De Ridder Karin

Adolescent health and the risk of school dropout and labour market exclusion in young adulthood:

A prospective family study of the Young-HUNT1 cohort

Thesis for the degree of Philosophiae Doctor

Trondheim, April 2014

Norwegian University of Science and Technology
Faculty of Medicine
Department of Public Health and General Practice

NTNU – Trondheim
Norwegian University of
Science and Technology

NTNU
Norwegian University of Science and Technology

Thesis for the degree of Philosophiae Doctor

Faculty of Medicine
Department of Public Health and General Practice

ISBN 978-82-326-0130-1 (printed ver.)
ISBN 978-82-326-0131-8 (electronic ver.)
ISSN 1503-8181

Doctoral theses at NTNU, 2014:103

Printed by NTNU-trykk

In memory of my dad,
Frans De Ridder
(1947-2009)

There are two things
we should give
our children:
One is roots.
The other is wings.

Hodding Carter, Jr
(borrowed from Henry W. Beecher)

Helse i ungdomstida og risiko for skolefrafall og arbeidsekskludering i ung voksen alder: en prospektiv familiestudie basert på Ung-HUNT1-kohorten.

I denne ph.d. avhandlingen har jeg undersøkt sammenhengen mellom helse og helserelatert sårbarhet i ungdomstida, skolefrafall og mottak av langvarige trygdeytelser i ung voksen alder. I tillegg undersøkte jeg hvordan disse sammenhengene var influert av foreldrenes sosioøkonomi og familien som kontekst mer generelt.

Studiene i avhandlingen tok utgangspunkt i den første Ung-HUNT (Helseundersøkelsen i Nord-Trøndelag) som foregikk i perioden 1995-97. Opplysninger om selvrapportert helse og livsstil fra nesten 9000 ungdommer (13-19 år) ble koblet til nasjonale registerdata. Slik var det mulig å få opplysninger om deltakernes utdanning og mottak av trygdeytelser i ung voksen alder, samt familiemedlemmer (foreldre og søsken). Alle studier er basert på kvantitative analyser.

I vår studiepopulasjon hadde nesten én av fem ungdommer ikke fullført videregående skole i en alder av 24 år. Det var en sterk sammenheng mellom helse (kroniske somatiske sykdommer og symptomer, symptomer på angst og depresjon, konsentrasjonsproblemer, insomni, selvrapportert dårlig helse, overvekt og fedme) og skolefrafall, uavhengig av justering for foreldrenes sosioøkonomiske posisjon. Disse resultatene ble også understøttet av analyser av søsken med ulik helsestatus (med unntak av en svak sammenheng mellom skolefrafall og kroniske somatiske sykdommer og symptomer på angst og depresjon blant søsken). Risikoen for skolefrafall var sterkt assosiert mellom søsken.

Om lag én av fem ungdommer var registrert med langvarige trygdeytelser i løpet av en femårsperiode mellom 24-28 år. Langvarige trygdeytelser ble definert som helserelaterte ytelser i mer enn seks måneder i løpet av ett kalenderår (sykmelding, rehabiliteringspenger, attføringspenger, uførepensjon) eller ikke-helserelaterte ytelser (dagpenger og sosialstønad). Ungdom som ikke hadde fullført videregående skole hadde en tre ganger så høyt risiko for å motta langvarige trygdeytelser sammenlignet med ungdom med fullført videregående skolegang. Også dårlig helse i ungdomstida økte risikoen for både medisinske og ikke-medisinske ytelser i ung voksen alder, uavhengig foreldrenes utdanningsnivå. Skolefrafall var

også sterkt assosiert med mottak av langvarige medisinske ytelser, til tross for en omfattende justering for ungdommens sårbarhet målt med foreldres sosioøkonomiske posisjon og ungdommens helse, helserelatert atferd, psykososiale faktorer og skoleproblemer. Risiko for mottak av langvarige medisinske trygdeytelser var også betydelig større for søsken som ikke fullførte videregående skole, sammenliknet med søsken som fullførte.

Denne avhandlingen bidrar til kunnskap om helserelaterte seleksjonsmekanismer i overgangen mellom ungdomstida og arbeidsliv i ung voksen alder. Våre funn viser at ungdom som ikke fullfører videregående skole har klart forhøyet risiko for å motta medisinske og ikke-medisinske trygdeytelser i ung voksen alder. Resultatene bygger videre opp under betydningen av tiltak for å forebygge skolefrafall så langt det er mulig, kombinert med økt oppmerksomhet på arbeidsintegrering og målrettet støtte for dem som ikke fullfører videregående skole. Resultatene gir også støtte til at ungdomshelse bør ha en sentral plass i folkehelsearbeidet.

Cand. med. Karin De Ridder
Institutt for samfunnsmedisin, NTNU

Hovedveileder: professor Johan Håkon Bjørngaard
Biveiledere: professor Roar Johnsen, professor Turid Lingaas Holmen, professor Steinar Westin

Ovennevnte avhandling er funnet verdig til å forsvares offentlig
for graden PhD i samfunnsmedisin.
Disputas finner sted i Auditoriet, Medisinsk-teknisk forskningssenter
tirsdag 8. april , kl. 12.15

Table of Contents

Acknowledgement

This thesis is based on work carried out at the Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology (NTNU) during the period 2010-2014. The work was financed by the Faculty of Medicine, NTNU. Additionally, I have received financial support from the Department of Research and Development, Nord-Trøndelag Health Trust, to publish paper I and II.

