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INTRODUCTION

Bully victimization is a common problem among children and adolescents, with prevalence rates of 5-20 percent in Scandinavian countries (1–3). Victims of bullying tend to experience more symptoms of depression and anxiety, loneliness and diminished self-esteem, and engage in self-harming behaviour more often than non-victimized peers (4–6). Further, prior studies have linked bully victimization in childhood to similar outcomes in adulthood, including depression, anxiety, suicide, and psychotic symptoms (4,7–10). Other negative correlates include lower income, impaired physical health, and problems in social relationships (11). Given the adverse outcomes associated with bully victimization, identifying children at particular risk of being bullied and the causal character of such links is important in designing risk assessment and prevention strategies.

Children with neurodevelopmental problems (NDPs) represent one such group of individuals, as they tend to experience more bully victimization than their normally developing peers (1), particularly those diagnosed with Autism Spectrum Disorder (ASD;(12,13) Attention Deficit/Hyperactivity Disorder (ADHD;(14,15) and motor coordination problems (16,17). Yet, it remains unclear if particular diagnoses or symptoms uniquely predict bully victimization, or whether NDPs in general are associated with bully victimization. NDPs, although described as separate conditions by the Diagnostic and Statistical Manual of Mental Disorder (DSM;(18)), have a high degree of symptom overlap and "pure" conditions are rare (19–21). Indeed, recent research into the structure of NDPs indicate that much of the co-variation among NDPs is accounted for by a broad general factor (22). This general factor indicates that symptoms classified as belonging to different diagnostic entities are influenced by the same genes. It may be that this general NDP factor is the primary risk factor for bully victimization rather than specific NDPs. Further, previous research has been limited by cross-sectional design, making conclusions regarding causality difficult. In the present study, we used a prospective design to assess children for a wide range of NDPs, making it possible to discern if associations with bully victimization at follow-up are general or unique to specific NDPs.

Furthermore, NDPs are highly heritable (23,24) and genetic factors may also account for more than two-thirds of the variation in bully victimization (25). As bully victimization is an exposure, rather than a trait or behaviour, the genetic influence could be a reflection of heritable characteristics that influence the vulnerability to bully victimization. By using a

twin design we are able to further investigate a possible association between NDPs and bully victimization and address to what extent this association is influenced by genetic and environmental factors. The twin design thus represents one way to control for unmeasured genetic and shared environmental factors. Specifically, within a monozygotic twin pair, if the twin with more NDPs tend to suffer from more bully victimization, then the association cannot be attributed to unmeasured genetic and shared environmental confounds. If, on the other hand, both twins within a pair suffer from an equal amount of bully victimization regardless of who had more NDPs in childhood, then a causal interpretation is untenable (26,27).   In other words, in such a case, the phenotypic association is driven by genes and/or shared environments. This is important to establish before designing intervention programs; if the association between NDPs and bully victimization is attributable to genes or the shared environment, then there is little or no reason to expect that interventions targeting NDPs would lead to decreased prevalence of bully victimization.

In sum, we aimed to investigate the association between NDPs in childhood and subsequent adolescent bully victimization. Specifically we aimed to answer the following questions:

a) Do children with parent-reported NDPs at age 9 or 12 self-report more bully victimization at age 15? Is this effect related to a general NDP factor, or is it unique to specific neurodevelopmental problems?

b) If an association between NDPs and later bully victimization exist, to what extent is it driven by genetic and environmental factors (shared and non-shared)?

MATERIALS AND METHODS

Participants

The Child and Adolescent Twin Study in Sweden (CATSS) started in 2004, and is an on-going prospective cohort study targeting all Swedish twins born from July 1992 and onwards. The overall aim is to prospectively study the development of physical and mental health from childhood to adolescence and young adulthood, with specific attention to NDPs. Parents are interviewed by telephone in conjunction with their twins' 9th or 12th birthday (CATSS-9/12) and when the twins are 15, they self-report data in web-questionnaires (CATSS-15). The telephone interviews are conducted by interviewers from a professional company `Intervjubolaget´, who, after a brief introduction in child and adolescent psychiatry

and twin research, use a computerized version of the interview. Up to December 31, 2012 CATSS-9/12 included 21,450 individuals (response rate: 80% of all twins born in Sweden in the mentioned timeframe) (28).

