The educational burden of disease: a cohort study

Magnus Nordmo, Jonas Minet Kinge, Bjørn-Atle Reme, Martin Flatø, Pål Surén, Jonathan Wörn, Per Magnus, Camilla Stoltenberg, Fartein Ask Torvik

Summary

Background Students with health disorders might be at risk of disengaging from education, which can reinforce socioeconomic inequalities in health. We aimed to evaluate the associations between 176 diseases and injuries and later school performance in Norwegian adolescents and to estimate the importance of each disorder using a novel measure for the educational burden of disease (EBoD).

Methods We used diagnostic information from government-funded health services for all Norwegian inhabitants who were born between Jan 1, 1995, and Dec 31, 2002, were registered as living in Norway at age 11–16 years, and were participating in compulsory education. School performance was assessed as grade point average at the end of compulsory education at age 16 years. We used a linear regression of school performance on disease in a fixed-effects sibling comparison model (113 411 families). The association (regression coefficients) between disease and school performance was multiplied by disease prevalence to estimate the proportional EBoD among 467 412 individuals participating in compulsory education.

Findings Overall, although most diseases were not meaningfully associated with grade point average (regression coefficients close to 0), some were strongly associated (eg, intellectual disability regression coefficients –1·2 for boys and –1·3 for girls). The total educational disease burden was slightly higher for girls (53·5%) than for boys (46·5%). Mental health disorders were associated with the largest educational burden among adolescents in Norway (total burden 44·6%; boys 24·6% *vs* girls 20·0%), of which hyperkinetic disorder contributed to 22·1% of the total burden (boys 14·6% *vs* girls 7·5%). Among somatic diseases, those with unknown causes and possibly mental causes were associated with the largest educational burden.

Interpretation The EBoD concept could provide a simple metric to guide researchers and policy makers. Because mental health disorders form a large component of the educational burden, investment in mental health might be particularly important for improving educational outcomes in adolescents.

Funding The Research Council of Norway.

Lancet Public Health 2022; 7: e549–56

Centre for Fertility and Health (M Nordmo PhD, J M Kinge PhD, B-A Reme PhD, M Flatø PhD, J Wörn PhD, Prof P Magnus PhD, F A Torvik PhD), **Norwegian Institute of Public Health, Oslo, Norway; Norwegian Institute of Public Health, Oslo, Norway** (P Surén PhD, Prof C Stoltenberg PhD); **Promenta Research Center, Department of Psychology, University of Oslo, Oslo, Norway** (F A Torvik); **Department of Global Public Health and Primary Care, University of Bergen, Bergen, Norway** (Prof C Stoltenberg)

Correspondence to:
Dr Magnus Nordmo, Centre for Fertility and Health, Norwegian Institute of Public Health, Oslo 0473, Norway
magnus.nordmo@fhi.no

Introduction

Despite the considerable evidence of the effects of education on health[1] and of health on education,[2] there is little understanding of how health in adolescence is linked to school performance. Studies have typically examined single disorders, such as diabetes,[3] depression,[4] anxiety disorders,[5] attention deficit hyperactivity disorder,[6] psychotic experiences,[7] or obsessive compulsive disorder.[8] One exception is a study of Danish adolescents, which assessed the associations between common mental health disorders and school performance.[9] The study concluded that all mental health disorders had a negative impact on grade point average (GPA). However, no attempt was made to quantify the relative importance of different disorders on reducing the school performance of the population or to compare mental health disorders with somatic diseases. Hence, disease has been reported to be negatively associated with primary school performance; however, little is known about the relative effects of different diseases and groups of diseases. Furthermore, there are differences in school performance in favour of adolescent girls,[10] which remain unexplained and could be linked to health.

The burden of disease concept[11] is crucial for estimating the effects of various diseases on different populations; therefore, the notion is an important input in discussions on health policy. This concept is usually represented by a measure of healthy years of life lost due to either premature mortality or to years lived with a disability, weighted by the severity of that disability.[12] Although the concept originally indicates the loss of healthy life-years, we apply it to an educational setting by estimating the associations between various diseases and educational performance. Compared with adults, adolescents in high-income countries have low rates of mortality and severe morbidity, yet the consequences of disease on education could be severe.

Our aim was to propose a new indicator—the educational burden of disease (EBoD)—that could quantify the association between health conditions and school performance in adolescents. This concept can be used as

Research in context

Evidence before this study

The negative association between disease and school performance has been reported. However, the current literature lacks breadth, control for confounders, and representativeness. Most investigations have focused on a single disease and not allowed for comparison across diseases. Studies have rarely sought to manage confounding variables, such as socioeconomic status. Therefore, we did not know the relative importance of various mental health disorders and somatic diseases, and which disorders contributed the most to the educational disease burden in the Norwegian population.

Added value of this study

This cohort study is based on a population-wide register of school performance and health conditions to overcome data limitations in most previous studies. These data allowed us to compare the relative burden of disease across the disease spectrum. The association (regression coefficients) between disease and school performance is multiplied by prevalence to estimate the educational burden of disease (EBoD). Our results showed that mental health disorders, particularly hyperkinetic disorder, contributed the most to the EBoD.