The Nord-Trøndelag Health Study (The HUNT Study) is a collaboration between HUNT Research Centre (Faculty of Medicine, NTNU), Nord-Trøndelag County Council, Central Norway Health Authority and the Norwegian Institute of Public Health. The thesis is based on data from the first Young-HUNT Study (Young-HUNT1) and I thank the HUNT Research Center for providing the data. I wish to thank all the participants of the Young-HUNT and HUNT Study, whose participation has made this study possible. This research was also only feasible thanks to the availability of high quality national register data, smoothly provided by Statistics Norway.

Further, I wish to express my gratitude to the following individuals for their contribution to this research work:
Professor Johan Håkon Bjørngaard, my supervisor, for your excellent supervision, enthusiasm and for always having the time to answer my questions. Thank you for guiding me in the field of epidemiology and scientific writing. Without your challenge for methodological renewal, this work would not have reached the same quality.
Professor (emeritus) Roar Johnsen, my co-supervisor, for introducing me to the world of public health, epidemiology and teaching. Thank you for believing in me and for your constructive criticism to my work.
Professor Turid Lingaas Holmen and Professor Steinar Westin, my co-supervisors, for critical reviewing my manuscripts.
Kristine Pape, PhD MD and co-author, for teaching me STATA software with your very helpful do-files and for carefully reviewing my manuscripts. Your previous work within this scientific field and previous data processing created an optimal start for my PhD engagement.

I want to express my deepest respect for Professor Jostein Holmen and Professor Turid Lingaas Holmen (and many others) for their contribution to and the achievements in the Young-HUNT and HUNT Surveys. Professor Jostein Holmen opened for me the doors to the field of epidemiology in 2007 when I contacted him with the question whether I could use The HUNT Survey to give policy advice to my former employers, Levanger and Verdal municipality.

I am thankful to my former employer, Department of Rehabilitation and Physical Medicine, Nord-Trøndelag Health Trust, Levanger who gave me the chance to start with my PhD

parallel with clinical practice. I will always remember the friendly atmosphere and the excellent teamwork.

Special thanks to Professor Karel Hoppenbrouwers, head of Youth Health Care, Department of Public Health and Primary Care, KU Leuven, Belgium for giving me the opportunity to continue my scientific work at his unit in the period 2013-2014.

I am very grateful to my colleagues at ISM (Institutt for samfunnsmedisin) in Norway and Youth Health Care in Belgium for their support and friendship.

Finally, last but not least, thanks to the most important cornerstone in my life: my family. I am very grateful to my husband Koenraad Cuypers, PhD MSc PT, for seducing me to the land of snow and ice, midnight light and darkness, sled dogs, horses, sheep and farming, hiking and fishing… Your enthusiasm and initiative made me discover and develop my (un)known talents: who would believe that horses could pave the way to research and our first scientific publication? Thank you for your critical review of my third paper, but above all: thank you for your love and support.

Special thanks to our children Stijn and Lieselotte for their unconditional love and for putting all the work and the content of this work in perspective. Thanks to Koenraad´s children, Jochem and Hannelore and their families, for their support and for being fine role models for Stijn and Lieselotte.

Thanks to my parents for encouraging education and I will always remember all they have resigned to give me and my sisters the best possible start in life.

Finally, on behalf of myself, Koenraad and the children, I want to thank our “norsk bestemor” Gjertine Skjesol for practical support and warm friendship.

Karin De Ridder, Rillaar (Belgium), November 2013

List of abbreviations

ADHD	Attention Deficit and Hyperkinetic Disorder
ATT	Average treatment effect on the treated
BMI	Body mass index
CI	Confidence interval
FD-Trygd	Forløpsdatabase Trygd (event database social security)
HBSC	Health Behaviour in School-aged Children study
HUNT	Helseundersøkelse i Nord-Trøndelag
ICC	Intraclass correlation coefficient
MICE	Multiple imputation by chained equations procedures
MOR	Median odds ratio
NAV	Ny Arbeids- og Velferdsforvaltning (Norwegian Labour and Welfare Administration)
NUDB	Nasjonal Utdanningsdatabase (Norwegian National Education Database)
NEET	Persons “neither in education nor in employment or training”
OECD	Organisation for Economic Cooperation and Development (international economic organization of 34 countries founded in 1961 to stimulate economic progress and world trade)
OR	Odds ratio
PAR	Population attributable risk
RD	Risk difference
SCL-5	Hopkins Symptom Checklist, 5-item short version
SEP	Socioeconomic position
SSB	Statistisk Sentralbyrå (Statistics Norway)

List of papers

This thesis is based on the following original papers, which will be referred to in the text by their Roman numerals.

Paper I

De Ridder KA, Pape K, Johnsen R, Westin S, Holmen TL, Bjørngaard JH. School dropout: a major public health challenge: a 10-year prospective study on medical and non-medical social insurance benefits in young adulthood, the Young-HUNT 1 Study (Norway). *J Epidemiol Community Health* 2012; 66: 995-1000.

doi: 10.1136/jech-2011-200047

Paper II

De Ridder KA, Pape K, Johnsen R, Holmen TL, Westin S, Bjørngaard JH. Adolescent health and high school dropout: A prospective cohort study of 9000 Norwegian adolescents (The Young-HUNT). *PLoS One* 2013; 8: e74954.

doi:10.1371/journal.pone.0074954

Paper III

De Ridder KA, Pape K, Cuypers K, Johnsen R, Holmen TL, Westin S, Bjørngaard JH. High school dropout and long-term sickness and disability in young adulthood: a prospective propensity score stratified cohort study (the Young-HUNT study). *BMC Public Health* 2013; 13: 941.

doi:10.1186/1471-2458-13-941

Summary

Background

High school dropout and problematic work integration in young adulthood represent an individual hazard and a societal challenge in most Western countries. Health problems with varying degree of functional limitations are common in adolescence, and there is some evidence to suggest a higher risk of sickness and disability among high school dropouts compared to high school completers. Prospective studies of the complex role of adolescent health, socioeconomic factors and family context for completion of secondary school and subsequent problematic labour market integration due to ill health are rare.

