection, or the extent to which a child is disliked by the peer group, can therefore be construed as a key social failure. The construct reflects the private sentiments of the peer group and is measured by asking children who they dislike or with whom they would not want to play with as either a friend or playmate (Bierman, 2004). Together, the model suggests that aggression is a socially inappropriate behavior that can lead to negative appraisals by the peer group.
Overtime, the negative appraisals contribute to dysphoria and internalizing symptoms (Morrow et al., 2006).

Although peer rejection is a major component of the causal mechanism posited by the failure model, Capaldi and Patterson (1990) suggest that it is far from the only one. The failure involves several other psychosocial failures that may link aggression to subsequent internalizing symptoms. Other relationships with parents, teachers, and siblings can play important roles in the development of internalizing symptoms. Further, according to the model, academic failures also play an independent indirect role. Aggressive children are thought to also encounter difficulty within academic settings (i.e., following instructions, abiding to classroom rules, etc.) which can reflect in poor academic performance, independently contributing to a rise in internalizing symptomology (Evans & Fite, 2019). The specificity of the association between early aggression and subsequent internalizing symptoms via psychosocial failures is also contested. While many theorists have homed in on the specific pathways existing between early aggression and subsequent internalizing symptoms occurs through peer rejection (Boutin et al., 2020; Panak & Garber, 1993), some have argued that the effects are more generalized. Functioning in one domain spreads out and may affect the course of functioning in other domains, resulting in a snowballing or cascading effect (Masten & Cicchetti, 2010). Behavioral problems (possibly arising from the family) carry into the school domain, reflecting in poor social and academic functioning, which in turn, contributes to internalizing problems and an exacerbation of antisocial tendencies. Thus, initial problems in one domain (i.e., aggression) spreads to a series of other domains, creating a chain reaction.
This dissertation, however, focuses specifically on the indirect effect of peer rejection, given its purported central theoretical role in the association between aggression and internalizing symptoms (Patterson & Capaldi, 1990).

**Indirect Effects Designs.** An influential interpretation of the failure model proposes that the path from aggression to internalizing symptoms is indirect. An indirect effect describes a process whereby the effect of one variable on another is mediated by a third variable. Indirect effects models (i.e., mediation) describe the extent to which a variable (i.e., a mediator) accounts for the association between a predictor and an outcome (Baron & Kenny, 1986). In complete mediation, the entirety of the effect from the predictor to the outcome occurs through the mediator, whereas in partial mediation, only part of the overall effect from the predictor to the outcome occurs through the mediator. Most mediation models in the social sciences are partially mediated, as it is unlikely for one variable to fully account for the variance shared between the predictor and the outcome (Baron & Kenny, 1986). Developmental psychologists recognize several forms of indirect effects designs, each with varying degrees of methodological strengths.

Cross-sectional mediation models explore mediation paths using variables collected at a single time point (Cerin & MacKinnon, 2009). The direct effect (i.e., effect from the predictor to the outcome) and the indirect effect (i.e., effect from the predictor to the outcome through the mediating variable) are the main paths of interest. Although traditionally, the established conditions for mediation were that (a) the predictor should be causally associated to the outcome, (b) the predictor should be causally associated to the mediator, and (c) the mediator should be causally associated to the outcome, authors have since indicated that (a) is not a requirement for a significant indirect effect (Hayes,
Cross-sectional mediation designs estimate these paths concurrently within a regression framework. Sequential designs are an alternative to cross-sectional models. The design abides by guidelines proposed by the MacArthur group, which stresses the importance of using distinct time points in mediation designs, as mediation is thought to be a process that should take time to unfold (Chmura Kraemer et al., 2008). Each component of the mediation is collected at a separate time point. Put succinctly, the predictor is measured prior to the mediator, which in turn, is measured prior to the outcome variable. Unlike true longitudinal designs, however, sequential models do not include autoregressive effects. Each measure is only included once in the analyses, each at a different time point. Thus, effects that each variable has on itself at later time points are not estimated. Despite their differences, neither cross-sectional nor sequential mediation designs accurately assess longitudinal mediation. Each provides biased estimates that can erroneously indicate the presence of an indirect effect.

Longitudinal models are preferred for mediation. Designs that employ two waves of data are termed half-longitudinal mediation models. In these models, either the predictor and the mediator are measured at two separate time points, or the mediator and the outcome are measured at separate two time points, but not both. Thus, associations between two of the three variables are concurrent (Maxwell & Cole, 2007). Autoregressive paths are included for both time points. However, because only two time points of data are included, either the effect of the predictor on the mediator must occur instantaneously or the effect of the mediator on the outcome must occur instantaneously to be able to measure the indirect effect of the predictor on the outcome. Because the purported effects are not instantaneous, this can bias the estimate of the mediated effect
(Fritz & MacKinnon, 2012). Full longitudinal mediation designs are the most effective way to test mediation. Designs that employ three (or more) waves of data are termed full longitudinal mediation models. Several strategies are available to test full longitudinal mediation, but the cross-lagged panel design is probably the most used approach (Jöreskog, 1979). In this approach, the three components of mediation (i.e., predictor, mediator, and outcome) are each measured at the same three time points. The mediation design assigns the predictor to Time 1, the mediator to Time 2, and the outcome to Time 3. Autoregressive paths for each variable are included in the model to measure change. Additional cross-lagged paths may be included in the model; constraints on all but mediation paths are not required (Cole & Maxwell, 2003). Similarly, path restrictions that imply constructs are only affected by constructs that are one lag removed (i.e., the previous time point), with the exception of the outcome, which is predicted by the predictor at one- and two lags removed (i.e., the first and second time point) are not strict.

Pathways from Aggression to Internalizing Symptoms: Central Research Findings

There is empirical evidence to substantiate both the direct effects and the indirect effects models. Each will be described below.

Direct Effects Models

Substantial evidence suggests that externalizing symptoms and internalizing symptoms are interrelated. Longitudinal effects from externalizing to internalizing symptoms are also well-established (Willner et al., 2016). A meta-analysis involving 263 studies with community samples suggested that child externalizing behavior precedes adult internalizing disorders (Loth et al., 2014). Research linking early-onset aggression, specifically, to subsequent internalizing symptoms is less robust. However, several
studies have found an association between the two. One study found that aggression problems among 3-year-olds predicted internalizing problems at age 10 (Mesman et al., 2001). Another found that among 4th graders, relational aggression was associated with longitudinal increases in internalizing symptoms (Murray-Close et al., 2007). Similar findings emerged in a clinical sample of boys, where oppositional defiant disorder predicted depressive symptoms and anxiety throughout childhood and adolescence (Burke et al., 2005).

Links between reactive aggression and internalizing symptoms have also been proposed (Card & Little, 2006). Concurrently, reactive aggression is associated with internalizing symptoms in childhood and early adolescence (Vitaro & Brendgen, 2011; Fite et al., 2012). Few longitudinal studies that have examined prospective associations between reactive aggression to internalizing symptoms. Those that have, focus on older childhood and adolescent samples. One study by Vitaro and colleagues (2002) found that reactive aggression in late childhood predicted increased levels of depressive symptoms in adolescence. Another study found that adolescent reactive aggression predicted increases in both depressive symptoms and anxiety, over the course of three years (Fite et al., 2014).

**Indirect Effects Models**

Several studies have explored indirect links from aggression to the development of internalizing symptoms. Many focus on peer rejection as an intermediary variable, consistent with the failure model. Three sets of findings will be reviewed. The first set of findings test the failure model with concurrent and longitudinal data. The second set of findings separately examine, with longitudinal data, each step in the failure model: (1)
from aggressive behavior to peer rejection (2) from peer rejection to internalizing symptoms.

**Indirect Effects between Aggressive Behavior and Internalizing Symptoms via Peer Rejection: The Full Failure Model.** Concurrent studies report findings consistent with the failure model, revealing an indirect effect from aggressive behavior to internalizing symptoms mediated by peer rejection. Two studies with the same sample of at-risk boys found associations between rejection and depressive symptoms, both examining students in the 4th grade (Patterson & Capaldi, 1990; Patterson & Stoolmiller, 1991). Similar results emerged from a community sample of school-aged children (Messer & Gross, 1994). One concurrent study focused specifically on the role of reactive aggression in the failure model. Peer rejection partially mediated the relation between reactive aggression (but not proactive aggression) and depression (Morrow et al., 2006).

Prospective studies testing the failure model have yielded inconclusive results. One study that followed late-elementary school children over the span of a year indicated that peer rejection partially mediated the association between aggression and depression (Panak & Garber, 1992). However, these findings have since been contested, as the reported indirect effect was minimal and the authors neglected to evaluate its statistical significance (Kiesner, 2002). A recent study also found that in community and clinical samples, kindergarten externalizing problems predicted internalizing symptoms two years later, mediated by 1st grade peer victimization (Boutin et al, 2020). Not all longitudinal tests of the failure model find evidence of mediation. Bornstein et al (2010) did not find
support for an indirect effect from age 4 externalizing symptoms to age 14 internalizing symptoms as mediated by age 10 social competence.

It is worth noting that most previous studies focus on externalizing symptoms as a predictor rather than aggression. Externalizing problems refer to a wide spectrum of behavioral symptoms describing disinhibited and externally focused behaviors (Willner et al., 2016). They are broadly characterized by three components: aggression, delinquency, and hyperactivity. Although aggression is one component of externalizing symptoms, it is far from the only one. Thus, aggression, and particularly reactive aggression, differs in important ways from externalizing difficulties. Reactive aggression refers specifically to intentionally harmful behaviors carried out in response to a perceived or real threat. Some have suggested that reactive aggression is the most theoretically appropriate predictor of internalizing symptoms (Evans & Fite, 2019). To date, only one prospective study has tested the failure model using reactive aggression as a predictor. In this study of primary school students, direct links emerged from grade 1 reactive aggression and grade 3 peer rejection, but not from grade 3 peer rejection to grade 5 depressive symptoms; further, there was no indirect effect from grade 1 reactive aggression to grade 5 depressive symptoms (Evans & Fite, 2019).

**Longitudinal Associations between Aggression and Peer Rejection: Step One of the Failure Model.** Few longitudinal studies test the failure model. Multiple studies, however, have separately examined each direct step of the failure model. Although these studies do not directly test indirect pathways, they can be informative because they assess whether effects exist from the predictor to the mediator, and from the mediator to the outcome, both of which are necessary to establish mediation (Fritz & MacKinnon, 2012).
The first step of the model posits a link from early aggressive behavior to peer rejection. Several studies report longitudinal associations from aggression to peer rejection. As early as preschool, patterns of aggressive behavior have been linked to later peer difficulties (Coie et al., 1990; Burt & Roisman, 2010). Although most studies have focused exclusively on boys (e.g., Coie et al., 1989; Pope & Bierman, 1999), similar patterns have emerged among girls (Little & Garber, 1995). Reactive aggression, in particular, has strong ties to subsequent peer rejection (Fite et al., 2007; Morrow et al., 2006).

The failure model posits that early aggression predicts increased peer rejection. Some have suggested, however, that the association is bidirectional (Vitaro et al., 2018). Children may aggress because they are rejected by peers, and they may be disliked because they aggress (Bierman et al., 1993). There is evidence for both. Among high-risk children, Miller-Johnson and colleagues (2002) found that conduct problems predicted longitudinal increases in peer rejection, and that peer rejection predicted increases in conduct problems. Similar findings are reported in community samples. Among early elementary school children, early externalizing problems predicted peer rejection across time and peer rejection predicted subsequent externalizing problems (Sturaro et al., 2011). These studies all examined general aggression. No studies have focused specifically on the bidirectional relations between reactive aggression and peer rejection (Hubbard et al., 2010).

**Longitudinal Associations between Peer Rejection and Internalizing Problems: Step Two of the Failure Model.** The second step of the failure model describes a link between peer rejection and subsequent internalizing symptoms. Here too,
longitudinal evidence supports the hypothesized association. Peer difficulties have been tied to a range of internalizing problems, including depressive symptoms (Boivin et al., 1994; Brendgen et al., 2009) and anxiety (Ginsberg et al., 1998). Although most prospective studies involve adolescent samples (e.g., Nolan et al., 2003; Prinstein & Aikins, 2004), those that include younger children yield similar findings (Gooren et al., 2011; van Lier & Koot, 2010). Among children aged 7-12 years, peer rejection predicted increases in teacher-rated internalizing symptoms over a four-year span (DeRosier et al., 1994). In another study, longitudinal associations from peer rejection to internalizing symptoms, increased with age from early childhood to adolescence (Ladd, 2006).

While the failure model posits a longitudinal link from rejection to internalizing symptoms, a dynamic, bidirectional interplay between the two constructs is also possible. Peer rejection can contribute to a rise in internalizing symptoms, which in turn may trigger even greater rejection (Sameroff & MacKenzie, 2003). Empirical studies testing longitudinal bidirectional effects are scarce and have yielded inconsistent findings. A study of adolescents found a bidirectional link between peer difficulties and anxiety across gender, but bidirectional links between peer difficulties and depressive symptoms emerged for boys only (Lester et al., 2012). Another study of children and adolescents found that internalizing problems were associated with later peer rejection, but not the reverse (Agoston & Rudolph, 2013).

The Role of Genetic and Environmental Factors in the Failure Model

The failure model proposes a causal pathway from aggression to internalizing symptoms via peer rejection. However, there is growing evidence suggesting that genetic factors, alongside environmental ones, are crucial to shaping children’s development
(Rutter et al., 2006). In addition, genetic and environmental influences are often interdependent. Although no study has yet examined the relative influence of genetic and environmental factors on the failure model, there is reason to suspect that in addition to environmental influences, genetic influences also account for the direct and indirect effects that are posited by the model.

Separate studies show the extent to which a specific variable has genetic and environmental influences. Classical twin research designs, partition behavioral traits into three main sources of variance: genetic factors, shared environmental factors, and nonshared environmental factors. Genetic factors describe heritable influences. Shared environmental factors describe environmental influences that are common to cohabitating members of the same family. These include family-related characteristics (e.g., socioeconomic status, household structure) and characteristics outside the family (e.g., neighborhood, school-related characteristics). Nonshared environmental factors describe environmental influences that are unique and not shared between members of the same family – whether within or outside the family (e.g., differential parental treatment, peer relationships, friendships; Brendgen et al., 2012). The latter also capture measurement error.

Although few studies examine the etiology of reactive aggression, those that do, consistently find that genetic factors are an importance source of variance. This finding is in line with research that suggests that reactive aggression has specific links to temperament (e.g., emotional negativity, irritability, low threshold for aggression; Vitaro et al., 2002, 2006), which are themselves heritable (Cyphers et al., 1990). However, there are mixed findings regarding the relative importance of the shared and nonshared
environment. A study of 6-year-old children found that teacher-reported reactive aggression was influenced by genetic and nonshared environmental factors, with no contribution from the shared environment. Baker and colleagues (2008), in turn, found that among 9 and 10-year-old twins, reactive aggression was influenced by genetic, nonshared, and shared environmental factors. Similarly, Tuvblad and colleagues (2009) found that genetic, shared, and nonshared environmental influences contributed to reactive aggression across middle to late childhood, with diminishing influence of the shared environment as children aged. Both shared- and non-shared environmental factors may play a role in reactive aggression. Harsh and hostile parenting is predictive of reactive aggression. Individuals who endure early physical abuse and are exposed to punitive home environments are at risk for developing hypervigilance to anger and a decreased threshold for frustration and angry outbursts (Dodge et al., 1997). Thus, it is possible that the shared environment plays an important role in reactive aggression. Similarly, the nonshared environment may be key. Children’s peer interactions are crucial to children’s development. Experiencing maltreatment or harassment from the peer group may place a child at an elevated risk for aggressive behavior (Brenden et al., 2006).

