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Genetic and Environmental Influences of Bullying Involvement: A Longitudinal Twin Study

Ellyn Dunbar
Virginia Commonwealth University

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A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

by

Ellyn Dunbar, B.S.
The University of Southern Mississippi, 2016

Director: Judy Silberg, Ph.D.
Associate Professor, Departments of Human and Molecular Genetics, and Psychiatry

Virginia Commonwealth University
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List of Abbreviations

-2LL	Maximum Likelihood Ratio
A	Additive Genetics (Standardized)
a	Additive Genetic Path Coefficient
ADHD	Attention Deficit Hyperactivity Disorder
AIC	Akaike's Information Criteria
ASPD	Antisocial Personality Disorder
Bullying involvement	Refers to Bully victimization, Bullies, Bully Victims
C	Common/Shared Environment (Standardized)
c	Common Environment Path Coefficient
CAPA-C/P	Child and Adolescent Psychiatric Assessment (Child/Parent)
CD	Conduct Disorder
CI	Confidence Interval
CMH	Cochran-Mantel-Haenszel
D	Dominance Genetics (Standardized)
d	Dominance Path Coefficient
df	Degrees of Freedom
DZ	Dizygotic Twins
DZF	Female DZ twins
DZM	Male DZ twins
DZO	Opposite Sex DZ twins
E	Unique Environment (Standardized)
e	Unique Environment Path Coefficient
EEA	Equal Environments Assumption
eq	Equal

MDD	Major Depressive Disorder
MZ	Monozygotic Twins
MZF	Female MZ twins
MZM	Male MZ twins
ne	Not Equal
OAD	Overanxious Disorder/Anxious Affect
ODD	Oppositional Defiant Disorder
OR	Odds Ratios
rDZ	DZ Correlation/Covariance
rMZ	MZ Correlation/Covariance
SAD	Separation Anxiety
SEM	Structural Equation Model
SOC ANX	Social Anxiety
VTSABD	Virginia Twin Study of Adolescent Behavioral Development
YAFU	Young Adult Follow-Up

Abstract

GENETIC AND ENVIRONMENTAL INFLUENCES OF BULLYING INVOLVEMENT: A LONGITUDINAL TWIN STUDY

By Ellyn Dunbar, B.S.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

Virginia Commonwealth University, 2018

Major Director: Judy Silberg, Ph.D., Associate Professor, Departments of Human and Molecular Genetics, and Psychiatry

Introduction—Bullying involvement is associated with many long-term adverse outcomes. Bullied children are at risk for internalizing disorders including anxiety, depression and suicidal behavior in childhood and adulthood. Bullies are also at risk for psychiatric disorders, specifically externalizing disorders. Bully victims—children who are both bullied and bullies—have a particularly poor prognosis, with a higher risk for internalizing and externalizing disorders. The purpose of this study is to study the epidemiology, risk of psychiatric disorders, and genetic and environmental influences of being bullied, a bully, and a bully victim—in the sample and individually in males and females.

Methods—Twins (N=2,844, aged 8-17) from the Virginia Twin Study of Adolescent Behavioral Development and the Young Adult Follow-Up were used to study bullying involvement. Child and mother responses from three waves of data collection were used to determine bullying involvement status and to diagnose internalizing and externalizing disorders. The epidemiology

of bullying involvement was examined. The odds ratios (OR) of being involved in bullying and having a psychiatric disorder were calculated. The twin methodology was used to estimate the genetic and environmental influences of bullying involvement.

Results—In the sample, 14.56% were bullied, 17.33% were bullies, and 10.69% were bully victims. Males are more often involved in bullying, but females are more severely affected by their involvement. Bullied children are at a higher risk for internalizing disorders, especially young adult depression (OR 1.29). Bullies are at a higher risk for externalizing disorders, and depression (OR 1.72). Bully victims are at a higher risk for nearly every disorder tested. Bullying involvement is heritable, and being bullied has a dominance genetic component. The heritability of being bullied, a bully, and a bully victim is 48.12%, 54.81%, and 62.62% respectively.

Conclusion—Individuals involved in bullying are at risk for serious and long-lasting psychiatric disorders. Interventions need to be developed that target each category of bullying involvement, and the specific disorders that these children are at risk for, while keeping in mind that their involvement is heritable.

1 Introduction

The saying "sticks and stones may break my bones, but words can never hurt me," may not be true as once believed—bullying can have adverse lifelong consequences. Bully victimization is defined as repetitive victimizing behavior with an imbalance of power between the victim and the bully (Olweus, 1993). Bullying is a world wide epidemic with approximately 33% of all children being bullied (Lereya et al. 2015). Thomas et al. used surveys and interviews of 2,967 children and 6,310 guardians to find a prevalence of victimization of 13.3% in adolescents in Australia (2017). Romo and Kelvin found a rate of 37.8% in Latin America based on survey data from 14,560 students from five countries (2016). Scandinavian countries have a prevalence of 5-20% (Törn et al. 2014). It is estimated that between 9 and 50% of children in the United States have been bullied (Ramirez et al. 2016).

Adolescence is a vulnerable developmental time when children are exposed to more sources of victimization other than bullying (Fisher et al. 2015). It is also during these years that children are at higher risk of developing symptoms of suicidality, depression, and anxiety, which can be exacerbated in the presence of bully victimization (Arango et al. 2016). Unfortunately, childhood and adolescence is also when most bullying occurs (Cosma et al. 2017).

It has repeatedly been shown that victims of bullying are at a higher risk for a plethora of mental health issues, especially internalizing disorders, than children not involved in bullying (Arseneault, 2017). Internalizing issues refers to one turning stress inwards resulting in anxiety and mood disorders (Kelly et al. 2015). Arseneault reviewed the results of three longitudinal cohorts from Finland, the US, and the UK that studied the effects of bullying. Bullied children have higher rates of suicidality, panic disorder, agoraphobia, depression, anxiety, and psychiatric

hospital treatments. All three cohorts controlled for preexisting mental disorders, inferring a cause and effect relationship between bullying and new mental disorders (2017).

The consequences of bullying can persist into adulthood, and extend from mental health concerns to employment issues (Lereya et al. 2015). Copeland et al. found that childhood bully victimization increases the risk of anxiety in adulthood (2013). Klomek, Sourander, and Elonheimo reviewed the results of several cohorts that assessed the adulthood outcomes of childhood bullying (2015). Common findings among the studies were the prevalence of depression and anxiety in victims. However, suicidality in adulthood was not found in every cohort, and some cohorts found that girls were at higher risk of suicidality than boys (2015).

Takizawa, Maughan, and Arseneault (2014) studied the effects of childhood bully victimization in mid-adulthood. The researchers used 7,771 participants from the British National Child Development Study. Participants were born during one week in 1958, and follow up interviews were conducted at ages 7, 11, 16, 23, 33, 42, 45, and 50 years. This study used data collected from interviews at ages 7, 11, 23, 45, and 50. Data on psychological distress, depressive and anxiety disorders, suicidality, and general health were collected in adulthood. Takizawa and colleagues found that children who were bullied had poorer health in adulthood (2014). Not only were the young adults at risk for psychological disorders, but the 50 year olds were at risk as well. At age 45, individuals who were frequently bullied had higher rates of depression, anxiety, and suicidality. Additionally, the frequently bullied children were less likely to obtain higher education, and the men were less likely to be employed. Takizawa emphasizes the need for bullying prevention, as the consequences are life long (2014). It is possible that bully victimization and adult outcomes are not causally related but could reflect an underlying liability to psychiatric disorders in adulthood that is expressed as an increased risk of being

bullied in childhood.

Bullying negatively affects both genders; however, boys and girls do tend to respond to bullying exposure differently. Many studies find greater lasting effects in bullied females (Klomek, Sourander, and Elonheimo, 2015). Arango and colleagues found that females experienced suicidal ideation in response to being bullied more than males. However, males were more severely affected by verbal bullying than females (2016). Additionally, girls are more likely to express their feelings and seek treatment before boys (Alavi et al. 2015).

Monozygotic and dizygotic twins have been used to study the genetic and environmental influences on bully victimization. Twin studies show that both genetic and environmental factors influence bully victimization; however, the extent of influence of each depends on the context in which bullying is studied. For example, Ball et al. used a univariate twin design to study bully victimization, bullying, and bully victims (2008). Twins were drawn from the Environmental Risk Longitudinal Twin Study in England and Wales. Bully victimization in this study was found to be 73% heritable (Ball et al. 2008). However, Fisher et al. studied victimization exposure in adolescents using the same data set as Ball et al. and found that bully victimization was only 34% heritable (2015, 2008). The difference between the two studies was that Ball et al. used data collected on bully victimization by asking mothers if their twins experienced bullying between the ages of 5 and 10; alternatively, Fisher et al. asked the twins (at age 18) directly about their bullying experiences between the ages of 12 and 18 (2008; 2015).

Another discrepancy in heritability arises when looking at bully victimization in association with mental health issues. Törn et al. studied the connection of neurodevelopmental issues in childhood and bully victimization later in life to find that bully victimization is 67% heritable (2015). Alternatively, Shakoor et al. studied bully victimization in adolescence as a risk

factor for experiencing psychotic experiences later in life, and found only a 35% heritability of bully victimization (2014). It can be concluded that bully victimization can be considered both a genetic risk factor and an environmental risk factor depending on the context of the study.

Victims are not the only children at risk; the bullies themselves and the children who are both bullies and victims (bully victims) are also affected (Kelly et al. 2015). As opposed to bullied children who are at a higher risk of internalizing disorders, bullies tend to experience more externalizing disorders. Externalizing disorders occur when stress manifests outwardly, such as conduct disorder and ADHD (Kelly et al. 2015). Similarly to bullied individuals, bullies are also at risk for depression, anxiety, and suicidality (Ball et al. 2008; Holt et al. 2015). Unique to bullies is an increased risk of antisocial personality disorder (Copeland et al. 2013; Klomek, Sourander, and Elonheimo, 2015).

Bully victims are a very vulnerable group. These children are at a higher risk of both internalizing and externalizing disorders than individuals not involved in bullying and those who are only bullied or bullies (Ball et al. 2008; Kelly et al. 2015). Co-occurrence of internalizing and externalizing disorders increases the severity of outcomes, including suicidality (Kelly et al. 2015; Klomek, Sourander, and Elonheimo, 2015).

Boys are significantly more likely to bully others and be bully victims than girls (Copeland et al. 2013; Ball et al. 2008). Data on gender specific risk of psychiatric disorders in bullies and bully victims is limited. Although, Copeland et al. did find that female bully victims were at a greater risk for agoraphobia and that male bully victims were at a greater risk for suicidality (2013).

Most of the existing literature on bullying involvement focuses on the victims, who are very important. However, the bullies and the bully victims are also at risk. The aim of this paper

is to study the epidemiology and effects of genes and environment on bullying involvement. We will use data from the Virginia Twin Study of Adolescent Behavioral Development (VTSABD) and the Young Adult Follow-Up (YAFU) and twin methodology.

1.1 Sources of Variation

All human characteristics show variation between individuals. Generally, variation in traits can be divided into two major categories of influence: genetic and environmental. The genetic influence on variation is referred to as heritability, and is further divided into two sub-categories: additive and non-additive. Environmental influences on variation can be categorized as common and unique (Neale and Cardon, 1992).

1.2 Twin Methods

The study of monozygotic (MZ) and dizygotic (DZ) twins is a powerful approach for estimating genetic and environmental influences on trait variation. Because MZ twins are genetically identical and share the same common environment (parents, home, etc.) and DZ twins share half of their genes and a common environment, the comparison of MZ and DZ twins is used to estimate the influence of genetics, shared environment, and unique environment on the variation observed in a particular trait, e.g., being bullied by peers. For example, if the DZ correlation for bully victimization is one half of the MZ correlation, the behavior is heritable. Conversely, if the DZ correlation for being bullied is equal to or greater than half the MZ correlation, some aspect(s) of their common environment causes the DZ twins to be more similar to each other than can be accounted for by their shared genes alone.

Additive genetic factors (A) refer to the cumulative effect of the alleles. Non-additive (dominance and epistasis) genetic effects are the deviations from the additive effects. Dominance and epistasis effects are usually estimated together as dominance effects (D). The common or

shared environment (C) is the environment shared by individuals in a family, and makes individuals in a family more similar. Conversely, the unique environment (E) influences each twin separately. Common environment and dominance effects are confounded—they cannot be estimated at the same time. Collectively the sources of variation of a trait are estimated using ACE (or ADE) models described later (Neale and Cardon, 1992).

Twin studies can be used to study a single trait (univariate), or twin studies can also be used to study the causes of association between multiple traits (multivariate).

1.3 Statistics Used for the Twin Method

1.3.1 Tetrachoric Correlation

Most of the variables used in twin calculations in this study are binary, meaning they are a "yes/no" or "present/absent" response coded as a 1 and 0 respectively. The data can then be described in contingency tables such as Table 1. Each cell of the contingency table contains the number of individuals with the corresponding responses. As long as the data has a bivariate normal distribution, a tetrachoric correlation can be used to describe the correlation between the traits (Neale and Cardon, 1992).

Table 1 Example of a Contingency Table

		Response 1	
		Yes	No
Response 2	Yes	100	50
	No	50	100

1.3.2 Structural Equation Modeling

Twin data can be fit to a structural equation model (SEM). SEM allows us to fit linear equations, making it possible to estimate parameters from the variances and covariances of

variables.

The diagram used in SEM is called a path diagram, and the model is fit by path analysis. The relationship between variables is determined by the path coefficients. Tracing rules define the equations used to calculate the relationships (Neale and Cardon, 1992).

In the diagram, arrows, squares, and circles are used to identify different components and relationships of the model. Squares are observed or measured variables, while circles are latent or unmeasured variables. Single-headed arrows are drawn between variables to represent a causal relationship and are called paths. Double-headed arrows represent covariance between variables. Double-headed arrows drawn from a variable back to itself represent the variance of the variable. Often the variance of the latent variables is standardized to one. When labeling the diagram, lowercase letters are used for path coefficients (single-headed arrows) and correlation coefficients (double-headed arrows). Uppercase letters are used for variables (Neale and Cardon, 1992).

The tracing rules of the path diagram allow the predicted variances and covariances between variables to be calculated. The basic tracing rules are: trace backwards, change directions, trace forwards. This means follow a single-headed arrow backwards from one variable to another, change directions over a two-headed arrow, and then follow a single-headed arrow to the final variable. The (co)variance is calculated by multiplying all the path coefficients along the trace together, and if there are multiple chains between variables, summing the products of these chains (Neale and Cardon, 1992).

1.4 Twin Model

The classic twin model is one of the most powerful tools for studying genetic and environmental effects on the variation of a trait. In the path diagram, the additive genetic factors

and shared and unique environmental effects are specified as latent variables.