The current analyses included 3,921 twin individuals (and their parents) who responded to both baseline interview and follow-up at age 15 (overall response rate to current study sample: 60.5%). Response rates were lower for children screened positive for neurodevelopmental disorders in the CATSS-9/12 parent interview (ADHD: 46%, ASD: 33%, Developmental Coordination Disorder [DCD]: 53%, Tic Disorder [TD]: 52%, Learning disorder [LD]: 47%) and also for children who were bullied according to the CATSS-9/12 parent interview (52.9%).

Zygosity was determined from DNA analyses in 86% of all same-sex twins. For twins without genetic assessments, an algorithm based on five questions of twin similarity derived from 571 pairs of twins with known zygosity was used to determine zygosity. Only twins with more than 95% probability of being correctly classified were assigned zygosity by this method. Twin analyses included 1,114 females (540 monozygotic and 574 same-sex dizygotic twins) and 1048 males (402 monozygotic and 646 same-sex dizygotic twins). All participants consented to the study and the Ethics Committee at Karolinska Institutet, Sweden granted ethical approval.

Measures

Autism – Tics, ADHD and other Comorbidities (A-TAC) interview

Neurodevelopmental problems were assessed with the A-TAC inventory which covers a broad range of neurodevelopmental symptoms and other common child psychiatric problems with high reliability and validity (29,30). A total of 96 items are arranged in 19 modules, each addressing a specific trait dimension. Items are worded to correspond to symptom definitions and diagnostic criteria of the DSM-IV-TR (18), answered in a lifetime perspective and in relation to similarly aged peers. Response options include "No" (0), "Yes to some extent" (0.5) and "Yes" (1).   We used modules that primarily tapped neurodevelopmental traits, including motor control; perception; concentration and attention; impulsiveness and hyper-activity; learning; memory, planning and organizing tasks; language; social interaction; flexibility and tics. The modules for *concentration and*

attention, and *impulsiveness and hyper-activity*, correspond to DSM criteria for ADHD, and modules *language*, *social interaction* and *flexibility* correspond to DSM criteria for ASD.

Olweus bully/victim scale

The Olweus bully/victim scale (31,32) is a self-report questionnaire of perceived bully experiences, which was filled out by participants at the age of 15. The questionnaire provides a definition of bullying and asks about such occurrences in the past couple of months. It consists of 11 items tapping verbal, physical, and internet bullying as well as social exclusion, rated on 5-point scales ranging from "it has not happened to me in the last months" (1) to "several times a week" (5). Because previous analyses have demonstrated that this is a unidimensional construct (3), we treated it as a single continuous scale. For the prevalence estimates presented in Table 1, a previously validated cut-off score using one global item was used (3).

Possible confounders

Socioeconomic factors also seem associated with bullying (33–35); hence, we included parental education level as a possible confound. To control for bully victimization at baseline, we used one item from the A-TAC parent interview in CATSS 9/12 that asked if the child had ever been bullied in school.

Statistical analysis

Observed associations between A-TAC NDP modules and bullying

We analysed self-reported bully victimization at age 15 as a function of parent-reported NDPs at age 9/12. In order to take measurement error and the non-normal distribution of the items into account, we created a latent continuous factor for each A-TAC NDP module and the bully victimization scale based on the polychoric correlations among the items. We regressed the continuous latent bully victimization variable on the continuous latent A-TAC NDP modules and then controlled for parent education level and parent-reported bully victimization at baseline. Figure 1 displays this model. Subsequently, we controlled for comorbidity by creating a latent general NDP factor . This latent general NDP factor included all A-TAC NDP modules, excluding the one module that was the target of

interest in each analysis. Thus, we examined if a given NDP module had a unique association with bully victimization after controlling for a general NDP factor. The full analyses thus address the independent effects of each specific A-TAC NDP module on adolescent bully victimization, while controlling for bully victimization at baseline, parent education and comorbid NDPs. We used robust standard errors to account for the non-independence among twin pairs. We analyzed boys and girls separately, as both bully victimization and NDPs were more common among boys (table 1, table 2, appendix table 1) and because gender differences have previously not been explored. .