Aims

The main objective of the thesis was to study, with a prospective design, the associations between adolescent health, high school dropout and long-term social insurance benefits in young adulthood. We investigated whether adolescent health and health-related vulnerability confounded the association between school dropout and long-term social insurance benefits. We studied the associations between several dimensions of self-reported health in adolescence and school dropout. For all these associations, we assessed the contribution of socioeconomic factors and the family context.

Methods

Our study population was 8795 school attending adolescents (13-21 years) who participated in the Young-Hunt1 Survey in the county Nord-Trøndelag, Norway (1995-1997). The information of the questionnaire was combined with national register based information on education and social insurance benefits from 1998 to 2007/2008. High school dropout or completion was defined with the Norwegian National Education Database (NUDB) in the

calendar year the participant turned 24 years old. We recorded long-term social insurance benefits, such as long-term (more than 180 days/calendar year) sickness absence, rehabilitation benefits, disability benefits, long-term unemployment, and social insurance support in the Norwegian Labour and Welfare registers (FD-Trygd). Parental information on socioeconomic position and linkage of siblings was obtained by the NUDB, FD-Trygd and the HUNT2 survey. The main adolescent health exposures from Young-Hunt1 Survey were chronic somatic disease, somatic symptoms, psychological distress, concentration difficulties, insomnia, self-reported health and overweight. Common risk factors that could confound the association between dropout and benefits were the various adolescent health dimensions, health-related behaviours, psychosocial factors, school problems, and parental socioeconomic position. Health-related vulnerability for dropout was estimated with a propensity score for high school dropout based on these common risk factors.

We assessed the associations between the different adolescent health dimensions and school dropout, and between school dropout and long-term social insurance benefits with descriptive statistics and various logistic regression models, both in the whole population and among siblings.

Results

In the Young-HUNT1 cohort, a total of 17% was registered as high school dropouts at age 24. The predicted 5-year risk for receiving long-term social insurance benefits between ages 24-28 was 21% (95% CI 20% to 23%). High school dropouts had about a 3-fold higher risk for receiving long-term benefits and poor self-rated health was associated with receiving both medical and non-medical benefits in the models adjusted for parental socioeconomic position. (Paper I)

All explored health dimensions were strongly associated with high school dropout, also in the models adjusted for parental socioeconomic position. The results were confirmed in the analyses comparing differentially exposed siblings, with the exception for chronic somatic disease and psychological distress. School dropout was strongly clustered within families. (Paper II)

High school dropouts have a strongly increased risk for long-term sickness benefits and disability pension between ages 24-29 across all vulnerability or risk factor level for school dropout, i.e. independent of own health, family and socioeconomic factors in adolescence. Strong associations between dropout and receipt of benefits were also present when comparing siblings. (Paper III)

Conclusions

High school dropout and receipt of long-term social insurance benefits in young adulthood are substantive problems both for the individual and society. All examined dimensions of health in adolescence were associated with school dropout. High school dropout increased the risk for sickness and disability regardless of the risk factor level present for school dropout. Although the family context was very important for both dropout and receipt of benefits, most associations were confirmed in sibling comparisons. Future research on educational attainment and labour market exclusion in young adulthood should also have attention to health-related mechanisms. For policy, our findings support the importance of prevention of school dropout where possible, in combination with increased attention to labour market integration and targeted support for those who fail to complete high school. The results also support that adolescent health should have a central role in public health policy.

1 Introduction

As stated in the first line of the protocol of this PhD-project, the main aim was to investigate "health related factors in adolescence, accomplishment of secondary school, and work inclusion in young adulthood". This broad research topic arised based on the recognition that the population receiving disability pension in the Nordic countries was growing and in Norway, the relative growth in disability pension was highest for the youngest adult age groups. At the same time, a review on the epidemiological research on disability benefits in Nordic countries revealed that most research focused on the older adult age groups and that this research mainly focused on individual risk factors.[1] However, disability pension before the age of 30 years still remains a rather rare phenomenon (less than 1.5% of that age group population) and the majority is related to serious diagnoses such as schizophrenia, mental retardation, congenital malformations and neural disorders.[2] The risk of work disability increases rapidly after the age of 40-50 years, and disability is often the final result of long-term problematic labour market integration process related to poor health.[3 4] At the start of this doctoral project in 2010, there was a sparse literature on young adults on the relation between adolescent health and high school completion or the possible interaction between adolescent health and high school completion in relation to adult health and work integration. However, a Norwegian study showed that the population of young adults at risk for long-term health-related labour market exclusion might be larger than the hard endpoint "disability pension" suggested.[5]

Problematic labour market integration has a multifactorial aetiology.[1 6-8] Both a life course and an eco-social framework with their respectively temporal and hierarchical approach are crucial to understand the trajectories into adulthood (Figure 1.1).[9-11] Adolescence is a critical phase during the life course, when there might be potentials for interventions in the health

services as well in the educational system. We know from previous studies that completion of high school is associated with better adult health, increased work opportunities and reduced risk of labour market exclusion.[12-16] There is, however, limited knowledge on the individual health-related vulnerability of high school dropouts and completers, as recorded by self-reported health, specific diseases, health-related behaviour, and psychosocial risk factors in adolescence.[17] The eco-social framework defines besides the *individual* level also three other levels: the *interpersonal* level with family, friends and peers, the *community* level including schools, neighbourhoods and work places, and the *societal* level such as the economic, social, educational, and political systems. In addition to individual vulnerability, contextual factors, such as family background, geography, work place and time periods, have impact on the risk of later labour market exclusion.[1] Hence, an important part of this thesis is to investigate how individual vulnerability might be modified by a specific context, namely the family background.