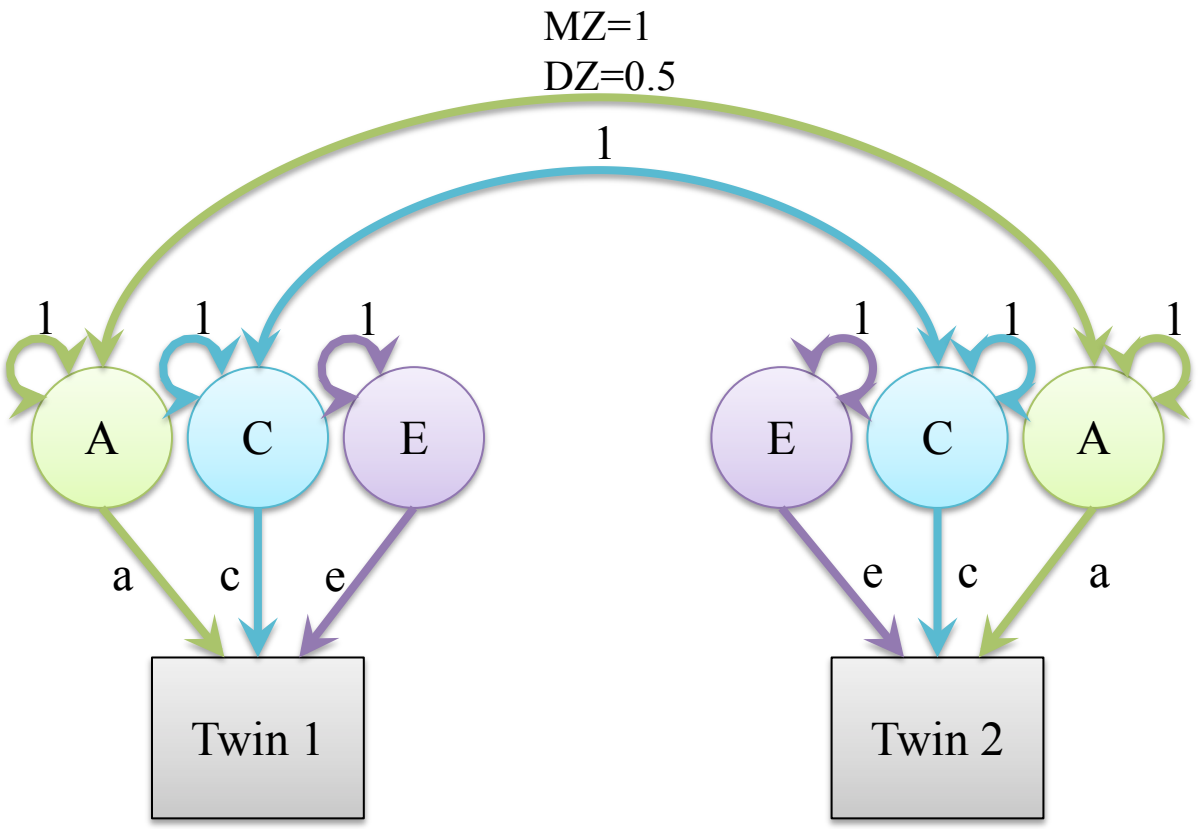

Figure 1 ACE Model. The green path denotes additive genetics, blue is common environment, and purple is unique environment.

To calculate the predicted MZ covariance (rMZ), we trace all the paths that connect twin 1 and twin 2 and sum them. Since only a and c (the green and blue paths in Figure 1) connect the twins, the MZ covariance is:

$$rMZ = a^2 + c^2 \quad (1)$$

Following the same rules, the DZ covariance (rDZ) is:

$$rDZ = 0.5a^2 + c^2 \quad (2)$$

To calculate the variance (V) of the trait—assuming that the variance in twin 1 is equal to the variance in twin 2—trace all the paths from twin 1 to the latent variables and back.

$$V = a^2 + c^2 + e^2 \quad (3)$$

Given Equations 1-3, we can estimate the parameters using the observed MZ and DZ variances

and covariances. Any differences between MZ twins should be caused by unique environmental effects and will be reflected by the difference of rMZ from unity—in the standardized case—as seen in Equation 4. Equation 5 is an estimate of the heritability (h^2) of the trait. These calculations only give rough estimates; however, fitting the model gives better estimates of the parameters.

$$V - rMZ = 1 - rMZ = e^2 \quad (4)$$

$$2(rMZ - rDZ) = a^2 = h^2 \quad (5)$$

$$rMZ - a^2 = c^2 \quad (6)$$

The variance of each component of the model is calculated by tracing from twin 1 to that component and back.

$$VA = a^2 \quad (7)$$

$$VC = c^2 \quad (8)$$

$$VE = e^2 \quad (9)$$

The components are standardized by dividing each variance component by the total variance (Neale and Cardon, 1992). Standardized components will be reported in this paper.

$$A = \frac{VA}{V} \quad (10)$$

$$C = \frac{VC}{V} \quad (11)$$

$$E = \frac{VE}{V} \quad (12)$$

1.4.1 The Best-Fit Model

After fitting the twin data to the full ACE model, parameters are "dropped" to test their significance in the model using goodness of fit statistics. A maximum likelihood value (-2LL) is generated for each model, and compared to that of the ACE model by taking the difference. The difference in likelihood of two models is distributed as a chi-squared (χ^2). The degrees of freedom (df) of the χ^2 test is the difference between the df of the sub-model and the df of the full

model. The sub-model is significant if the χ^2 value is greater than the critical value at that df and if the associated p value is lower than 0.05. However, when fitting models, a significant likelihood ratio test means that the sub-model is significantly different or fits significantly worse than the full model. Therefore, the best-fit model is identified by a non-significant χ^2, which means that the sub-model is not significantly different than the full model and is more parsimonious by using fewer variables to explain the variance. Additionally, the Akaike's Information Criteria (AIC) fit index is also used to help identify the best-fit model. The most negative AIC indicates the better-fitting model. This is helpful when two sub-models are both non-significant (Neale and Cardon, 1992).

2 Methods

2.1 Virginia Twin Study of Adolescent Behavioral Development and Young Adult Follow-Up

The Virginia Twin Study of Adolescent Behavioral Development (VTSABD) is a longitudinal, developmentally informed genetic study of Caucasian twin families residing in Virginia. A total of 1,442 MZ and DZ twin pairs between the ages of 8 and 17 were included in the study. Psychiatric and environmental information was collected across three waves of study from twins remaining under the age of 17 (Silberg et al. 2016; Hewitt et al. 1997; Meyer et al. 1996).

Table 2 VTSABD Stats by Wave

	Wave1	Wave2	Wave3
Males*	1337	1005	741
Females	1547	1101	795
Age Range	8-17	9-17	12-17
MZ**	749	562	452
MZM	328	254	205
MZF	421	308	247
DZ	687	489	377
DZM	186	139	116
DZF	198	131	103
DZO	303	219	158

*Gender is by individual twins

**Zygosity is by twin pair

In the Young Adult Follow-Up (YAFU) 2,307 individuals (1,079 complete twin pairs, 82% cooperation rate) from the original VTSABD sample were assessed as young adults. Twins in the YAFU were between ages 18 and 25 (Silberg et al. 2016).

2.2 Data Collection

2.2.1 VTSABD

Data was collected from parents and twins using the Child and Adolescent Psychiatric Assessment (CAPA) (Angold and Costello, 2000). The CAPA records details about relevant psychiatric and behavioral symptoms based upon DSM-III criteria. Assessed in the CAPA was suicidal behavior and self-harm, depression, separation anxiety, anxious affect and worries, and social anxiety. Also included was an assessment of oppositional and conduct disorders, attention deficient hyperactivity disorder (ADHD), food related behaviors, school behavior and performance, sleep problems, and substance use. Environmental measures included family structure, peer relationships, social and personal functioning, and pubertal stage. Individuals were asked if they experienced symptoms within the three months prior to the interview. The CAPA was designed to accommodate criteria for diagnosis found in the DSM-III-R and can also be applied to the DSM-IV.

Box 1: Psychiatric Disorders Studied in the VTSABD
• Suicidal Ideation and Behavior • Depression (MDD) • Social anxiety (SOC ANX) • Separation anxiety (SAD) • Anxious affect (OAD) • Oppositional (ODD) and conduct disorders (CD) • Attention Deficit Hyperactivity Disorder (ADHD)

The CAPA-C was administered to each twin, and the primary parent was administered the CAPA-P regarding each twin. The parent interviews also included information on child ADHD, early health of the twins, and zygosity. Interviews were conducted in the families' homes, with different interviewers interviewing each twin simultaneously in different parts of the home.

In addition to the CAPA, the families were also given self-report questionnaires. These questionnaires covered a variety of topics including the Rutter Scale Parent Questionnaire and Teacher Questionnaire, which collected information on children bullying others from the parents and teachers (Hewitt et al. 1997).

2.2.2 The Young Adult Follow-Up

The twins from the original VTSABD sample were re-assessed using telephone interviews to diagnose common psychiatric disorders. The Young Adult Follow-Up used the Structured Clinical Interview based on DSM-III-R criteria for diagnosis. Disorders included in the YAFU were generalized anxiety, antisocial personality disorder, substance use, panic attacks, depression, and suicidal ideation (Silberg et al. 2016).

2.3 Soft and Hardware

Statistical analysis was conducted on a MacBook Pro (2017) running macOS Sierra. Software used included SAS® University Edition (Base 3.7) and R version 3.3.1 (2016-06-21) using the OpenMx package.

2.4 Organization of Data

Epidemiological calculations were based on individual twins. These calculations required each twin to have their own row. An example of the basic epidemiological organization scheme is shown in Table 3.

Table 3 Organization of Epidemiological Data

Family	Twin Number	Variable
1	1	1
1	2	0

In order to calculate the twin correlations, the data had to be organized as such that each twin pair was on a single row in the table. Variables were labeled to distinguish between twin 1 and twin 2. Converting the data from twin pairs to individuals (or vice versa) involved sub-setting the data into twin 1 and twin 2, renaming variables of interest, and merging the sets back together. An example of the structure of twin data used for twin correlations and modeling is in Table 4.

Table 4 Organization of Twin Data

Family	Twin 1 Variable	Twin 2 Variable
1	1	0
2	0	1

2.5 Definition of Variables

The variables for bullying involvement and psychiatric disorders are all binary. These binary variables were coded such that "0" means that the specific trait is "absent" and "1" means

"present." Categorical variables mentioned in code later are defined in Table 5.

Table 5 Categorical Variables

Variable	Coding	
Sex	1	Male
	2	Female
Zygosity	1	MZ
	2	DZ
Zyg	1	MZ males (MZM)
	2	MZ females (MZF)
	3	DZ males (DZM)
	4	DZ females (DZF)
	5	Opposite sex (DZO)
famno	De-identified Family Number	
Wave	Wave of data collection	
twid	1	Twin 1
	2	Twin 2

2.5.1 Bullying Involvement

2.5.1.1 The Bullied

In the CAPA being bullied is defined as "Subject is a *particular object* of mockery or physical attacks or threats by peers." Additional probes used by the interviewer to determine if the child was bullied are listed in Box 2. Reports from the child and the mother were used to identify bullied children. Individuals included in the calculations were restricted to those who

were only involved in bully victimization; those who were also bullies (called bully victims) were separated into their own category to control for confounding effects (Sourander et al. 2007).

Box 2: Probes of Bully Victimization in CAPA

• Do you get teased or bullied at all?
  o Is it friendly teasing, or mean teasing?
  o Is that more than other children?
• Are other boys and girls mean to you?
  o How much?
  o Tell me about the last time.
  o Who does it?
  o Why do they do it?
  o Why do they pick on you?
  o What do you do about it?

2.5.1.2 The Bully

The CAPA defines bullying behavior as "Mocking, taunting behavior of others with the intention of hurting their feelings or frightening them, but without threats of physical violence…Does include minor pushing or shoving." The Rutter Scales asked if a child bullies other children. Parents' and teachers' responses to the Rutter Scales were used to determine if the child was a bully. Again, individuals who are bully victims were not included in these calculations.

2.5.1.3 The Bully Victims

Bully victims are those individuals who are bullied and also bully others. This variable was coded as a "1" if the child was both bullied and a bully. Bully victims were removed from the bullied and bully categories.

2.5.2 Psychiatric Disorders

2.5.2.1 Depression: Childhood and Young adult

Depression or major depressive disorder (MDD) is characterized by "a period of at least 2

weeks during which there is either depressed mood or the loss of interest or pleasure in nearly all activities" (American Psychiatric Association (APA), 2013). Depressed mood can manifest as irritability in children and adolescents (APA, 2013). Depression was considered to be diagnosed if 5 out of 9 symptoms listed in Box 3 were present in 2 or more activities within the last 3 months during the CAPA interviews and when the depressed mood was at its worst since turning 18 for the YAFU.

Box 3: Symptoms of MDD
• Depressed Mood (can be irritability in children/adolescents) • Subjective Agitation • Loss of interest/Anhedonia • Feels unloved/self-depreciation and self-hatred • Subjective anergia • Subjective motor slowing • Subjective complaints about thoughts • Significant weight change • Suicidal ideation

2.5.2.2 Social Anxiety

Social anxiety (SOC ANX) in the CAPA is defined as "Subjective Anxious Affect specific to social interactions. There is desire for involvement with familiar people. Can include peers and/or adults." The main probe used by the interviewer was, "Do you ever get really 'nervous' or 'frightened' when you have to talk to new people?" Social anxiety was considered diagnosable if it was present in two or more activities within the last three months.

2.5.2.3 Separation Anxiety

The CAPA defines separation anxiety (labeled SAD) as "Excessive worries or fear concerning separation from the persons [parental figure(s)] to whom the affected child is attached. Do not include co-twin." Nine symptoms were assessed (see Box 4), and separation anxiety was considered diagnosed if three or more of these symptoms were present in two or

more activities within the last three months.

Box 4: Symptoms of Separation Anxiety
• Worries/Anxiety about possible harm
• Worries/Anxiety about calamitous separation
• Reluctance to sleep alone
• Avoidance of sleeping away from family
• Separation Dreams
• Avoidance of being alone
• Anticipatory distress
• Withdrawal when attachment figure absent
• Actual distress when attachment figure absent

2.5.2.4 Overanxious Disorder

Overanxious disorder (OAD) is related to general anxiety. Anxiety is the "anticipation of future threat." (APA, 2013) Symptoms include excessive worries, need for reassurance, and nervous tension. Four out of seven symptoms had to have occurred in more than two activities within the last three months and be uncontrollable to be considered diagnostic.

2.5.2.5 Oppositional Defiant Disorder

Oppositional defiant disorder (ODD) "is a frequent and persistent pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness" (APA, 2013). Oppositional defiant disorder was considered diagnosable if 5 or more of the 9 symptoms (see Box 5) were present in at least 2 activities and subject to admonition by an authority figure in the last 3 months.

Box 5: Symptoms of ODD
• Strained Parental Relationships
• Strained Teacher Relationships
• Rule Breaking
• Disobedience
• Annoying Behavior
• Angry or Resentful
• Bullying
• Spiteful or Vindictive
• Swearing

2.5.2.6 Conduct Disorder

Conduct disorder (CD) is characterized by "repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated" (APA, 2013). Conduct disorder was diagnosed if 3 of the 12 symptoms listed in Box 6 were present in the last 3 months.

Box 6: Symptoms of Conduct Disorder
• Stealing • Lying • Running Away from Home • Conduct Problems involving Violence • Staying out Late • Fire setting • Violence against persons • Assault and Cruelty • Police Contact • Delinquency • Tobacco Use • Alcohol Use

2.5.2.7 Attention Deficit Hyperactivity Disorder

Attention Deficit Hyperactivity Disorder (ADHD) is characterized by "a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development" (APA, 2013). Symptoms of ADHD were collected from parents during the CAPA-P interview process. Three main symptoms were used for the diagnosis of ADHD: over/hyper-activity, inattention, and impulsivity. The presence of at least two of these symptoms is diagnostic. Some of the prompts given to parents during the CAPA-P are listed in Box 7.