Insert Figure 1 about here

Twin analyses – Cholesky decomposition

After inspecting the observed associations between NDP modules and bully victimization, we applied so-called Cholesky decompositions, which examine how much of the observed associations that could be attributed to shared genes (A), shared environment (C), and non-shared (unique) environment (E). Shared genes represents the inherited additive effects of different alleles; the shared environment represents non-genetic components making twins within a pair similar; and the non-shared environment represents non-genetic components making twins within a pair dissimilar. Monozygotic twins are expected to share, on the average, all of their segregating genes, all of the shared environment (because they grow up in the same household), and, by definition, none of the non-shared environment. Dizygotic twins differ from monozygotic twins only in that they share, on the average, half of their segregating genes.

More specifically, the Cholesky decomposition capitalizes on differences in correlations between NDP in twin one within a pair with bully victimization in twin two within the same pair. To the extent that correlation is stronger for monozygotic twins compared to dizygotic twins, it implies the influence of shared genes. To the extent it is similar across monozygotic and dizygotic twins, it implies the influence of the shared environment. To the extent this association is not unity within pairs, it implies the influence of the non-shared environment. As mentioned in the introduction, the non-shared environmental effect (E) may be interpreted as a quasi-causal parameter, that is, an association that persists after controlling for all potential genetic and shared environmental confounds (26,27). In the context of this study, a significant E parameter would indicate that

within a twin pair, the twin who displayed more NDPs at age 9 would also be bullied more at age 15, which cannot solely be attributed to genetic or shared environmental factors.

Because there were too few observations to analyse the data at the item level (as in the observed analyses) when examining MZ and DZ separately, the Cholesky decompositions were carried out at the scale level (i.e., items were summed to create scale scores). We used STATA for descriptive statistics (36) and Mplus (37) for the observed associations and the Cholesky decompositions.

RESULTS

Descriptive statistics

Descriptive statistics are displayed in Table 1. Mean scores for the A-TAC NDP modules are presented in Table 2. (Prevalence of neurodevelopmental diagnoses presented in the appendix Table 1)

Insert Table 1 and Table 2 about here

Observed associations between childhood general NDPs and adolescent bully victimization.

Observed (phenotypic) associations between NDPs at age 9/12 and bully victimization at age 15 are presented in Table 3. In boys, six NDP modules were weakly but significantly prospectively associated with adolescent bully victimization (β ranged from 0.09 to 0.21; Table 3, unadjusted). This effect only remained significant for the NDP modules social interaction (β = 0.17, 95% CI: 0.04-0.30) and flexibility (β = 0.14, 95% CI: 0.03-0.25) when controlling for bully victimization at baseline and parent education (Table 3, adjusted 1). In girls, all ten NDP modules were significantly prospectively associated with adolescent bully victimization (β ranged from 0.10 to 0.42; Table 3, adjusted 1) and these effects remained for all modules when controlling for bully victimization at baseline and parent education (β ranged from 0.08 to 0.39) (Table 3, adjusted 1).

The general NDP factor, which included all A-TAC trait modules, was itself significantly associated with bully victimization in girls (β=0.30, 95% CI: 0.21-0.38) and boys (β=0.11, 95% CI: 0.03-0.18) (Table 3, unadjusted). This effect remained only in girls when controlling for bully victimization at baseline and parental education (β=0.27, 95% CI: 0.18-0.36) (Table 3, adjusted 1).

Observed associations of childhood unique NDPs and adolescent bully victimization.

We then proceeded to control for the general NDP factor to examine if NDP modules would remain uniquely associated with bully victimization. For boys, when controlling for the general NDP factor, no specific NDP module uniquely predicted bully victimization (Table 3, adjusted 2). In girls, however, when controlling for the general NDP factor, social interaction (β=0.54; 95% CI: 0.08-0.99) and motor control problems (β=0.10, 95% CI: 0.02-0.17) remained significantly related to adolescent bully victimization (Table 3, adjusted 2).

Finally, for ADHD and ASD, we also conducted analyses with diagnoses rather than scale scores; the resulting pattern was analogous (appendix Table 3).