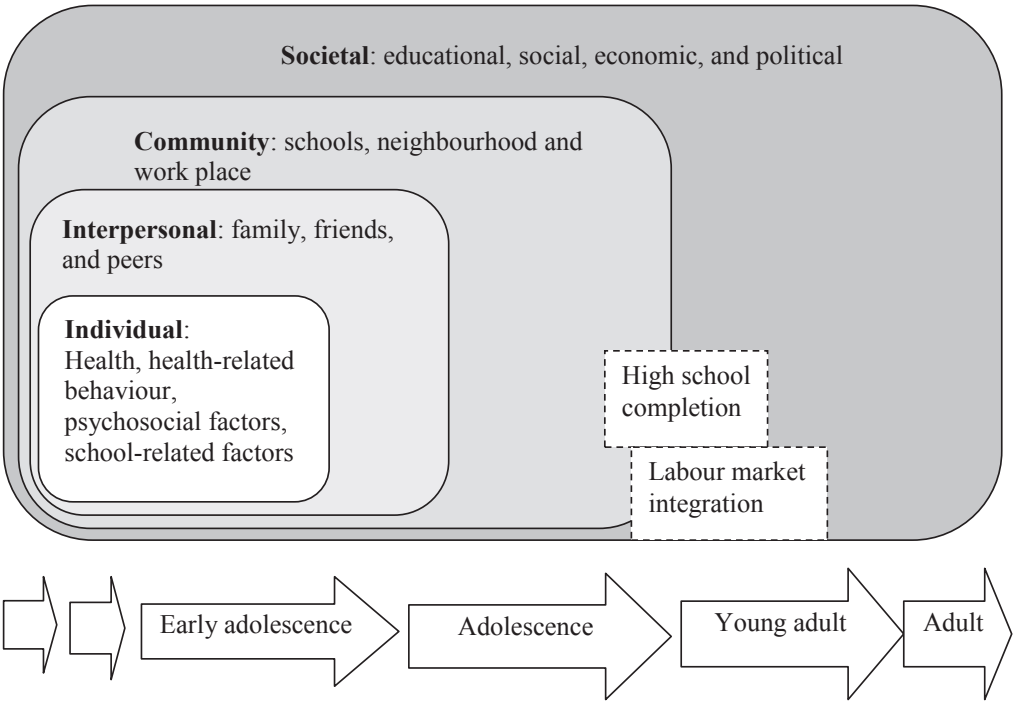


Figure 1.1 Schematic overview, adapted from Bronfenbrenner (1977).[11]

2 Background

2.1 Key concepts

2.1.1 Adolescence and future health

According to a definition by the World Health Organisation (WHO), adolescence can be defined as the period between ages 10 and 19 years, which can be subdivided into early adolescence (age 10-14 years) and late adolescence (age 15-19 years).[18] About 12% of the population in Norway is between ages 10-19 years (www.ssb.no/statistikkbanken/).

A life course epidemiology approach implies to study the long-term effects on later health or disease risk of physical or social exposures during gestation, childhood, adolescence, young adulthood and later adult life.[19] Earlier determinants are known to be related to aspects of adolescent health, but less emphasis has been placed on how health outcomes in adolescence may have a sustained effect on the future health of this young people. Sawyer et al.[20] visualized in a recent article in the Lancet how the life course perspective on adolescent health (horizontal flow) and social determinants of health (vertical flow) can be united (Figure 2.1).

Adolescence, starting with the onset of puberty, is a period characterized by rapid physical growth, sexual maturation, and high importance of peer relationships and ends with key social-role transitions such as completion of education, employment, marriage and/or childrearing. Adolescence is a sensitive period not only because the timing of puberty is associated with the increase of health-related behaviours, but also because of a rise in psychosocial health problems.[21] However, the age-gap between the achievement of physical maturity and social-role maturity has widened substantially the last 4-5 decades.