Box 7: Symptoms and Probes of ADHD

• **Over activity**
  o Fidgetiness
  o How much does s/he squirm or wiggle in his/her seat?
  o How much does s/he fidget with his/her hands or feet?
  o Difficulty remaining seated when required
  o Can s/he usually remain in his/her seat when s/he's supposed to?
  o Does s/he get up much more than other children (young people)?
• **Inattention**
  o Difficulty concentrating on tasks requiring sustained attention.
  o Is s/he able to concentrate on things s/he has to?
  o Does s/he have more problems concentrating than other children (young people) his/her age?
  o Difficulty following through instructions from others.
  o How good is s/he at following through instructions from others?
  o Does s/he tend to complete things s/he's been asked to do?
• **Impulsivity**
  o Often acts before thinking.
  o Does s/he usually think about things before s/he does them?
  o Or does s/he tend to jump straight in impulsively without thinking about what might happen?
  o Difficulty waiting for turn in games or group situations.
  o Can s/he wait his/her turn for things?
  o As well as most children?

2.5.2.8 Suicidality: Childhood and Young Adult

Childhood suicidality was defined in the CAPA as "Thoughts specifically about killing oneself, by whatever means…Do you ever think about ending it all?"

Young adult suicidality was assessed as part of the section of the YAFU inquiring about times of major depression. Individuals first had to meet the diagnostic criteria for depression before being asked about suicidality. The specific question about suicidality was: "were you thinking a lot about death or about hurting yourself?"

2.5.2.9 Antisocial Personality Disorder

Antisocial personality disorder (ASPD) is a "disregard for, and violation of, the rights of others" (APA, 2013). ASPD is diagnosable after the individual has turned 18, and has exhibited symptoms since the age of 15 (APA, 2013). ASPD was diagnosed using the YAFU. Three or

more of the symptoms of ASPD from the DSM-V listed in Box 8 are needed for a diagnosis (APA, 2013).

Box 8: Symptoms of ASPD
• Failure to respect social norms/lawful behaviors • Deceitfulness • Impulsivity • Irritability/aggressiveness • Reckless disregard for safety of self or others • Irresponsibility • Lack of remorse

2.6 Generation of Variables

In order to include all available information from all waves of data collection, an `if/then` scheme was used. Using `bullied` as an example, responses from each child (identified with a `c`) and mother (identified with a `p`) within each wave were combined into a single variable based on the "`or`" rule (SAS, 2017). The following is a portion of the code used to generate a single `bullied` variable from wave 1 of the VTSABD.

```
data wave1; merge wave1c wave1p; by famno twid;
if w1c_bullied eq 1 or w1p_bullied eq 1 then w1_bullied = 1;
if w1c_bullied eq 0 and w1p_bullied eq 0 then w1_bullied = 0;
if w1c_bullied eq . and w1p_bullied ne . then w1_bullied =
w1p_bullied;
if w1c_bullied ne . and w1p_bullied eq . then w1_bullied =
w1c_bullied;
run;
```

The variables generated at each wave were used for epidemiological calculations of age and gender trends. After mother and child responses (parent and teacher responses for the `bully` variable) from each wave were combined, the variables from each of the waves were combined into `ever` variables. The `ever` variables tracked if that individual had experienced the trait in the 3 months prior to any wave of data collection using the "`or`" rule. The `ever` variables did not reflect the number of times the individual experienced the trait, just its presence across the

waves. These variables were used for whole sample calculations and (after reorganization, see section 2.4) twin correlations. An example of the coding scheme used for the `bullied_ever` variable follows:

```
data bullied; merge wave1 wave2 wave3; by famno twid;
if w1_bullied eq 1 or w2_bullied eq 1 or w3_bullied eq 1 then
bullied_ever = 1;
if w1_bullied eq . and w2_bullied eq . and w3_bullied eq . then
bullied_ever = 2;
if bullied_ever eq . then bullied_ever=0;
if bullied_ever eq 2 then bullied_ever=.;
run;
```

The variables for the psychiatric disorders were previously generated using DSM-III-R guidelines and the same coding schemes listed above (Silberg et al. 2016).

2.7 Epidemiology

The `ever` variables (see section 2.6) based on responses across waves were used to examine the sample trends of bullying involvement (bullied, bullies, and bully victims). The `PROC FREQ` procedure was used to generate odds ratios (OR) and Fisher's exact tests to test the over all significance of gender in bullying involvement (SAS, 2017). The code used to generate these exact tests and odds ratios for bullied boys and girls is bellow.

```
proc freq; tables bullied_ever*sex/chisq relrisk; exact pchi or;
run;
```

The variables generated for each wave were used to describe the epidemiology of bullying involvement in more detail. The age and sex of each twin was attached to the `bullied`, `bully`, and `bully_victim` variables to observe age and gender trends at each age. It should be noted that for this scheme, an individual twin was counted several times but at different ages to obtain the trends. An example of the data organization is shown in Table 6.

Table 6 Organization of Epidemiological Data for Age Trends

Family	Twin	Age	Bullied	Wave	Sex
1	1	9	yes	1	1
1	1	10	no	2	1
1	1	11	yes	3	1
1	2	9	no	1	2
1	2	10	yes	2	2
1	2	11	no	3	2

Once the data was organized correctly, the `PROC FREQ` procedure was used to analyze age and gender trends of bullying involvement. Bellow is the code used to view the rates of being bullied at each age in the sample. The `all` option requests that all statistics associated with the `PROC FREQ` command be printed. In this situation, a chi-squared test was used to test the null hypothesis that bullying and age are independent. If the reported χ^2 value is higher than the critical value for the degrees of freedom (df) of the test and an alpha of 0.05, then the bullying variable is not independent of age.

```
proc freq; tables bullied*age /all;
run;
```

Gender trends were viewed similarly, with the addition of a `by` statement to run separate calculations for each of the genders.

```
proc freq; tables bullied*age; by sex;
run;
```

The chi-squared test, produced by the following code, was used to test for significant differences between boys and girls involved in bullying at each age. The `exact` option requests

exact p values to be calculated (SAS, 2017). Again, if the χ^2 value is greater than the critical value, there is a significant difference between boys and girls being bullied at that specific age.

```
proc freq; tables sex*bullied/chisq exact; by age;
run;
```

2.8 Psychiatric Disorders

Individuals involved in bullying were assessed for an increased risk of co-occurrence of psychiatric disorders in both child and young adulthood. The `ever` variables (see section 2.6) were used in these calculations. In addition to sample risk, the effect of gender on the risk of being involved in bullying and having a psychiatric disorder was also studied. The risk (odds ratios) of being involved in bullying and having a disorder for boys and girls was calculated using the `PROC GENMOD` logistic scheme (SAS, 2017). This procedure controls for the non-independence of twins and fits the data to a generalized linear model. The code used to calculate the risk of depression in bullied children is shown bellow. Significance of the odds ratios was also determined using the `PROC GENMOD` procedure. Addition of a `by sex` statement was used to calculate odds ratios separately for each gender.

```
proc genmod descending; class famno; model bullied_ever=MDD/dist=b
link=logit;
repeated subject=famno/type=ind;
estimate "log O.R. MDD" MDD 1 / exp;
run;
```

A Cochran-Mantel-Haenszel (CMH) statistical test was used to determine if the association between bullying involvement and the psychiatric disorder remained significant after controlling for sex. The following code was used to generate the statistic.

```
proc freq; tables sex*bullied_ever*MDD/chisq cmh;
run;
```

2.9 Twin Correlations

Twin correlations were produced using the `PROC FREQ` procedure to calculate tetrachoric correlations. The sample was sorted by zygosity to calculate MZ and DZ correlations separately. The MZ and DZ correlations for boys and girls were calculated by replacing the `zygosity` variable with the `zyg` variable (see Table 5). The following code was used:

```
proc freq; tables twin1*twin2/plcorr exact; by zygosity;
run;
```

2.10 Model Fitting

ACE and ADE models were fit using OpenMx run in R. Dr. Hermine Maes wrote the script used to fit the binary models (Maes, 2015). The data was exported from SAS as a .csv and then imported into R. Instructions found in the script and online were followed to run the models (The OpenMX Project, 2017). The full AC(D)E model was used as the base model to compare the fit of AE, C(D)E, and E models. A sub-model was considered the best fit if the associated χ^2 and p values, comparing it to the full model were non-significant. A non-significant value meant that the model did not have a significantly worse fit than the full model, and was a more parsimonious explanation of the data. ACE models were fit for the sample of twins involved in bullying and for each gender. ADE models were fit to the sample and each gender of twins involved in bullying when dominance effects were estimated based on the twin correlations.

The models were specified using matrix algebra. A full explanation of the code can be found in the OpenMX documentation at http://openmx.psyc.virginia.edu/docs/OpenMx/latest/GeneticEpi_Matrix.html.

3 Results

3.1 Epidemiology

Data on bullying involvement was available for 2,844 individuals representing 1,419 complete twin pairs. Of these individuals, 1,317 were male and 1,527 were female. The percent of the sample and percent of each gender involved in bullying are listed in Table 7. Using a chi-squared test of association, being bullied (χ^2=4.2285, df=1, p=0.0398) and being a bully (χ^2=11.2089, df=1, p=0.0008) are significantly associated with gender. Fisher's exact tests were used to test the alternative hypothesis that males were more likely to be involved in bullying. Boys were significantly more likely to be bullied (χ^2=4.2285, p=0.0227, OR (95% CI)=1.2433 (1.0100-1.5329)) than girls. Similarly, boys were more likely to be bullies (χ^2=11.20889, p=0.0005, OR (95% CI)=1.3933 (1.1468-1.6927)). Although gender was not associated with bully victims overall, of those who were bully victims more were boys (χ^2=3.4332, p=0.0367, OR (95% CI)=1.2520 (0.9867-1.5885)).

Table 7 Prevalence of Bully Involvement in VTSABD

	Bullied	Bullies	Bully Victims
Sample	14.56%	17.33%	10.69%
Male	16.02%	19.89%	11.85%
Female	13.29%	15.13%	9.69%

3.1.1 The Bullied

3.1.1.1 Age Trends

The frequency of children being bullied at each age in the sample is graphed in Figure 2. Each bar represents the percentage of children bullied out of all of the children at that age in the sample. The highest rates of bully victimization in the VTSABD are found in the 8 and 9 year olds with a frequency of 18.52% and 17.74% respectively. At 9 df and a significance level of 0.05 the critical χ^2 value is 16.919. The χ^2 value of `bullied` by `age` was 112.1815. This value is well above the critical value, suggesting that the frequency of bullied children is dependent on age. Visual observation of the graph suggests that the frequency of being bullied decreases as children age.

3.1.1.2 Gender Effects

The effect of gender on being bullied is graphed in Figure 3 in the same manner as the age trends in Figure 2. There were no significant differences in being bullied in boys and girls at any age.

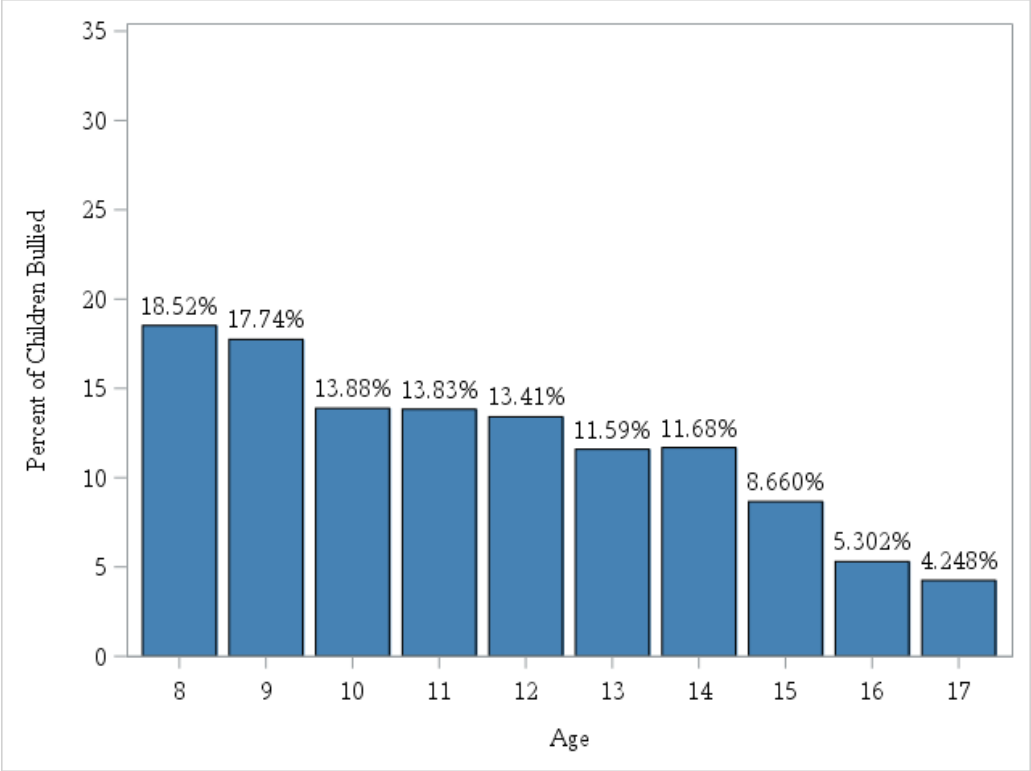

Figure 2 Prevalence of Bullying Across Age. Each bar represents the percentage of children in each age group who were bullied.

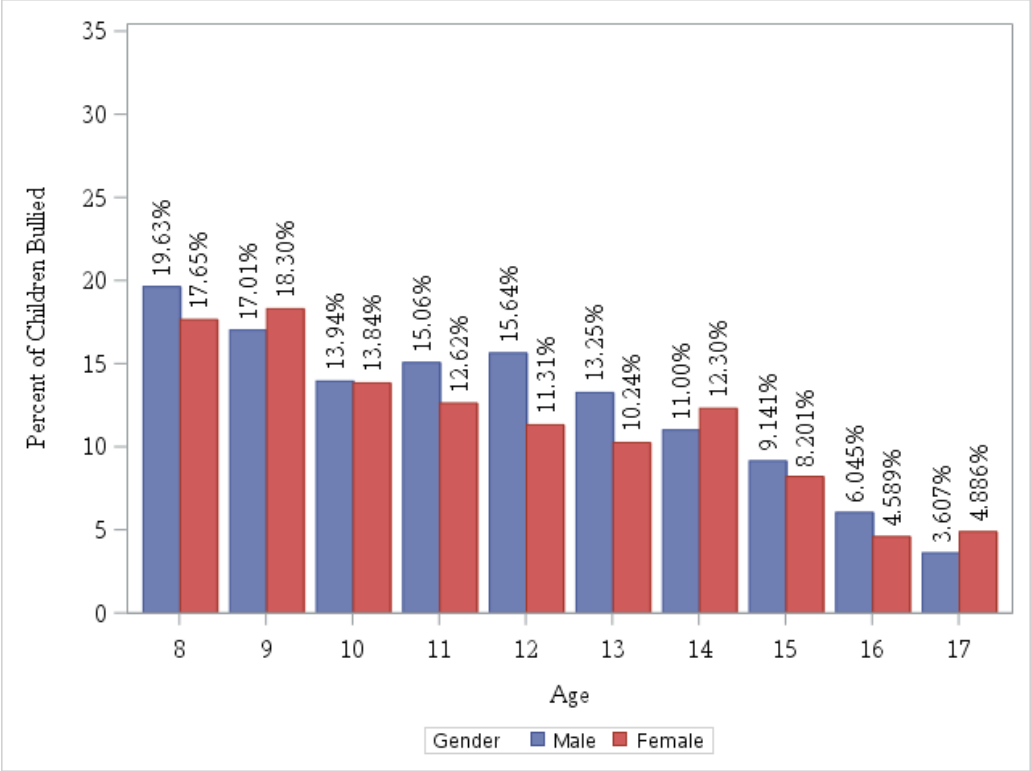

Figure 3 Prevalence of Bullying in Each Gender Across Age. Each bar represents the percentage of children bullied in each age group.