Insert Table 3 about here

Genetic and environmental contributions to observed associations

We proceeded to examine to what extent the observed association could be explained by genetic and environmental factors. Because the observed associations between NDPs and bully victimization were significant only among girls, we only performed Cholesky decompositions on the female subsample. In order to control for a general NDP factor, we used a trivariate Cholesky decomposition, that is, we included three variables (the general factor, motor/social problems, and victimization of bullying). We entered the general NDP factor first and then added the respective NDP modules that evidenced an additional significant observed association with bully victimization (social interaction, Figure 2a, and motor control, Figure 2b). Thus, the trivariate Cholesky decomposition essentially represents a twin version of the observed associations conducted above in that we examined the general effect of having any problem (i.e., the general NDP factor) in addition to a specific effect associated with the social interaction and motor control modules, respectively. We were primarily interested in examining if paths from the general NDP factor and the motor/social problems E paths influenced bully victimization. If both of these regression coefficients were to be positive and significant, it would indicate that the twin within a pair with more general NDPs and motor/social problems also tended to suffer from more bully victimization (i.e., that the associations were not confounded by genetic and shared environment confounds). Because a univariate analysis demonstrated that the shared environment (C) did not influence variation in the outcome (comparing an ACE to an AE model revealed that the latter did not fit worse, $\Delta\chi^2(1) = 1.09, p = .30$), this component was dropped from the analyses. Univariate analyses of the predictors (i.e., the general NDP

factor, motor problems, and social problems) also demonstrated that AE models did not fit worse than ACE models (across all three ACE models, the C estimate converged at zero; subsequently, the chi-square differences between the ACE and AE models were not significant). As a consequence, we fit trivariate AE Cholesky decompositions.

The observed association between the general NDP factor and bully victimization was influenced by both genetic factors (65% averaged across the two analyses displayed in figures 2a and 2b) and unique environmental factors (35% averaged across the two analyses in figures 2a and 2b). After controlling for the general factor, the observed association between motor problems and later bully victimization was primarily influenced by genetic (76%) rather than unique environmental (24%) factors. The observed association between social interaction and later bully victimization was primarily influenced by unique environmental (84%) rather than genetic (16%) factors, after controlling for the general factor.

Our results indicate that the E-regression path between the general NDP factor and bully victimization was significant in both decompositions (Figure 2a: $\beta = 0.22$, 95% CI: 0.12, 0.32; Figure 2b: $\beta = 0.25$, 95% CI: 0.15, 0.35). The E-regression path was also significant between social interaction and bully victimization ($\beta = 0.11$; 95%CI: 0.04, 0.18; Figure 2a). This means that within female twin pairs, the girl with more general NDPs or social interaction problems also self-reported more bully victimization. For motor control problems, however, the E-regression path to bully victimization was not significant, that is, within a twin pair, knowing which twin had more motor control problems provided no information about which twin was more likely to report adolescent bully victimization.

By summing all the genetics paths leading into bully victimization, one can compute an estimate of its heritability. This shows that the heritability is 67%, in line with previous research (25).

Insert Figure 2 about here.

DISCUSSION

The general NDP factor and bully victimization

In this population-based prospective study of almost 4,000 twins, children with more parent-reported NDPs at age 9/12 more often self-reported bully victimization in mid-adolescence (age 15). The largest effect could be attributed to a general factor underlying all

specific NDPs. This suggests that the amount, rather than the specific nature of NDPs, is the primary driving force behind the observed association between childhood NDPs and adolescent bullying. The genetic part of this association could be mediated through a host of currently unknown variables, including problems and disorders outside the neurodevelopmental domain. Although speculative, one possibility is that the same genes that predispose individuals to neurodevelopmental problems also lead to somewhat different personality traits. Though individuals with such traits may manage fairly well during childhood, they may risk bullying once fitting in with peers grows increasingly important during adolescence.

The finding that the amount appears more important than the specific nature of NDPs dovetails with other recent research highlighting the importance of a general NDP common to all or most neurodevelopmental diagnostic entities. Past studies have demonstrated substantial overlap among specific NDPs (20,22,23,28), and Gillberg (38) has argued for more comprehensive assessment and treatment for children with NDPs than what the current diagnostic system allows. The current results might be seen as corroborating this argument, in that a general NDP factor appeared stronger in predicting bullying victimization than specific neurodevelopmental problems or diagnoses.