Although research examining the etiological factors of peer rejection is limited, there is also evidence suggesting both genetic and environmental factors contribute to differences in peer rejection and peer difficulties, more broadly. One study examining Kindergarteners found that most of the variance in peer rejection was explained by genetic and non-shared environmental factors. The shared environment played a small (albeit significant) role in explaining peer rejection (Brendgen et al., 2009). Studies
examining peer difficulties, and peer victimization have similar findings. Boivin and colleagues (2013) found that genetic factors accounted for most of the variance in peer difficulties, while the rest of the variance was attributed to nonshared environmental factors. Although genes cannot code for the reactions of others, genetic factors can influence peer rejection through a mediated environmental mechanism (Burt & Donnellan, 2015). Peer rejection may be evoked through a series of heritable behavioral traits and tendencies (e.g., disruptiveness, antagonism; e.g., Dodge, 1983). Temperament may also play a role. Heritable temperamental characteristics like irritability, impulsivity and emotional reactivity are known to predict interpersonal difficulties (Newman et al., 1997). Shared environmental influences may include maladaptive parenting behaviors. The way in which parents relate to their children may affect children’s peer interactions (Dickson et al., 2019). Children who experience peer difficulties are more likely to experience punitive, overprotective, and abusive parenting (Dodge et al., 1997).

Nonshared environmental influences may include the social context at school. The availability of friends, social relationships with teachers, children own attitudes and beliefs about peer maltreatment have all been proposed to influence interpersonal difficulties (Brendgen et al., 2008).

Finally, genetic studies examining the etiology of internalizing symptoms also suggest genetic and environmental underpinnings. In a large study, genetic and nonshared environmental components emerged as significant contributors to differences in internalizing symptoms, across development (from ages 8-20 years), with the shared environment playing no role (Patterson et al., 2018). Similar findings were reported by Brendgen and colleagues (2009) who found that genetic and non-shared environmental
factors were the only significant contributors to children’s depressive symptoms. However, one study examining 12-year-olds, found that genetic, shared, and non-shared environmental factors all significantly contributed to the variance in internalizing symptoms. Genetic factors that contribute to internalizing symptoms may involve temperamental characteristics such as emotional reactivity and difficulties with effortful control (Crawford et al., 2010). High levels of reactivity encompass feelings like anger, sadness, tension, nervousness, and rejection (Watson & Clark, 1984). Effortful control is reflected in decreased self-regulation and impulsivity. Influences from the shared may include factors attributed to the family environment like parental conflict, marital discord, which all been linked to child internalizing symptoms (Lucia & Breslau, 2006). Nonshared environmental factors such as children’s attitudes, friendships, and peer interactions have also been implicated (Crosnoe & Elder, 2002; Prinstein et al., 2005).

Taken together, the evidence clearly suggests that genetic factors (in addition to environmental ones) make important contributions to the separate components of the failure model.

**Possible Mechanisms of Genetic Influence in the Failure Model**

Although studies examining the extent to which genes and the environment contribute to separate study variable are important, they do not inform on the extent to which genetic influences are shared between variables (Brendgen et al., 2012). However, there is evidence suggesting that many behavioral traits share some of the same underlying genetic factors (Sullivan et al., 2015). This finding is likely to extend to the failure model. Common genetic influences that are shared amongst the different components of the failure model likely exist and can be problematic because they muddle
conclusions about the causal paths that the model posits. The extent to which genetic influences on different traits are correlated is termed ‘pleiotropy’ (Trzaskowski et al., 2013), with high correlations indicating that the same genes affect the phenotypes. There are several mechanisms that can explain the overlap in genetic factors across distinct traits. Although the concept has been mostly applied to biological disorders and explained in a molecular genetic context, its broader implications are applicable to the current study. Biological pleiotropy occurs when a specific genetic factor directly affects multiple phenotypes (e.g., genetic mutations for the genetic disorder phenylketonuria (PKU) directly affect intellectual disability, pigmentation, and metabolism; Donovan & Owen, 2016). Mediated pleiotropy occurs when a genetic factor influences two traits. However, unlike biological pleiotropy, genetic influences on one trait are direct, while the influences on the other one are indirect. Specific genetic factors directly affect a trait, which in turn, affect another trait (e.g., a genetic variation causes nicotine dependence, which in turn, leads to lung cancer; Gage et al., 2016). It is noteworthy that mediated pleiotropy can involve gene-environment interplay. Specifically, environmentally-mediated pleiotropy or gene-environment-trait correlation (rGET) is an often-overlooked form of mediated pleiotropy that suggests a genetically-influenced trait predisposes an individual to experiencing a particular environment, which in turn, affects a second trait. The mechanism builds upon evidence suggesting environmental experiences can also be partially heritable, through a process termed gene-environment correlation (rGE). An rGE occurs when exposure to an environment is non-random and directed by underlying genetic factors. Scarr and McCartney (1983) describe three main processes whereby an rGE can arise. A passive rGE occurs when biological parents construct a child’s
environment based on the parents’ own genetically influenced characteristics. Because children share genes with parents, a child’s genotype is correlated with the environment. An evocative rGE occurs when a child’s genetically linked characteristics (i.e., characteristics with genetic underpinnings) elicit specific reactions from the environment. Selective rGE arises when a child actively chooses environments based on their genetically linked characteristics (Brendgen et al., 2018a). Of course, pleiotropy can also arise spuriously when misclassification or biases falsely inflate genetic overlap in different traits. Genetic influences explaining the direct and indirect paths of the failure model are described below. Developmental considerations are also detailed.

Genetic Influences on the Direct Effect from Reactive Aggression to Internalizing Symptoms. Although genetic links between aggression (or reactive aggression, more specifically) and internalizing symptoms has not yet been examined, there are studies that suggest that, at least concurrently, externalizing and internalizing symptoms are genetically related (e.g., Pesenti-Gritti et al., 2008). One study examining 5-year old children found that the association between externalizing and internalizing symptoms could be best explained by common genetic vulnerabilities (Jaffee et al., 2002). A study examining the nature of the association between antisocial behaviors and depressive symptoms yielded similar findings, with 45% of the observed association accounted for by common genetic factors. Given that no longitudinal studies have examined the genetic overlap between the two constructs, the directionality of the results remains unclear. However, it is likely that genetic factors explain both aggression and internalizing symptoms via biological pleiotropy. Both externalizing and internalizing symptoms may be directly predicted by the same underlying factors.
Genetic Influences on the Direct Effect from Reactive Aggression to Peer Rejection. Heritable factors associated with aggressive behavior may contribute to the risk of peer rejection. Although there are no studies that examine genetic overlap in aggression and peer rejection, research on externalizing problems (e.g., aggressive behavior, disruptive behaviors) and peer difficulties suggests that the link between the two may be partially due to \( r \text{GE} \). Similar findings emerge from studies examining associations between aggressive behavior and victimization (Ball et al., 2008; Brendgen et al., 2011). One prospective study examined the genetic underpinnings of the longitudinal association between early disruptive problems and peer difficulties in school-aged children. The findings indicate that (a) genetic factors explained a sizeable portion of the variance in disruptive behaviors and peer difficulties, and (b) observed longitudinal associations from disruptive behaviors to peer difficulties were largely a product of \( r \text{GE} \) (Boivin et al., 2013a). The \( r \text{GE} \) overlap between externalizing problems and peer difficulties is probably a product of evocative \( r \text{GE} \); it is unlikely that children would actively choose to be rejected or victimized (Brendgen et al., 2018a). Thus, the findings suggest that children who are genetically predisposed to disruptiveness, evoke problems with peers during the early school years (Boivin et al., 2013a). Previous research has focused on verbal aggression (Ball et al., 2008). However, it is unclear if a similar \( r \text{GE} \) process explains the link between reactive aggression and peer rejection (Brendgen et al., 2018a).

Genetic Influences on the Direct Effect from Peer Rejection to Internalizing Symptoms. The direct effect from peer rejection to internalizing symptoms may be partially attributed to common genetic influences. Empirical research examining genetic
links between aggression and internalizing symptoms remains scarce. To date, there are no longitudinal studies examining the association. However, concurrent studies have examined the genetic overlap between peer difficulties (peer rejection, victimization) and internalizing symptoms (depressive symptoms, anxiety). One study found that in early childhood, a significant portion of the heritable factors influencing rejection were also associated with depressive symptoms (Brendgen et al., 2009). Another study found a genetic link between adolescent victimization and anxiety problems (Brendgen et al., 2015). Because of the cross-sectional nature of both studies, however, directionality could not be determined. The findings may suggest that children with genetic predispositions to depression and anxiety are more likely to experience maltreatment and dislike from the peer group, indicating an evocative rGE (Brendgen et al., 2006, 2009). However, this would imply that depressive symptoms/anxiety precede the development of internalizing symptoms (Boivin et al., 2013a). The findings could also be explained by pleiotropy; a common genetic factor may evoke peer difficulties and internalizing symptoms. A retrospective study examining the association between adolescent victimization and adult depressive symptoms, suggested this may be the case, as it found that nearly 40% of the association between initial victimization and subsequent depression could be explained by shared genetic effects (Kretschmer et al., 2018).

Unaccounted genetic factors (e.g., behavioral, language, physical characteristics) that evoke peer difficulties may exacerbate children’s internalizing symptoms over time.

**Genetic Influences on the Indirect Effect of Reactive Aggression and Internalizing Symptoms via Peer Rejection.** Longitudinal studies that examine the genetic underpinnings of the direct paths proposed by the failure model are scarce. Those
examining genetic influences on the indirect path posited by the model (i.e., mediating
effect of peer rejection on the link between reactive aggression and internalizing
symptoms) are virtually non-existent. However, evidence suggests externalizing and
internalizing symptoms are genetically linked (e.g., Pesenti-Gritti et al., 2008). The
association likely extends to reactive aggression and internalizing symptoms, which may
also share a genetic link. Peer rejection may act as a mediator of a genetic link between
reactive aggression and internalizing symptoms. Recent work by Avinun (2020) proposes
that an environment can act as a mediator for genetic correlations between two distinct
phenotypes, in a process termed environmentally-mediated pleiotropy or gene-
environment trait correlations ($r$GET). The mechanism stems from the concept of a gene-
environment correlation.

Peer rejection has been found to be genetically influenced (Brendgen et al., 2009).
That is, genetically influenced traits can affect the degree to which a child experiences
rejected by the peer group. Reactive aggression, which itself is heritable (Tuvblad et al.,
2009), is associated to peer rejection. The association between reactive aggression and
peer rejection may occur partially as a result of an evocative $r$GE (Boivin et al., 2013a),
with reactively aggressive children evoking dislike from the peer group. Peer rejection, in
turn, is also associated with internalizing symptoms, both of which are genetically linked
(Brendgen et al., 2009). Together, these findings indicate that the association between
initial reactive aggression and subsequent internalizing symptoms is genetically mediated
by peer rejection.

_Developmental Considerations in Genetic Influence_
Although children are born with all their genes, heritability tends to increase as children get older (Plomin, 1986). The trend has been identified in domains that encompass aggression (Miles & Carey, 1997), peer difficulties (St. Pourcain et al., 2014) and internalizing problems (Bergen et al., 2007; Scourfield et al., 2003). Therefore, identifying genetic contributions at a single time point does not provide a full picture of genotypic expression. Two possible explanations for age-dependent increases in heritability have been advanced: innovation and amplification (Plomin, 1986). Heritability increases as a result of innovation are due to novel genetic influences that arise as children develop. New biological (e.g., puberty) and environmental (e.g., moving from home) occurrences may activate new genes. The newly activated genes work with previously activated ones and together increase genetic influences on a trait (Briley & Tucker-Drob, 2013). In contrast, heritability increases as a result of amplification occur when early genetic influences do not newly arise with age, but instead become more important as children get older. The mechanisms behind amplification is related to rGE. As children age, their transactions with the environment become more commonplace. Children select, shape, and evoke environments that complement their genetic tendencies. In doing so, genetic differences are magnified (Briley & Tucker-Drob, 2013).

The Present Study

The present study aims to disentangle environmental contributions from those that are genetic. The discordant MZ-twin difference method is a novel tool used to isolating the effects of the nonshared environment by controlling for the influence of genetic and shared environmental factors in associations between behavioral characteristics and social outcomes (Vitaro et al., 2009). Monozygotic (MZ) twins share 100% of their
genetic makeup. When reared together, they also share many environmental circumstances (e.g., certain family experiences, school, community, socio-economic status). The MZ twin difference method isolates influence specific to nonshared aspects of the social environment (e.g., peer relationships, teacher-child relationships) from genetic and nonshared environmental contributions.

The basis of the MZ twin method is straightforward: Within-pair differences (i.e., twin 1 score – twin 2 score) are calculated for each variable of interest. The difference between the twins on a variable represents a score that is free of genetic and shared environmental influences.

A necessary precondition to the use of the MZ twin difference method is that genetic factors explain some of the variance in the variables of interest. When each separate variable has a significant genetic component, it justifies the need for controlling for its genetic influences. ACE decomposition analysis is the preferred strategy for identifying genetic contributions to a variable (Neale & Cardon, 1992). ACE models partition the variance of a trait into three components: additive genetic effects (A; heritable factors), shared environmental effects (C; environmental factors shared by members of the same family), and non-shared environmental effects (E; environmental factors distinct to members of the same family). The latter also capture measurement error (Brendgen et al., 2012).

A significant genetic component indicates that a study variable is influenced by genetic factors, a necessary precondition to using MZ twin difference model (Vitaro et al., 2009). ACE modeling assumes that genetic and environmental effects are independent (Brendgen et al., 2012), thereby ignoring possible gene-environment
interplay. To identify common genetic influences, an additional set of genetic modeling is required. Bivariate Cholesky models identify genetic factors that are shared across two variables.

To illustrate, Brendgen and colleagues (2009) used a series of univariate ACE models to partition the variance of rejection and depressive symptoms into respective genetic, shared, and non-shared components. The results indicated that 30% of the variance in rejection was explained by genetic factors, 15% was explained by shared environmental factors, and a remaining 55% was explained by nonshared environmental influences. For depressive symptoms, 30% of the variance was explained by additive genetic factors, 3% was explained by shared environmental sources, and 67% was explained by environmental influences. Then, using the Bivariate Cholesky framework, rejection and acceptance were partitioned into (1) common latent factors influencing both variables, and (2) unique latent factors influencing only depressive symptoms. The results indicated that part of the association between depressive symptoms and peer rejection could be attributed to evocative rGE.

**Study Overview**

The failure model, proposed by Patterson and Capaldi (1990) is an influential conceptual model used to explain links between externalizing and internalizing symptoms (Evans & Fite, 2019). However, despite its influence, empirical support for the failure model remains inconclusive (Gooren et al., 2011; Kiesner, 2002). In addition, although the failure model examines associations between variables known to be genetically influenced (e.g., Boivin et al., 2011; Brendgen et al., 2009), the model has not been tested in a genetically controlled design to isolate the effects of the nonshared environment.
This is the first study to assess longitudinal associations between reactive aggression and internalizing symptoms, as mediated by peer rejection, independent of genetic and family-wide (i.e., shared) environmental effects.

**Study Objectives**

The current study had three primary objectives. The first objective was to determine the genetic contributions to each study variable of the failure model (i.e., reactive aggression, rejection, and internalizing symptoms), as well as the genetic influences that are shared between the study variables during the kindergarten and grade school years, both necessary preconditions to genetically controlled research. The second objective was to determine (a) the direct longitudinal effects from reactive aggression to internalizing symptoms, and (b) the direct longitudinal effects in each of the separate steps in the failure model (i.e., from reactive aggression to peer rejection and from peer rejection to internalizing symptoms). Although these paths were of main interest, all bidirectional longitudinal associations were examined. In other words, longitudinal paths in the reverse direction were also tested (i.e., from internalizing symptoms to reactive aggression, from peer rejection to reactive aggression, and from internalizing symptoms to peer rejection). Results from conventional analyses were compared with those from genetically controlled analyses to identify the degree to which genetic and shared environmental factors are responsible for previous findings that suggest longitudinal links from early childhood aggression to increases in internalizing difficulties. The third objective was to test the failure model, which posits an indirect longitudinal association from early childhood reactive aggression to changes in internalizing symptoms, as mediated by peer rejection. Here too, results from conventional analyses were compared
to those obtained from genetically controlled analyses, isolating effects to nonshared environmental factors (e.g., peer experiences), and removing confounds arising from common genetic factors and gene-environment interplay (e.g., rGE, rGET).