3.1.1.3 Psychiatric Disorders

The potential increased risk of psychiatric disorders in bullied children was assessed using the `PROC GENMOD` logistic scheme. Odds ratios (OR), 95% confidence intervals (CI), and p values for the sample of bullied children as well as male and female bullied children are reported in Table 8. Additionally, the OR and significance are graphed in Figure 4. A significance level of 0.05 was used.

In the sample, bullied children are at a higher risk of social anxiety, separation anxiety, overanxious disorder, ADHD, and suicidal ideation than children who were not bullied. As compared to non-bullied individuals, young adults who were bullied as children are at a higher risk for both depression and suicidality.

3.1.1.4 Psychiatric Disorders by Gender

The OR and CI of each disorder in boys and girls are reported in Table 8. In Figure 5 asterisks mark the disorders in which the risk of being bullied and having that disorder was significant after accounting for gender as tested by the CMH test.

The disorders that all bullied children are at a higher risk for remained significant when considering gender. Bullied boys and girls are both at a higher risk of social anxiety than their non-bullied peers of the same gender. Bullied girls are significantly more at risk for separation anxiety, overanxious disorder, childhood and young adult suicidality, and young adult depression than girls who were not bullied. By the CMH test, being bullied and ADHD remain significantly associated when gender is considered; however, neither bullied boys nor girls are at a significantly higher risk of having ADHD than boys and girls who were not bullied.

Table 8 Odds Ratios of Psychiatric Disorders in Bullied Children. Significant values are bolded.

	Sample		Males		Females	
Disorder	OR(95% CI)	p	OR(95% CI)	p	OR(95% CI)	p
MDD	1.62 (0.96-2.75)	0.07	1.00 (0.38-2.63)	0.99	**2.18 (1.15-4.12)**	0.02
SOC ANX	**2.19 (1.71-2.80)**	<1.0e-4	**2.13 (1.47-3.09)**	<1.0e-4	**2.34 (1.67-3.28)**	<1.0e-4
SAD	**1.94 (1.41-2.67)**	<1.0e-4	1.24 (0.65-2.35)	0.51	**2.59 (1.76-3.80)**	<1.0e-4
OAD	**1.47 (1.17-1.84)**	8.0e-4	1.28 (0.90-1.82)	0.17	**1.75 (1.30-2.37)**	3.0e-4
ODD	1.42 (0.99-2.03)	0.06	1.21 (0.74-2.0)	0.42	1.61 (0.96-2.70)	0.07
CD	1.05 (0.73-1.52)	0.80	0.72 (0.43-1.19)	0.20	1.68 (0.996-2.84)	0.05
ADHD	**1.92 (1.21-3.04)**	5.4e-3	1.68 (0.97-2.91)	0.07	2.24 (0.97-5.16)	0.06
C-SUICIDALITY	**2.54 (1.66-3.01)**	<1.0e-4	1.08 (0.44-2.66)	0.86	**3.83 (2.32-6.30)**	<1.0e-4
Y-MDD	**1.29 (1.01-1.66)**	0.04	1.08 (0.74-1.58)	0.67	**1.55 (1.10-2.18)**	0.01
ASPD	0.81 (0.44-1.48)	0.49	0.85 (0.43-1.70)	0.65	0.52 (0.13-2.14)	0.36
Y-SUICIDALITY	**1.90 (1.28-2.82)**	1.4e-3	1.63 (0.87-3.05)	0.13	**2.16 (1.30-3.60)**	3.0e-3

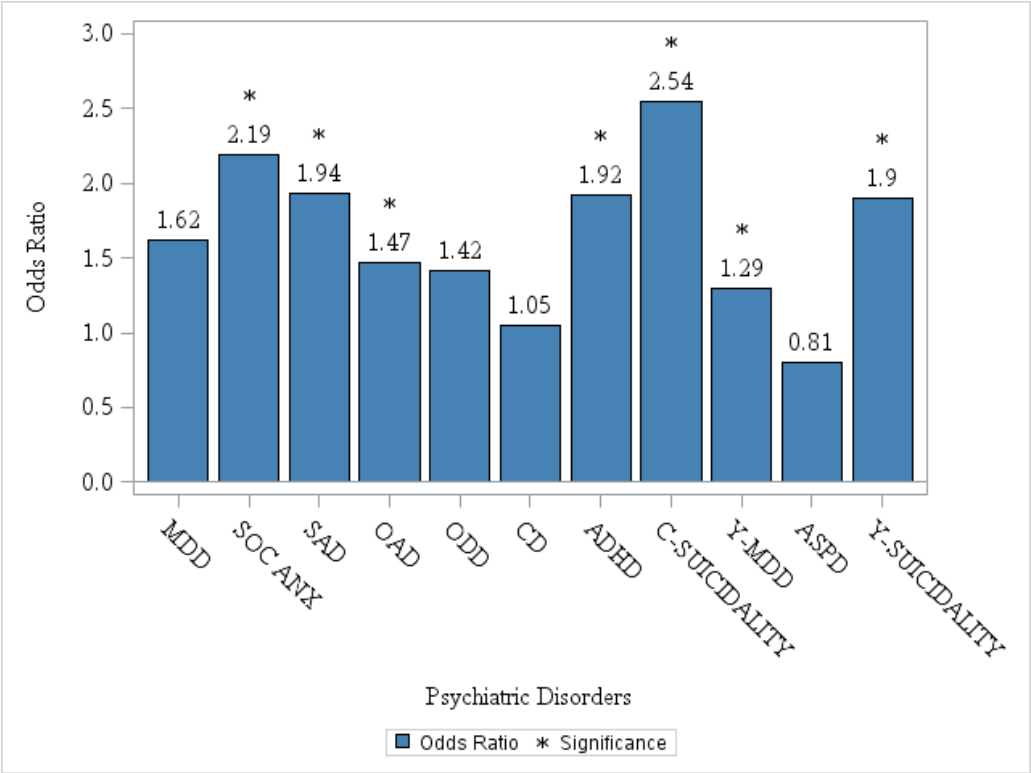

Figure 4 Odds Ratios of Psychiatric Disorders in Bullied Children. Bars with * are disorders significant in bullied children.

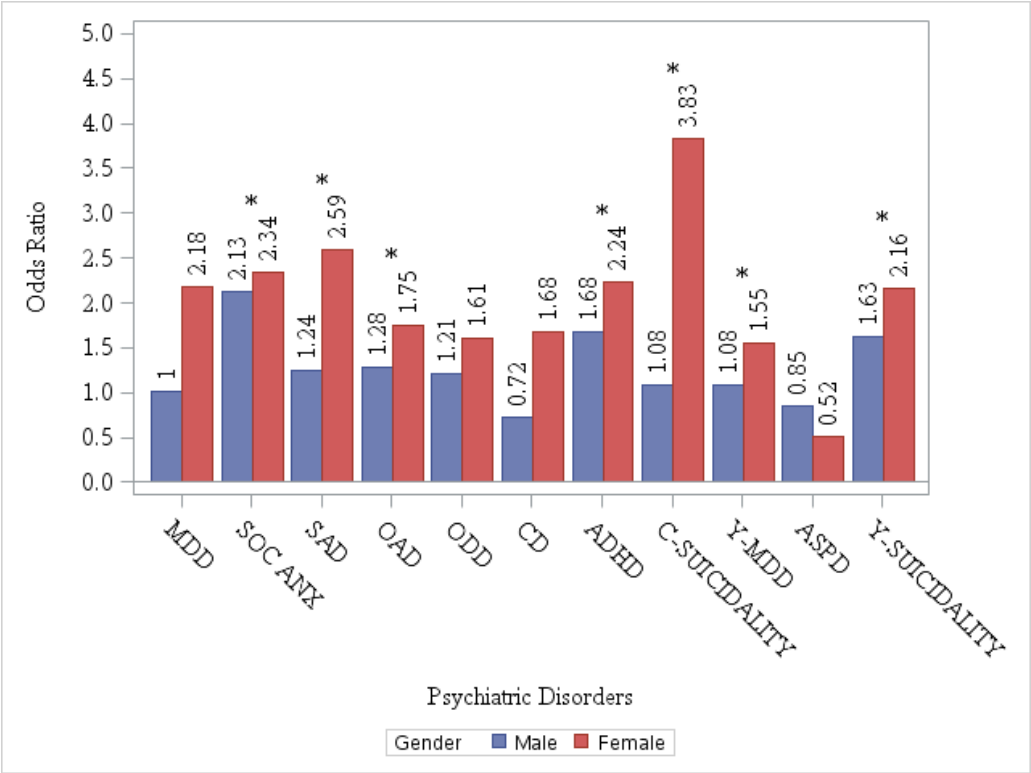

Figure 5 Odds Ratios of Psychiatric Disorders in Bullied Boys and Girls. Disorders with * are significant when controlling for gender (CMH).

3.1.2 The Bully

3.1.2.1 Age Trends

The age trends of bullies are graphed in Figure 6. The age with the highest percentage of bullies is age 10 with a prevalence of 16.70%. The χ^2 value was 45.2433 with 9 df, indicating that the prevalence of bullies is dependent on age. There is a (visual) general decrease in the frequency of bullies as children age past 10.

3.1.2.2 Gender Effects

The prevalence rates and significance (*) are graphed across the age range for boys and girls in Figure 7. Ages with a significant difference in the prevalence of bullies in boys as compared to girls, as determined by a chi-squared test with an alpha at 0.05, are reported in Table 9. At ages 13, 15, and 16 a higher percentage of boys were bullies compared to girls at those ages.

Table 9 Ages with a Significantly Different Prevalence of Bullies in Boys and Girls

Age	χ^2	p value
13	5.4847	0.0192
15	4.9275	0.0264
16	5.3718	0.0205

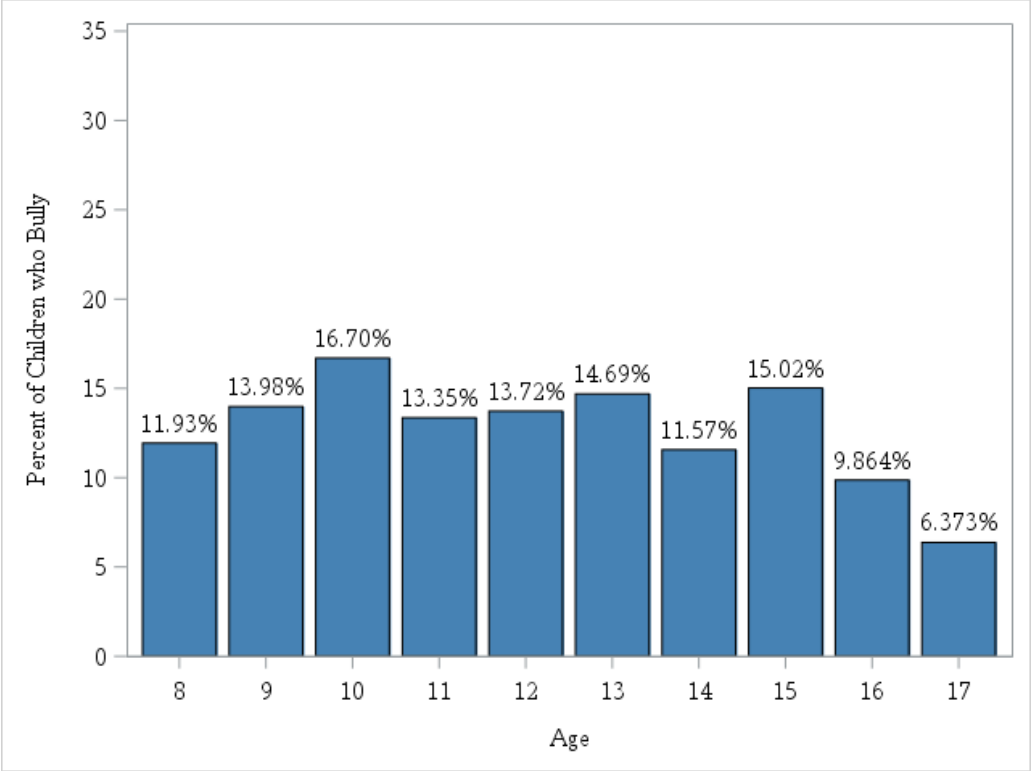

Figure 6 Prevalence of Bullies Across Age. Bars represent the percentage of children in each age group who were bullies.

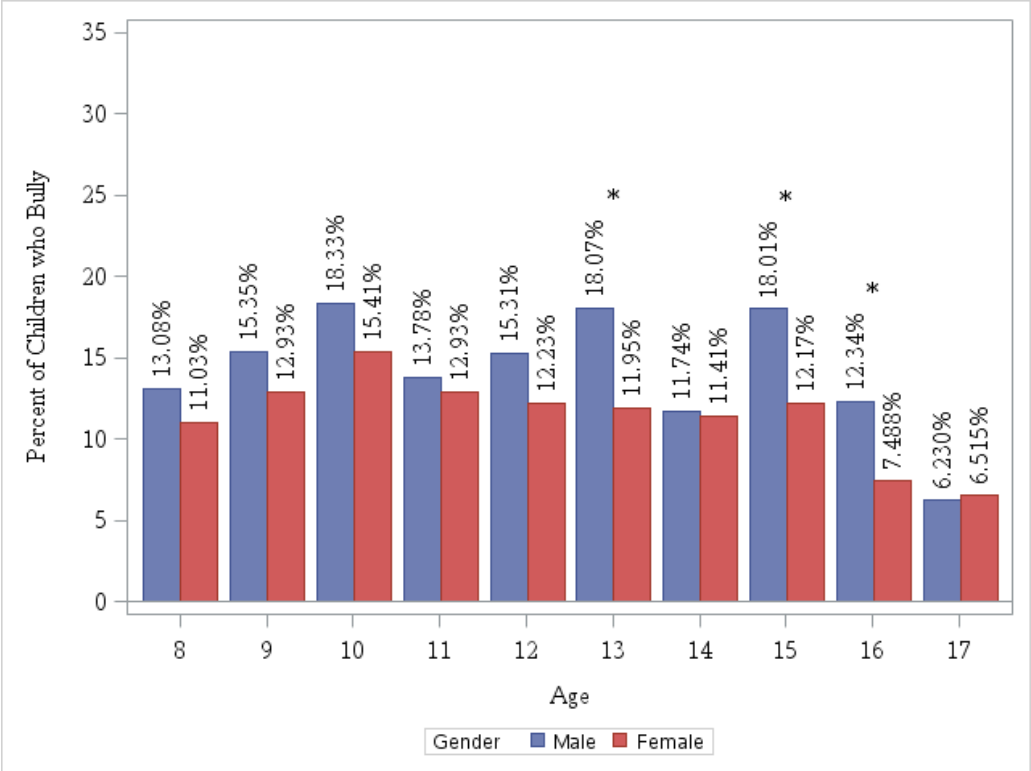

Figure 7 Prevalence of Bullies in Each Gender Across Age. Ages with * have a significant difference between the prevalence of bullies in each gender.

3.1.2.3 Psychiatric Disorders

The potential increased risk of psychiatric disorders in children who were bullies was assessed using the `PROC GENMOD` logistic scheme. Odds ratios, confidence intervals, and p values for the sample of bullies as well as male and female bullies are reported in Table 10. The OR and significance of the sample are graphed in Figure 8.