Implications of all the available evidence

Prioritising treatment and prevention of mental health disorders in adolescence could positively affect school performance. Alternatively, strategies to improve education for adolescents with disease might yield benefits. Compared with mental illness, most somatic diseases did not substantially correlate with school performance in the Norwegian population. The concept of EBoD provides a transparent and simple metric that can guide researchers and policy makers.

an age-appropriate indicator of disease burden. Additionally, this indicator could be used in health policy debates, given that education is a modifiable social determinant of health. A precise educational estimate of the burden of disease can quantify the association between disease and education on an individual level and a societal scale.

We aimed to evaluate the associations between 176 diseases and injuries and later school performance in Norwegian adolescents, identifying the most important health conditions at the individual level; to quantify the EBoD imposed by each health condition, thus identifying the most important health conditions at the population level; and to investigate sex differences in these associations.

Methods

Study design and participants

In this cohort study, we used diagnostic information from government-funded health services for all Norwegian inhabitants who were born between Jan 1, 1995, and Dec 31, 2002, alive at age 16 years, registered as living in Norway at age 11–16 years, participating in compulsory education (ie, primary and lower secondary education), and registered with a GPA between age 15 years and 17 years. The analytical sample of 467 412 individuals (figure 1) belonged to 337 722 different family clusters, of whom 113 411 families included two or more siblings fulfilling the inclusion criteria. We identified family clusters using birth records, in which each child had received a family-unique identification number based on having the same mother and father.

Procedures

Norwegian students are evaluated at the end of 10 years of compulsory education, usually in the year they reach age 16 years. Among the 467 412 individuals with a valid GPA, 462 037 (98·9%) had the GPA registered the year they reached age 16 years (and 1488 [0·3%] in the year reaching age 15 years and 3887 [0·8%] in the year reaching age 17 years). GPA scores obtained at age 18 years or older were not used (figure 1). These grades have marks from 1 to 6, in which 6 is best. The GPA is calculated as the average of externally graded exams and a teacher-assessed grade, the latter of which can be potentially affected by teacher subjectivity in grading. The GPA score is used for ranking students who apply for admission to upper secondary education and is the most decisive early-life predictor for completion of upper secondary education.[13] Therefore, students have an incentive to perform well. Due to its relevance in further education, we chose the GPA rather than standardised test results or measures of intelligence. We standardised the GPA score (mean 0 [SD 1]) for each graduation year cohort to adjust for grade inflation and to ease interpretation.

Unlike in many other countries, it is not possible to fail the compulsory education in Norway and there is no grade retention. Even the lowest grades contribute to GPA, as well as those that would not be considered a pass at a higher level of education. Consequently, nearly all students (467 412 [95·9%] of 487 395) have a valid GPA. A school subject is marked as not evaluated if the teacher cannot evaluate the student due to absence, or in special circumstances related to learning difficulties or non-native speakers. If more than half of the subjects are not evaluated, the student will not receive a GPA score. We ran supplementary analyses replacing missing GPA scores with the lowest possible valid GPA score and tested other model specifications, including models with comorbidity and demographic adjustments.

All individuals who legally reside in Norway are assigned a general practitioner. Most patients are registered with the general practitioner because use of specialist health

care typically requires a referral from the general practitioner. The service is free of charge for children and adolescents younger than 16 years. General practitioners send billing information to The Norwegian Health Economics Administration, along with a diagnosis or reason for the visit to receive reimbursements. It is unlikely that visits to general practitioners go unreported. Diagnostic information is coded according to the International Classification of Primary Care, 2nd edn (ICPC-2; appendix p 2)[14] and is registered in a database. We had access to the database covering information from 2006 to 2019. We extracted all general practitioner visits with valid diagnostic codes occurring between the ages of 11 years and 15 years, and recorded whether each child had at least one visit recorded with each of the possible 365 diagnostic codes. We removed diagnoses of conditions with a prevalence below 0·1% in the general Norwegian population and the diagnostic codes A97 (ie, no disease) and A99 (ie, general disease not otherwise specified). 176 dichotomous variables remained, indicating who had ever visited a general practitioner for a specific reason between the ages of 11 years and 15 years.

Statistical analysis

We estimated the association ($\beta_{s,D}$) between diagnostic status of each disease (D; dummy coded as 0 or 1) and GPA separately for each sex (s) for individuals (i), using an ordinary least squares linear regression.

$$GPA_{is}=\alpha_s+\beta_{s,D}D_{is}+\varepsilon_{is}$$

This step was done separately for each diagnosis and sex, yielding 352 unadjusted bivariate coefficients. Next, we repeated this procedure, while adjusting for characteristics shared by siblings from the same family (f) using a family fixed-effects model, and controlled for birth order (BOi):

$$GPA_{isf}=\alpha_s+\alpha_f+\beta_{s,D}D_{is}+BO_i+\varepsilon_{isf}$$

where the constant (α_s) is by sex and (α_f) is by each family. The estimates from this procedure showed the statistical effect of having a diagnosis on GPA, relative to a sibling that did not have the same diagnosis. Therefore, the estimates are adjusted for experiences and risk factors that siblings from the same family share and that can affect both health and school performance as confounders. Importantly, this equation also adjusts for parental health-care seeking behaviour, and the degree to which this is stable between siblings. Additionally, we performed several sensitivity analyses to check the robustness of our main findings (appendix p 2).