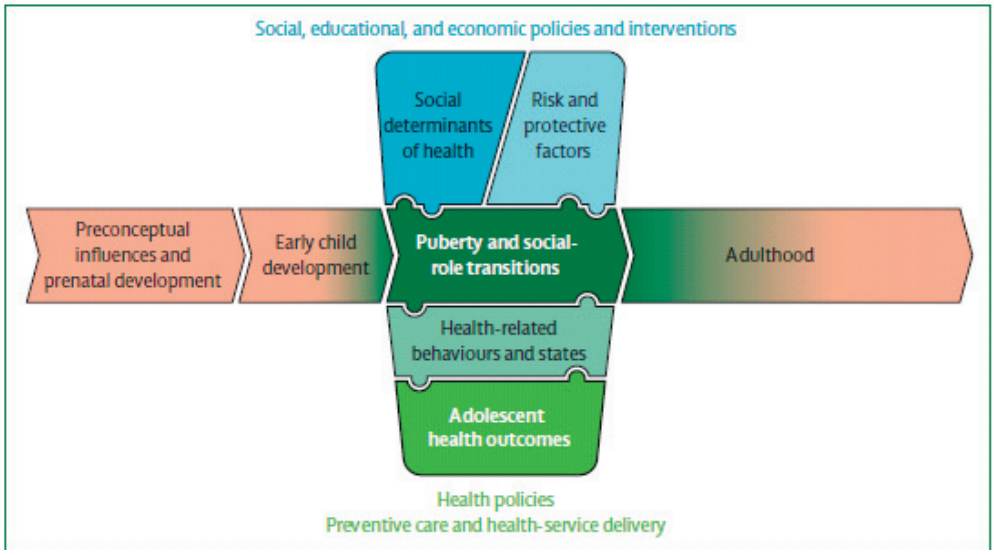


Figure 2.1 The conceptual framework for adolescent health based on the life course perspective and the importance of the social determinants of health by Sawyer et al.[20]

The vertical axis (the social determinants of health) intersects around the unique characteristics of adolescence (puberty and social-role transitions) to emphasize how these factors affect adolescent health-related behaviour and states.[22] The WHO has defined the social determinants of health as "the conditions in which people are born, grow, live, work and age".[12] These conditions are shaped by interpersonal relations (families, peers) and communities (schools, work place) and by the structural determinants such as the distribution of national wealth, access to education and health care. Risk and protective factors operate within the individual and their family, peers, and school.

The complex interaction between the physical changes of puberty, the social-role transitions, social determinants of health, and risk and protective factors can result in highly heterogeneous life experiences and health outcomes for adolescents within the same country.

2.1.2 Labour market exclusion

The end of adolescence usually is characterized with the completion of an education, and with access to a social institution, the labour market. In most OECD countries, graduation from secondary school is viewed as the minimum level of educational attainment needed for successful participation of young people in further study and work (Figure 2.2).[23] In most nations, secondary education is needed to enter university (college) and it also prepares for labour market entry. Low educational attainment is associated with general health impairment, lower work participation and higher work disability.[5 13 15]

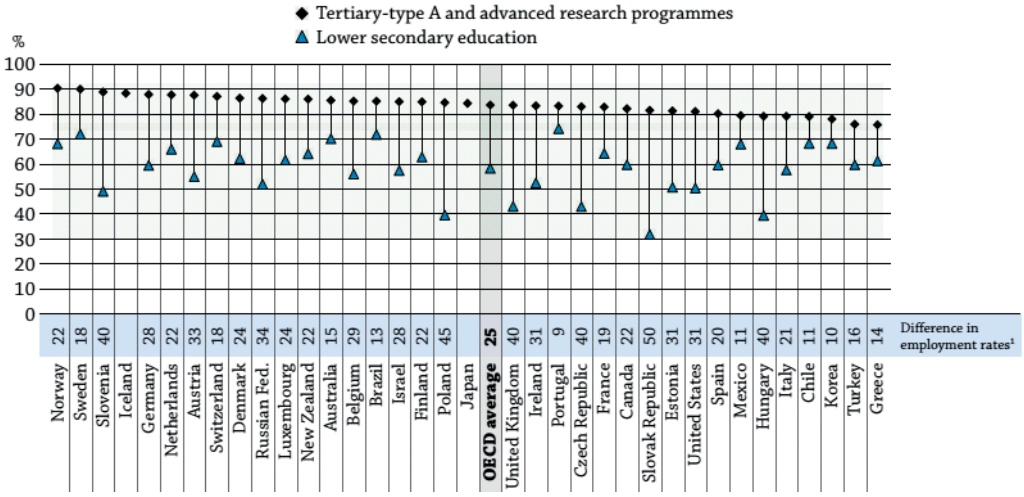


Figure 2.2 The difference in employment (in percentage points) between tertiary-educated adults and those with only lower secondary education (2011). (Source: OECD, Education at a glance 2013, page 76[23]).

Not everybody is able to fully participate in the labour market, as a result of unemployment, ill health, disability or other factors. In these situations, the general term "social exclusion" (refers to resource deficiencies in various components of personal welfare such as

employment, education, health, etc.) or more specific "labour market exclusion" are often used in the literature.[24] The term "exclusion" means "the act of forcing someone out or not allowing someone to take part in an activity". People are excluded not just because they are currently without a job or income, but because they have little prospects for the future.[25]

Although research has indicated increased vulnerability among young adults with short breaks from work,[5] such breaks are not the scope of our research. We were specifically interested in labour market exclusion related to ill health. Long-term medical benefits such as sickness benefits, rehabilitation or disability pension are conditionally associated with ill health and might reflect problems with sustaining labour market integration because of ill health. Health problems in childhood and adolescence are associated with such subsequent work disability.[13] Additionally, chronic disease in childhood and adolescence might affect either entrance into or maintenance on the labour market and in this way increase also the risk for non-medical benefits such as unemployment or social insurance support.[15]

Since high school dropout and impaired health in adolescence seem to be associated with higher risk for health-related labour market exclusion, it is important to consider both factors and their interaction during the transition from adolescence to young adulthood with a life course perspective.[26]

2.1.3 Pathways to health-related labour market exclusion

Graham and Power have integrated the literature based knowledge on social inequalities of health into a life-course framework (Figure 2.3).[27] Social inequalities in health denote the situation when poor health is distributed unequally across socioeconomic groups: worse