Children who were bullies are at a significantly increased risk of childhood and young adult depression, social anxiety, oppositional defiant disorder, conduct disorder, and ASPD.

3.1.2.4 Psychiatric Disorders by Gender

The OR and significance of male and female bullies and each disorder are listed in Table 10. After controlling for gender (CMH test), all disorders—except young adult depression—that were significant in the sample remained significantly associated with bullying others. Boy and girl bullies are at a significantly higher risk of ODD and conduct disorder than boys and girls who were not bullies. Females who were bullies have a higher risk of young adult depression than females who were not bullies. Males who were bullies have higher rates of childhood depression and ASPD than males who were not bullies.

Table 10 Odds Ratios of Psychiatric Disorders in Bullies. Significant values are bolded.

	Sample		Males		Females	
Disorder	OR(95% CI)	p	OR(95% CI)	p	OR(95% CI)	p
MDD	**1.72 (1.04-2.85)**	0.03	**2.28 (1.08-4.82)**	0.03	1.49 (0.73-3.06)	0.27
SOC ANX	**0.72 (0.53-0.97)**	0.03	0.68 (0.43-1.09)	0.11	0.79 (0.53-1.17)	0.24
SAD	0.77 (0.53-1.13)	0.18	0.57 (0.29-1.12)	0.10	0.98 (0.62-1.56)	0.94
OAD	0.94 (0.75-1.18)	0.60	1.00 (0.72-1.41)	0.97	0.97 (0.72-1.32)	0.85
ODD	**1.91 (1.36-2.68)**	2.0e-4	**1.93 (1.23-3.02)**	4.2e-3	**1.87 (1.13-3.10)**	0.02
CD	**1.98 (1.44-2.72)**	<1.0e-4	**1.85 (1.25-2.74)**	2.0e-3	**1.95 (1.12-3.4)**	0.02
ADHD	1.43 (0.89-2.30)	0.13	1.54 (0.88-2.67)	0.13	0.93 (0.36-2.46)	0.89
C-SUICIDALITY	0.66 (0.37-1.20)	0.18	0.24 (0.06-1.01)	0.05	1.02 (0.52-1.99)	0.95
Y-MDD	**0.74 (0.57-0.96)**	0.02	1.00 (0.68-1.45)	0.99	**0.62 (0.44-0.88)**	7.8e-3
ASPD	**1.82 (1.13-2.93)**	0.01	**1.95 (1.11-3.41)**	0.02	1.04 (0.39-2.74)	0.94
Y-SUICIDALITY	0.68 (0.42-1.10)	0.12	0.85 (0.42-1.72)	0.64	0.61 (0.31-1.2)	0.15

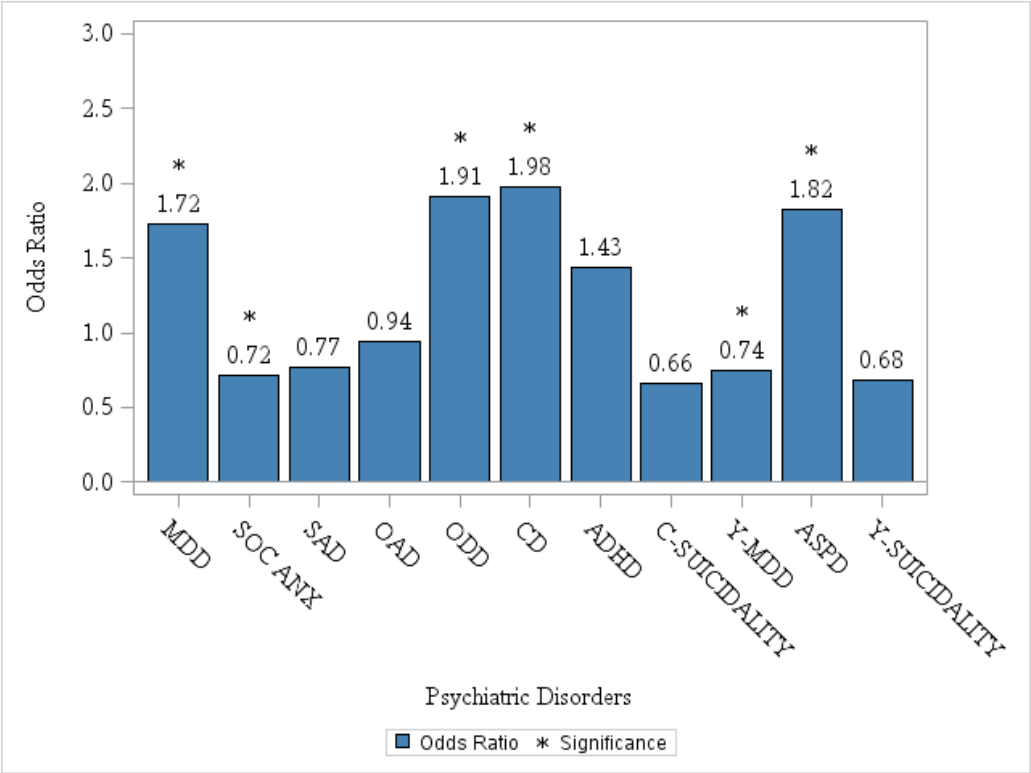

Figure 8 Odds Ratios of Psychiatric Disorders in Bullies. Bars with * are disorders significant in bullies.

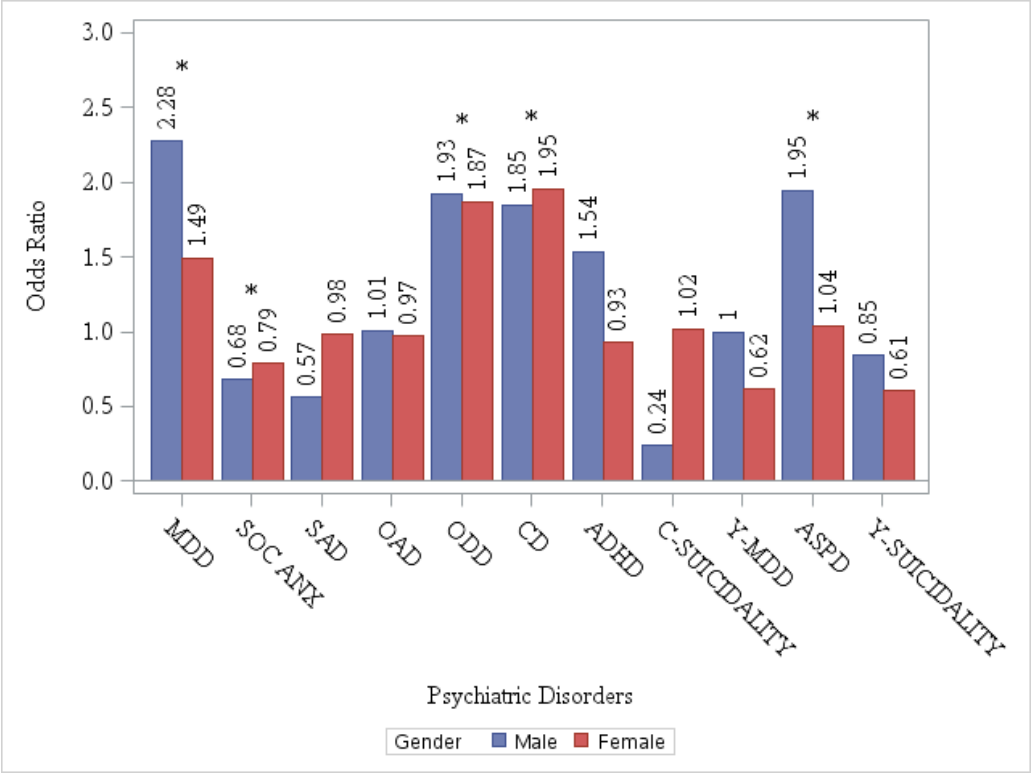

Figure 9 Odds Ratios of Psychiatric Disorders in Boy and Girl Bullies. Disorders with * are significant when controlling for gender (CMH).

3.1.3 The Bully Victims

3.1.3.1 Age Trends

The percentage of bully victims at each age in the sample is shown in Figure 10. The 10 year olds have the highest percentage of bully victims at 9.666%. At 9 df and a χ^2 value of 112.6326, being a bully victim is dependent on age. The prevalence of bully victims appears to decrease as children age past 10.

3.1.3.2 Gender Effects

The prevalence of bully victims in each gender at each age and any significant (*) differences between genders are graphed in Figure 11. At age 16 there is a significant difference between the prevalence of boy and girl bully victims with a χ^2 value of 4.1365 (1 df, $p = 0.0420$). The boys have the higher percentage of bully victims.

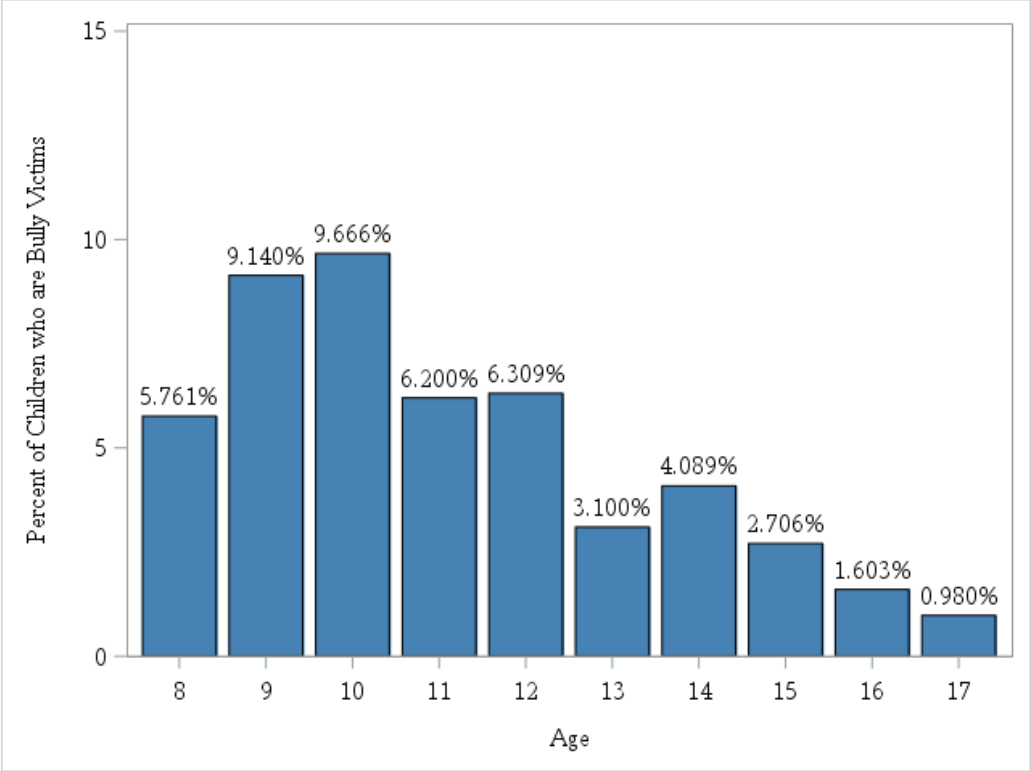

Figure 10 Prevalence of Bully Victims Across Age. Bars represent the percentage of children in each age group who were bully victims.

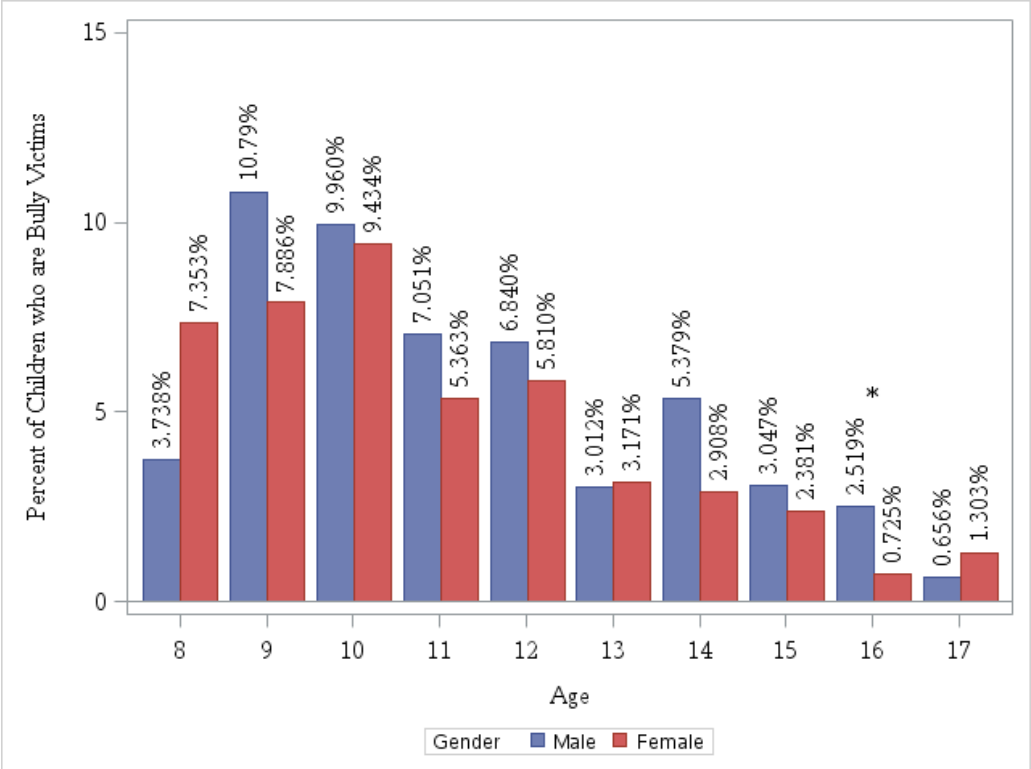

Figure 11 Prevalence of Bully Victims in Each Gender Across Age. Ages with * have a significant difference in prevalence between genders.

3.1.3.3 Psychiatric Disorders

The potential increased risk of psychiatric disorders in children who were bully victims was assessed using the `PROC GENMOD` logistic scheme. Odds ratios, 95% confidence intervals, and p values for the sample of bully victims as well as the male and female bully victims are reported in Table 11. Additionally, the OR and significance of psychiatric disorders of bully victims in the sample are graphed in Figure 12.

In the sample, bully victims are at a significantly higher risk of every psychiatric disorder tested except young adulthood depression.

3.1.3.4 Psychiatric Disorders by Gender

The odds ratios of boy and girl bully victims and any significant associations (*) when controlling for gender are graphed in Figure 13. Both boy and girl bully victims are at a higher risk for depression, separation anxiety, ODD, conduct disorder, ADHD, childhood suicidality, and ASPD. Girl bully victims are at a higher risk for social anxiety and young adult suicidality than girls who were not bully victims. Boy bully victims are at a higher risk for overanxious disorder than boys who were not bully victims.

Table 11 Odds Ratios of Psychiatric Disorders in Bully Victims. Significant values are bolded.