The EBoD for each disorder was estimated by multiplying the prevalence (proportion of individuals affected) of each health condition (p) with an impairment weight (b) equal to the regression coefficient from the fixed-effects model. The calculation is inspired by years lived with disability, but does not incorporate duration of the disease.[15] The proportion of burden associated with a condition was obtained by dividing this estimate by the sum of burdens across all health conditions:

$$EBoD_{d,s}=\frac{p_{d,s}b_{d,s}}{\sum_{j=1}^{176}\sum_{s=0}^{1}(p_{j,s}b_{j,s})}$$

where (d) is an indicator of a specific disease, (j) includes the full set of included diseases, and (s) denominates sex. We calculated the EBoDs for each sex individually (0 for girls and 1 for boys), with separate regression coefficients and prevalences. A traditional attributable risk factor score would not allow for a continuous dependent variable. The total EBoD for an individual diagnosis is the sum of the burden for boys and the burden for girls.

We excluded diagnoses that had a positive regression coefficient in the calculation of educational burden of each disorder to eliminate their influence. This step was necessary to avoid the effects of several small positive associations counterweighting the burden of disease. A positive regression coefficient indicated that having a disease was associated with improved school

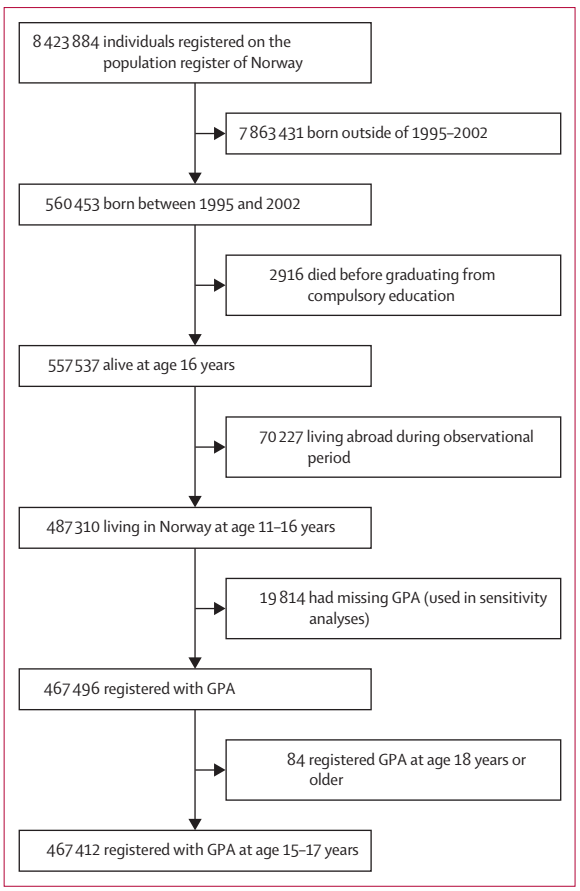

Figure 1: **Flowchart of sample selection from the population register**
GPA=grade point average.

See **Online** for appendix

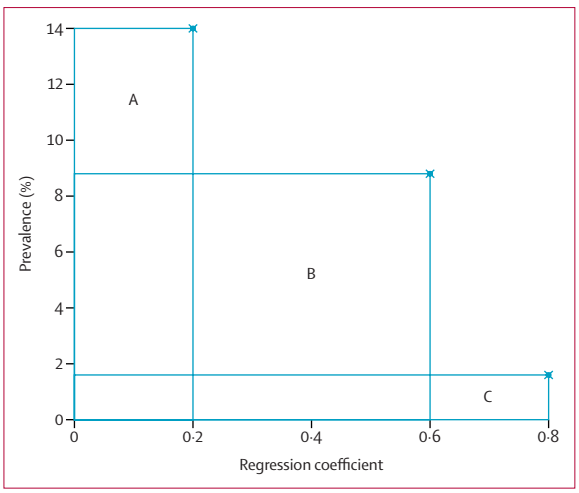

Figure 2: **Calculating the EBoD**
The EBoD was calculated as prevalence × regression coefficient. This estimate has the geometric property as the area of the rectangle. The hypothetical disease B has the highest total burden with a considerable prevalence and regression coefficient. By contrast, the product of prevalence and the coefficient is less for diseases A and C. EBoD=educational burden of disease.

performance. Importantly, all positive regression coefficients were small and could be categorised as statistical noise. The calculation of EBoD was a product of the prevalence and the regression coefficient (figure 2). We estimated 95% CIs on regression coefficients (1·96×SE) for each disorder. These two estimates were used to calculate the lower and upper bounds of the EBoD confidence interval.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

445 444 (95·3%) of 467 412 adolescents were registered with a general practitioner and had made at least one visit during the 5-year observational period, of whom 218 708 (95·6%) of 228 848 were girls and 227 058 (95·2%) of 238 564 were boys. Regression coefficients, disease prevalence, educational burdens of disease, and sex-specific educational burdens of disease are provided in the appendix (pp 3, 11–19).