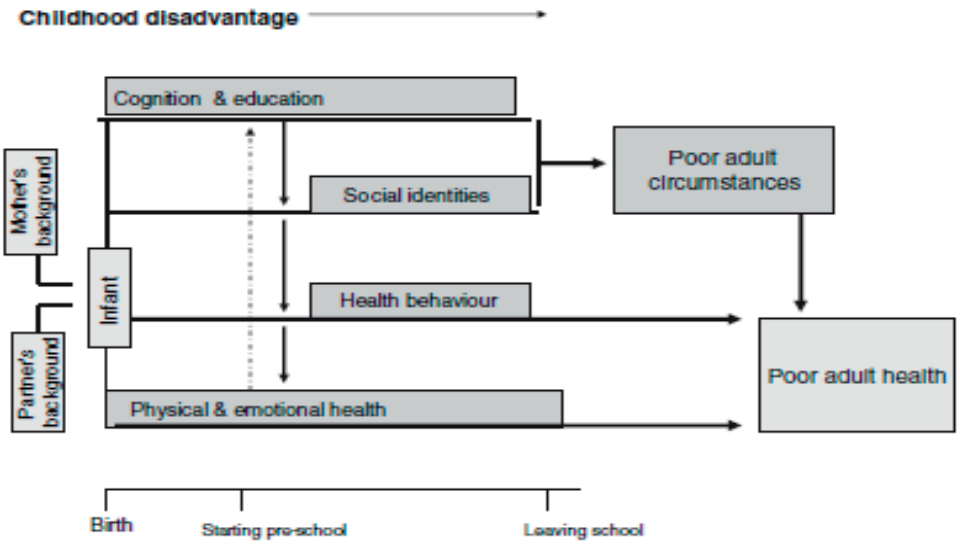


Figure 2.3 Lifecourse framework linking poor childhood circumstances to poor adult health. (Source: Graham H and Power C, 2004[27]).

health for those who are at a lower level of socioeconomic position (measured by educational attainment, income, occupation).[28] It should be noted that it is not the objective of this thesis and papers to study the presence of social inequalities of *health* per se. However, since there is a strong association between health and long term sickness absence/disability pension, it is reasonable to hypothesize similar effects as presented in the life course framework by Graham and Power.

In this schematic representation, childhood disadvantage affects both socio-economic circumstances (such as education, employment, wealth) and health in adulthood through several key processes: developmental health (physical, emotional and cognitive) and health behaviours, together with educational and social trajectories. In the view of the papers included in this thesis, some comments to this schematic representation are added.

First, the social causation theory has been accepted as a major explanation for social inequalities in health. It refers to the concept that socioeconomic conditions are defined as the origin of health distribution.[29] There is a huge amount of articles related to this theory with many different definitions for the socioeconomic conditions, including a conceptual overlap of socioeconomic position (SEP) and the social determinants of health.[30] In paper I, we used the term "socioeconomic status", but we decided to use the term "socioeconomic position" in paper II and III because it reflects not only status or rank in social hierarchy, but also the access to the material and social resources.[31] In the papers, the measure of parental socioeconomic position (and status) was mainly defined by education (see methods, 4.5.4).

Second, the schematic representation recognizes, but simultaneously attributes less impact to, another process, often called "health selection" or "health-related social mobility". During a sensitive period in life course, the transition from adolescence to adulthood, or more precisely the period where educational, training and occupational processes occur, health selection may have a important impact.[32] The earliest literature focused mainly on the impact of health selection on change between class of origin (parents) and achieved social class, the *inter*generational social mobility.[33-35] This literature is less extensive than the literature on social causation, and the findings remain contested: some studies reported no impact of illness on intergenerational social mobility,[33] while others found a negative impact of illness.[34 35] However, it is questionable whether it is important if you at least can maintain your social class of origin (e.g. for those with unskilled parents without high school education) in a society where upper secondary school is viewed as the minimum level of educational attainment needed for successful participation of young people (please, see 2.1.2). Indeed, newer research with focus on educational attainment suggests that adolescent health may play a role,[36-39] but at the same time it remains contested whether parental socioeconomic

position (SEP) is a common cause to both adolescent health and educational attainment or whether adolescent health independently of parental SEP influences educational attainment and subsequently labour market exclusion.

Third, as suggested in the schematic representation, the main life course causal model is based on an accumulation model with risk clustering: different types of exposures *clustered by parental SEP* may cause long term damage, such as lower educational attainment, problematic labour market integration and poorer adult health.[19] Such a model implies that parental SEP should lose its impact on adult outcomes (education, labour market exclusion) as intervening variables are entered to the models. An alternative accumulation model is the *"chains of risk" with additive effects*: each exposure not only increases the risk of subsequent exposure, but has also an independent effect on the adult outcome irrespective of the later exposure.[19 40] The literature examining specifically the latter model in relation to the following chain of risk (parental SEP, adolescent risk factors, educational attainment, and subsequently labour market exclusion) is scarce. A Swedish study suggests that both the cluster model and the "chains of risk" complementary influence sickness absence in adulthood.[41] This study also found evidence for not only additive effects in the "chain of risks", but also cumulative effect of adverse childhood circumstances and adult SEP (statistical interactions): poor childhood conditions created a greater vulnerability to pressures in adulthood.[40 41] Another life course model is based on *sensitive periods*: an exposure has a stronger effect on development and subsequent disease in a specific time period than it would in other times.[19] This thesis is not able to assess the latter model because the exposures (ill health) were registered only at one point in one time period, adolescence.