Disorder	Sample		Males		Females	
	OR(95% CI)	p	OR(95% CI)	p	OR(95% CI)	p
MDD	**3.31 (2.04-5.36)**	<1.0e-4	**3.16 (1.37-6.94)**	4.2e-3	**3.56 (1.89-6.69)**	<1.0e-4
SOC ANX	**1.38 (1.03-1.84)**	0.03	1.25 (0.79-1.96)	0.34	**1.55 (1.05-2.27)**	0.03
SAD	**2.55 (1.82-3.57)**	<1.0e-4	**2.52 (1.44-4.41)**	1.2e-3	**2.78 (1.80-4.29)**	<1.0e-4
OAD	**1.46 (1.12-1.89)**	4.9e-3	**1.64 (1.12-2.39)**	0.01	1.41 (0.97-2.04)	0.07
ODD	**3.99 (2.77-5.73)**	<1.0e-4	**3.73 (2.26-6.15)**	<1.0e-4	**4.21 (2.55-6.94)**	<1.0e-4
CD	**2.91 (2.06-4.09)**	<1.0e-4	**2.89 (1.88-4.44)**	<1.0e-4	**2.71 (1.59-4.61)**	2.0e-4
ADHD	**4.62 (3.07-6.96)**	<1.0e-4	**3.67 (2.19-6.15)**	<1.0e-4	**6.6 (3.27-13.32)**	<1.0e-4
C-SUICIDALITY	**4.49 (2.99-6.75)**	<1.0e-4	**7.75 (3.87-15.53)**	<1.0e-4	**3.56 (2.08-6.07)**	<1.0e-4
Y-MDD	1.14 (0.85-1.54)	0.39	1.08 (0.69-1.7)	0.72	1.3 (0.86-1.94)	0.21
ASPD	**2.66 (1.558-4.57)**	4.0e-4	**2.30 (1.20-4.40)**	0.01	**2.99 (1.16-7.69)**	0.02
Y-SUICIDALITY	**2.08 (1.34-3.23)**	1.1e-3	2.03 (0.97-4.25)	0.06	**2.27 (1.33-3.89)**	2.7e-3

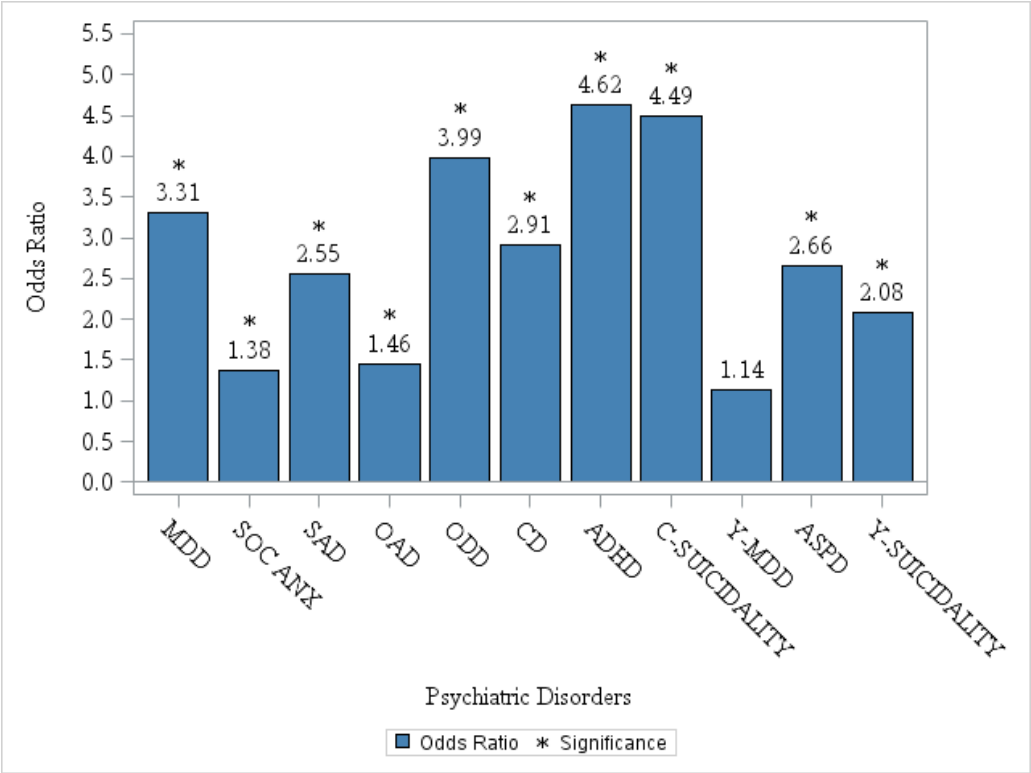

Figure 12 Odds Ratios of Psychiatric Disorders in Bully Victims. Bars with * are significant disorders in bully victims.

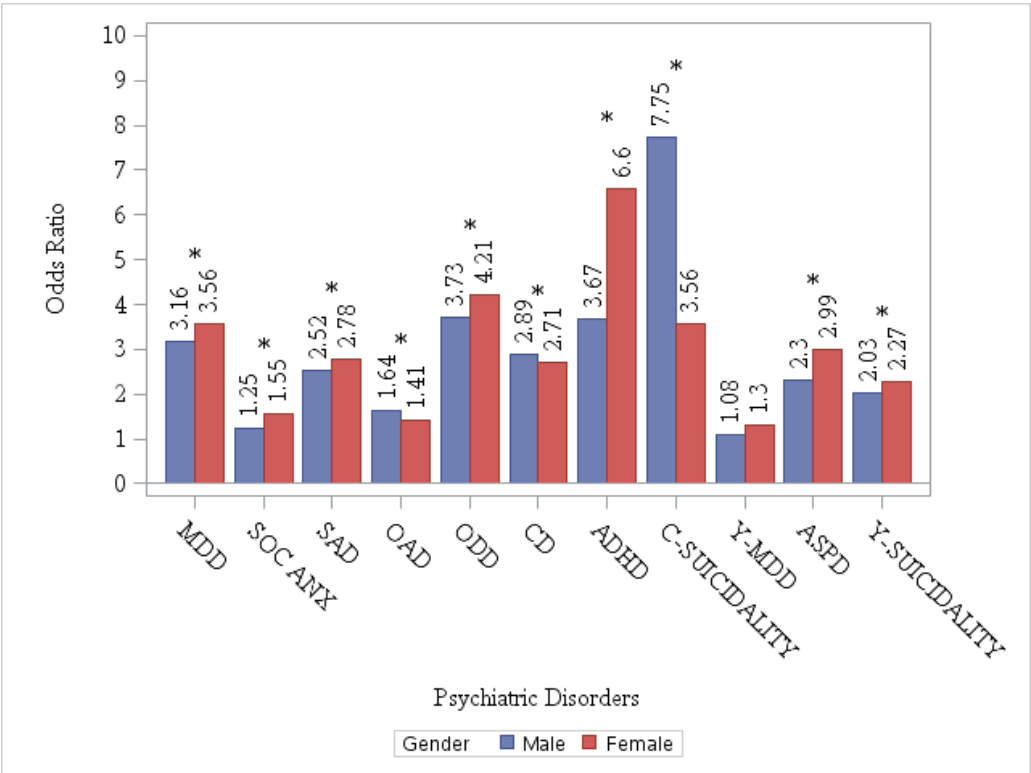

Figure 13 Odds Ratios of Psychiatric Disorders in Boy and Girl Bully Victims. Disorders with * are significant when controlling for gender (CMH).

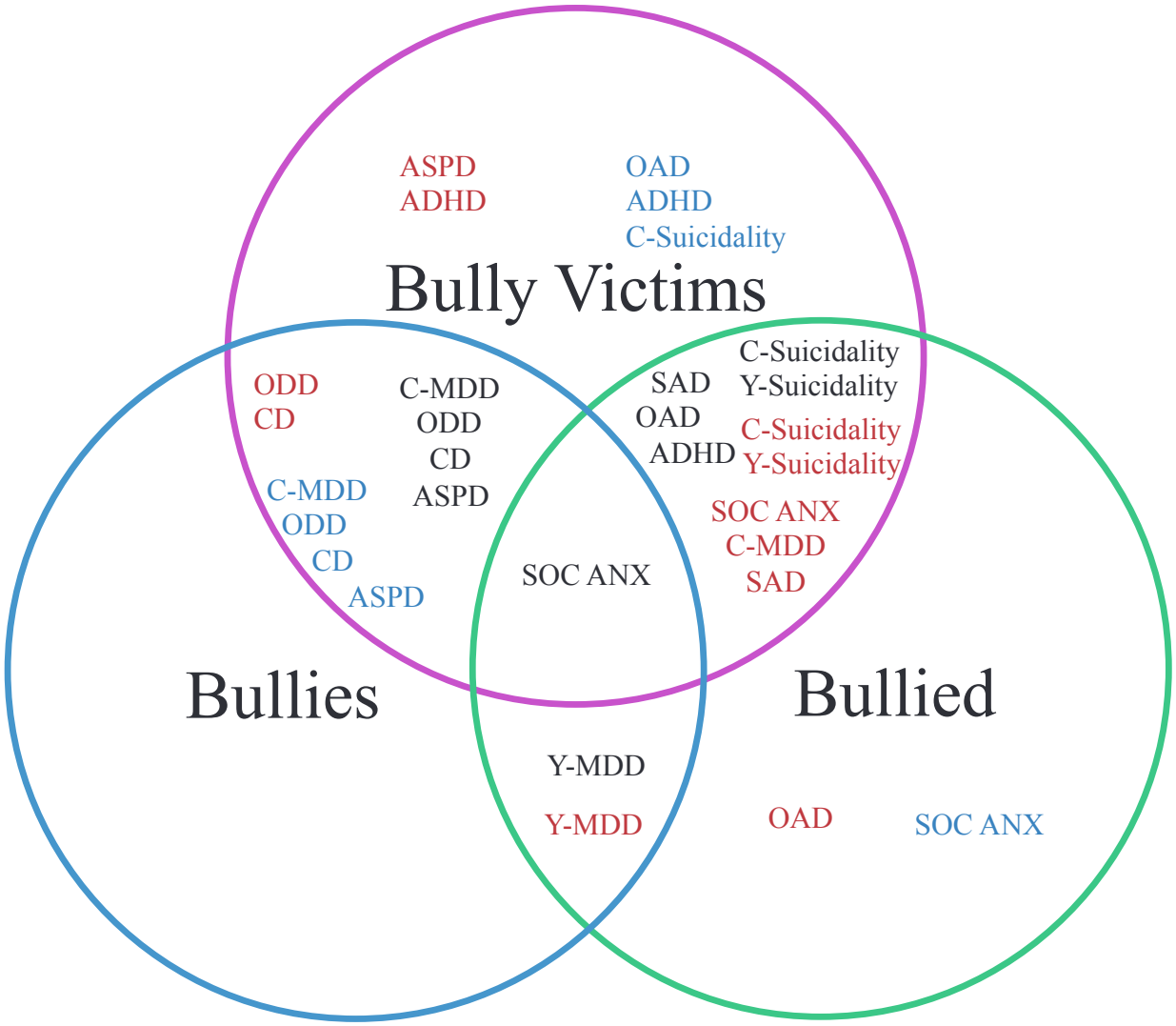

Figure 14 Venn Diagram Showing Relationship of Psychiatric Disorders Across Bullying Involvement. Disorders listed in black are significant in the sample. Disorders listed in red are significant in females, and disorders listed in blue are significant in males.

3.3 Twin Correlations and Model Fitting

3.3.1 The Bullied

The twin correlations for being bullied are listed in Table 12. Given that the DZ correlation is less than ½ of the MZ correlation, it was estimated that non-additive genetic (dominance) effects influenced the variance of being bullied and that the common environment was not significant. The unique environment was also estimated to influence the variance of the trait, based on the deviation from unity of the MZ twins (1-rMZ). This estimation holds true for the sample and for each gender. To determine the effect of genetic and environmental factors on being bullied, the data was fit to both ACE and ADE models.

Table 12 Twin Correlations of Bullied Children

	Total		**Males**		**Females**		**Opposite**
Zygosity	MZ	DZ	MZ	DZ	MZ	DZ	DZ
Correlation	0.4903	0.0660	0.4325	-0.1389	0.5369	0.1168	0.1714

The best-fitting ACE model is the AE model, where C is dropped. The results of fitting the ACE model and path coefficients are listed in Table 13.

Table 13 ACE Model Fitting Bullied Children. * marks the best-fit model.

Model	**a**	**c**	**e**	**-2LL**	**df**	**AIC**	χ^2	**Δdf**	**p**
ACE	0.664	0	0.748	2318.183	2831	-3343.817	—	—	—
AE*	0.664	0	0.748	2318.183	2832	-3345.817	-5.457e-12	1	1.000
CE	0	0.555	0.832	2328.859	2832	-3335.141	10.677	1	1.085e-3
E	0	0	1.000	2357.064	2833	-3308.936	38.881	2	3.607e-9

The data was then fit to an ADE model since the DZ correlation was less than ½ of the MZ correlation, suggesting dominance genetic effects. The results of fitting the ADE model and the path coefficients are listed in Table 14. Statistically the DE model fits the data the best.

Table 14 ADE Model Fitting Bullied Children. * marks the best-fit model.

Model	a	d	e	-2LL	df	AIC	χ^2	Δdf	p
ADE	0	0.694	0.720	2314.990	2831	-3347.010	—	—	—
AE	0.664	0	0.748	2318.183	2832	-3345.817	3.193	1	7.394e-2
DE*	0	0.694	0.720	2314.990	2832	-3349.010	-6.207e-10	1	1.000
E	0	0	1.00	2357.064	2833	-3308.936	42.074	2	7.307e-10

The comparison of the best-fit ACE and ADE models is in Table 15. Under the ACE model, being bullied is 44.13% heritable, and under the ADE model the trait is 48.12% heritable. Based on the χ^2 and AIC values, the best-fit model is the DE model. Figure 15 is the graphical representation of the best-fit model for being bullied.