Social interaction and motor control as unique predictors

Social interaction and motor problems had independent, additional predictive effects on bully victimization risk among girls (but not boys) in this study. Poor social competence has previously been suggested as an important risk factor for bully victimization in non-clinical populations (34,39,40). Also, children with ASD report alarmingly high rates of bully victimization (12,13,41,42). Besides difficulties with social interaction, ASD also includes behavioural problems such as inflexibility, repetitive, stereotypic actions, and communication difficulties. However, our results indicate that only social skill problems relate uniquely to bully victimization. Although two prior studies did not find social skills problems to be uniquely associated to bully victimization (12,43) our findings agree with other studies suggesting that "social vulnerability" including gullibility and credulity is an independent risk factor for bully victimization in school-aged children with ASD (44) and that better social skills protect against bully victimization in ASD youths (13). Although comparing

studies is difficult due to differences in design and study population, our study is the largest in this area and controls for a wide range of comorbid NDPs.

Motor clumsiness has previously been suggested as a risk factor for bully victimization (16,17,45,46). This was supported by our data among girls (but not boys) and influenced by shared genes predisposing individuals to both motor coordination problems and bully victimization.

The unique environmental effect

Aside from genes, the association between NDPs and bully victimization was also influenced by the unique environment, particularly so for the general NDP factor and social interaction module among girls. As discussed above, a significant unique environmental effect (E) indicates that this association cannot be attributed solely to genetic or familial factors and, thus, is consistent with an interpretation of NDPs causing a higher risk of being subjected to bully victimization. Accordingly, interventions focused on reducing general NDPs and social interaction problems among girls could potentially reduce the individual risk of being subjected to bully victimization in adolescence. In contrast, motor problems did not exhibit a causal effect on bully victimization. Hence, though motor clumsiness could be used as a marker of risk of being bullied, interventions directed towards motor skills might not decrease that risk.

The current literature on social skills training in relation to bully victimization is limited. In one randomly controlled study the efficacy of a generic social skills intervention, Social Skills GRoup INtervention (S.S.GRIN), for children with social difficulties was assessed. It revealed that S.S.GRIN moderately increased peer liking, enhanced self-esteem and self-efficacy, and decreased social anxiety. However, no effect on bully victimization was seen (47).

Recent reviews on social skills training for children with ASD (48,49) do not include any studies using bully victimization as an outcome.

Gender difference

According to our results, there is a trend, although statistically non-significant, that the association between NDPs and bully victimization is stronger for girls than boys, indicating that girls may be more vulnerable to be subjected to bully victimization than are

boys with similar amounts of NDPs. Although more research on this topic is needed before drawing firm conclusions, the same pattern was seen in a large longitudinal study demonstrating that delinquent behaviour predicted indirect (non-physical) bully victimization more strongly in girls than boys (50). Furthermore, Bacchini et al found that ADHD symptoms in females predicted bully victimization whereas the same symptoms predicted bully perpetration among boys (51). Thus, one hypothesis to the gender differences we find is that NDPs may lead to bully victimization among girls, but bully perpetration among boys, perhaps due to a gender difference in which actions are considered socially accepted and gender appropriate. A further possibility is that females need a higher liability to develop the disease (or a female protective effect). For example, siblings of girls with ASD had a higher risk of the disorder compared to siblings of boys (52) and rare mutations in females with ASD are larger and more disruptive than those in males with ASD (53). Of course, other both biological and/or social factors are also possible, so it would be interesting to investigate the possible mechanisms in future studies.

Strengths and limitations

Strengths of this study include, first, that we used a large nationwide, population-based cohort that included both children who met diagnostic criteria and children with sub-diagnostic NDP levels. Second, because diagnoses can be rather heterogeneous, we analysed more specific NDP components. Third, we accounted for the extensive comorbidity among NDPs. Fourth, by assessing twins prospectively from ages 9/12 to 15, and fifth, by modelling genetic and environmental contributions to associations, we controlled unmeasured confounds and approached causal interpretability.

Nevertheless, our results should be viewed in light of some important limitations. The study experienced attrition at follow-up, particularly among children with more problems, although this may rather underestimate the associations studied. Further, NDPs and baseline bullying were both assessed by parental report and outcome bully victimization tapped only by self-report. Clinical examination and/or multiple informants might provide more accurate information. However, the instruments we used have been validated previously (3,29,30). A small number of studies have showed that adolescents with ASD do not interpret social situations correctly, raising the question whether self-reported bully victimization can be used as a reliable source of information (54,55). The findings from a recent study using

multiple informants however indicate that the perception of adolescents with ASD on bullying behaviour is likely to be accurate (56). Additionally, although results from twin studies on bully victimization should be generalized with some caution to non-twin samples, previous twin studies suggest similar bully victimization prevalence as in singletons (33) and there is no reason to suspect that associations between risk factors and bully victimization should operate differently in twins than among non-twins. Lastly, the latent factors in the phenotypic analyses were based on items, whereas we had to revert to scale level (i.e., sum scores) when we conducted the Cholesky decompositions due to lack of observations. Because sum scores include measurement error, these analyses had less power to detect any association.