2.2 Norwegian labour market and welfare system – a short overview

2.2.1 The Norwegian labour market

The Norwegian labour market is characterized by a high participation rate and low unemployment rate (Table 2.1). However, Norwegian youth (15-24) were relatively more affected by unemployment compared to older adults (25-64) than average in the OECD

Table 2.1 Characteristics of the Norwegian labour market in 2001 and 2011 compared with OECD averages. (Source: OECD, list of key indicators; http://www.oecd.org/statistics/)

	Norway		OECD	
	2001	**2011**	**2001**	**2011**
POPULATION				
Employment rate (% of population (15-64))	77.5%	75.3%	65.4%	64.8%
Employment rate women (% of female population (15-64))	73.8%	73.3%	55.3%	56.6%
Unemployment rate (% of labour force)	3.5%	3.3%	6.4%	8.2%
YOUTH aged 15-24				
Employment rate (% of age group)	56.5%	51.4%	43.3%	37.8%
Unemployment rate (% of the labour force)	10.5%	8.6%	14.5%	19.0%
Unemployment to population ratio (% of the age group)	6.6%	4.9%	6.7%	8.1%
Relative unemployment rate Youth/adult (15-24)/(25-64)	4.0	3.1	2.6	2.7
NEET rate* (% of the age group)	4.8%	6.7%	13.2%	12.8%
Relative unemployment rate Low skills/high skills (<upper secondary education/≥upper secondary education)	2.7	3.4	2.5	2.2

*NEET: neither in education nor in employment or training. Percentages for 1999 and 2009

countries. Compared to the average in the OECD countries, Norwegian youth with less education were more often unemployed than youth with upper secondary education. The percentage of youth not in education, nor employment or training (NEET) is relatively low. The OECD average for 15-29 years old NEET youth was 16% in 2011, and was below 10% in Norway.[23] The size of the NEET population may reflect declining economic situations, but some of this NEET population may deliberately take a time off or raise a family (especially women).

In Norway, primary industry (agriculture, forestry and fishing) declines, while secondary (industry) and tertiary industry (transport, trade, stores, school system, health care) are growing.(https://www.ssb.no/arbeid-og-lonn/statistikker/regsys)

2.2.2 The Norwegian welfare system

In brief, a welfare system is based on the governmental redistribution of resources in order to protect and promote economic and social well-being of its citizens.[42] The welfare model of the Nordic countries have been categorized as a social democratic regime type, that includes social transfers (income maintenance programs) and welfare services (education, health care, social services) granted for everyone based on social citizenship (universalism).[42 43] A strongly interventionist state promotes social equality through a redistributive social security system and it promotes an equality of the *highest* standards for welfare services and social transfers. The highly redistributive system is based on a progressive tax system combined with vertical (from rich to poor) and horizontal redistribution (e.g. from working adults to pensioners), and the endeavor to realize "full employment" by both men and women. Because of a commitment to full employment and income protection, this regime type provides *highly* decommodifying programs, which means that individuals to a large extent can maintain acceptable standard of living regardless of their market performance.

Existing research literature on health differences suggests that the degree of redistributive policy and the level of generosity of the welfare program are positively correlated with population health outcomes.[44-46] Additionally, the risk for non-employment in the presence of reported limiting longstanding illness in combination with low educational level was lowest in the social democratic regime compared to other welfare state regimes.[47]

2.2.3 The Norwegian Labour and Welfare Administration (NAV)

In 2006, the Norwegian Labour and Welfare administration (NAV) was established as a collaboration of the former social security agency National Insurance Service (Folketrygden), employment agency (Aetat) and certain parts of the municipal social services. NAV has a local office in most municipalities and administers the Norwegian social security schemes and a large proportion of the welfare benefits, such as sickness benefits, work assessment allowance, disability pension, unemployment benefits, social assistance, pensions and family related benefits. While NAV also provides (non-economical) services tailored to the user´s needs and circumstances on the labour market, table 2.2 summarizes the social security benefits (used in the papers) which give people *economical* support in case of sickness, unemployment or financial problems. The terminology used in the table is based on the available benefits relevant for the time period studied in the papers (1998-2008), but later changes in terminology and definitions are mentioned.

2.2.4 Trends in unemployment and medical benefits

In general, economic recessions were initially associated with increases in unemployment and (with some time lag) disability benefits.[48 49] But while the prevalence of unemployment fluctuates cyclical, the prevalence of disability benefits is very little cyclical in all countries,

Table 2.2 The social security benefits used in the papers (Source: www.nav.no/English)

Social security benefit	Definition
Sickness benefits	Compensate for loss of income to insured people who are occupationally disabled due to illness or injury. Requirements: occupational activity at least four weeks + minimum annual income of 1/2G* Coverage: 100% income replacement (maximum of 6G) for a maximum of 52 weeks
Disability pension	Compensate for loss of income to insured people with *permanently* impaired earning capacity due to illness or injury. Requirements: insured minimum three years; be between 18 and 67 years old; working capacity permanently impaired by minimum 50% due to illness, injury or disability; completed appropriate work-oriented measures to access work Coverage: consists of a basic pension and a supplementary pension (dependent on previous salary and number of working years). About 52-90% permanent income replacement
Other long-term medical benefits	Compensate for loss of income to insured people (18 and 67 years) due to at least 50% impaired work ability due to illness or injury + see subgroups Coverage: 66% income replacement for up to four years (maximum of 6G)
- Rehabilitation allowance**	+ undergoing active medical treatment
- Vocational rehabilitation** allowance	+ participating in employment schemes or work testing
- Time limited disability pension**	+ unsuccessful rehabilitation and/or vocational rehabilitation, but has still a certain possibility to become able to work/be employed
Unemployment benefit	Partial compensation for loss of income when unemployed. Requirements: genuine jobseeker; working hours reduced at least 50%; registered as jobseeker with the Labour and Welfare Service; not studying; minimum income of 1,5G last year or 3G in the last 3 years Coverage: about 62,5% of income up to 104 weeks
Social insurance support (financial)	Financial assistance to ensure enough money to cover basic subsistence costs. Requirements: all other options for support are considered (gainful employment, spending own savings and other financial rights) Coverage: The amount is based on specific and individual needs

* G=grunnbeløp= basic amount used to determine benefits in the National Insurance scheme. 1G is NOK 85 245 on May 01. 2013 and is annually adjusted. The maximum coverage for benefits is usually limited to 6G.