Table 15 Best-Fit Models and Standardized Components of Bullied Children

Best-Fit models	A	C	D	E	-2LL	df	AIC	χ^2	Δdf
AE	0.4413	0	—	0.5587	2318.183	2832	-3345.817	—	—
DE	0	—	0.4812	0.5188	2314.990	2832	-3349.010	-3.193	0

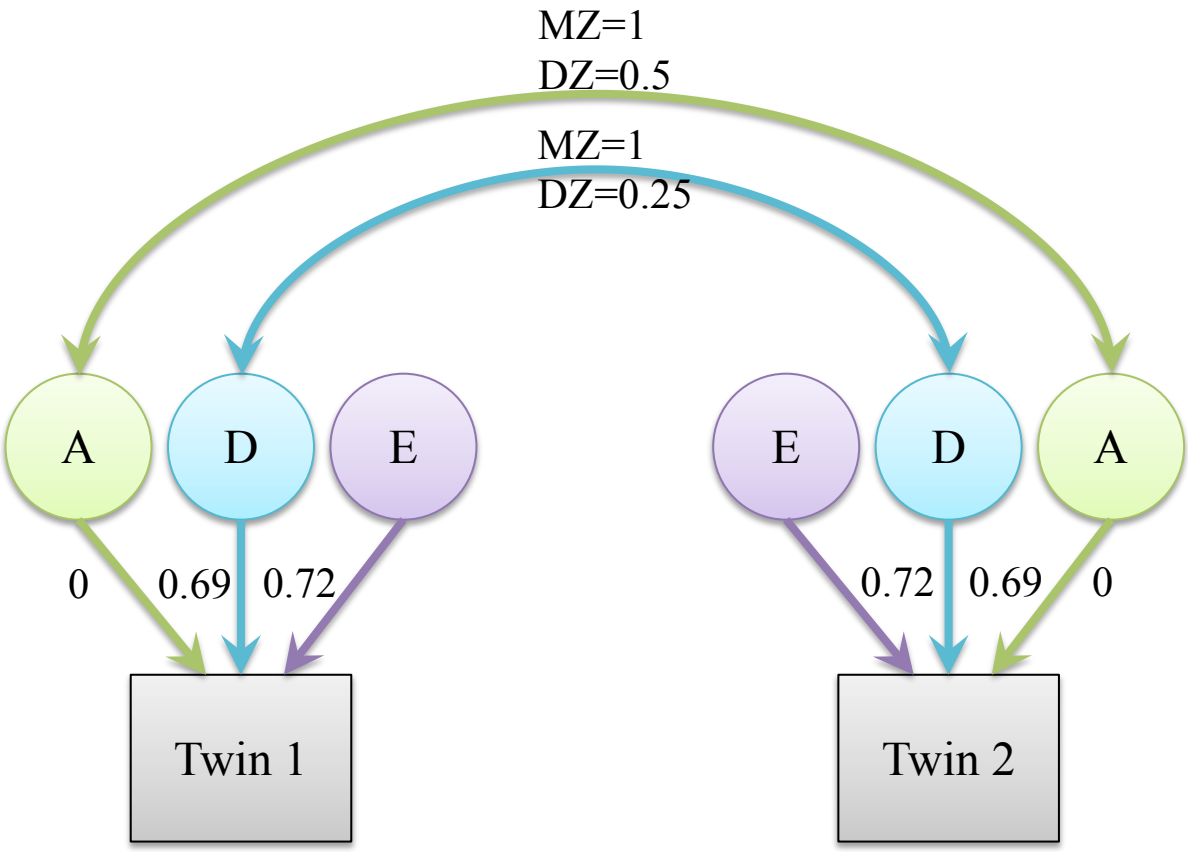

Figure 15 Best-Fit ADE Model of Bullied Children

3.3.1.1 Males

Based on the twin correlations for male twin pairs listed in Table 12, the common environment should not be a significant source of variation in being bullied, but dominance genetic effects should be present. The best-fit ACE model is the AE model; C does not significantly contribute to the variance of being bullied.

Table 16 ACE Model Fitting of Bullied Boys. * marks the best-fit model.

Model	a	c	e	-2LL	df	AIC	χ^2	Δdf	p
ACE	0.603	0	0.798	879.190	1008	-1136.810	—	—	—
AE*	0.603	0	0.798	879.190	1009	-1138.810	-1.683e-11	1	1.00
CE	0	0.488	0.873	883.513	1009	-1134.487	4.323	1	0.038
E	0	0	1.000	889.808	1010	-1130.192	10.618	2	0.005

Given the relationship between rMZ and rDZ, dominance genetic effects were suspected for being bullied in male children. The results of fitting the ADE models are in Table 17. Based on the AIC, the DE model provides the better-fit to the data.

Table 17 ADE Model Fitting of Bullied Boys. * marks the best-fit model.

Model	a	d	e	-2LL	df	AIC	χ^2	Δdf	p
ADE	0	0.643	0.755	877.037	1008	-1138.963	—	—	—
AE	-0.603	0	0.798	879.190	1009	-1138.810	2.153	1	0.142
DE*	0	0.643	0.766	877.037	1009	-1140.963	-6.821e-13	1	1.000
E	0	0	1.000	889.808	1010	-1130.192	12.771	2	1.686e-3

Under the best-fit ACE model (AE) being bullied is 36.39% heritable in VTSABD males, and 63.61% environmental. The best-fit ADE model, the DE model, gave a 41.28% heritability for being bullied in boys. Comparing the two best-fitting models (Table 18), suggests that the DE model explains the variance in the trait better than the AE model.

Table 18 Best-Fit Model and Standardized Components of Bullied Boys

Best-Fit models	A	C	D	E	-2LL	df	AIC	χ^2	Δdf
AE	0.3639	0	—	0.6361	879.190	1009	-1138.810	—	—
DE	0	—	0.4128	0.5872	877.0368	1009	-1140.963	-2.153	0

3.3.1.2 Females

Similar to the males, the twin correlations for female twin pairs suggested that genes (additive and dominance) and unique environment contribute to the variance of the trait. The best-fit ACE model (AE) corroborates this estimation.

Table 19 ACE Model Fitting for Bullied Girls. * marks the best-fit model.

Model	a	c	e	-2LL	df	AIC	χ^2	Δdf	p
ACE	0.715	0	0.699	939.588	1220	-1500.412	—	—	—
AE*	0.715	0	0.699	939.588	1221	-1502.412	-1.819e-12	1	1.000
CE	0	0.655	0.755	943.490	1221	-1498.510	3.902	1	4.822e-2
E	0	0	1.000	967.693	1222	-1476.307	28.105	2	7.889e-7

To estimate the effects of dominance genetics on girls being bullied, the ADE model was fit. The best-fit model is the DE given the more negative AIC.

Table 20 ADE Model Fitting for Bullied Girls. * marks the best-fit model.

Model	a	d	e	-2LL	df	AIC	χ^2	Δdf	p
ADE	0	0.726	0.688	938.983	1220	-1501.017	—	—	—
AE	-0.715	0	0.699	939.588	1221	-1502.412	0.605	1	0.437
DE*	0	0.726	0.688	938.983	1221	-1503.017	-2.240e-11	1	1.00
E	0	0	1.000	967.693	1222	-1476.307	28.710	2	5.829e-7

In female children, being a victim of bullying is 51.18% heritable, and 48.82% due to unique environmental influences under the AE model. The heritability under the DE model is 52.73%. The DE model fits the data better than the AE model (Table 21).

Table 21 Best-Fit Models and Standardized Components for Bullied Girls

Best-Fit models	A	C	D	E	-2LL	df	AIC	χ^2	Δdf
AE	0.5118	0	—	0.4882	939.5879	1221	-1502.412	—	—
DE	0	—	0.5273	0.4727	938.9826	1221	-1503.017	-0.605	0

3.3.3 The Bully

The twin correlations for being a bully are listed in Table 22. The DZ correlation is close to ½ of the MZ correlation; therefore, it was estimated that additive genetic factors are the predominate sources of variation. Dominance genetic effects were not expected based on the correlations; therefore, only ACE models were fit.

Table 22 Twin Correlations for Bullies

	Total		**Males**		**Females**		**Opposite**
Zygosity	MZ	DZ	MZ	DZ	MZ	DZ	DZ
Correlation	0.5687	0.1953	0.6098	0.3524	0.5255	0.2071	0.0791

The ACE model fitting results are listed in Table 23. The best-fit model to the bullying data is AE, dropping C. In the best-fitting model, being a bully is 54.81% heritable and 45.19% the result of unique environment (Table 26). The best-fit model for the VTSABD bullies is diagramed in Figure 16.

Table 23 ACE Model Fitting for Bullies. * marks the best-fit model.

Model	**a**	**c**	**e**	**-2LL**	**df**	**AIC**	χ^2	**Δdf**	**p**
ACE	0.740	0	0.672	2538.415	2831	-3123.585	—	—	—
AE*	0.740	0	0.672	2538.415	2832	-3125.585	-4.002e-11	1	1.000
CE	0	0.631	0.776	2552.785	2832	-3111.215	14.370	1	1.502e-4
E	0	0	1.000	2609.863	2833	-3056.137	71.447	2	3.058e-16

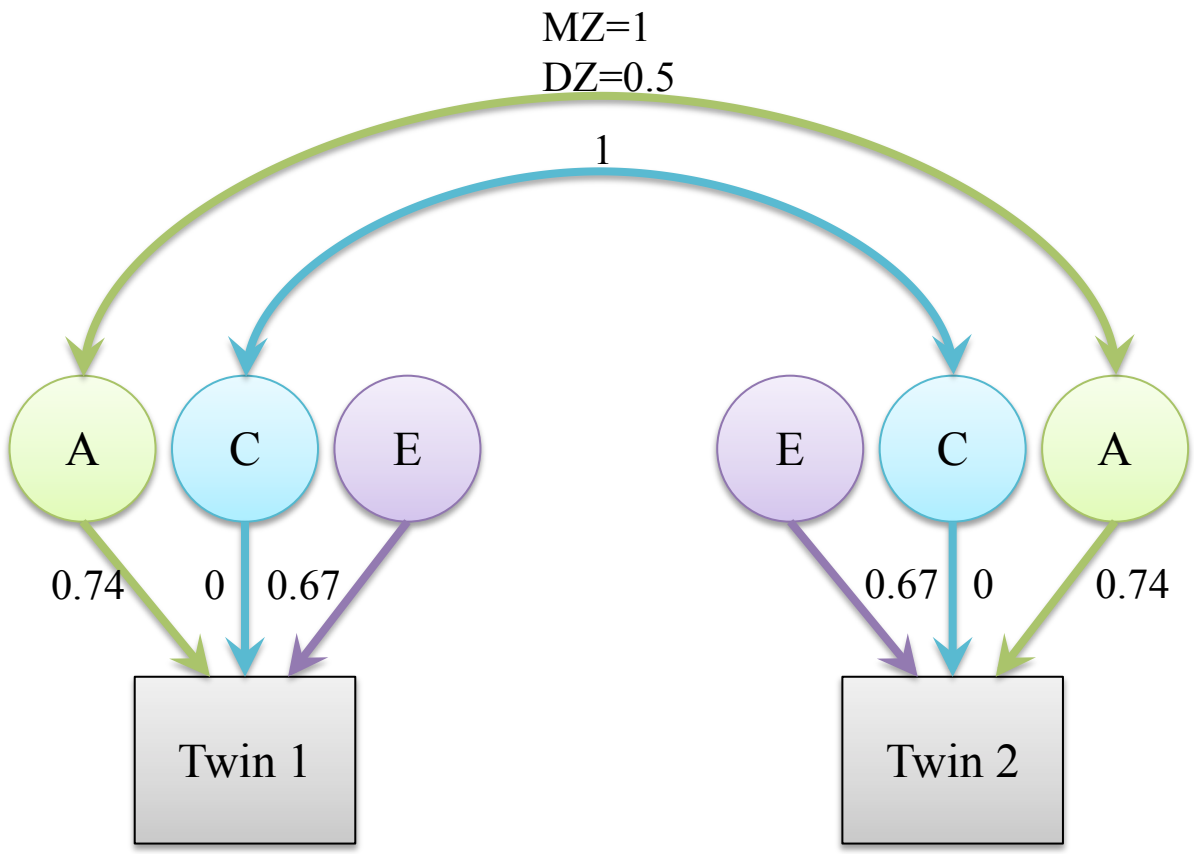

Figure 16 Best-Fit Model for Bullies

3.3.3.1 Males

The twin correlations for male bullies suggested that A, C, and E should have an influence on the variation of bullying. This is seen in the full ACE model; however, it is the AE model that fits the data the best, suggesting that C is not a significant source of variation. Being a bully is 61.57% heritable in males (Table 26).

Table 24 ACE Model Fitting for Male Bullies. * marks the best-fit model.

Model	a	c	e	-2LL	df	AIC	χ^2	Δdf	p
ACE	0.741	0.250	0.624	931.051	1008	-1084.949	—	—	—
AE*	0.785	0	0.620	931.100	1009	-1086.900	0.049	1	0.826
CE	0	0.720	0.694	934.350	1009	-1083.650	3.299	1	6.932e-2
E	0	0	1.000	974.120	1010	-1045.880	43.069	2	4.443e-10

3.3.3.2 Females

The twin correlations for female bullies suggested an influence of genes and unique environment on the trait variance. The best-fit model, AE, supports this estimation. The heritability of being a female bully is 51.99% (Table 26).

Table 25 ACE Model Fitting for Female Bullies. * marks the best-fit model.

Model	a	c	e	-2LL	df	AIC	χ^2	Δdf	p
ACE	0.721	0	0.693	1025.809	1220	-1414.191	—	—	—
AE*	0.721	0	0.693	1025.809	1221	-1416.191	-4.093e-12	1	1.000
CE	0	0.655	0.756	1029.536	1221	-1412.464	3.727	1	5.351e-2
E	0	0	1.000	1056.397	1222	-1387.603	30.588	2	2.279e-7

Table 26 Standardized Components of Best-Fit Models of Bullies

Components	Sample	Males	Female
A	0.5481	0.6157	0.5199
C	0	0	0
E	0.4519	0.3843	0.4801

3.3.4 The Bully Victims

In bully victims, the DZ correlation is greater than ½ the MZ correlation, suggesting an influence of common environment on the variation of the trait. This is true for males and to a lesser extent for females as well.

Table 27 Twin Correlations of Bully Victims

	Total		**Males**		**Females**		**Opposite**
Zygosity	MZ	DZ	MZ	DZ	MZ	DZ	DZ
Correlation	0.5994	0.3448	0.4724	0.3627	0.6933	0.3550	0.3305

The model that fits the data the best is the AE model, meaning that common environment contributes non-significantly to the variation of being a bully victim.

Table 28 ACE Model Fitting for Bully Victims. * marks the best-fit model.

Model	**a**	**c**	**e**	**-2LL**	**df**	**AIC**	χ^2	**Δdf**	**p**
ACE	0.748	0.243	0.618	1858.089	2831	-3803.911	—	—	—
AE*	0.791	0	0.611	1858.197	2832	-3805.803	0.108	1	.742
CE	0	0.689	0.725	1864.698	2832	-3799.302	6.609	1	1.015e-2
E	0	0	1.000	1922.976	2833	-3743.024	64.887	2	8.128e-15

Being a bully victim in the VTSABD is 62.62% heritable, and 37.38% due to unique environment (Table 31). The best-fit model is shown in Figure 17.

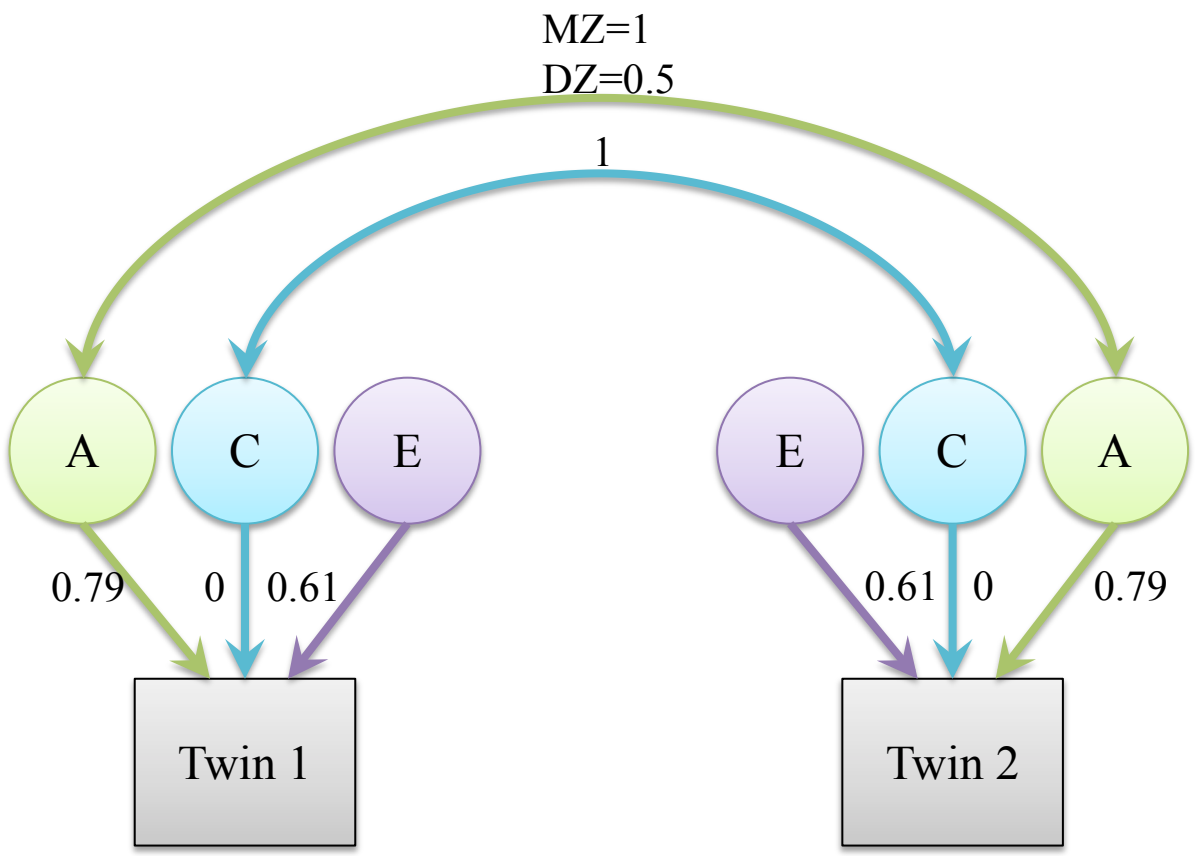

Figure 17 Best-Fit Model for Bully Victims

3.3.4.1 Males

Based on the twin correlations, it was estimated that the common environment of males should influence the variation of being a bully victim. In sub-model comparisons (Table 29), both the AE and CE models have non-significant χ^2 and p values, meaning that both models are not significantly different from the ACE model. However, the AE model has the more negative fit statistic (AIC), suggesting that it is the better-fit model. The heritability of being a male bully victim is 51.28% with 48.72% due to unique environment (Table 31).