Conclusion and implication

Given that bully victimization comes at great individual, familial, and societal costs, it is imperative to understand the developmental processes behind it to guide risk assessment and prevention strategies. In this study we identified child characteristics, including neurodevelopmental problems in general and social interaction and motor control problems in particular, that put individuals at risk for adolescent bully victimization. This study focuses on the child´s individual characteristics and we acknowledge the ethical dilemma in pointing out presumed individual traits as risk factor for being bullied, which can be misunderstood as "blaming the victim". Bullying happens in a social context and it is also influenced by contextual factors, which are likely to greatly influence both the prevalence of bully victimization and the consequences of bully victimization for the individual child. However, individual factors also influence this risk and need to be addressed in order to maximize the effort of reducing bully victimization. Our results indicate that reducing childhood NDPs in general, and social interaction problems among girls in particular, may be one important step in reducing a child's risk of being subjected to adolescent bully victimization.

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CONFLICTS OF INTEREST

On behalf of all authors, the corresponding author states that there is no conflict of interest.

REFERENCES

1.   Nordhagen R, Nielsen A, Stigum H, Köhler L (2005) Parental reported bullying among Nordic children: a population-based study. Child Care Health Dev 31(6):693–701.

2.   Craig W, Harel-Fisch Y, Fogel-Grinvald H, Dostaler S, Hetland J, Simons-Morton B, et al (2009) A cross-national profile of bullying and victimization among adolescents in 40 countries. Int J Public Health 54 Suppl 2:216–24.

3.   Solberg ME, Olweus D (2003) Prevalence estimation of school bullying with the Olweus Bully/Victim Questionnaire. Aggress Behav 29(3):239–68.

4.   Arseneault L, Bowes L, Shakoor S (2009) Bullying victimization in youths and mental health problems: "Much ado about nothing"? Psychol Med 40(05):717-727.

5.   Fisher HL, Moffitt TE, Houts RM, Belsky DW, Arseneault L, Caspi A (2012) Bullying victimisation and risk of self harm in early adolescence: longitudinal cohort study. BMJ 26(344):e2683–e2683.

6.   Hawker DS, Boulton MJ (2000) Twenty years' research on peer victimization and psychosocial maladjustment: a meta-analytic review of cross-sectional studies. J Child Psychol Psychiatry. 41(4):441–55.

7.   Copeland WE, Wolke D, Angold A, Costello EJ (2013) Adult psychiatric outcomes of bullying and being bullied by peers in childhood and adolescence. JAMA Psychiatry 70(4):419–26.

8.   Kim YS, Leventhal B (2008) Bullying and suicide. A review. Int J Adolesc Med Health 20(2):133–54.

9.   Klomek AB, Sourander A, Niemelä S, Kumpulainen K, Piha J, Tamminen T, et al (2009) Childhood Bullying Behaviors as a Risk for Suicide Attempts and Completed Suicides: A Population-Based Birth Cohort Study. J Am Acad Child Adolesc Psychiatry 48(3):254–61.

10.   Wolke D, Lereya ST, Fisher HL, Lewis G, Zammit S (2014) Bullying in elementary school and psychotic experiences at 18 years: a longitudinal, population-based cohort study. Psychol Med 44(10); 2199-2211.

11.   Wolke D, Copeland WE, Angold A, Costello EJ (2013) Impact of Bullying in Childhood on Adult Health, Wealth, Crime, and Social Outcomes. Psychol Sci 19;1958–70.

12.   Cappadocia MC, Weiss JA, Pepler D (2012) Bullying experiences among children and youth with autism spectrum disorders. J Autism Dev Disord 42(2):266–77.

13. Sterzing PR, Shattuck PT, Narendorf SC, Wagner M, Cooper BP (2012) Bullying involvement and autism spectrum disorders: prevalence and correlates of bullying involvement among adolescents with an autism spectrum disorder. Arch Pediatr Adolesc Med 166(11):1058–64.