The first objective addressed the question: \textit{To what extent do genetic influences contribute to reactive aggression, peer rejection, and internalizing symptoms and to what extent are the same genetic influences shared between the study variables?} Identifying genetic contributions to each study variable and determining that to some extent these genetic contributions are common to the different study variables are necessary preconditions to employing the MZ twin difference method testing the failure model. To this end, I conducted two sets of analyses to determine the degree to which study variables, separately and jointly, were influenced by genetic, shared environmental, and non-shared environmental factors. First, a series of univariate ACE models were used to decompose each study variable into genetic and environmental components. Given past findings suggesting reactive aggression, peer rejection, and internalizing symptoms each have a significant genetic component (Boivin et al., 2013a; Brendgen et al., 2006, 2009), I predicted that the present study would find that a significant portion of the variance for each study variable was explained by genetic factors. Second, Bivariate Cholesky models were used to identify common underlying genetic factors that influenced paired associations between reactive aggression and rejection, internalizing symptoms and rejection, and reactive aggression and internalizing symptoms. Prior studies indicate that a portion of genetic factors are shared between aggression, peer difficulties, and internalizing symptoms (e.g., Boivin et al., 2013a; Brendgen et al., 2009, 2015). Thus, I predicted that, in line with past findings, some of the same genetic factors would be
shared between a) reactive aggression and internalizing symptoms, b) reactive aggression and peer rejection, and c) peer rejection and internalizing symptoms.

The second objective addressed two sets of questions: (1) *Are there longitudinal associations between the different components of the failure model?* and (2) *Do these associations remain after controlling for genetic (and shared environmental) influences when using a genetically controlled design?* To this end, longitudinal associations were identified between reactive aggression and internalizing symptoms, between reactive aggression and peer rejection, and between peer rejection and internalizing symptoms. Bidirectional paths were also included in the model, given findings that suggest reciprocal associations between behavioral adjustment and peer rejection (Miller-Johnson, 2002; Ladd, 2006). To remove the potential contribution of common genetic factors (including gene-environment interplay; e.g., *rGE*) and shared environment factors, the analyses were repeated using the MZ twin difference method. Prior findings that are non-genetically controlled, provide evidence for the direct longitudinal associations that form part of the failure model (e.g., Burt & Roisman 2010; Fite et al., 2014; Gooren et al., 2011). However, genetic models suggest that these associations may be under partial genetic influence (Boivin et al., 2013a; Brendgen, 2006, 2009). As such, I predicted that longitudinal associations would be substantial when genetic factors were not controlled for. However, the magnitude of the associations would be reduced when controlling for genetic and shared environmental influences.

The third objective addressed two questions: (1) *Does peer rejection mediate the association between reactive aggression and internalizing symptoms?* and (2) *Does the association remain after controlling for possible common genetic (and shared*
environmental factors). To this end, a full longitudinal mediation model (Fritz and MacKinnon (2007) was estimated to test the failure model. To remove the potential contribution common genetic factors (including gene-environment interplay; rGET) and shared environmental factors, the analyses were repeated using the MZ twin difference method. Although few non-genetically controlled studies have tested empirically tested the failure model in a longitudinal framework, those examining direct paths associated with the failure model provide initial evidence for an indirect effect (e.g., Gooren et al., 2011; van Lier & Koot., 2010). Further, recent prospective studies have found a significant mediation effect (e.g., Boutin, 2020). Given these findings, I predicted that peer rejection would mediate the association between reactive aggression and internalizing symptoms in non-genetically controlled analyses. However, given findings suggesting that some of the paths in the failure model may be a product of common genetic influences (e.g., Boivin et al., 2013a; Brendgen et al., 2006, 2009), I predicted that the magnitude of the indirect effect would be reduced when using the MZ twin difference design.

Bayesian estimation procedures were used in analyses that addressed the second and third objectives. Bayesian estimation differs from traditional approaches mainly in that Bayesian estimation assumes that each parameter is a variable that can be described using a probability distribution (Van de Schoot et al., 2015), unlike traditional (i.e., frequentist) perspectives, which treat unknown parameters of interest as a fixed - albeit-unknown value. Bayesian estimation provides an advantage to traditional, maximum-likelihood (ML) approaches when using small samples and non-normally distributed data (Van de Schoot et al, 2015). Maximum-likelihood (ML) estimation requires large sample
sizes and asymptomatic normality to yield unbiased estimates. In contrast, Bayesian estimation does not require these assumptions be followed to produce accurate results. Instead, Bayesian approaches use sampling-based methods (e.g., Markov chain Monte Carlo (MCMC). Thus, the quality of inference is not determined by the sample size approaching infinity but instead by number of \textit{samples taken} approaching infinity (McNeish, 2016).
METHOD

Participants

Participants included 682 twin pairs (237 male twin pairs, 235 female twin pairs, and 210 mixed-sex twin pairs) from the Quebec Newborn Twin Study (see Boivin, et al., 2013a), an ongoing longitudinal study of a population sample of twins born between 1995 and 1998 in the Greater Montreal area (Boivin et al., 2013a). Data were collected when children were in Kindergarten ($M=6.04$ years, $SD=0.27$), Grade 1 ($M=7.07$ years, $SD = 0.27$), and Grade 4 ($M=10.00$ years, $SD= 0.28$). A total of 257 participating twin-pairs were monozygotic (128 male twin pairs, 129 female twin pairs) and 425 were dizygotic (109 male twin pairs, 106 female twin pairs, 210 other sex twin pairs). Zygosity was assessed via analyses of 8-10 highly polymorphous genetic markers. Monozygotic twins were designated as such when concordant for every genetic marker. If there was insufficient or unavailable genetic material due to parental refusal (43% of cases), zygosity was determined through physical resemblance questionnaires at 18 months and again at 9 years (Goldsmith, 1991; Spitz et al., 1996).

Demographic characteristics of the twin families when children were 5 months of age did not differ from those of a sample of singletons representative of large urban centers in the province of Quebec. Of the families participating, 87.9% were Caucasian, 4.2% were of African descent, 2.2% were of Asian descent, and 0.3% were Native North Americans. Most children lived in a two-parent home (92.1%). Participants’ birthweight was, on average, 2.47 kg ($SD= 0.54$). Most households (69.8%) had an income of 30,000
Canadian dollars or higher at the outset and a majority of twins’ mothers had completed high school (82.4%).

**Procedure**

Data collection was approved by the Institutional Review Boards of the University of Quebec in Montreal and the St.-Justine Hospital Research Center. Instruments were administered in French or English, depending on the language spoken at home. Trained research assistants used back-translation procedures to ensure semantic similarity. Children had different teachers at each time point. Most of their classmates were also different. In Kindergarten, 74.4% of twins attended separate classrooms, compared to 69.5% in 1st grade, and 59.7% in 4th grade. For MZ twin pairs, the percent attending separate classrooms was 70% in kindergarten, 73% in 1st grade, and 67% in 4th grade.

**Measures**

All scores were standardized by sex and zygosity in order to control for mean level differences (see Arseneault et al., 2003; Van den Oord et al., 2000).

**Peer Nominations**

In Kindergarten, Grade 1 and Grade 4, participants completed a standard peer nomination inventory. Children were given a roster with all the students in the classroom. Peer rejection was assessed by asking children to select three classmates whom they “least liked to play with” (see Vitaro et al., 2012). The number of received nominations was summed, then z standardized within classroom (Coie et al., 1982).
In Kindergarten, Grade 1, and Grade 4, teachers completed a 7-item measure of aggression (Coie & Dodge, 1987), rating participants on a 3-point scale, ranging from 0 (never) to 2 (often). *Reactive aggression* included 4-items (Mean α=.88) describing the use of hot-blooded or defensive aggression in response to actual or perceived provocations (See Appendix A). *Proactive aggression* included 3-items (Mean α=.86) describing the instrumental use of aggression (See Appendix B).

**Internalizing Symptoms**

In Kindergarten, Grade 1, and Grade 4, teachers completed a 4-item assessment of depressive symptoms (see Appendix C) from the *Emotional Disorder Scale of the Ontario Child Health Study* (Offord et al., 1989), and a 3-item assessment of anxiety symptoms (See Appendix D) from the *Social Behavior Questionnaire* (SBQ; Tremblay et al., 1991). Items were rated on a scale ranging from 0 (does not apply) to 2 (applies often). Internal reliability was acceptable for depressive symptoms (Mean α =.81) and anxiety (Mean α =.76).

**Potential Confounding Variables**

*Birthweight* was obtained from birth records. Birthweight was included as a control variable in the analyses, because children with lower birthweight tend to display higher levels of behavioral disturbances (Hines et al., 2020). Maternal education and household income were also included as control variables, because each assays socioeconomic status, which has been linked to socioemotional adjustment (Achenbach, 1990)

**Plan of Analysis**
The first set of analyses identified sources of variance for each of the study variables. Separate univariate ACE models, conducted for reactive aggression, peer rejection, and internalizing symptoms, examined the relative contribution of genetic, shared, and nonshared environmental factors to each variable. Bivariate Cholesky ACE models examined common underlying genetic, shared, and nonshared environmental components to the phenotypic associations between (1) reactive aggression and internalizing symptoms (2) reactive aggression and peer rejection, and (3) peer rejection and internalizing symptoms. The latter determine the degree to which associations between the study variables is a result of shared genetic factors, which, if present, point toward the need for genetically controlled statistical analyses. In other words, ACE analyses that partition sources of variance are useful prerequisites to research designed to examine peer influence processes, because they can signal the need for research designs that isolate nonshared environmental processes.

The second set of analyses examined reciprocal longitudinal associations between study variables. Two sets of path models examined the direct longitudinal associations between (1) reactive aggression and internalizing symptoms, (2) reactive aggression and peer rejection, and (3) peer rejection and internalizing symptoms. Each was conducted with a traditional and a genetically controlled design. Analyses with a traditional design (i.e., singleton analyses) were conducted on one randomly selected twin member from each monozygotic (MZ) twin pair. These analyses mirrored conventional longitudinal analyses that explore the origins of internalizing symptoms, in that they make no account of genetic or shared family contributions (e.g., Gooren et al., 2011, van Lier & Kloot, 2010). The second set of analyses employed a genetically controlled design. In these
analyses, within-pair twin difference scores were calculated for each variable, in order to
the effects of genes (including rGE), shared environmental contributions (Vitaro et al.,
2009). In so doing, the results capture only non-shared environmental experiences.

The third set of analyses examined the role of peer rejection as a mediator. Full
longitudinal mediation models examined whether peer rejection mediated the
longitudinal association between initial levels of reactive aggression and changes in
internalizing symptoms. The analyses were conducted twice, with a traditional and a
genetically controlled design. Traditional analyses utilize one randomly selected member
of each twin pair, in a test of the failure model (Patterson & Capaldi, 1990). The second
set of analyses tests the failure model in a genetically controlled framework, to determine
if previously reported results are a product of genes, family-wide contributions, and rGE.

**Objective 1: Identifying Sources of Variance**

Sources of variance were identified for each of the study variables, and for the
bivariate associations between reactive aggression, peer rejection, and internalizing
symptoms. Decomposing the variance components for the study variables illustrates the
relative contribution of genetics and the environment (both shared, and unique). In doing
so, it is possible to determine the extent to which genetic influences, shared-, and non-
shared environmental factors explain the variance in peer rejection, reactive aggression,
and internalizing symptoms and their associations. Two sets of analyses were conducted.
The first set described the proportion of variance accounted for by genetic and
environmental factors in each study variable. The second set described the degree to
which phenotypic associations between peer rejection and reactive aggression, peer
rejection and internalizing symptoms, and reactive aggression and internalizing
symptoms are explained by common underlying factors. All analyses were conducted using Mplus 8.2 (Muthén & Muthén, 1998-2018).

Standard fit indices assessed model fit. The root mean square error of approximation (RMSEA) should be equal or less than .06; and the comparative fit index (CFI) should exceed .95 (Kline, 2005).

**Univariate ACE models.** Univariate ACE models determined the relative contribution of genetic and environmental factors for reactive aggression, peer rejection, and internalizing symptoms (see Figure 1 for measurement model). Separate analyses were conducted at each time point (Kindergarten, Grade 1, Grade 4), resulting in a total of 6 models. The analyses were conducted according to conventions established by Neale and Cardon (1992).

By comparing within-pair correlations for MZ twins (who share 100% of their genetic makeup) with same-sex DZ twins (who, on average, share 50% of their genetic makeup), sources of variability in each variable (phenotype) can be estimated as latent additive genetic (A), latent shared environmental (C), and latent nonshared environmental factors (E). Within-twin pair correlations of the latent genetic factors (A) were fixed to 1.0 for MZ twins and 0.5 for DZ twins. Within-twin pair correlations of the latent nonshared environmental factors were fixed to 0 for MZ and DZ twins. The coefficients a, c, and e were fixed to be equal across the two twins in a pair and across MZ and DZ twins. Each coefficient represents factor loadings that provide information about the relative contribution of the latent factors A, C, and E to the total variance of each phenotype. That is, the variance of each phenotype is broken down in three parts: a provides information regarding the relative contribution of genetic factors (A), c provides
information regarding the relative contribution of shared environmental factors (C), and $e$ provides information regarding the relative contribution of non-shared environmental factors (E). Measurement error is included in the E variance component.

Latent factors that do not reach statistical significance are removed from a model if model fit improves or parsimony improves without sacrificing model fit. Univariate models were chosen on the basis of parsimony and model fit. Chi-square difference ($\Delta \chi^2$) tests are used for model comparison and selection, when a model (e.g., AE model) is nested within another (e.g., ACE model). A statistically significant $\chi^2$ difference indicates that the fit of the nested model is poorer than that of the full model and thus the full model must be retained. Otherwise, the nested model is considered to have better fit in terms of parsimony, given its fewer parameters (Kline, 2015).

**Bivariate Cholesky models.** Bivariate Cholesky ACE models (Neale & Cardon, 1992) were specified to identify common factors underlying associations between (a) reactive aggression and internalizing symptoms, (b) reactive aggression and peer rejection, and (c) peer rejection and internalizing problems. Figure 2 presents a measurement model. Separate models were conducted for Kindergarten, Grade 1, and Grade 4, resulting in a total of nine models.

Each bivariate Cholesky model included two components: (1) common latent factors, $A_C, C_C,$ and $E_C,$ that influence the two phenotypes included in the model (i.e., reactive aggression and internalizing; reactive aggression and peer rejection; internalizing symptoms and peer rejection) and (2) unique latent factors $A_U, C_U,$ and $E_U$ specific to peer rejection (Figures 5-7, Figures 8-10) or internalizing symptoms (Figures 11-13).
In bivariate Cholesky models examining reactive aggression and peer rejection, coefficients $A_{CX}, C_{CX}$, and $E_{CX}$ represent the factor loadings of reactive aggression on the “common” factors $A_C, C_C$, and $E_C$. Respectively, each describes the effects of genetic, shared, and non-shared environmental factors on reactive aggression. A significant coefficient $A_{CX}$ indicates that reactive aggression is significantly influenced by genetic factors, a necessary precondition for testing $rGE$ with peer rejection. Coefficients $A_{CY}, C_{CY}$, and $E_{CY}$ represent the factor loadings of peer rejection on the “common” latent factors $A_C, C_C$, and $E_C$. A significant $A_{CY}$ indicates that both peer rejection and reactive aggression are influenced by the same genetic factors. Because peer rejection is an environmental construct, a significant $A_{CY}$ component may indicate the presence of $rGE$. Significant coefficients $C_{CY}$ or $E_{CY}$ suggest that peer rejection and reactive aggression are influenced by the same shared or nonshared environmental factors, respectively.