Overall, most diseases have a negligible EBoD. Mental health disorders had the largest contribution to the total EBoD (44·6%; boys 24·6% *vs* girls 20·0%), followed by the endocrine, metabolic, and nutritional diseases (4·6% *vs* 4·0%), and general and unspecified disease (3·4% *vs* 5·2%; figure 3; appendix p 3). The next groups with the largest contribution to the total EBoD were neurological (3·0% *vs* 3·1%), musculoskeletal (2·9% *vs* 3·8%), respiratory (1·2% *vs* 4·8%), ear (1·7% *vs* 3·2%), urological (0·4% *vs* 3·3%), digestive (1·4% *vs* 1·8%), skin (1·5% *vs* 1·7%), and eye (1·4% *vs* 1·4%) diseases.

Most of the disease groups included one or two prominent diagnoses that contributed to most of the educational burden. The most important diagnoses, which accounted for at least 2% when combining data for boys and girls, were hyperkinetic disorder (boys 14·6% *vs* girls 7·5%), depressive disorder (1·8% *vs* 4·3%), psychological disorder not otherwise specified (3·5% *vs* 2·1%), intellectual disability (ICPC-2 diagnostic code P85; 2·6% *vs* 2·3%), vitamin or nutritional deficiency (1·9% *vs* 1·8%), cystitis or urinary infection (0·3% *vs* 2·8%), overweight (1·5% *vs* 1·3%), epilepsy (1·4% *vs* 1·4%), and musculoskeletal injury (0·9% *vs* 1·1%; figure 4; appendix pp 11–19). An exception to this pattern was the psychological group, in which all but one diagnostic codes were associated with reduced school performance. The highest burden was found in hyperkinetic disorder, which had both high prevalence (boys 5·7% *vs* girls 2·4%) and a large negative regression coefficient (–0·6 *vs* –0·7; figure 4). Eating disorders (anorexia and bulimia) was the only group that did not contribute substantially to educational burden (0·0% *vs* 0·2%).

Some diseases showed high correlations with GPA, but had a small educational burden because of low prevalence. Intellectual disability (similar to International Classification of Diseases, tenth revision [code P85]) was most negatively associated with GPA (regression coefficient –1·2 for boys *vs* –1·3 for girls); however, due to a low prevalence (0·5% *vs* 0·4%), the educational burden was moderate (2·6% *vs* 2·3%). Similarly, the regression coefficients for suicide attempt (–0·4 *vs* –0·5) and personality disorder (–0·4 *vs* –0·5) were large, yet prevalence for both was low (suicide attempt 0·1% *vs* 0·4%; personality disorder 0·1% *vs* 0·1%). The pattern of large negative associations but low prevalence was also seen in some somatic diagnoses, such as poisoning by a medical agent (regression coefficient –0·3 *vs* –0·5; prevalence 0·1% *vs* 0·3%), neurological disease (–0·3 *vs* –0·4; 0·5% *vs* 0·4%), deafness (–0·2 *vs* –0·3; 0·3% *vs* 0·2%), and congenital cardiovascular anomaly (–0·2 *vs* –0·2; 0·1% *vs* 0·1%; appendix pp 11–19). Most diseases were not meaningfully associated with GPA and had regression coefficients close to 0. The results from the supplementary analyses supported the robustness of our procedure (appendix p 2). These results were similar to the fixed-effect models.

There were substantial sex differences in school performance, whereby boys had lower GPA scores than did girls (Cohen's *d* effect size of –0·51). This difference did not change after adjusting for all 176 diagnoses, indicating that different prevalence of health conditions was not a major explanation of the sex differences in school performance. The total educational disease burden was slightly higher for girls (53·5%) than for boys (46·5%). The greater burden among girls was due to a higher mean prevalence (girls 1·7% *vs* boys 1·6%) and larger negative mean regression coefficient (–0·08 *vs* –0·05). The burden of hyperkinetic disorder

was substantial for both boys and girls; girls had a lower prevalence but a higher regression coefficient. Boys had a higher burden of psychological problems than did girls (24·6% *vs* 20·0%) due to the high impact of hyperkinetic disorder.

Discussion

Three major conclusions can be drawn for our results. First, mental illness is associated with the greatest EBoD for adolescents in Norway. By contrast, most somatic diseases do not affect educational burden due to low prevalence. Second, among diagnoses of mental illness, hyperkinetic disorder was associated with an unparalleled educational burden for both boys and girls. Third, we found that analysing variation in GPA and its association with disease resulted in a transparent metric for estimating EBoD in adolescence.

Adolescence represents a key developmental phase with considerable physical development, new social and emotional challenges, and challenges related to identity formation.[16] The effects of disease in adolescence can play a part in establishing social inequalities, given that someone's educational trajectory has a powerful impact on life outcomes.[1,17] This understanding is reflected in UNESCO's policy of health-promoting schools and its goal of promoting health among young people in the school setting.[18] We used the burden of disease framework on education to gain insight into the population perspective of adolescent health, which can help to guide policy and research. A key finding from our investigation is that mental illness dominates the EBoD. This finding reflects the observation that mental health disorders are more prevalent and have larger negative associations with GPA than do somatic diseases. Our estimate that 44·6% of the educational burden is due to mental health disorders is arguably conservative because mental health problems can also underlie somatic diseases with an unknown cause.[19] For example, vitamin deficiency and cystitis stand out as influential somatic diagnoses but have been shown to also be closely related to depression[20] and anxiety,[21] respectively. Similarly, the large negative association of poisoning by medical agent is plausibly related to self-harm.