14. Holmberg K, Hjern A (2008) Bullying and attention-deficit- hyperactivity disorder in 10-year-olds in a Swedish community. Dev Med Child Neurol 50(2):134–8.

15. Unnever JD, Cornell DG (2003) Bullying, Self-Control, and Adhd. J Interpers Violence 1;18(2):129–47.

16. Bejerot S, Edgar J, Humble MB (2011) Poor performance in physical education - a risk factor for bully victimization. A case-control study. Acta Paediatr 100(3):413–9.

17. Bejerot S, Humble MB (2013) Childhood clumsiness and peer victimization: a case–control study of psychiatric patients. BMC Psychiatry 13(1):68.

18. American Psychiatric Association, American Psychiatric Association (2000) Diagnostic and statistical manual of mental disorders: DSM-IV-TR. 4th ed., text revision. Washington, DC: American Psychiatric Association.

19. Gillberg C, Billstedt E (2000) Autism and Asperger syndrome: coexistence with other clinical disorders. Acta Psychiatr Scand 102(5):321–30.

20. Murray MJ (2010) Attention-deficit/Hyperactivity Disorder in the context of Autism spectrum disorders. Curr Psychiatry Rep 12(5):382–8.

21. Reiersen AM, Constantino JN, Todd RD (2008) Co-occurrence of motor problems and autistic symptoms in attention-deficit/hyperactivity disorder. J Am Acad Child Adolesc Psychiatry 47(6):662–72.

22. Pettersson E, Anckarsäter H, Gillberg C, Lichtenstein P (2013) Different neurodevelopmental symptoms have a common genetic etiology. J Child Psychol Psychiatry 54(12):1356–65.

23. Lichtenstein P, Carlström E, Råstam M, Gillberg C, Anckarsäter H (2010) The genetics of autism spectrum disorders and related neuropsychiatric disorders in childhood. Am J Psychiatry 167(11):1357–63.

24. Posthuma D, Polderman TJC (2013) What have we learned from recent twin studies about the etiology of neurodevelopmental disorders? Curr Opin Neurol 26(2):111–21.

25. Ball HA, Arseneault L, Taylor A, Maughan B, Caspi A, Moffitt TE (2008) Genetic and environmental influences on victims, bullies and bully-victims in childhood. J Child Psychol Psychiatry 49(1):104–12.

26. Shadish WR (2001) Experimental and quasi-experimental designs for generalized causal inference. Houghton Mifflin, Boston.

27. D'Onofrio BM, Lahey BB, Turkheimer E, Lichtenstein P (2013) Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. Am J Public Health 103 Suppl 1:S46–55.

28. Anckarsäter H, Lundström S, Kollberg L, Kerekes N, Palm C, Carlström E, et al (2011) The Child and Adolescent Twin Study in Sweden (CATSS). Twin Res Hum Genet Off J Int Soc Twin Stud 14(6):495–508.

29. Larson T, Anckarsäter H, Gillberg C, St\aahlberg O, Carlström E, Kadesjö B, et al (2010) The Autism-Tics, AD/HD and other Comorbidities inventory (A-TAC): further validation of a telephone interview for epidemiological research. BMC Psychiatry 10(1):1.

30. Larson T, Lundström S, Nilsson T, Selinus EN, Råstam M, Lichtenstein P, et al (2013) Predictive properties of the A-TAC inventory when screening for childhood-onset neurodevelopmental problems in a population-based sample. BMC Psychiatry 13:233.

31. Olweus D (1994) Bullying at school: basic facts and effects of a school based intervention program. J Child Psychol Psychiatry 35(7):1171–90.

32. Olweus D (1996) The Revised Olweus Bully/Victim Questionnaire. Bergen, Norway: Research Center for Health Promotion, University of Bergen.

33. Bowes L, Arseneault L, Maughan B, Taylor A, Caspi A, Moffitt TE (2009) School, Neighborhood, and Family Factors Are Associated With Children's Bullying Involvement: A Nationally Representative Longitudinal Study. J Am Acad Child Adolesc Psychiatry 48(5):545–53.

34. Cook CR, Williams KR, Guerra NG, Kim TE, Sadek S (2010) Predictors of bullying and victimization in childhood and adolescence: A meta-analytic investigation. Sch Psychol Q 25(2):65–83.