Coefficients $A_{UY}, C_{UY}$ and $E_{UY}$ represent the factor loadings of peer rejection (Y) on the unique latent factors $A_U, C_U$ and $E_U$. Respectively, these coefficients indicate the extent to which peer rejection is influenced by genetic, shared, and non-shared environmental factors unrelated to reactive aggression.

In bivariate Cholesky models examining peer rejection and internalizing symptoms, $A_{CX}, C_{CX}$, and $E_{CX}$ represent the factor loadings of internalizing symptoms on the “common” factors $A_C, C_C$, and $E_C$, with a significant $A_{CX}$ coefficient indicating that internalizing symptoms are significantly influenced by genetic factors. Coefficients $A_{CY}, C_{CY}$, and $E_{CY}$ represent the factor loadings of peer rejection on the “common” latent factors $A_C, C_C$, and $E_C$, with a significant $A_{CY}$ indicating that both peer rejection and internalizing symptoms are influenced by the same genetic factors. Because peer
rejection is an environmental construct, a significant $A_{CY}$ component may indicate the presence of $rGE$. Significant coefficients $C_{CY}$ or $E_{CY}$ suggest that peer rejection and internalizing symptoms are influenced by the same shared or nonshared environmental factors, respectively. Coefficients $A_{UY}$, $C_{UY}$ and $E_{UY}$ represent the factor loadings of peer rejection ($Y$) on the unique latent factors $A_U$, $C_U$ and $E_U$.

Bivariate Cholesky models were constructed by combining the best-fitting models estimated in univariate analyses. Nonsignificant latent factor effects in the univariate models were removed to maximize model parsimony and statistical power. Bivariate Cholesky models only estimated overlap for latent factors that were statistically significant in the univariate models. The bivariate results represent the best-fitting, most parsimonious models (Vitaro et al., 2014).

**Objective 2: Bivariate Longitudinal Associations Between (1) Reactive Aggression and Internalizing Symptoms, (2) Reactive Aggression and Peer rejection, and (3) Peer Rejection and Internalizing Symptoms**

Path analyses were conducted to examine bivariate reciprocal associations between reactive aggression and internalizing symptoms and the associations pertaining to the paths posited by the failure model (i.e., reactive aggression and peer rejection, peer rejection and internalizing symptoms). The analyses were conducted twice, with traditional analyses and with a genetically controlled design, to examine whether longitudinal bivariate associations remain after removing genetic and shared environmental influences. Findings from the latter analyses provide evidence for probable causal pathways between reactive aggression and internalizing symptoms,
reactive aggression and peer rejection and between peer rejection and internalizing symptoms.

Analyses were conducted using Bayesian Structural Equation Modeling (BSEM) using Markov chain Monte Carlo (MCMC) algorithms. Models were estimated using two chains, as per the default setting on Mplus v.8.2 (Muthén, 1998-2017). Bayesian statistics incorporate uncertainty of parameter values, and therefore provide a probability distribution (i.e., interval) of a parameter value rather than a single true value. Probability distributions of a parameter estimate (“posterior distributions”) are composed of (a) prior distributions (i.e., existing information on the data that is built into the model) and (b) data likelihood. When prior information about the parameters in the sample is inconclusive, the current data alone can be used inform the probability distribution (Van der Schoot et al., 2014). In the current study, prior information was not sufficiently informative and thus priors were not inserted into the analyses to avoid biasing results. Instead, default uninformative priors were used. In this case, distributions are centered at zero, therefore allowing for greater variance. In large samples, results obtained using uninformative priors are expected to be close to those obtained using maximum-likelihood approaches (Browne & Draper, 2006).

Point estimates in Bayesian statistics, analogous to regression coefficients, refer to the median of the posterior distribution. Standardized estimates are reported. One-tailed p-value describe the direction of posterior distribution. For positive beta point estimates, the p-value represents the proportion of the posterior distribution that is below zero. For negative estimates, the p-value represents the proportion of the posterior distribution that is above zero. Credibility intervals, analogous to confidence intervals, describe the
uncertainty of parameter values. A 95% Bayesian credibility interval spans the 2.5 and 97.5 percentiles of the posterior distributions. Credibility intervals that do not include zero imply a statistically significant effect (Muthen, 2010).

To improve power, temporal constraints (Widaman & Thompson, 2003) were added to analogous influence paths at consecutive time points [e.g., Kindergarten reactive aggression to Grade 1 peer rejection \((a_{1})\) and Grade 1 reactive aggression to Grade 4 peer rejection \((a_{12})\)]. Constraints were retained if they did not significantly worsen fit \((p<.05)\). Because reactive and proactive aggression are known to be highly associated (Poulin & Boivin, 2000), Kindergarten proactive aggression was used as a Time 1 covariate.

Model fit was estimated using the posterior predictive p-value (PPP) and the confidence interval for the difference between the observed and the replicated chi-square values. A PPP above .05 and a negative lower limit 95% confidence interval indicate acceptable model fit (Muthén & Asparouhov, 2012).

**Singleton analyses.** One randomly selected MZ twin’s score was selected for inclusion in the singleton model (see Figure 3). The results represent (a) the direct influence of behavioral adjustment on peer rejection, including genetic and shared environmental influences and (b) direct influence of peer rejection on behavioral adjustment, including genetic and shared environmental influences.

Paths \(a_{1}\) and \(a_{12}\) represent the longitudinal association between initial levels of reactive aggression and changes in peer rejection. Paths \(b_{1}\) and \(b_{12}\) represent the longitudinal association between initial levels of peer rejection and overtime changes in reactive aggression. Paths \(d_{1}\) and \(d_{12}\) represent the longitudinal association between initial levels of peer rejection and changes in internalizing symptoms. Paths \(e_{1}\) and \(e_{12}\)
represent the longitudinal association between initial levels of internalizing symptoms and changes in peer rejection.

**MZ twin differences.** To identify the role of common genetic influences (and gene-environment interplay; rGE) in the direct paths posited by the failure model, the discordant MZ twin difference method was employed. These analyses include difference scores derived from both twins (see Figure 3). The analyses control for genetic effects and for effects that stem from the shared environment to determine a) the unique nonshared environmental effects between reactive aggression and internalizing symptoms, after removing potential confounding contributions of genetic and shared environmental effects, b) the unique nonshared environmental effects between reactive aggression and peer rejection, after removing potential confounding contributions of genetic and shared environmental influences, and c) the unique nonshared environmental effects between peer rejection and internalizing symptoms, after removing potential confounding contributions of genetic and shared environmental influences.

Because MZ twins share 100% of their genetic background, as well as a sizable portion of their environment, differences between twin members should be attributable to their unique environmental experiences, assessed via within-pair differences. The difference score strategy uses the relative differences between the two members of an MZ dyad as a construct (Vitaro et al., 2009). Twins are first randomly assigned as Twin 1 and Twin 2. A twin’s score is then subtracted from the score of the co-twin (e.g., $\Delta_{\text{reactive aggression}}, \Delta_{\text{peer rejection}}, \Delta_{\text{internalizing problems}}$). Associations between MZ twin difference scores are, by definition, a result of non-shared environmental processes. The MZ twin difference scores used were directional (not absolute values); higher values indicate
greater levels of a variable relative to the co-twin, while lower values indicate lower levels of a variable relative to the co-twin.

The analyses determine whether (a) intra-pair differences in reactive aggression are associated with intra-pair differences in internalizing symptoms, (b) intra-pair differences in reactive aggression are associated with intra-pair differences in peer rejection, and (c) intra-pair differences in peer rejection are association with intra-pair differences in internalizing symptoms. Paths s1-s6 represent the stability of intra-pair differences on the same variable over time. Paths a1-a12 represent the longitudinal associations between initial intra-pair differences on reactive aggression and changes in intra-pair differences on peer rejection. Paths b1-b12 represent the longitudinal associations between initial intra-pair differences on peer rejection and changes in intra-pair differences on reactive aggression. Paths c1-c12 represent longitudinal associations between initial intra-pair differences on peer rejection and changes in intra-pair difference on internalizing symptoms. Paths d1-d12 represent longitudinal associations between initial intra-pair differences on internalizing symptoms and over-time changes in intra-pair differences on peer rejection.

**Objective 3: Longitudinal Association from Kindergarten Reactive Aggression to Grade 4 Internalizing Symptoms, Mediated by Grade 1 Peer rejection**

A series of full longitudinal mediational model were conducted to examine the indirect effect of peer rejection in the association between reactive aggression and internalizing symptoms, as posited by the failure model (Patterson & Capaldi, 1990). The analyses were conducted twice, with traditional analyses and with a genetically controlled design, to examine whether indirect effects remain after removing influence from genetic
factors (and gene-environment interplay; rGET) and shared environmental factors. Findings from the latter analyses provide evidence for a probable causal pathway between reactive aggression and internalizing symptoms via peer rejection.

**Singleton analyses.** Path analyses that were used to examine bidirectional effects of reactive aggression, peer rejection, and internalizing symptoms were adjusted to examine the hypothesized indirect effect. Figure 4 presents the measurement model. A full longitudinal design to mediation analyses (Fritz & MacKinnon, 2012) was applied. A statistically significant association from the Time 1 predictor (i.e., Kindergarten reactive aggression) to the Time 3 outcome (i.e., Grade 4 internalizing symptoms) is not a precondition for mediation; preliminary tests of the association are not recommended (Shrout & Bolger 2002). Mediation was tested using Bayesian estimation, which constructs credible intervals (CI) for indirect effects with potentially skewed data (Yuan & MacKinnon, 2009).

**MZ differences.** A full mediational model was conducted using the MZ twin-difference score method. Figure 4 presents the measurement model. Path analyses examining the bidirectional effects of the intra-pair MZ twin differences for reactive aggression, peer rejection, and internalizing symptoms were adapted to examine the hypothesized indirect effect. Intra-pair difference scores from both twins were placed use as constructs in order to methodologically control for the role of genetic (and shared environmental) influences in the mediation between of Kindergarten reactive aggression and Grade 4 internalizing symptoms, via Grade 1 peer rejection.

*Missing Data*
Missing item-level data were minimal, averaging 1.6% in Kindergarten (Range: 1.3-4.5%), 2.8% in Grade 1 (Range: 1.3-2.5%) and 4.3% in Grade 4 (Range: 0.9-5.7%). An average of 23% of participants were missing at each wave. Little’s MCAR test indicated that data were missing completely at random, $\chi(1106) = 10956.692$, $p = .76$. To account of wave and item-level missingness among twin-pair dyads in the study, analyses were conducted with multiple imputations generated in SPSS, using an expectation maximization (EM) algorithm with 25 iterations. Multiple imputation is a robust and accurate estimator of results when less than 50% of data are missing completely at random (Graham, 2009). Parameter estimates were averaged over the imputed data sets (Rubin, 1987).

**Supplemental Analyses**

A series of three supplemental analyses were conducted to isolate the unique contributions of variables included from other potentially confounding factors. Supplemental models included the addition of control variables known to correlate influence behavioral differences in twin members (i.e., child birthweight) and demographic controls (i.e. maternal education, income). Each control was separately added to each model (singleton analyses and MZ difference scores), as a covariate in Kindergarten to determine if the same pattern of statistically significant indirect effects were maintained.

Multiple group models were used to examine sex differences in patterns of associations, using Wald tests of parameter equality constraints. Multiple group models also tested whether class enrollment (i.e., whether twins were in the same class in school) affected the pattern of associations. In addition, given that main path analyses were each
conducted using proactive aggression as a concurrent covariate in Kindergarten, all analyses were repeated without proactive aggression.

Finally, the direction of effects in the mediation model was confirmed by comparing the indirect effects models with statistically plausible alternative mediation models (Little, 2013). An alternative model in which the effect of Kindergarten internalizing symptoms to Grade 4 reactive aggression was mediated by Grade 1 peer rejection was tested by adding the direct path from Kindergarten internalizing symptoms to Grade 4 reactive aggression and testing its indirect effect. The alternative mediation model was tested in both the singleton and MZ twin differences analyses. A final alternative model tested reverse causal effects (Judd & Sadler, 2008). The outcome (i.e., internalizing symptoms) and mediator (i.e., peer rejection) are switched, such that Kindergarten reactive aggression predicts Grade 4 peer rejection, via internalizing symptoms. The alternative mediation model was tested using both the singleton and MZ twin difference designs.
RESULTS

Preliminary Analyses

Concurrent bivariate correlations, means, and standard deviations for study variables are presented in Tables 1-3. Correlations for one randomly selected member of each twin dyad (i.e. singletons) are presented above the diagonal. The same pattern of results emerged in analyses with the other member of the twin dyad. Correlation contrasts failed to reveal any statistically significant ($p<.05$) differences between samples in any of the correlations. Among singletons, all study variables were concurrently correlated (except between internalizing symptoms and proactive aggression during Kindergarten, and between internalizing symptoms and peer rejection during Kindergarten).

Correlations for MZ twin difference scores are presented below the diagonal. Reactive aggression was correlated with internalizing symptoms at each time point. Reactive aggression was correlated with proactive aggression in Kindergarten, and reactive aggression was correlated peer rejection in Grade 1.

Autocorrelations are summarized in Table 4. All singleton autocorrelations were statistically significant. There were statistically significant autocorrelations for MZ twin difference scores for reactive aggression from Kindergarten to Grade 1 and from Grade 1 to Grade 4. Autocorrelations for peer rejection were significant from Kindergarten to Grade 4, and autocorrelations for internalizing symptoms were significant from Kindergarten to Grade 1. Table 4 also summarizes longitudinal correlations between study variables. All over-time singleton correlations were significant (except for those
between initial internalizing symptoms and later peer rejection and between Grade 1 internalizing symptoms and Grade 4 reactive aggression). For twin difference scores, Kindergarten reactive aggression was correlated with Grade 1 internalizing symptoms, and Kindergarten peer rejection was correlated with Grade 4 internalizing problems.

**Objective 1: Partitioning Sources of Variance**

Genetic analyses were conducted to partition sources of variance. Univariate ACE models estimated the relative contribution of genetic and environmental factors to reactive aggression, peer rejection, and internalizing problems at each time point (Kindergarten, Grade 1, and Grade 4). Using information from MZ and DZ twins, the analyses provide information regarding the degree to which each (separate) variable is influenced by genetic and environmental factors. Upon partitioning the variance for each variable separately, bivariate Cholesky models were conducted to gain a better understanding of the underlying genetic and environmental variance that is shared between reactive aggression and peer rejection, internalizing symptoms and peer rejection, and reactive aggression and internalizing symptoms. (e.g., aggression, internalizing symptoms), common underlying genetic factors between the two traits can be attributed to a gene-environment correlation (Jaffi, 2016). If a significant portion of the shared variance between peer rejection and (1) reactive aggression or (2) internalizing symptoms can be attributed to common genetic factors, the presence of rGE can be implied. This is possible because peer rejection is a putative environmental experience.

**Univariate ACE Model Results**

Tables 5-7 summarize the results of the univariate ACE models describing reactive aggression, peer rejection and internalizing problems at each time point.
For reactive aggression, genetic factors explained 52-56% of the variance, shared environmental factors explained 0-4% of the variance, and nonshared environmental factors explained 43-48% of the variance. For peer rejection, genetic factors explained 34-56% of the variance, shared environmental factors explained 0-10% of the variance, and nonshared environmental factors explained 44-59% of the variance. For internalizing symptoms, genetic factors explained 21-47% of the variance, shared environmental factors explained 0-12% of the variance, and nonshared environmental factors explained 53-68% of the variance.

Because the effect of the shared environmental components in each model was non-significant and estimated to be zero (or nearly zero) in each of the univariate ACE models, additional analyses were conducted in which the C component was omitted from the model. Specifically, shared environmental effects were fixed to zero in a series of AE models, following procedures recommended by Brendgen and colleagues (Brendgen et al., 2012). A series of nested $\chi^2$ comparison tests revealed no statistically significant change in model fit between any of the ACE and AE models estimated, $\chi^2(1) = 0.00-0.26$, $p = .26-1.00$. The nested AE models, with fewer parameters are more parsimonious, and therefore have better fit. The results indicate that reactive aggression, peer rejection, and internalizing symptoms are each partially explained by genetic and nonshared environmental factors, in roughly equal parts, with no shared environmental contribution.