We also found that most of the severely debilitating somatic diseases were associated with a low educational burden due to low prevalence. Previous studies have found negative effects of specific disorders,[3–7,9,22] or sets of disorders;[10] however, we are not aware of any study comparing across different classes of disorders, as presented in this study. Of note, many somatic diseases were filtered out of the analyses due to having a prevalence below 0·1%. Nevertheless, some of these diseases could be strongly related to GPA, which slightly lessens the effects of several somatic categories. The negative association between hyperkinetic disorder and GPA score is consistent with previous investigations.[7] However, this association should be cautiously interpreted because we do not account for intelligence. Some studies have found that attention deficit hyperactivity disorder was associated with lower intelligence quotient scores, whereas others have not found this association.[23] Additionally, we cannot exclude the possibility that poor school performance increases the risk of receiving a diagnosis of hyperkinetic disorder. Problems with attention and hyperactivity in school is the most common cause for referral to the specialist health services that diagnose hyperkinetic disorder. Furthermore, it is important to note that most Norwegian children diagnosed with hyperkinetic disorder also receive treatment, potentially reducing its impact. Compared with Norway, few countries in the world invest more funding in health care and educational

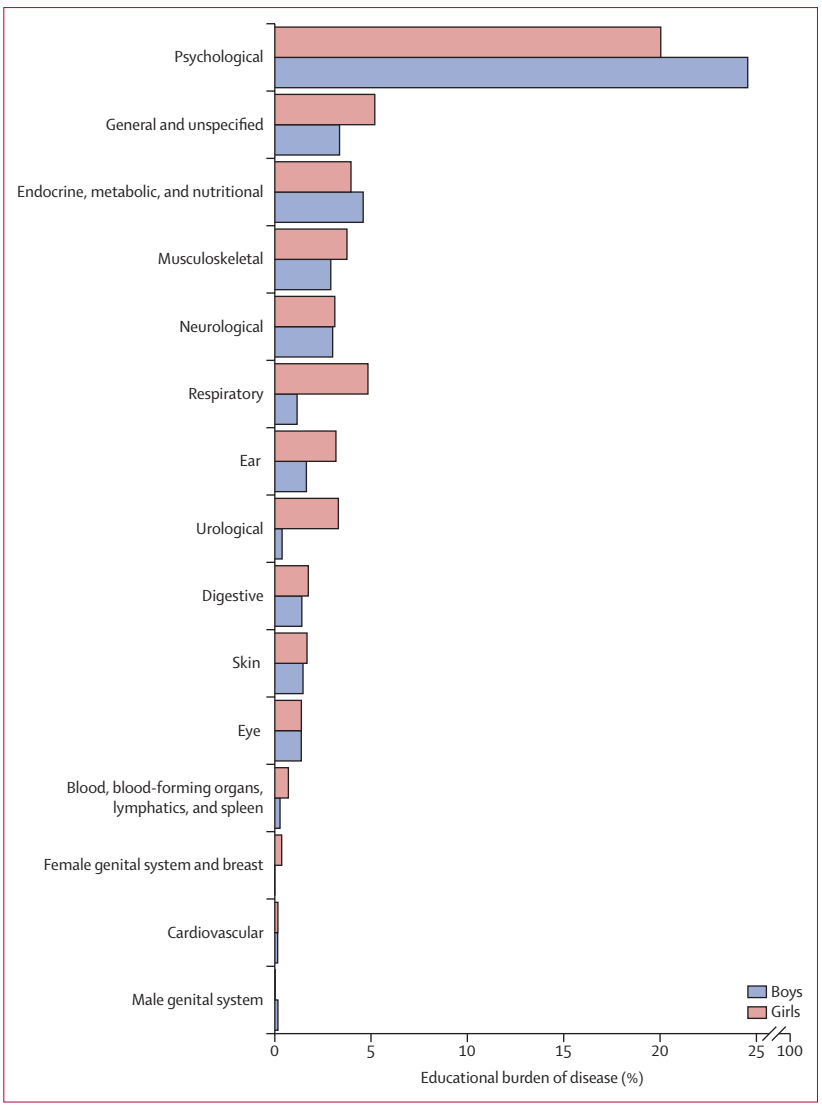

Figure 3: **Relative contributions of each ICPC-2 disease chapter to the educational burden of disease**
Disease chapters are constituted by their respective ICPC-2 diagnoses. Two chapters, pregnancy, child bearing, and family planning; and social problems, are excluded because they had an educational burden of approximately 0%. EBoD=educational burden of disease. ICPC-2=International Classification of Primary Care (2nd edn).