35. Wolke D, Woods S, Stanford K, Schulz H (2001) Bullying and victimization of primary school children in England and Germany: prevalence and school factors. Br J Psychol 92(Pt 4):673–96.

36. StataCorp (2013) Stata Statistical Software: Release 13. College Station,TX. StataCorp LP.

37. Muthén LK, Muthén BO (1998) Mplus User's Guide. Seventh Edition. Muthén & Muthén, Los Angeles.

38. Gillberg C (2010) The ESSENCE in child psychiatry: Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations. Res Dev Disabil 31(6):1543–51.

39. Crawford AM, Manassis K (2011) Anxiety, social skills, friendship quality, and peer victimization: an integrated model. J Anxiety Disord 25(7):924–31.

40. Egan SK, Perry DG (1998) Does low self-regard invite victimization? Dev Psychol. 34(2):299–309.

41. Hebron J, Humphrey N (2014) Exposure to bullying among students with autism spectrum conditions: A multi-informant analysis of risk and protective factors. Autism: the international journal of research and practice 18(6): 618-630.

42. Roekel E, Scholte RHJ, Didden R (2009) Bullying Among Adolescents With Autism Spectrum Disorders: Prevalence and Perception. J Autism Dev Disord 8;40(1):63–73.

43. Storch EA, Larson MJ, Ehrenreich-May J, Arnold EB, Jones AM, Renno P, et al (2012) Peer Victimization in Youth with Autism Spectrum Disorders and Co-occurring Anxiety: Relations with Psychopathology and Loneliness. J Dev Phys Disabil 21;24(6):575–90.

44. Sofronoff K, Dark E, Stone V (2011) Social vulnerability and bullying in children with Asperger syndrome. Autism: the international journal of research and practice 15(3):355–72.

45. Jansen DE, Veenstra R, Ormel J, Verhulst FC, Reijneveld SA (2011) Early risk factors for being a bully, victim, or bully/victim in late elementary and early secondary education. The longitudinal TRAILS study. BMC Public Health 11(1):440.

46. Stephenson EA, Chesson RA (2008) "Always the guiding hand": parents' accounts of the long-term implications of developmental co-ordination disorder for their children and families. Child Care Health Dev34(3):335–43.

47. DeRosier ME (2004) Building relationships and combating bullying: effectiveness of a school-based social skills group intervention. J Clin Child Adolesc Psychol Off J Soc Clin Child Adolesc Psychol Am Psychol Assoc Div 53. 33(1):196–201.

48. Reichow B, Steiner AM, Volkmar F (2012) Social skills groups for people aged 6 to 21 with autism spectrum disorders (ASD). Cochrane Database Syst Rev. 7:CD008511.

49. McMahon CM, Lerner MD, Britton N (2013) Group-based social skills interventions for adolescents with higher-functioning autism spectrum disorder: a review and looking to the future. Adolesc Health Med Ther. 22;(4):23–8.

50. Carbone-Lopez K, Esbensen F-A, Brick BT (2010) Correlates and Consequences of Peer Victimization: Gender Differences in Direct and Indirect Forms of Bullying. Youth Violence Juv Justice 22;8(4):332–50.

51. Bacchini D, Affuso G, Trotta T (2008) Temperament, ADHD and peer relations among schoolchildren: the mediating role of school bullying. Aggress Behav 34(5):447–59.

52. Robinson EB, Lichtenstein P, Anckarsäter H, Happé F, Ronald A (2013) Examining and interpreting the female protective effect against autistic behavior. Proc Natl Acad Sci U S A. 26;110(13):5258–62.

53. Gilman SR, Iossifov I, Levy D, Ronemus M, Wigler M, Vitkup D (2011) Rare de novo variants associated with autism implicate a large functional network of genes involved in formation and function of synapses. Neuron 9;70(5):898–907.

54. Pierce K, Glad KS, Schreibman L (1997) Social perception in children with autism: an attentional deficit? J Autism Dev Disord 27(3):265–82.

55. Loveland KA, Pearson DA, Tunali-Kotoski B, Ortegon J, Gibbs MC (2001) Judgments of social appropriateness by children and adolescents with autism. J Autism Dev Disord. 31(4):367–76.

56. Van Roekel E, Scholte RHJ, Didden R (2010) Bullying among adolescents with autism spectrum disorders: prevalence and perception. J Autism Dev Disord 40(1):63–73.