**Bivariate Cholesky ACE Model Results**

A series of bivariate ACE Cholesky models were conducted to identify common underlying sources of variance between each pair of variables. Three sets of analyses were conducted at each time point: (1) associations between peer rejection and reactive
aggression (Figure 5-7), (2) associations between peer rejection and internalizing symptoms (Figure 8-10) and (3) associations between reactive aggression and internalizing symptoms (Figure 11-13).

**Reactive aggression and peer rejection.** Bivariate Cholesky models examining the association between peer rejection and reactive aggression included two components: (1) common latent factors $A_C$, $C_C$, and $E_C$ that influence both peer rejection and reactive aggression, and (2) unique latent factors, $A_{UY}$, $C_{UY}$, and $E_{UY}$ specific to peer rejection (see Figures 5-7). Because shared environmental effects were not statistically significant in the univariate analyses of peer rejection and reactive aggression, the C component of reactive aggression ($C_{CX}$), the C component of peer rejection ($C_{UY}$), and the C overlap parameters ($C_{CY}$) were fixed to be zero in the bivariate model (Neale & Cardon, 1992). Model fits were acceptable, $\chi^2(20) = 6.99$ -22.43, $p$.99-1.00, CFI =.99-1.00, RMSEA = .00-.02.

Figure 5 presents the results for Kindergarten. The model revealed that approximately 31.5% (.38$^2$/(.38$^2$ + .56$^2$) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. There was no nonshared environmental overlap between peer rejection and reactive aggression. The shared environment covariance path was fixed to zero and was therefore not estimated.

The Grade 1 model (see Figure 6) revealed that approximately 29.8% (.41$^2$/(.41$^2$ +.63$^2$) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. There was a small, but significant, amount of overlap in the nonshared environmental factors predicting peer rejection and reactive aggression.
2.2% \((.10^2 / (.10^2 + .66^2))\). The shared environment covariance path was fixed to zero and was not estimated.

The Grade 4 model (see Figure 7) revealed that 32.8% \((.37^2 / (.37^2 + .53^2))\) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. There was no nonshared environmental overlap in the factors predicting peer rejection and reactive aggression. The shared environment covariance path was fixed to zero and was not estimated.

**Peer rejection and internalizing symptoms.** Bivariate Cholesky models examining the association between peer rejection and internalizing symptoms included two components: (1) common latent factors \(A_C, C_C\), and \(E_C\) that influence both peer rejection and internalizing symptoms, and (2) unique latent factors, \(A_UY\), \(C_{UY}\), and \(E_{UY}\) specific to peer rejection (see Figures 8-10). Because shared environmental effects were not statistically significant in the univariate analyses of peer rejection and internalizing symptoms, the C component of internalizing symptoms (\(C_{CX}\)), the C component of peer rejection (\(C_{UY}\)), and the C overlap parameters (\(C_{CY}\)) were fixed to be zero in the bivariate model. The models fit the data well, \(\chi^2(20) = 6.64 - 25.09, p = .20 - 1.00, CFI = .97 - 1.00, RMSEA = .00 - .03\).

Figure 8 presents the results for Kindergarten. The model revealed that genetic variance in children’s peer rejection could not explain influences in internalizing symptoms. There was a small, but significant, amount of overlap in the nonshared environmental factors predicting peer rejection and reactive aggression \(1.5% \((.09^2 / (.09^2 + .74^2))\). The shared environment covariance path was fixed to zero and was therefore not estimated.
The Grade 1 model (see Figure 9) revealed that approximately 13.8% \((.28^2/ (.28^2 + .70^2))\) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. There was no nonshared environmental overlap in the factors predicting peer rejection and reactive aggression. The shared environment covariance path was fixed to zero and was not estimated.

The Grade 4 model (see Figure 10) revealed that 17.3% \((.27^2/ (.27^2 + .59^2))\) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. There was no overlap in the nonshared environmental factors predicting peer rejection and reactive aggression. The shared environment covariance path was fixed to zero and was not estimated.

**Reactive aggression and internalizing symptoms.** Bivariate Cholesky models examining the association between reactive aggression and internalizing symptoms included two components: (1) common latent factors \(A_C, C_C, E_C\) that influence both reactive aggression and internalizing symptoms, and (2) unique latent factors, \(A_{UY}, C_{UY}, E_{UY}\) specific to internalizing symptoms (see Figures 11-13). Because shared environmental effects were not statistically significant in the univariate analyses of reactive aggression and internalizing symptoms, the \(C\) component of reactive aggression (\(C_{CX}\)), the \(C\) component of internalizing symptoms (\(C_{UY}\)), and the \(C\) overlap parameters (\(C_{CY}\)) were fixed to be zero in the bivariate model. The models fit the data well, \(\chi^2(20) = 3.72-22.96, p=.29-1.00, CFI = .99-1.00, RMSEA = .00-.02.\)

Figure 11 presents the results for Kindergarten. The model revealed that genetic variance in children’s internalizing symptoms explained a small, albeit significant amount of variance in reactive aggression, 4.8% \((.13^2/ (.13^2 + .58^2))\). There was also a
small, but significant, amount of overlap in the nonshared environmental factors predicting reactive aggression and internalizing symptoms, 3.9% ($0.16^2 / (0.16^2 + 0.79^2)$). The shared environment covariance path was fixed to zero and was therefore not estimated.

The Grade 1 model (see Figure 12) revealed that approximately 15.9% ($0.27^2 / (0.27^2 + 0.62^2)$) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. There was a small, but significant, amount of overlap in the nonshared environmental factors predicting peer rejection and reactive aggression 3.6% ($0.14^2 / (0.14^2 + 0.72^2)$), but the majority of nonshared environmental influences were specific to peer rejection. The shared environment covariance path was fixed to zero and was not estimated.

The Grade 4 model (see Figure 13) revealed that 21.1% ($0.31^2 / (0.31^2 + 0.60^2)$) of the genetic variance in children’s peer rejection was explained by factors that also influenced reactive aggression. Most of the variance attributed to genetic factors was unique to peer rejection. There was a small, but significant, amount of overlap of nonshared environmental factors predicting peer rejection and reactive aggression 3.2% ($0.13^2 / (0.13^2 + 0.72^2)$), but the majority of nonshared environmental influences were specific to peer rejection. The shared environment covariance path was fixed to zero and was thereby not estimated.

In sum, the analyses suggest that peer rejection, reactive aggression and internalizing symptoms are each influenced by genetic and non-shared environmental factors. A sizeable portion of the genetic influence is shared between study variables. Genetic factors that influence both reactive aggression and internalizing symptoms explain variance in peer rejection. Genetic factors that influence reactive aggression also
explain a statistically significant (albeit small) portion of the variance in internalizing symptoms. The results suggest that associations between study variables are at least partly a product of rGEs and common genetic factors.

**Objective 2: Longitudinal Associations between Behavioral Adjustment and Peer Rejection**

The next set of analyses were designed to test reciprocal longitudinal associations between peer rejection and (1) reactive aggression and (2) internalizing symptoms. Figures 14 and 15 depict results. Two sets of analyses were conducted. The first set examined longitudinal associations among singletons (i.e., without removing genetic contributions). The second set examined longitudinal associations among MZ twin difference scores (i.e., after controlling for genetic contributions). Model fit was acceptable, with posterior predictive p values (PPP) equal to .58 for the singleton analyses and .32 for the MZ twin difference score model.

**Singleton Analyses**

Higher initial levels of peer rejection predicted increases in reactive aggression, from Kindergarten to Grade 1 ($\beta=.19$, 95% CI [.10, 26]), and from Grade 1 to Grade 4 ($\beta=.18$, 95% CI [.10, .27]). The reciprocal effect was also significant: Higher initial levels of reactive aggression predicted increases in peer rejection from Kindergarten to Grade 1 ($\beta=.12$, 95% CI [.03, 21]), and from Grade 1 to Grade 4 ($\beta=.11$, 95% CI [.03, 21]).

Higher initial levels of peer rejection predicted increases in internalizing symptoms from Kindergarten to Grade 1 ($\beta=.18$, 95% CI [.07, 25]), and from Grade 1 to Grade 4 ($\beta=.18$, 95% CI [.07, 27]). However, the reciprocal effect was not statistically significant: Internalizing symptoms did not predict changes in peer rejection from Kindergarten to
Grade 1 (β = -.02, 95% CI [-.09, .08]), or from Grade 1 to Grade 4 (β = -.02, 95% CI [-.09, .08]).

Reactive aggression did not predict changes in internalizing symptoms from Kindergarten to Grade 1 (β=.07, 95% CI [.04, .14]), or from Grade 1 to Grade 4 (β=.07, 95% CI [.04, .16]). Reciprocal paths were also nonsignificant: internalizing symptoms did not predict reactive aggression from Kindergarten to Grade 1 (β=.07, 95% CI [.04, .14]), or from Grade 1 to Grade 4 (β=.07, 95% CI [.04, .16]). The results are depicted in Figure 14.

**MZ Twin Difference Score Analyses**

Twin differences in peer rejection did not predict changes in twin differences in reactive aggression from Kindergarten to Grade 1 (β=.03, 95% CI [.05, .12]), or from Grade 1 to Grade 4 (β=.03, 95% CI [.04, .11]). Reciprocal paths were also nonsignificant: Twin differences in reactive aggression did not predict changes in twin differences in peer rejection from Kindergarten to Grade 1 (β=.01, 95% CI [.08, .11]), or from Grade 1 to Grade 4 (β=.01, 95% CI [.07, .08]).

Twin differences in peer rejection did not predict changes in twin differences in internalizing symptoms, from Kindergarten to Grade 1 (β=.07, 95% CI [.03, .15]) or from Grade 1 to Grade 4 (β=.06, 95% CI [.03, .14]). The reciprocal effect was also nonsignificant: Twin differences in internalizing symptoms did not predict changes in twin differences in peer rejection, from Kindergarten to Grade 1 (β=.03, 95% CI [.12, .05]) or from Grade 1 to Grade 4 (β=.03, 95% CI [.11, .04]).

Twin differences in reactive aggression did not predict changes in twin differences in internalizing symptoms from Kindergarten to Grade 1 (β=.07, 95% CI [.03, .15]).
.03, 15]) or from Grade 1 to Grade 4 (β=.07, 95% CI [-.03, 17]). The reciprocal effect was also nonsignificant: Twin differences in internalizing symptoms did not predict changes in twin differences in reactive aggression from Kindergarten to Grade 1 (β=-.03, 95% CI [-.12, 05]) or from Grade 1 to Grade 4 (β=-.03, 95% CI [-.11, 04]). The results are depicted in Figure 15.

Objective 3: Indirect Effects of Reactive Aggression on Internalizing Symptoms, via Peer rejection

The next set of analyses examined Grade 1 peer rejection as a mediator of the association between Kindergarten reactive aggression and Grade 4 internalizing symptoms. Figures 16 and 17 depict full longitudinal mediation models. Two sets of analyses were conducted. The first set examined longitudinal associations among singletons (i.e., without removing genetic contributions), testing the failure model posited by Patterson and colleagues (1990). The second set examined longitudinal associations among MZ twin difference scores (i.e., after controlling for genetic contributions), to determine whether associations identified in the failure model are a product of genetic influences. Model fit was acceptable, with a posterior predictive p values (PPP) equal to .58 for the singleton model, and .27 for the MZ twin difference score model.

Singleton Analyses

Kindergarten reactive aggression was positively associated with Grade 1 peer rejection (β=.11, 95% CI [.03, .19]) which, in turn, was positively associated with Grade 4 internalizing symptoms (β=.18, 95% CI [.09, .28]). The indirect effect through Grade 1 peer rejection was statistically significant (β=.02, 95% CI [.004, .04]). Higher levels of reactive aggression predicted increases in internalizing symptoms, mediated by peer
rejection. The addition of the mediated path did not change the pattern of statistically significant reciprocated effects: peer rejection and reactive aggression predicted one another, across each lag. Peer rejection was associated with internalizing symptoms across each lag, but internalizing symptoms did not predict peer rejection. Reactive aggression and internalizing symptoms were not significantly associated in either lag. The results are depicted in Figure 14.

**MZ Twin Difference Score Analyses**

Kindergarten reactive aggression was not associated with Grade 1 peer rejection ($\beta=-.004, 95\% \text{ CI } [-.09, .08]$), and Grade 1 peer rejection was not associated with Grade 4 internalizing symptoms ($\beta=.06, 95\% \text{ CI } [.02, .15]$). The indirect effect for Grade 1 peer rejection was not statistically significant ($\beta=.00, 95\% \text{ CI } [.01, .01]$). The addition of the mediated path did not change the reciprocated effects: Associations between peer rejection and reactive aggression, peer rejection and internalizing symptoms, and reactive aggression and internalizing symptoms were not statistically significant in either lag.

**Summary**

Path analyses using singleton scores revealed significant bidirectional effects between peer rejection and reactive aggression. Higher levels of peer rejection predicted increases in reactive aggression. Higher levels of reactive aggression predicted increases in peer rejection. However, the association between peer rejection and internalizing symptoms was unidirectional. While higher levels of peer rejection predicted increases in internalizing symptoms, the reverse was not true. Internalizing symptoms were not associated with changes in peer rejection. Findings were consistent across both time lags. When repeating the analyses using MZ twin difference scores, direct effects were no
longer significant. Peer rejection was not associated with either reactive aggression or internalizing symptoms across either time lag. The full longitudinal mediation model using singleton scores revealed a significant indirect of peer rejection on the association between reactive aggression and internalizing symptoms. However, when analyses were repeated using MZ twin difference scores, the indirect effect was no longer significant. The results indicate that that shared genetic influences are responsible for bidirectional associations between peer rejection and reactive aggression, the unidirectional association between initial levels of peer rejection and changes in internalizing symptoms, and the indirect effect of Kindergarten reactive aggression to Grade 4 internalizing symptoms through Grade 1 peer rejection. When MZ twin difference score analyses fail to replicate singleton analyses, it is assumed that rGE is responsible for the singleton associations, especially given the large presence of shared genetic influence on peer rejection and (1) reactive aggression and (2) internalizing symptoms.

**Supplemental Analyses**

Separate analyses were conducted to control for the contribution of variables known to contribute to disturbance to socioemotional adjustment (i.e., birthweight, maternal education, household income; Achenbach; Chatterji et al., 2014). Each was separately added to the model as a concurrent Kindergarten covariate. In each case, the same pattern of statistically significant results emerged.

Wald tests of parameter equality constraints revealed no statistically significant sex differences in patterns of associations. The same tests also found no statistically significant differences in patterns of associations between children who attended the same class in school as their co-twin and those who attended different classes, ruling out
the possibility that heritability was inflated due to shared source bias (Towers et al., 2000).

Follow-up analyses explored alternative mediation models, to confirm the direction of effects (Little, 2013). Singleton and MZ twin difference models tested an indirect path from Kindergarten internalizing symptoms to Grade 4 reactive aggression, via Grade 1 peer rejection. Neither set of indirect effects were statistically significant (Singletons: $\beta = -.03$, 95% CI [-.02, 01]; MZ twin differences: $\beta = .00$, 95% CI [-.01, 00]).