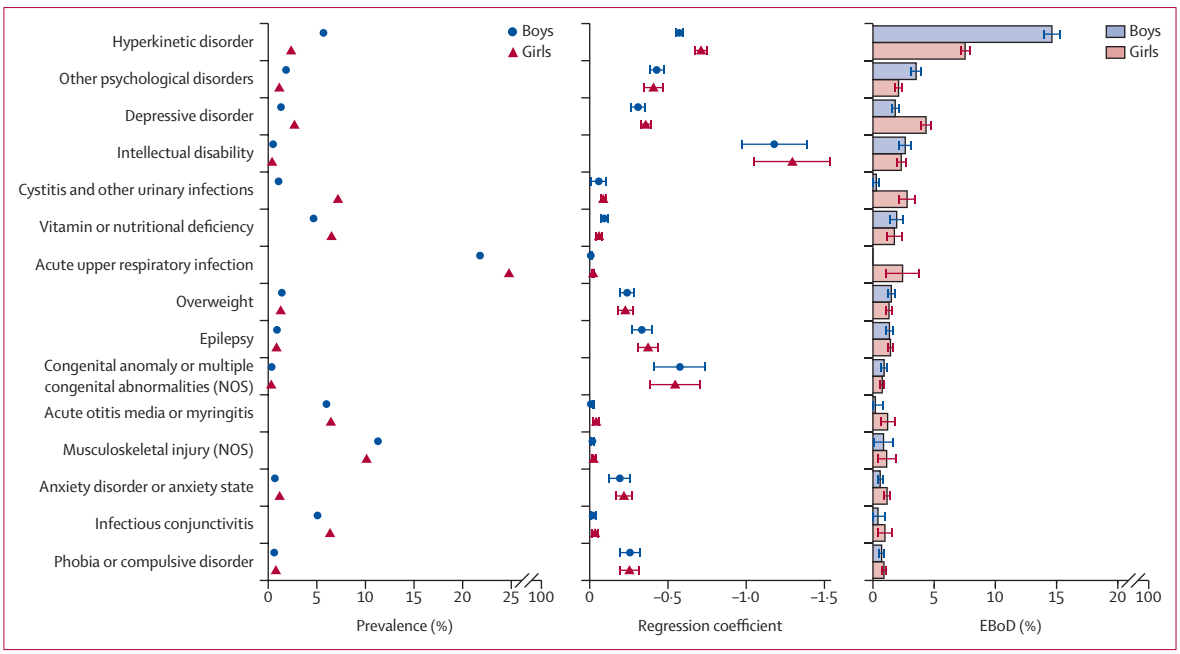

Figure 4: **Prevalence, regression coefficients, and educational burden of ICPC-2 disease chapters**
The prevalence, regression coefficients, and EBoD are provided for the 15 ICPC-2 diagnoses with the highest burden among this cohort. The EBoD is the product of prevalence and regression coefficient, divided by the relative contribution for each disease. Error bars indicate 95% CIs. EBoD=educational burden of disease. ICPC-2=International Classification of Primary Care (2nd edn). NOS=not otherwise specified.

interventions. Therefore, a higher burden of hyperkinetic disorder might be expected in countries that have less infrastructure aimed at managing the condition. Future research could apply the EBoD concept to other medical and social settings. Similarly, including the severity of disease and dose-response analyses is another useful perspective.

Adolescent development has been highlighted as a key area for developing and sustaining public health.[24,25] but there is a need for high-quality data and data analysis to make visible the challenges in adolescent health. We believe that the EBoD concept can aid this effort. Quantifying the burden of disease in terms of school performance allows for a broad, transparent, and equitable description of how disease affects adolescents. This concept can serve to guide health and educational interventions. The combination of disease prevalence and their associations with educational outcomes can also be used to analyse the effects of disease on the societal level.

Overall, our results suggest that the EBoD is greater for adolescent girls than for adolescent boys. This finding is in line with investigations showing a higher prevalence of mental health disorders and somatic disease in adolescent girls.[26] In addition, we show that the association with GPA is larger for girls, conditional on a diagnosis. Hence, the larger educational burden on girls is not simply a matter of higher disease prevalence. Hyperkinetic disorder is an exception to this pattern, with a prevalence that is more than two times higher in boys than in girls. However, our results indicate that there is a larger negative association between hyperkinetic disorder and GPA score in girls. This observation should encourage education and health policies that cover all sexes. The substantial sex differences in school performance were large in the context of educational outcomes, which can shape developmental trajectories.

Major strengths of this study are the use of a total population sample with no attrition and the use of data from health services that cover various health conditions. The association between disease and educational outcome is temporally separated with the exposure preceding the outcome.

This study addresses the two main weaknesses in the current literature on health and education. First, most studies include small and unrepresentative samples. By contrast, our results are based on population-wide registers. The registers are uniquely comprehensive with GPA measures for 96% of the Norwegian population. Furthermore, these registers include complete records of consultations with general practitioners, which are free of charge. These records allow us to address the second limitation—namely, that no previous study has covered the full range of both somatic disease and mental health disorders. By contrast, our data allowed us to evaluate the importance of each diagnosis on the population level and to compare the effects of both somatic diseases and mental health disorders on educational performance.