Table 29 ACE Model Fitting for Male Bully Victims. * marks the best-fit model.

Model	a	c	e	-2LL	df	AIC	χ^2	Δdf	p
ACE	0.518	0.465	0.718	694.752	1008	-1321.248	—	—	—
AE*	0.716	0	0.698	695.134	1009	-1322.866	0.381	1	0.537
CE	0	0.653	0.757	695.299	1009	-1322.701	0.547	1	0.459
E	0	0	1.000	712.242	1010	-1307.758	17.490	2	1.592e-4

3.3.4.2 Females

The DZ correlation in female bully victims is only slightly greater than ½ the MZ correlation, suggesting a possible influence of common environment on the trait. However, the best-fit model (AE) does drop C. Being a female bully victim is 71% heritable and 29% due to the unique environment.

Table 30 ACE Model Fitting for Female Bully Victims. * marks the best-fit model.

Model	a	c	e	-2LL	df	AIC	χ^2	Δdf	p
ACE	0.843	0	0.539	749.169	1220	-1690.831	—	—	—
AE*	0.843	0	0.539	749.169	1221	-1692.831	-6.594e-12	1	1.000
CE	0	0.762	0.647	754.336	1221	-1687.665	5.167	1	2.302e-2
E	0	0	1.000	793.834	1222	-1650.166	44.665	2	2.000e-10

Table 31 Standardized Components of Best-Fit Models of Bully Victims

Components	Sample	Males	Female
A	0.6262	0.5128	0.71
C	0	0	0
E	0.3738	0.4872	0.29

4 Discussion

The purpose of this paper is to expand available information regarding the genetic and environmental impacts of bullying involvement. Bullying involvement includes individuals who are bullied, those who are bullies, and those who are both (bully victims). To date and to our knowledge, most of the existing literature focuses on bullied individuals. Although this focus is necessary, bullies and bully victims are also at a higher risk for psychiatric disorders than children not involved with bullying. It is necessary to identify the risks associated with all types of bullying involvement so that intervention methods can be developed to target all vulnerable individuals before lasting damage occurs.

4.1 Sample: Epidemiology/Psychiatric Disorders

In the VTSABD sample, more individuals were bullies (17.33%) than victims (14.56%) and bully victims (10.69%). Children were most likely to be bullied at ages 8 and 9, and most likely to be bullies and bully victims at age 10. It is possible that the 10 year olds are bullying the younger 8 and 9 year olds. However, all bullying decreases as children age past 10.

Bullied children are predominantly susceptible to internalizing disorders including: social anxiety, separation anxiety, overanxious disorder, and childhood suicidality. Notably, individuals bullied as children are at an increased risk of depression and suicidality in young adulthood. Copeland et al. also saw an increase of depression and suicidality in bullied individuals (2013). Bullied children are also at a higher risk for ADHD.

The children who bully others are at a higher risk for the externalizing disorders tested, and a few of the internalizing disorders. As expected by definition, bullies are at a higher risk for oppositional defiant and conduct disorder in childhood, and antisocial personality disorder in

young adulthood (APA, 2013). ASPD has been reported in bullies in the literature (Copeland et al. 2013). Bullies are at an increased risk of social anxiety and depression both as children and young adults.

The individuals who are both bullies and victims were expected to be at a higher risk for both internalizing and externalizing disorders, and this is seen in the VTSABD sample (Kelly et al. 2015). Bully victims are at a higher risk for childhood depression, social anxiety, separation anxiety, overanxious disorder, oppositional defiant disorder, conduct disorder, ADHD, antisocial personality disorder, and suicidality in childhood and young adulthood. The only disorder tested that these individuals are not at a higher risk for is young adult depression.

Across the sample, all the psychiatric disorders tested are significant in at least two of the bullying involvement categories. Children who were bullied or who were bullies are at an increased risk for depression as young adults. Children who were bullied and children who were bully victims are both at a higher risk for separation anxiety, overanxious disorder, ADHD, and childhood and young adult suicidality. Children who were bullies and children who were bully victims are at a higher risk for childhood depression, ODD, conduct disorder, and ASPD. Finally, children who were involved in bullying in any capacity are at a higher risk for social anxiety. Our results replicate those of Kelly et al. who found that individuals with internalizing disorders were more likely to be bullied, students with externalizing disorders were more likely to be bullies, and all disorders they examined increased the risk of being a bully victim (2015). (See Figure 14 for a graphical display of these results.)

4.2 Sample: Twin Methodology

The twin correlations for being bullied suggested an effect of dominance genetic factors (or epistasis) on the variance of being victimized. The best-fit model for being bullied is the DE

model, suggesting that additive genetic factors and the common environment do not significantly contribute to the variance of being bullied. As a result, the heritability of being bullied is 48.12%. Our heritability of bully victimization is close to the heritability found by Fisher et al. and Shakoor et al. (2015, 2014). Fisher et al. used data on bully victimization occurring between the ages of 12 and 18, and reported a heritability of 34% (2015). Shakoor et al. measured bully victimization at 16 years old and reported a heritability of 35% (2014).

The relationship of the MZ and DZ correlations in bullies did not suggest an effect of dominance genetic factors on the trait, rather that additive genetic factors were the predominate influence. The best-fit AE model confirmed that the variance of being a bully is due to additive genetic and unique environmental effects. Being a bully is more heritable than being bullied, at 54.81%. Our heritability is similar to Ball et al. who reported a heritability of 61% for 9 and 10 year old bullies (2008).

The common environment was estimated to effect the variation of being a bully victim; however, the best-fit model is the AE model. The common environment is not a significant source of variation in being a bully victim. The heritability of being a bully victim is 62.62%. The heritability of being a bully victim is closest to that of being a bully. Other studies using twin data and SEM to estimate heritability of being a bully victim have not been found to date.

4.3 Gender: Epidemiology/Psychiatric Disorders

Boys were more likely to be involved in bullying than girls in this sample. However, female victims and bully victims are at risk for more psychiatric disorders than the males; replicating gender trends reported in the literature (Klomek, Sourander, Elonheimo, 2015).

Bullied girls are at a higher risk for depression, social anxiety, separation anxiety, overanxious disorder and suicidality in childhood than girls who were not bullied. Furthermore,

females bullied as children are more likely to experience depression and suicidality in young adulthood than females who were not bullied. Increased suicidality in females has been replicated in the literature (Stewart et al. 2017). Boys who were bullied are only more likely to experience social anxiety.

Boys were significantly more likely to be bullies in the sample, especially at ages 13, 15, and 16. Boys who bully others are at a higher risk for depression in childhood than boys who do not bully others. Girls who were bullies are at a higher risk for young adult depression than girls who were not bullies. Both boys are girls who bully others are at a higher risk for the externalizing disorders oppositional defiant disorder and conduct disorder. Males who bullied others are at a higher risk for ASPD in young adulthood.

Overall, more boys are bully victims than girls, with significantly more boys involved at age 16. As seen in the total sample, bully victims are at a higher risk for both internalizing and externalizing disorders. The female bully victims are more likely to experience depression, social anxiety, separation anxiety, ODD, conduct disorder, ADHD, and suicidality during childhood. In young adulthood, females who were bully victims are at a higher risk for ASPD and suicidality. Boys are at a higher risk for depression, overanxious disorder, ODD, conduct disorder, ADHD, and suicidality in childhood. As adults, male bully victims are at a higher risk for ASPD than males not involved in bullying.

As a whole, females involved with bullying in any capacity are at higher risk for more psychiatric disorders than males. Females who were bullied or were bully victims are at a higher risk for depression, social anxiety, separation anxiety, and childhood and young adult suicidality. Females who were bullies or bully victims are at a higher risk for ODD and conduct disorder. Females who were bullied or bullies are at risk for young adult depression. An increased risk for

overanxious disorder is unique to bullied females. Similarly, ADHD and ASPD are only increased in those females who were bully victims, which differs from the results of Copeland et al., who found that ASPD was increased in boy and girl bullies, not bully victims (2015). However, the longer lasting effects related to bullying involvement in females were expected (Cosma et al. 2017).

None of the psychiatric disorders tested are significant in males across all three bullying involvement categories. Regardless, male bullies and bully victims are both more likely to have childhood depression, ODD, conduct disorder, and ASPD. Overanxious disorder, ADHD, and childhood suicidality are significantly more likely in only male bully victims, and social anxiety is only significant in males who were bullied.

4.4 Gender: Twin Methodology

The relationship between the MZ and DZ correlations in male and female children who were bullied suggested an effect of dominance genetic factors on the variation of the trait. Thus both ACE and ADE models were fit to the bullied data. The best-fit model for both males and females is the DE sub-model of the ADE model. Based on this model, the heritability of being bullied in males is 41.28%, and the heritability in females is 51.18%.

Dominance effects were not suspected as sources of variation in being a bully—only ACE models were fit. The best-fit model for both males and females is the AE sub-model. The heritability of being a bully in males is 61.57% and 51.99% in females.

The twin correlations for bully victims suggested common environmental effects; however, both males and females fit best to the AE sub-model. Based on the AE model heritability of being a bully victim is 51.28% in males and 71% in females.

4.5 Limitations

Using binary responses may not fully capture any dose-response effects in the relationship of bullying involvement and psychiatric disorders (Singham et al. 2017; Thomas et al. 2017). The `bullied` variable was based on one "umbrella" measure of bullying; the CAPA does not address different types of bullying (verbal, physical, etc.) separately. Additionally, the data did not include cyber bullying. The MZ twins in the VTSABD are more similar for measures (sharing the same room, dressing alike) vital to the equal environments assumption (EEA) than the DZ twins. There is little evidence for violation of the EEA in studies of psychiatric disorders in twins (Rutter, 2006; Kendler et al. 1992). Another limitation of this study is that all the participants were Caucasian and from one state; therefore, the results may not be generalizable to the general population (Bowes et al. 2009). The low DZ correlation as compared to the MZ correlation for being bullied may be explained by mechanisms other than genetic dominance or epistasis. A reciprocal influence between the twins for victimization ("contrast" or "competition") can lead to a lowered DZ correlation. Moreover, mothers contrasting the twins, making the twins less similar than they really are, can also result in a DZ correlation less than one half the MZ correlation. This contrast effect has been shown for Attention Deficit Hyperactivity Disorder as rated by mothers (Eaves et al. 1997). Greater focus on contrast models of sibling interaction and rater effects should be an important follow-up analysis of this study. Finally, we did not directly test for significant differences in the magnitude of genetic and environmental effects on bullying involvement between the males and females. Having data on the same sex MZ and DZ pairs and opposite sex pairs will allow us to test for differences in the effect of genes and environment on being involved in bullying and whether the same or different genes explain variation in the boys and the girls in future analyses (Neale and Cardon, 1992).

5 Conclusion

Being bullied has both short-term and long-term impacts on children's mental health. Boys and girls of all ages are at risk for internalizing disorders concurrent with and the result of being bullied (Silberg et al. 2016). Individuals bullied as children are at a higher risk for depression in young adulthood than individuals who were not bullied. Our results are consistent with conclusions drawn from Klomek, Sourander, and Elonheimo's examination of several longitudinal studies (2015) on bully victimization and with the results of Copeland et al. (2013). The literature is in agreement that bully victimization is associated with serious, sometimes clinical consequences and requires better intervention methods.

The bullies are at risk for just as many psychiatric disorders as those who are bullied—but the pattern of disorders is different. The bullies in the VTSABD were more often diagnosed with externalizing disorders, a result replicated in the limited literature (Thomas et al. 2017; Bowes et al. 2009; Ball et al. 2008; Klomek, Sourander, Elonheimo 2015; Kelly et al. 2015). It is pertinent that intervention plans specific to bullies are developed and improved, not only to prevent bullying before it occurs, but also because the bullies are at risk for childhood depression. These children may not just be acting out; they are at risk for serious childhood problems as well.

Finally, studies on bully victims are even scarcer than studies on the bullies, even though the numbers of bully victims are not insignificant. Nearly 11% of our sample was a bully victim. Bully victims are at a higher risk for nearly all the psychiatric disorders tested, both internalizing and externalizing. This result is briefly reported in the literature (Thomas et al. 2017; Bowes et al. 2009; Ball et al. 2008; Klomek, Sourander, Elonheimo 2015; Kelly et al. 2015). Intervention

methods aimed at the bullies and/or the bullied children should identify the bully victims; however, treating these children as one or the other could result in an incomplete assessment of the psychiatric issues being addressed. In addition to missing the disorders significant to the other category of bullying involvement, interventions designed only for bullies and/or victims might also miss the disorders unique to the bully victims. Female bully victims are at a uniquely higher risk for ADHD and ASPD and male bully victims are at a higher risk for OAD, ADHD, and childhood suicidality.

Many twin studies have been conducted on bully victimization and the consequences thereof (see Introduction). To our knowledge, only one twin study has looked specifically at all three categories of bullying involvement not in relation to psychiatric disorders, and then only reported heritability of bully victimization and bullies (Ball et al. 2008). Our study expands the current information on the heritability of bully victimization and bullying, and provides a new estimate of the heritability of being a bully victim.

Future directions of this research include expanding on the twin method to use discordant MZ twins to study any causal relationships of psychiatric disorders in bullies and bully victims. Silberg et al. has already found a significant impact on social anxiety, separation anxiety, and young adult suicidality from bully victimization using discordant MZ twins from the VTSABD and YAFU sample (2016). Also, any dose-response effects of bullying exposure should be explored.

6 References

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7 Vita

Ellyn Dunbar was born September 15, 1995, in Hattiesburg, Mississippi. She was home schooled and completed her high school education in 2012. She graduated Summa Cum Laude from the University of Southern Mississippi in 2016 earning a Bachelor of Science in an American Chemical Society accredited Chemistry (Biochemistry emphasis) program. She will receive a Master of Science in Human Genetics in 2018 from Virginia Commonwealth University.