A Levene’s test for equality of variances determined that there were no statistically significant differences between the variance in any of the variables when using traditional designs and the MZ twin difference design, with the exception of Kindergarten reactive aggression ($F=17.35, p<.01$).
DISCUSSION

The current study tested adjustment pathways posited by the failure model, which hypothesized longitudinal associations from aggression to internalizing symptoms mediated by peer rejection (Patterson & Capaldi, 1990). As expected, ACE models revealed considerable genetic contributions to each variable and Cholesky models suggested that some of these genetic contributions were shared between variables, which can bias observed estimates in longitudinal path analyses when left unaddressed (Vitaro & Brendgen, 2016). Divergent findings emerged when the failure model was tested twice, once using a traditional framework (i.e., without accounting for genetics), and again using the MZ twin difference method, which controls for the influence of genetic factors. Results from conventional analyses were consistent with the failure model, but those from genetically controlled analyses were not. Specifically, traditional analyses identified direct longitudinal associations between each of the specific sequential steps in the failure model (i.e., from reactive aggression to increases in peer rejection, and from peer rejection to increases in internalizing symptoms), as well as for the indirect association from reactive aggression to internalizing symptoms via peer rejection. In genetically controlled analyses, none of these associations were statistically significant. Together, the results suggest that the causal paths previously identified as support for the failure model, may be, in large part, an artifact of common genetic factors and gene-environment interplay. The findings call into question the mechanisms underlying this
interpretation of the failure model and suggest that assumptions about the role of the peer
environment in the development of internalizing symptoms need to be revisited.

**Relative Contributions of Genetic Factors and Environmental Factors**

The first aim of the study sought to determine genetic contributions to reactive
aggression, peer rejection, and internalizing symptoms during the early primary school
years. A series of ACE and Cholesky models revealed substantial genetic influences to
each of the study variables and to their bivariate associations.

**Contributions of Genetic Factors and Environmental Factors to Each Study Variable**

Univariate ACE models identified genetic and environmental contributions to
each the constructs involved in the failure model (i.e., reactive aggression, peer rejection,
and internalizing symptoms) to ascertain the need for genetically controlled analyses. As
hypothesized, genetic factors contributed a significant portion of the variance in each
study variable. The rest of the variance was explained by nonshared environmental
factors, which include peer relationships, but can also encompass attitudes, parental
treatment and perceptions of relationships with siblings and teachers; Brendgen et al.,
2012). The shared environment (i.e., environmental factors common to family members
who live together) did not play a significant role in any variable. Together, the findings
suggest that reactive aggression, peer rejection and internalizing symptoms are each
influenced by heritable factors. While these findings do not specify whether the same
genetic factors influence the distinct components of the failure model, they serve as a
primary indication that genetic influences may play an important role in model and
should therefore be controlled for in empirical tests of the failure model.
Reactive Aggression

The results for reactive aggression were consistent with those from prior genetic studies during the early school years (Brendgen et al., 2006; Baker et al., 2007; Tuvblaud et al., 2009). A sizeable portion of the variance in reactive aggression was attributed to genetic factors. The genetic underpinnings of reactive aggression may be related to specific temperamental dispositions. Reactive aggression has been linked to a difficult childhood temperament (Vitaro et al., 2002). Given the highly heritable nature of infant temperament, it is likely that temperamental factors may explain genetic contributions to reactive aggression (Goldsmith, 1996). Specifically, heritable temperamentally-linked characteristics such as emotional negativity, irritability, and low tolerance for frustration, may be relevant (Vitaro et al., 2006).

This study found no significant contributions stemming from the shared environment. While the findings are concordant with those from a prior study that used subset of the current data (Brendgen et al., 2006; Paquin et al., 2014), they are inconsistent with others (e.g., Tuvblaud et al., 2009). My findings contradict research tracing reactive aggression to poor family environments (e.g., elevated levels of physical abuse, harsh parenting; Dodge et al., 1997). Experiencing harsh and hostile discipline has been postulated to increase levels of hypervigilance and lower thresholds for anger and frustration, characteristics that are typically observed in reactively aggressive children (Dodge et al., 1997; Vitaro et al., 2006). While these experiences are traditionally thought to be shared by cohabitating family members, the null findings do not necessarily mean that parental maltreatment is unimportant. Factors that are traditionally attributed to the shared environment, such as parenting behaviors, may instead be attributable to the
nonshared environment. Children, even when raised together, often experience
differential parental treatment (Paquin et al., 2014). The discrepant findings may also
have been a result of reduced shared-reporter bias. Teachers – rather than parents (as in
Tuvblad et al., 2009) – reported on children’s reactive aggression, which may have
minimized shared environmental effects otherwise inflated by shared-reporter bias
(Tuvblad et al., 2009).

In accordance to past studies, the nonshared environment significantly
contributed to differences in reactive aggression (Brendgen et al., 2006; Baker et al,
2007; Tuvblad et al., 2009). In addition to the home environment, it is more likely that
social experiences stemming from the peer group are important nonshared environmental
influences. Reactively aggressive children are often maltreated, isolated, victimized, and
experience a lower availability of friendships (Card & Little, 2006). Interpersonal
difficulties may aggravate children’s angry outbursts and can reflect in reactive
aggression (Dodge et al., 1997).

Genetic and non-shared environmental contributions remained significant and
substantial across the different ages. Because ACE models were conducted within time,
the analyses cannot inform on the overtime stability of genetic and environmental factors
influencing reactive aggression (Francic et al., 2010). However, they provide initial
evidence that the magnitude of effects varies little across the early school years. The
heritability of reactive aggression ranged from 52.2-55.8% between Kindergarten and 4th
grade, while the influence from the nonshared environment varied from 42.4-47.8%. While increases in the genetic influence of reactive aggression have been reported from
middle childhood to adolescence (Tuvblad et al., 2009; Baker et al., 2007), it is possible that such increases are not observed across the early primary school years.

**Peer Rejection**

The findings, consistent with a prior study that used a subset of this sample (Brendgen et al., 2009), indicated a significant genetic contribution to peer rejection. Because peer rejection is an environmental experience, genetic influences on peer rejection must occur through an evocative rGE process, whereby behavioral traits and tendencies predispose a child to experience dislike from the peer group (Burt & Donnellan, 2015). Heritable behavioral tendencies like disruptiveness and antagonism are known predictors of peer rejection (Dodge, 1983) and may partially explain the genetic underpinnings of the construct. In addition, temperamental characteristics linked to interpersonal functioning in adulthood that may also play a role. Heritable temperamental characteristics in early childhood (e.g., lack of effortful control, irritability, impulsivity, and emotionality lability) associated with interpersonal difficulties later in life (Newman et al., 1997).

In contrast to findings by Brendgen et al (2009), shared environmental factors did not significantly contribute to peer rejection. However, null findings are concordant with another study (also using a subset of the current sample; Boivin et al., 2013a) that examined group-related peer difficulties. While parental maltreatment has been postulated to affect peer functioning (Dickson et al., 2019), this study did not find evidence for such influences. Of course, it is possible that parental treatment may be a part of the nonshared environment. Thus, parenting may still affect peer rejection through nonshared environmental factors.
Consistent with prior studies examining peer rejection (Brendgen et al., 2009) and similar group-related peer difficulties (e.g., Ball et al., 2008; Boivin et al., 2013), nonshared environmental factors were responsible for a sizeable portion of the variance in peer rejection. Nonshared environmental influences may include the social context at school (e.g., relationships with teachers, peers), attitudes and reactions towards peer maltreatment, as well as the general availability of friendships (Brendgen et al., 2008).

Across different age groups, the magnitude of effects from genetic and nonshared environmental influences remained consistent, ranging from 41.3-56.5% and 43.5-58.7%, respectively, from Kindergarten to 4th grade. Although caution is required when drawing conclusions from the results given their concurrent nature, the findings are one of the first to suggest that the genetic influences of peer rejection may be consistent throughout the early school years (see Boivin et al., 2013b for another example).

**Internalizing Symptoms**

In line with prior studies, heritable factors significantly contributed to the variance in internalizing symptoms (e.g., Brendgen et al., 2009; Happonen et al., 2002; Scaini et al., 2014). Temperamental characteristics like reduced effortful control and negative affect, both highly heritable dispositions, may play a role in explaining the variance in internalizing symptoms (Crawford et al., 2010). Negative affect typically reflects in an elevated intensity and frequency in displays of anger, sadness, discomfort, and fear (Watson & Clark, 1984), while decreased effortful control can be reflected in poor attention and difficulties inhibiting behaviors (Corsnoe & Elder, 2002). In addition, consistent with past studies (e.g., Brendgen et al., 2009; Patterson et al., 2018), the shared environment did not play a role in explaining differences in internalizing symptoms.
While non-genetically informed studies have suggested that characteristics associated with the family environment including parental conflict and marital problems are associated with internalizing symptoms, these may be confounded with genetic factors or factors of the non-shared environment. Nonshared environmental factors did significantly contributed to the variance found in internalizing symptoms. This is consistent with prior findings (e.g., Brendgen et al., 2009; Patterson et al., 2018; Scaini et al., 2014) and suggests that factors associated with the peer context (i.e., attitudes about the social hierarchy, friendship opportunities, group-related experiences; Prinstein et al., 2005) may be important. It is also like that parent-child relationships, which are often cited as predictors of depressive and anxiety symptoms may also play a role as nonshared environmental factors (Crawford et al., 2010).

Longitudinal increases in heritability for internalizing symptoms are unlikely to occur through amplification processes (i.e., as children age, they have more autonomy to increased “niche-fitting”, thereby amplifying genetic influences; Scarr & McCartney, 1983) given the nature of internalizing symptoms. Instead, increases in heritability may be the result of innovative genetic influences. Genes that were not previously affecting internalizing problems at an earlier age may become do so at later periods. (Briley & Tucker-Drob, 2013). Declines in the nonshared environment are difficult to interpret given it reflects both nonshared environmental variance and measurement error (Knafo & Plomin, 2006).

**Shared Genetic and Environmental Contributions between Study Variables**

As expected, bivariate analyses indicated that reactive aggression, peer rejection, and internalizing symptoms shared some of the same underlying genetic factors,
underscoring the importance of using a genetically-controlled design to test the failure model. Specifically, genetic factors contributed to associations between a) reactive aggression and internalizing symptoms, b) reactive aggression and peer rejection, and c) peer rejection and internalizing symptoms. Bivariate associations could not be explained by the shared environment. However, a small portion of the link between reactive aggression and peer rejection was explained by nonshared environmental factors in Grade 1 and Grade 4. Similarly, common nonshared environmental factors explained a small portion of the link between rejection and internalizing symptoms in Kindergarten, and a moderate portion of the link between reactive aggression and internalizing symptoms across the Kindergarten, Grade 1, and Grade 4.

**Reactive Aggression and Internalizing Symptoms**

There was a significant genetic contribution to the association between reactive aggression and internalizing symptoms, with the degree of overlap seemingly increasing with age. Specifically, the overlap in genetic factors associated with both reactive aggression and internalizing symptoms was 4.8% in Kindergarten, 15.9% in Grade 1, and 21.2% in Grade 4. The findings are consistent with studies that suggest links between externalizing and internalizing problems can be partially attributed to common genetic factors (Pesenti-Gritti et al., 2008; Rappaport et al., 2018). Genetic overlap between reactive aggression and internalizing symptoms can be explained by two possible mechanisms. First, biological pleiotropy may responsible; the same genetic source may affect both reactive aggression and internalizing symptoms. For instance, genetically originating, temperamentally-linked characteristics (e.g., irritability, emotional dysregulation, anxiety, or emotional negativity) may give rise to both reactive aggression
(Dodge & Coie, 1987; Dodge et al., 1999) and internalizing symptoms (Anthony et al., 2002; Eisenberg, 2005; Vitaro et al., 2012). Second, it is possible that the association is a product of a complex, masked gene-environment interplay, wherein peer rejection acts as the mediating (heritable) environment between the two traits. Cholesky models involving peer rejection (a) reactive aggression and (b) internalizing symptoms addressed the latter possibility.

**Reactive Aggression and Peer Rejection**

A substantial portion of the association between reactive aggression and peer rejection was attributable to heritability. Specifically, the overlap in genetic factors involved in both reactive aggression and peer rejection was 31.5% in Kindergarten, 29.8% in 1st grade, and 32.8% in 4th grade. The findings are consistent with studies that report that some of the same genetic factors are common to aggression and peer difficulties (Ball et al., 2008; Brendgen et al., 2015. Two mechanisms may be at play. First, genetic factors associated with aggression may increase a child’s propensity to experience peer harassment or maltreatment (Boivin et al., 2013; Brendgen et al., 2015). Genetically-linked characteristics of reactive aggression (e.g., irritability, negative emotionality, impulsivity; Vitaro & Brendgen, 2005) may elicit dislike from peer group, reflecting in higher levels of peer rejection. This would suggest the presence an evocative rGE, implying that reactive aggression precedes peer rejection (Boivin et al., 2013). However, it is also possible that both peer rejection and reactive aggression are predicted by common genetic influences. Undefined behavioral, language or physical characteristics may both evoke peer difficulties and influence reactive aggression (Boivin et al., 2013a).
Peer Rejection and Internalizing Symptoms

A modest part of the association between peer rejection and internalizing symptoms is due to a shared genetic contribution. Although there was no shared genetic overlap in Kindergarten, the overlap increased to 13.8% in 1st grade, and 17.3% in 4th grade. The results were concordant with previous findings. Brendgen and colleagues (2009) found a genetic overlap between peer rejection and depressive symptoms in Kindergarten. Two mechanisms can explain the genetic overlap between peer rejection and internalizing symptoms. First, the overlap may be a result of an evocative rGE. Children who are predisposed to internalizing symptoms (e.g., anxiety, depressive behavior) may tend to elicit peer rejection (Brendgen et al., 2009). Internalizing symptoms have been linked to temperamental characteristics like negative emotionality, and behavioral inhibition (Rubin et al., 2012), heritable characteristics that have also been associated with increased levels of peer rejection (Eisenberg et al., 2000). It is possible that a genetic vulnerability to internalizing symptomology may reflect in conflict-ridden interpersonal behaviors and an inability to collaborate, which can evoke maltreatment from the peer group (Altman & Gotlib, 1998; Brendgen et al., 2009). Similarly, An evocative rGE would imply that internalizing symptoms precede peer rejection. It is also possible that both peer rejection and internalizing symptoms are the product of common underlying genetic factors (i.e., environmentally-mediated pleiotropy; Avinun, 2020). The underlying genetic factors (e.g., emotional reactivity, irritability, disruptiveness) that may evoke peer rejection from reactive aggression eventually contribute to a rise in internalizing symptoms.

Empirical Tests of The Failure Model
The second and third aims of the study focused on empirically testing the direct and indirect effects posited by the failure model. In response to results that revealed a significant genetic contribution to each of the study variables (and their bivariate associations), I used both traditional (i.e., non-genetically controlled) and MZ twin difference analyses (i.e., genetically controlled) to isolate nonshared environmental effects and to rule out the possibility that the pathways posited by the failure model were a result of unrecognized genetic influences. As hypothesized, traditional analyses supported the failure model. Peer rejection in 1st grade mediated the association between Kindergarten reactive aggression and 4th grade internalizing symptoms. However, when the MZ twin difference method was employed, the effects, both direct and indirect, were no longer significant and the variance explained (i.e., r-squared) was dramatically reduced. Although, I hypothesized that controlling for genetic influences would result in muted longitudinal associations, I did not anticipate a complete absence of statistically significant results.

The results from traditional analyses were consistent with prior studies that support both direct and indirect effects posited by the failure model. In line with prior studies examining direct effects of the model, reactive aggression significantly predicted increases in peer rejection (van Lier & Koot, 2010; Evans & Fite, 2019; Gooren et al., 2011), which in turn predicted internalizing symptoms (Ladd, 2006; Gooren et al., 2011; van Lier & Koot, 2010). The indirect effect of reactive aggression on internalizing symptoms via peer rejection was also significant (Boutin et al., 2020; Panak & Garber, 1992). Had additional genetically-controlled analyses not been conducted, I would have concluded that children who exhibit reactive aggression are apt to experience social
failures like peer rejection, which through the wear and tear, can ultimately contribute to increased internalizing symptoms (Patterson & Capaldi, 1990).

The present study is noteworthy because the analyses were also conducted in a genetically controlled framework. When controlling for genetic influences, the results paint a different picture, one at odds with the traditional analyses I conducted and with results from several other studies that do not control for genetic influences. There was no indirect effect of peer rejection on the association between reactive aggression and internalizing symptoms (e.g., Gooren et al., 2011). Neither were there any direct effects between reactive aggression and peer rejection, between peer rejection and internalizing symptoms, or between reactive aggression and internalizing symptoms (or vice versa).