Nevertheless, some limitations must be noted. First, although the term EBoD implies a causal effect, the analyses are based on non-experimental associations

between disease and GPA score; therefore, we cannot establish causality. However, use of sibling fixed effects isolates the variation within families. This type of analysis allowed us to control for confounders between families that could be otherwise overlooked in traditional models, in which all confounders must be specified directly. Of note, non-shared sibling confounders, such as adverse childhood experiences and other factors that differ between siblings, cannot be accounted for in this study.[27] Additionally, the sensitivity analyses did not rely on sibling comparison but found similar results. Furthermore, we did not explore the possible effects of economic variables on the EBoD. Future research could investigate these effects further by stratifying socioeconomic status. Second, this study used diagnoses that were registered by general practitioners as part of patient treatment. Adolescents mainly receive a diagnosis when they require referral to treatment or for administrative purposes, such as registration of a parent's sick leave or need for medical reports for insurance companies. However, our use of sibling comparisons controlled for parental help-seeking behaviour to the degree that these were stable for each child in the family. Third, registered diagnoses do not capture the entirety of disease experienced by the patient; instead, they are more likely to capture the main disease that serves as the impetus for a referral or statement. This situation means that diseases that are prevalent, but seldom require or receive treatment, are undervalued in their EBoD, which can introduce bias because multimorbidity is under-reported. For instance, representative samples suggest that the prevalence of childhood obesity is 18·0%[28] and not 1·3%, as shown in our data. However, the fact that these diagnoses are rarely used by general practitioners is itself an indication of their limited effects on adolescent functioning. A further implication of low reported prevalence is that individuals with a diagnosis are unlikely to be representative of all children with the condition, which is a source of bias in the association between the disease and GPA score. If individuals with a diagnosis are more severely affected by the disease than are individuals with undiagnosed disease, overestimated disability weight might compensate for an underestimated prevalence in the EBoD; however, it is uncertain whether the two sources of bias cancel each other out. Of note, the prevalence of hyperkinetic disorder in this study is close to identical to that reported in community prevalence studies.[29] Conversely, the prevalence of internalised mental health disorders (ie, anxiety and depression) is somewhat lower.[30] Taking all factors into consideration, we cannot exclude the possibility of biases that arise due to the imperfect capture of diagnostic information. The limitations of this study warrant further investigation into the effects of mental health disorders on educational outcomes. These main conclusions should be regarded as provisional until our results are replicated using data with complete diagnostic information.

Applying the concept of EBoD to school performance has allowed for the quantification of what are possibly the most important health drivers of educational inequality in the Norwegian population. This framework has shown that mental health disorders, particularly hyperkinetic disorder, represent the largest EBoD in adolescence. Effective treatment or prevention of mental health disorders in adolescence might positively affect school performance and reduce socioeconomic differences in health. Additionally, educational interventions could seek to minimise the impact of mental health disorders in this age group.

Contributors

FAT conceived the EBoD concept. All authors contributed to a collaborative effort to initialise the main idea behind the concept, to write and revise the manuscript, and to interpret the results. MN and FAT conceptualised the study, formalised the research project, evaluated the results, responded to reviewers, verified the data, and carried out the main data analyses. JMK, B-AR, MF, PS, JW, PM, and CS formalised the research project, evaluated the results, and responded to reviewers. All authors had full access to all the data in the study. MN had final responsibility for the decision to submit for publication.

Declaration of interests

All authors declare no competing interests.

Data sharing

The data for this study encompass educational outcomes and primary care records for entire cohorts of the Norwegian population. Researchers can access the data by application to the Regional Committees for Medical and Health Research Ethics and the data owners (Statistics Norway and the Norwegian Directorate of Health). The authors cannot share these data with other researchers due to the sensitive nature and potential for identification. However, other researchers can contact the authors if they have questions concerning the data or overlapping research projects.

Acknowledgments

This work was supported by the Research Council of Norway (grant numbers 273659, 300668, and 262700). This work was partly supported by the Research Council of Norway through its Centers of Excellence funding scheme (grant number 262700). We wish to thank Øystein Kravdal, Kåre Bævre, and Fredrik Swift for their excellent support and guidance.

References

1 Davies NM, Dickson M, Davey Smith G, van den Berg GJ, Windmeijer F. The causal effects of education on health outcomes in the UK Biobank. *Nat Hum Behav* 2018; **2:** 117–25.
2 Leopold L. Education and physical health trajectories in later life: a comparative study. *Demography* 2018; **55:** 901–27.
3 Skipper N, Gaulke A, Sildorf SM, Eriksen TM, Nielsen NF, Svensson J. Association of type 1 diabetes with standardized test scores of Danish schoolchildren. *JAMA* 2019; **321:** 484–92.
4 Thapar A, Collishaw S, Pine DS, Thapar AK. Depression in adolescence. *Lancet* 2012; **379:** 1056–67.
5 de Lijster JM, Dieleman GC, Utens EMWJ, et al. Social and academic functioning in adolescents with anxiety disorders: a systematic review. *J Affect Disord* 2018; **230:** 108–17.
6 Arnold LE, Hodgkins P, Kahle J, Madhoo M, Kewley G. Long-term outcomes of ADHD: academic achievement and performance. *J Atten Disord* 2020; **24:** 73–85.
7 Steenkamp LR, Bolhuis K, Blanken LME, et al. Psychotic experiences and future school performance in childhood: a population-based cohort study. *J Child Psychol Psychiatry* 2021; **62:** 357–65.
8 Pérez-Vigil A, Fernández de la Cruz L, Brander G, et al. Association of obsessive-compulsive disorder with objective indicators of educational attainment: a nationwide register-based sibling control study. *JAMA Psychiatry* 2018; **75:** 47–55.