Although this is the first genetically-controlled study to empirically test the mediation component of failure model, the direct effect findings align with prior genetically-informed longitudinal (Boivin et al., 2013; Kretschmer et al., 2018) and concurrent (Ball et al., 2008; Brendgen et al., 2009, 2011) research. Longitudinal findings from reactive aggression to peer rejection align with results from Boivin et al (2013), which indicated that Kindergarten disruptive behaviors predicted subsequent peer difficulties in 1st grade (i.e., peer rejection and victimization), with the association mainly being accounted for by genetic factors. Longitudinal findings from peer rejection to internalizing symptoms mirror results from an adolescent study suggesting that nearly half of the association between adolescent victimization and adult depressive symptoms was explained by common genetic factors.

Unlike prior studies that examine individual components of the failure model, this is the first full longitudinal mediation that uses a MZ twin difference design to test
whether reactive aggression predicts changes in internalizing symptoms via peer rejection using both singletons and difference scores. In doing so, the study indicates that findings vary dramatically in dependence of whether genetic influences are controlled for or not. Using traditional analyses, the full mediation would suggest that reactive aggression predicts peer rejection, which in turn, contributes to a rise in internalizing symptoms. However, because the findings are not replicated using MZ twin difference scores (when genetic and shared environmental influences are controlled for), the effects are likely due to an environmentally-mediated pleiotropic effect (rGE; Avinum, 2020), a mechanism whereby underlying genetic factors affecting one trait can predispose an individual to experiencing an environment (i.e., rGE), which in turn, can affects a second trait (Avinum, 2020). Genetic influences on reactive aggression (e.g., irritability, emotional overreactivity) may evoke peer rejection (i.e., evocative rGE; Boivin et al., 2013; Brendgen et al., 2011), which in turn, affect the likelihood of experiencing internalizing symptoms (Kretschmer et al., 2018). Although mediated pleiotropy has been discussed in the context of one trait mediating a genetic effect on another trait, it has been postulated that the mechanism can be extended to associations involving gene-environment interplay (Avinum, 2002). Underlying genetic factors associated with a behavioral trait can predispose an individual to a particular environmental experience (e.g., peer rejection), which will ultimately contribute to a rise in a secondary trait. In other words, through shared genetic factors, an environment links two behavioral traits. The current study is one of the first to provide evidence of an environmentally-mediated pleiotropy in a full longitudinal mediation framework (Avinum, 2020). The findings
suggest that environmental mechanisms that give rise to internalizing symptoms through reactive aggression need to be revisited.

The current study also examined direct effects posited by the failure model using a bidirectional framework. Using traditional analyses, the longitudinal effects from reactive aggression to peer rejection and from peer rejection to reactive aggression were each significant at both time lags. However, neither result remained upon controlling for genetic influences. Thus, findings suggest that genetic influences accounted for both effects. Longitudinal findings from reactive aggression to peer rejection are likely the byproduct of an evocative rGE (as previously described). However, the longitudinal association from peer rejection to reactive aggression may be the result of common genetic factors that predict both increases in reactive aggression and peer rejection (Boivin et al., 2013). Conventional analyses also suggested that while peer rejection was associated with subsequent increases in internalizing symptoms at both time lags, internalizing symptoms were not associated with subsequent changes in peer rejection. When using MZ twin difference scores, neither of the two paths were significant. It is noteworthy that while longitudinal findings were observed from peer rejection to internalizing symptoms, the reverse effects were not significant. Higher genetic predispositions to internalizing symptoms did not reflect in subsequent peer rejection. It is possible that genetic factors specific to internalizing symptoms may not evoke maltreatment from the group during the early school years. Rather, genetically-linked characteristics (possibly those also related to reactive aggression) that evoke peer rejection may contribute to subsequent internalizing symptoms. Finally, direct effects from reactive aggression to internalizing symptoms and from internalizing symptoms to
reactive aggression were not found to be significant in traditional analyses or using the MZ twin difference design. Although reactive aggression and internalizing symptoms are concurrently linked in our study, and others (Card & Little, 2006; Fite et al., 2009), the results suggest that neither is predictive. This is consistent with findings that suggest reactive aggression is linked to the development of internalizing symptoms both directly (Vitaro et al., 2002) and through impoverished peer relations (Morrow et al., 2006).

**Limitations**

The present study is not without limitations. While the sample size (and thereby statistical power) was sufficient to produce significant findings for conventional analyses, the power limitations prevented the examination of dual mediators. Although Patterson and Capaldi’s (1990) dual failure model posits that both social and academic failures mediate the association between aggression and internalizing symptoms, given a limited sample size, the current study focused on testing the indirect effect of peer rejection. I opted to focus on peer rejection given its central theoretical role. In addition, available assessments on children’s academic functioning (like those of reactive aggression and internalizing symptoms) were based on teacher reports. Results obtained from this data would have therefore led to biases, given inflated shared-reporter bias (Brendgen et al., 2012). Future studies, however, should examine the role of academic functioning, particularly when taking into consideration the influence of genetic factors. Although academic functioning has been previously linked to aggression and social competence (Chen et al., 2010), no study has yet controlled for possible rGE in examining the indirect effects of academic functioning in associations between aggression and internalizing symptoms. In addition, although this study examines the indirect effect of peer rejection,
it is far from the only social difficulty that children face. Children’s unique perceptions of their relationships with teachers, parents, and siblings may play an important role in contributing to internalizing symptoms (Morrow et al., 2006). There are also concerns regarding the generalizability of the findings given the sample used. The study assessed a homogenous French-Canadian population of twins, whose results may not generalize to more diverse populations. More importantly, longitudinal paths were tested using twins, whose social experiences may differ from those of singletons. Although evidence suggests that twins are not systemically different from non-twins in terms of their behaviors or development (Barnes & Boutswell, 2014), having a co-twin may provide unique socialization experiences, potentially protecting children against peer difficulties (Hodges et al., 1999). Students had different teachers and some different classmates each year, which may play a role in the stability of teacher-reported variables (e.g., reactive aggression, internalizing symptoms). The characteristics of informants can influence reports, which may result in lower stability coefficients and cross-lagged effects (Bierg-Nielson et al., 2011). While the current findings indicate a high degree of stability in traditional analyses, stability coefficients for genetically-controlled analyses were weak (and some non-significant). It is possible that the extended amount of time elapsed between the second and third time period contributed to poor stability as decreased effects in the nonshared environmental component (Burt et al., 2010). Time lags that are too long may cause the estimated effects to disappear (Taris et al., 2014).

Importantly, null findings should be interpreted with caution, particularly when comparing singleton scores with those obtained from MZ twin difference analysis. The latter results use a highly conservative design that methodologically removes familial and
heritable influences from the associations. Thus, resulting discordant scores can have little variance, compromising statistical power and potentially muting otherwise significant findings. While a Levene’s test of equality of variance suggested that the variable variances were equivalent (with one exception) when using traditional analyses that using singletons and those using MZ twin difference scores, providing a certain degree of confidence to these findings, future studies should use larger sample sizes in order to optimize the amount of variance available for analyses (Vitaro et al., 2012).

**Implications**

The findings have important implications for researchers. The present study provides evidence suggesting lack of empirical support for the failure model. Although the model is highly influential (Evans & Fite, 2019), no study to date had tested its purported adjustment paths using a genetically controlled framework. The removal of genetic influences bears significance because doing so revealed that there were no predictive paths from reactive aggression to internalizing symptoms via peer rejection. Had the current study not controlled for possible genetic influences (including rGE and pleiotropic effects), the results would have supported the failure model. However, these conclusions would have been erroneous, given no effect of reactive aggression on internalizing symptoms via peer rejection was found when using MZ twin difference scores (i.e., after controlling for genetic and shared environmental influences on reactive aggression, peer rejection and internalizing symptoms).

Together, the findings suggest that the genetic underpinnings behind the failure model are responsible for apparent longitudinal causal pathways from reactive aggression to internalizing symptoms. In other words, reactive aggression does not constitute a
unique risk to the development of internalizing symptoms via peer rejection. Instead, underlying genetic factors are responsible for the direct and indirect effects postulated by the model. Through an environmentally-mediated pleiotropy, \((r_{GET})\), heritable characteristics associated with reactive aggression evoke peer rejection, which ultimately contributes to a rise in internalizing symptoms.

Although not central to my study, the findings also provide insight onto the genetic (and environmental) underpinnings of reactive aggression. While a growing body of literature suggests that reactive aggression can be explained by genetic and environmental factors, this is the first study to find that some of the same genetic factors that contribute to reactive aggression also contribute to those in peer rejection and internalizing symptoms. Temperamental characteristics such as emotional negativity or sensitivity to stressors may predispose children to both forms of maladjustment (Vitaro et al., 2002).

Conclusions

The present study applied both traditional and genetically-controlled research designs to empirically test the adjustment pathways posited by the failure model in a sample of twins followed from Kindergarten to grade 4. Despite traditional analyses finding evidence that peer rejection mediates the association between reactive aggression and internalizing symptoms, genetically controlled analyses did not yield such findings. The environmental contingencies posited by the failure model appear to be an artifact of common genetic influences and gene-environment interplay, providing a powerful alternative explanation for the assumption that nonshared environmental experiences are responsible for the early development of internalizing symptoms.
### Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>(M (SD))</th>
</tr>
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<tr>
<td>1 Reactive Aggression</td>
<td>--</td>
<td>.54**</td>
<td>.33**</td>
<td>.20**</td>
<td>0.40 (0.40)</td>
</tr>
<tr>
<td>2 Proactive Aggression</td>
<td>.39**</td>
<td>--</td>
<td>.17**</td>
<td>-.01</td>
<td>0.27 (0.28)</td>
</tr>
<tr>
<td>3 Peer Rejection</td>
<td>.03</td>
<td>.04</td>
<td>--</td>
<td>.07</td>
<td>-0.20 (0.65)</td>
</tr>
<tr>
<td>4 Internalizing Symptoms</td>
<td>.20**</td>
<td>.06</td>
<td>.02</td>
<td>--</td>
<td>0.57 (0.35)</td>
</tr>
</tbody>
</table>

| \(M (SD)\) | <0.01 (0.36) | -0.01 (0.29) | -0.01 (0.77) | 0.03 (0.41) |

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*Concurrent Correlations, Means and Standard Deviations: Kindergarten*

*Note. N=257 randomly selected MZ twin singletons (singleton analyses) or N=257 twin pairs (MZ twin difference score analyses). Results for singletons are presented above the diagonal. Results for MZ twin difference scores are presented below the diagonal. Reactive aggression, proactive aggression, and internalizing symptoms were standardized by zygosity and sex for correlation analyses; means and standard deviations describe raw scores. Reactive Aggression, proactive aggression and internalizing symptoms were rated on a scale from 0 (*never or not true*) to 2 (*often or very true*). Peer rejection scores represent nominations received from classmates. Rejection scores were standardized by sex, zygosity and class size for correlation analyses; means and standard deviations represent scores standardized by class size. Rejection scores ranged from -1.85 to 2.68. \(p < .05\), \(**p < .001\), two-tailed.*
Table 2

Concurrent Correlations, Means and Standard Deviations: Grade 1

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>M (SD)</th>
</tr>
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<tr>
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<td>--</td>
<td>.17**</td>
<td>-.01</td>
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</tr>
<tr>
<td>3. Peer Rejection</td>
<td>.03</td>
<td>.04</td>
<td>--</td>
<td>.07</td>
<td>-0.20 (0.65)</td>
</tr>
<tr>
<td>4. Internalizing Symptoms</td>
<td>.20**</td>
<td>.06</td>
<td>.02</td>
<td>--</td>
<td>0.57 (0.35)</td>
</tr>
<tr>
<td>M (SD)</td>
<td>&lt;0.01 (0.36)</td>
<td>-0.01 (0.29)</td>
<td>-0.01 (0.77)</td>
<td>0.03 (0.41)</td>
<td></td>
</tr>
</tbody>
</table>

Note. N=257 randomly selected MZ twin singletons (singleton analyses) or N=257 twin pairs (MZ twin difference score analyses).

Results for singletons are presented above the diagonal. Results for MZ twin difference scores are presented below the diagonal.

Reactive aggression, proactive aggression, and internalizing symptoms were standardized by zygosity and sex for correlation analyses; means and standard deviations describe raw scores. Reactive Aggression, proactive aggression and internalizing symptoms were rated on a scale from 0 (never or not true) to 2 (Often or very true). Peer rejection scores represent nominations received from classmates. Rejection scores were standardized by sex, zygosity and class size for correlation analyses; means and standard deviations represent scores standardized by class size. Rejection scores ranged from -2.04 to 2.88. \( p < .05 \), \( **p < .001 \), two-tailed.
Table 3

Concurrent Correlations, Means and Standard Deviations: Grade 4

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Reactive Aggression</td>
<td>--</td>
<td>.28**</td>
<td>.34**</td>
<td>0.40 (0.43)</td>
</tr>
<tr>
<td>2. Peer Rejection</td>
<td>.07</td>
<td>--</td>
<td>.18**</td>
<td>-0.04 (0.77)</td>
</tr>
<tr>
<td>3. Internalizing Symptoms</td>
<td>.22**</td>
<td>-.02</td>
<td>--</td>
<td>0.56 (0.34)</td>
</tr>
</tbody>
</table>

M (SD) 0.01 (0.38) 0.01 (0.81) <-0.01 (0.35)

Note. N=257 randomly selected MZ twin singletons (singleton analyses) or N=257 twin pairs (MZ twin difference score analyses). Results for singletons are presented above the diagonal. Results for MZ twin difference scores are presented below the diagonal. Reactive aggression, proactive aggression, and internalizing symptoms were standardized by zygosity and sex for correlation analyses; means and standard deviations describe raw scores. Reactive Aggression, proactive aggression and internalizing symptoms were rated on a scale from 0 (*never or not true*) to 2 (*Often or very true*). Peer rejection scores represent nominations received from classmates. Rejection scores were standardized by sex, zygosity and class size for correlation analyses; means and standard deviations represent scores standardized by class size. Rejection scores ranged from -1.31 to 3.54. \( p < .05, **p < .001, \) two-tailed.
Table 4. Longitudinal Correlations

<table>
<thead>
<tr>
<th></th>
<th>Reactive Aggression</th>
<th>Peer Rejection</th>
<th>Internalizing Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grade 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade K Reactive Aggression</td>
<td>.40**/.15**</td>
<td>.24**/.05</td>
<td>.17**/.13*</td>
</tr>
<tr>
<td>Grade K Peer Rejection</td>
<td>.32**/.07</td>
<td>.44**/.12</td>
<td>.19**/.08</td>
</tr>
<tr>
<td>Grade K Internalizing Symptoms</td>
<td>.15**/.02</td>
<td>.08/.02</td>
<td>.33**/.15*</td>
</tr>
<tr>
<td><strong>Grade 4</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 1 Reactive Aggression</td>
<td>.45**/.19**</td>
<td>.24**/.04</td>
<td>.24**/.05</td>
</tr>
<tr>
<td>Grade 1 Peer Rejection</td>
<td>.33**/.03</td>
<td>.30**/.08</td>
<td>.30**/.08</td>
</tr>
<tr>
<td>Grade 1 Internalizing Symptoms</td>
<td>.10/.02</td>
<td>.04/.06</td>
<td>.31**/.02</td>
</tr>
</tbody>
</table>

*Note. N=257 randomly selected MZ twin singletons (singleton analyses) or N=257 twin pairs (MZ twin difference score analyses). Correlations between singletons are presented to the left of the slash Correlations between MZ twin difference scores are presented to the right of the slash. All study variables are standardized by zygosity and sex. \( p < .05 \), **\( p < .001 \), two-tailed.*
Table 5. Model Fit and Variance Explained: Results from Kindergarten Univariate ACE and AE Models