9 Dalsgaard S, McGrath J, Østergaard SD, et al. Association of mental disorder in childhood and adolescence with subsequent educational achievement. *JAMA Psychiatry* 2020; **77:** 797–805.

10 Stoet G, Geary DC. Gender differences in the pathways to higher education. *Proc Natl Acad Sci USA* 2020; **117:** 14073–76.

11 Vos T, Barber RM, Bell B, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 2015; **386:** 743–800.

12 Lopez AD, Murray CC. The global burden of disease, 1990–2020. *Nat Med* 1998; **4:** 1241–43.

13 Markussen E, Frøseth MW, Sandberg N, Lødding B, Borgen JS. Early leaving, non-completion and completion in upper secondary education in Norway. In: Lamb S, Markussen E, Teese R, Polesel J, Sandberg N, eds. School dropout and completion. Dordrecht: Springer, 2011.

14 Verbeke M, Schrans D, Deroose S, De Maeseneer J. The International Classification of Primary Care (ICPC-2): an essential tool in the EPR of the GP. *Stud Health Technol Inform* 2006; **124:** 809–14.

15 Devleesschauwer B, Havelaar AH, Maertens de Noordhout C, et al. Calculating disability-adjusted life years to quantify burden of disease. *Int J Public Health* 2014; **59:** 565–69.

16 Becht AI, Nelemans SA, Branje SJT, et al. The quest for identity in adolescence: heterogeneity in daily identity formation and psychosocial adjustment across 5 years. *Dev Psychol* 2016; **52:** 2010–21.

17 Galama T, Lleras-Muney A, Kippersluis H. The effect of education on health and mortality: a review of experimental and quasi-experimental evidence. Oxford: Oxford University Press, 2018.

18 WHO, UNESCO. Making every school a health-promoting school: global standards and indicators. Geneva: World Health Organization and the United Nations Educational, Scientific and Cultural Organization, 2021.

19 Bohman H, Låftman SB, Cleland N, Lundberg M, Päären A, Jonsson U. Somatic symptoms in adolescence as a predictor of severe mental illness in adulthood: a long-term community-based follow-up study. *Child Adolesc Psychiatry Ment Health* 2018; **12:** 42.

20 Vulser H, Wiernik E, Hoertel N, et al. Association between depression and anemia in otherwise healthy adults. *Acta Psychiatr Scand* 2016; **134:** 150–60.

21 McKernan LC, Walsh CG, Reynolds WS, Crofford LJ, Dmochowski RR, Williams DA. Psychosocial co-morbidities in interstitial cystitis/bladder pain syndrome (IC/BPS): a systematic review. *Neurourol Urodyn* 2018; **37:** 926–41.

22 Steenkamp LR, Bolhuis K, Blanken LME, et al. Psychotic experiences and future school performance in childhood: a population-based cohort study. *J Child Psychol Psychiatry* 2021; **62:** 357–65.

23 Herrera-Gutiérrez E, Gómez-Amor J, López-Ortuño J, Navarro-Noguera M, Villanueva-Blasco VJ. Cognitive and personality differences between adolescents with and without attention deficit/hyperactivity disorder. *Acta Psychol (Amst)* 2021; **219:** 103386.

24 Patton GC, Sawyer SM, Santelli JS, et al. Our future: a *Lancet* commission on adolescent health and wellbeing. *Lancet* 2016; **387:** 2423–78.

25 Resnick MD, Catalano RF, Sawyer SM, Viner R, Patton GC. Seizing the opportunities of adolescent health. *Lancet* 2012; **379:** 1564–67.

26 Sweeting H. Reversals of fortune? Sex differences in health in childhood and adolescence. *Soc Sci Med* 1995; **40:** 77–90.

27 Frisell T, Öberg S, Kuja-Halkola R, Sjölander A. Sibling comparison designs: bias from non-shared confounders and measurement error. *Epidemiology* 2012; **23:** 713–20.

28 Biehl A, Hovengen R, Grøholt EK, Hjelmesæth J, Strand BH, Meyer HE. Parental marital status and childhood overweight and obesity in Norway: a nationally representative cross-sectional study. *BMJ Open* 2014; **4:** e004502.

29 Mohammadi MR, Zarafshan H, Khaleghi A, et al. Prevalence of ADHD and its comorbidities in a population-based sample. *J Atten Disord* 2021; **25:** 1058–67.

30 Erskine HE, Baxter AJ, Patton G, et al. The global coverage of prevalence data for mental disorders in children and adolescents. *Epidemiol Psychiatr Sci* 2017; **26:** 395–402.