<table>
<thead>
<tr>
<th></th>
<th>Grade K Reactive Aggression</th>
<th>Grade K Peer Rejection</th>
<th>Grade K Internalizing Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%a²</td>
<td>%c²</td>
<td>%e²</td>
</tr>
<tr>
<td>ACE</td>
<td>53.6**</td>
<td>3.5</td>
<td>42.9**</td>
</tr>
<tr>
<td></td>
<td>(.32, .76)</td>
<td>(.15, .22)</td>
<td>(.35, .50)</td>
</tr>
<tr>
<td>AE</td>
<td>57.6**</td>
<td>--</td>
<td>42.4**</td>
</tr>
<tr>
<td></td>
<td>(.51, .65)</td>
<td>--</td>
<td>(.35, .49)</td>
</tr>
<tr>
<td></td>
<td>33.6**</td>
<td>9.8</td>
<td>56.6**</td>
</tr>
<tr>
<td></td>
<td>(.09, .59)</td>
<td>(.10, .29)</td>
<td>(.47, .66)</td>
</tr>
<tr>
<td>AE</td>
<td>45.4**</td>
<td>--</td>
<td>54.6**</td>
</tr>
<tr>
<td></td>
<td>(.37, .55)</td>
<td>--</td>
<td>(.47, .63)</td>
</tr>
<tr>
<td></td>
<td>20.7</td>
<td>11.9</td>
<td>67.5**</td>
</tr>
<tr>
<td></td>
<td>(-.07, .48)</td>
<td>(-.09, .32)</td>
<td>(.57, .78)</td>
</tr>
<tr>
<td>AE</td>
<td>35.3**</td>
<td>--</td>
<td>64.7**</td>
</tr>
<tr>
<td></td>
<td>(.26, .44)</td>
<td>--</td>
<td>(.56, .74)</td>
</tr>
</tbody>
</table>

Note. N=682 twin pairs (nMZ= 257 twin pairs, nDZ= 425 twin pairs). Confidence intervals for parameter estimates are given in parentheses. %a² = additive genetic effects. %c² = shared environmental effects. %e² = nonshared environmental effects. Best-fitting models are bolded. p < .05, **p < .001, two-tailed.
### Table 6. Model Fit and Variance Explained: Results from Grade 1 Univariate ACE and AE Model

<table>
<thead>
<tr>
<th></th>
<th>%a^2</th>
<th>%c^2</th>
<th>%e^2</th>
<th>χ^2</th>
<th>df</th>
<th>p</th>
<th>Δχ^2</th>
<th>Δdf</th>
<th>Δp</th>
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<tbody>
<tr>
<td><strong>Grade 1 Reactive Aggression</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>52.2**</td>
<td>0.9</td>
<td>47.8**</td>
<td>1.39</td>
<td>6</td>
<td>0.97</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.44, .60)</td>
<td>(.00, .00)</td>
<td>(.40, .56)</td>
<td></td>
<td></td>
<td></td>
<td>(.40, .56)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE</td>
<td>52.2**</td>
<td>--</td>
<td>47.8**</td>
<td><strong>1.39</strong></td>
<td>7</td>
<td><strong>0.99</strong></td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>(.44, .60)</td>
<td>--</td>
<td>(.37, .56)</td>
<td></td>
<td></td>
<td></td>
<td>(.37, .56)</td>
<td></td>
<td></td>
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<tr>
<td><strong>Grade 1 Peer Rejection</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>56.5**</td>
<td>0</td>
<td>43.5**</td>
<td>2.54</td>
<td>6</td>
<td>0.86</td>
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<tr>
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<td>(.49, .63)</td>
<td>(.00, .00)</td>
<td>(.36, .51)</td>
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<td></td>
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<td>(.36, .51)</td>
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<tr>
<td>AE</td>
<td>56.5**</td>
<td>--</td>
<td><strong>43.5</strong></td>
<td><strong>2.54</strong></td>
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<td></td>
<td>(.49, .64)</td>
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<td>(.36, .51)</td>
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<td></td>
<td></td>
<td>(.36, .51)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Grade 1 Internalizing Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>ACE</td>
<td>45.7**</td>
<td>0</td>
<td>54.3**</td>
<td>1.52</td>
<td>6</td>
<td>0.96</td>
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<td>(.37, .53)</td>
<td>(.00, .00)</td>
<td>(.43, .63)</td>
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<td>(.43, .63)</td>
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<tr>
<td>AE</td>
<td>45.7**</td>
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<td><strong>54.3</strong></td>
<td><strong>1.52</strong></td>
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<tr>
<td></td>
<td>(.35, .54)</td>
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<td>(.46, .63)</td>
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<td>(.46, .63)</td>
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</tbody>
</table>

**Note.** N=682 twin pairs (nMZ= 257 twin pairs, nDZ= 425 twin pairs). Confidence intervals for parameter estimates are given in parentheses. %a^2 = additive genetic effects. %c^2 = shared environmental effects. %e^2 = nonshared environmental effects. Best-fitting models are bolded. p < .05, **p < .001, two-tailed.
Table 7. Model Fit and Variance Explained: Results from Grade 4 Univariate ACE and AE Model.

<table>
<thead>
<tr>
<th></th>
<th>%a²</th>
<th>%c²</th>
<th>%e²</th>
<th>χ²</th>
<th>df</th>
<th>p</th>
<th>Δχ²</th>
<th>Δdf</th>
<th>Δp</th>
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<tr>
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<td>55.8**</td>
<td>0</td>
<td>44.2**</td>
<td>0.76</td>
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<td>0.99</td>
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<td>(.37, .52)</td>
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<td>AE</td>
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<td>--</td>
<td>44.2**</td>
<td>0.76</td>
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<td>Grade 4 Peer Rejection</td>
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<td>58.7**</td>
<td>0.27</td>
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<td>58.7**</td>
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<tr>
<td>ACE</td>
<td>46.6**</td>
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<td>53.4**</td>
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<td>0.94</td>
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</tr>
</tbody>
</table>

Note. N=682 twin pairs (nMZ= 257 twin pairs, nDZ= 425 twin pairs). Confidence intervals for parameter estimates are given in parentheses. %a² = additive genetic effects. %c² = shared environmental effects. %e² = nonshared environmental effects. The most parsimonious models with the lowest Akaike’s Information Criterion (AIC) were bolded. p < .05, **p < .001, two-tailed.
**Figure 1**

*Univariate ACE Measurement Model.*

Note: Parameters A, C, and E refer to additive genetic, shared environmental, and nonshared environmental factors, respectively. Separate models were conducted for reactive aggression, rejection, and internalizing problems, at three time points (kindergarten, Grade 1, Grade 4).
Note. The model partitions variance into (1) common latent factors $A_C$, $C_C$, and $E_C$, that influence both phenotypes (study variables X and Y), and (2) unique latent factors $A_{UY}$, $C_{UY}$, and $E_{UY}$, specific to the Y variable. Separate models were conducted for associations between (1) rejection and reactive aggression (2) rejection and internalizing symptoms, and (3) reactive aggression and internalizing symptoms. Separate models were conducted at three time points: Kindergarten, Grade 1, Grade 4. Coefficients $A_{CX}$, $C_{CX}$, and $E_{CX}$ represent the factor loadings of study variable X on the common latent factors $A_C$, $C_C$, and $E_C$, respectively. Coefficients $A_{CY}$, $C_{CY}$, and $E_{CY}$ represent the factor loadings of study variable Y on the common latent factor loadings $A_C$, $C_C$, and $E_C$, respectively. Coefficients $A_{UY}$, $C_{UY}$, and $E_{UY}$ represent the factor loadings of study variable Y on the unique latent factors $A_{UY}$, $C_{UY}$, and $E_{UY}$, respectively.
Longitudinal Associations between Peer Rejection and (a) Reactive Aggression and (b) Internalizing Symptoms: Measurement Model.

Note. Separate analyses were run for singletons and MZ twin difference scores.
Figure 4

Longitudinal Association from Kindergarten Reactive Aggression to Grade 4 Internalizing Symptoms, Mediated by Grade 1 Rejection:

Measurement Model

Note. Separate analyses were run for singletons and MZ twin difference scores.
Figure 5

*Common Genetic and Environmental Influences on the Association between Reactive Aggression and Rejection in Kindergarten*

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses revealed no statistically significant shared environmental effect for reactive aggression or rejection, so the C component of reactive aggression (C_C), the C component of rejection (C_UY), and C overlap (C_CY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 22.43, p=.32, CFI = .99, RMSEA = .02$. *p < .05, **p < .001, two-tailed.*
Common Genetic and Environmental Influences on the Association between Reactive Aggression and Rejection in Grade 1

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Grade 1 reactive aggression or rejection, so the C component of reactive aggression (C_C), the C component of rejection (C_UY), and C overlap (C_CY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: \( \chi^2(20) = 12.72 \), p=.89, CFI = 1.00, RMSEA = .00. *p < .05, **p < .001, two-tailed.
Figure 7

Common Genetic and Environmental Influences on the Association between Reactive Aggression and Rejection in Grade 4

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Grade 4 reactive aggression or rejection, so the C component of reactive aggression (C_C), the C component of rejection (C_UY), and C overlap (C_CY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 6.99$, $p=.99$, CFI = 1.00, RMSEA = .00. *$p < .05$, **$p < .001$, two-tailed.
Note. N=682 twin pairs (nMZ= 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Kindergarten internalizing symptoms or rejection, so the C component of internalizing symptoms (Cc), the C component of rejection (C_UY), and C overlap (C_CY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: χ²(20)= 25.09, p=.20, CFI = .97, RMSEA = .03. *p < .05, **p < .001, two-tailed.
Figure 9

Common Genetic and Environmental Influences on the Association between Internalizing Symptoms and Rejection in Grade 1

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Grade 1 internalizing symptoms or rejection, so the C component of internalizing symptoms (C_C), the C component of rejection (C_UY), and C overlap (C_CY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 6.64, p=1.00$, CFI = 1.00, RMSEA = .00. *$p < .05$, **$p < .001$, two-tailed.
Figure 10

Common Genetic and Environmental Influences on the Association between Internalizing Symptoms and Rejection in Grade 4

Note: $N=682$ twin pairs ($n_{MZ}=257$ twin pairs, $n_{DZ}=425$ twin pairs). Univariate analyses found no statistically significant shared environmental effect on Grade 4 internalizing symptoms or rejection, so the C component of internalizing symptoms ($C_C$), the C component of rejection ($C_{UY}$), and C overlap ($C_{CY}$) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 14.62, p=1.00, CFI = 1.00, RMSEA = .00$. *$p < .05$, **$p < .001$, two-tailed.
Figure 11

Common Genetic and Environmental Influences on the Association between Reactive Aggression and Internalizing Symptoms in Kindergarten

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Kindergarten reactive aggression or internalizing symptoms, so the C component of reactive aggression (C_C), the C component of internalizing symptoms (C_UY), and C overlap (C_CY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 3.72$, $p=1.00$, CFI = 1.00, RMSEA = .00. *$p < .05$, **$p < .001$, two-tailed.
Figure 12

*Common Genetic and Environmental Influences on the Association between Reactive Aggression and Internalizing Symptoms in Grade 1*

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Grade 1 reactive aggression or internalizing symptoms, so the C component of reactive aggression (CC), the C component of internalizing symptoms (CUY), and C overlap (CCY) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 8.23, p = 1.00$, CFI = 1.00, RMSEA = .00. *p < .05, **p < .001, two-tailed.
Figure 13

*Common Genetic and Environmental Influences on the Association between Reactive Aggression and Internalizing Symptoms in Grade 4*

Note: N=682 twin pairs (nMZ = 257 twin pairs, nDZ = 425 twin pairs). Univariate analyses found no statistically significant shared environmental effect on Grade 4 reactive aggression or internalizing symptoms, so the C component of reactive aggression (C), the C component of internalizing symptoms (C_{UY}), and C overlap (C_{CY}) parameters were fixed to zero, as denoted by the dotted lines. Model fit was adequate: $\chi^2(20) = 22.96$, $p = .29$, CFI = .99, RMSEA = .02. *$p < .05$, **$p < .001$, two-tailed.
Figure 14

Longitudinal Associations between Peer Rejection and (a) Reactive Aggression and (b) Internalizing Symptoms: Singleton Results

Note: N=257 randomly selected members of MZ twin dyads. PPP= .58 (−23.33, 17.30). Cross-lagged paths between reactive aggression and internalizing symptoms (across both Kindergarten to Grade 1 and Grade 1 to Grade 4 time-lags) were not statistically significant; they were included in the model but are not depicted in the figure. Concurrent associations between variables at Grade 1 were included in the model but are not depicted. *p < .05, **p < .001, two-tailed.
Figure 15. Longitudinal Associations between Peer Rejection and (a) Reactive Aggression and (b) Internalizing Symptoms: MZ Twin Difference Results

Note: N=257 MZ twin dyads. PPP = .32 (-15.29, 42.26). Cross-lagged paths between reactive aggression and internalizing symptoms (across both Kindergarten to Grade 1 and Grade 1 to Grade 4 time-lags) were not statistically significant; they were included in the model but are not depicted in the figure. *p < .05, **p < .001, two-tailed.
Figure 16. Longitudinal Association from Kindergarten Reactive Aggression to Grade 4 Internalizing Symptoms, Mediated by Grade 1 Peer Rejection: Singleton Results

Note. N=257 randomly selected members of MZ dyads. PPP = .58 (-23.33, 17.30). Cross-lagged paths between reactive aggression and internalizing symptoms (across both Kindergarten to Grade 1 and Grade 1 to Grade 4 time-lags) were not statistically significant; they were included in the model but are not depicted in the figure. The dotted line represents the indirect effect of K reactive aggression on Grade 4 internalizing problems through Grade 1 peer rejection. *p < .05, **p < .001, two-tailed.
Figure 17. Reciprocated and Mediated Associations between Reactive Aggression, Peer Rejection, and Internalizing Symptoms:
Results from the MZ Twin Difference Model

Note: N=257 randomly selected members of MZ dyads. PPP = .27(-15.09, 34.67) Cross-lagged paths between reactive aggression and internalizing symptoms (across both Kindergarten to Grade 1 and Grade 1 to Grade 4 time-lags) were not statistically significant; they were included in the model but are not depicted in the figure. The dotted line represents the indirect effect of K reactive aggression to Grade 4 internalizing problems through Grade 1 peer rejection. *p < .05, **p < .001, two-tailed.
Appendix A

*Teacher Ratings of Reactive Aggression* (Coie & Dodge, 1987)

Within the past 6 months, how many times would you say that the child:

1. …reacted in an aggressive manner when teased?

2. …when somebody accidentally hurt him/her (such as bumping into him/her), he/she reacted with anger and fighting?

3. …reacted in an aggressive manner when something was taken away from him/her?

4. …reacted in an aggressive manner when contradicted?

Scale:
1 = Never or not true
2 = Sometimes or a little true
3 = Often or very true
Appendix B

*Teacher Ratings of Proactive Aggression* (Coie & Dodge, 1987)

Within the past 6 months, how many times would you say that the child:

1. …tries to dominate others
2. …encourages others to pick on another child
3. …threatens others to get what he or she wants

Scale:

1 = *Never or not true*
2 = *Sometimes or a little true*
3 = *Often or very true*
Appendix C

*Emotional Disorder Scale of the Ontario Child Health Study* (Offord, Boyle, & Racine, 1989)

Within the past 6 months, how many times would you say that the child:

1. …seemed to be unhappy or sad?
2. …was not as happy as other children?
3. …had no energy, was feeling tired?
4. … had trouble enjoying him/herself?

Scale:
1 = *Never or not true*
2 = *Sometimes or a little true*
3 = *Often or very true*
Appendix D

Social Behavior Questionnaire Tremblay et al., 1991

Within the past 6 months, how many times would you say that the child:

1. …worried about many things?
2. …tended to be fearful or afraid of new things or new situations?
3. …cried easily?

Scale:
1 = Never or not true
2 = Sometimes or a little true
3 = Often or very true
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