** Replaced by work assessment allowance (AAP=arbeidsavklaringspenger) on March 01. 2010

probably because people does not move off disability benefit rolls during economic expansion periods.[48]

The prevalence of sickness absence and disability benefit in Norway is the highest in the entire OECD, while the number of unemployed is among the lowest.[48] The number of people on disability benefits has steadily risen since 1970 (6%), and in 2008 (10.3%), the number was about 4 times higher than the number of unemployed (2.5%). The percentage of youth (18-29 years old) receiving disability benefits has doubled since 1990. In 2006, the proportion of those receiving sickness- or disability-related benefits (4.4%) six years after school leaving is almost double of those who are unemployed or participating in Active Labour Market Programme (2.5%).[50] It should be noted that school leavers often are not entitled to unemployment benefits because it is conditional on prior work participation and past income requirements.

Nearly two thirds of people who received rehabilitation allowance in 2008 and 63% of the people on disability benefits in 2011 had either a mental illness or a musculoskeletal disorder.[2 51] Among youth (18-29), the main diagnoses for disability pension were related to mental retardation, neuroses, personality disorders, schizophrenia/paranoia, congenital malformations and neural disorders (in total 86%).[2] Sickness benefits related to mental illness increased with 20% from 2000 to 2011, while benefits related to musculoskeletal disorders sank.[52] Especially sickness benefits for minor mental disorders such as situational mental imbalance, psychological distress and insomnia increased substantially for all ages, whereas benefits for anxiety and depression decreased.[52]

2.3 High school dropout

2.3.1 Definition

Choosing a terminology and defining the measure was more difficult and challenging than expected.[53] The term "dropout" is used mainly in USA and Canada and refers to people who leave school without obtaining a high school degree. Other countries have similar concepts, such as "early school leaving", "non-completion" or "not in education, employment or training" (NEET), but they are measured differently: an early leaver describes young people leaving upper secondary school without finishing all the years required to complete and non-completion refers to staying the entire time but without passing all of the required exams.[54] The notion of "school completion" had similar challenges: in some context referring to "graduation", in others to "retention to the final year" or "obtaining an upper secondary certificate or equivalent". In some countries (England and Scotland) there is even no standard or benchmark to define the concept of school completion.[53] In Norway, dropout is defined as "leaving upper secondary education before the final year or remaining to the end, but failing to fulfill the graduation requirements". This leads us to the next challenge: official statistics on dropout and completion in Norway are often based on the cohort of students *entering* upper secondary education.[55] In some systems (USA and Canada) concepts such as "lower secondary" and "higher secondary" do not have meaning because high school covers both (partially) levels (Grades 9-12). In this thesis, the term "high school dropout" is used for *all* young people who never obtained a certificate of secondary school and included hereby also those who never entered upper secondary school or even did not finish lower secondary school.

Also the point at which dropout is measured is important. One option is to use the modal completion age, such as 19 years of age. Another alternative is to define completion 5 years

after entering upper secondary school, as used by Statistics Norway. In both situations, this might overestimate dropout rates because of the flexibility in study options, in length of time to complete, and in provision of alternative adult settings outside the secondary school system. It is also possible to measure dropout at a later point estimate, such as 24 years of age, which will reveal lower dropout rates. It makes international comparisons easier because it is less dependent of the national school structure.[53] In the papers, we have opted for the last solution.

2.3.2 The Norwegian upper secondary school system

In Norway, the first 10 years of education (primary school and lower secondary education) are compulsory. Virtually, everybody graduates from lower secondary school, even with very low grades.[54] Upper secondary education has had several major reforms over the last 30 years. The most important reform in the light of this thesis is Reform 94 and one of the main features was to give every 15- to 16-year old the statutory right to 3 years of upper secondary education. Upper secondary education consists of general and vocational tracks. The general tracks are 3 years of education and result in qualification for higher education. Most vocational tracks are 2 years of schooling and 2 years of apprenticeship and result in a vocational qualification. The system allows student to switch pathways.[53]

2.3.3 Prevalence of high school dropout

Of all Norwegian students entering upper secondary school in 1998, 71% completed, 24% did not complete/dropped out and 5% was still at school and had not yet completed within a 5 year period.[56] Similar results for students started in 2005 were for the general tracks respectively 84%, 14%, 2%, and for the vocational tracks respectively 61%, 35%, 4%.[55] Because the students are distributed almost fifty-fifty over general and vocational tracks, the

proportion dropouts within 5 years after entering upper secondary school is stable during the last decade: about 24%, or one on four students. About 5% of all students did not start in upper secondary school.[54 57] The OECD average for lifetime completion of upper secondary education is estimated to be 83%, compared to 89% in Norway.[23] Compared with other industrialized countries, the Norwegian upper secondary school graduation rate below 25 years old is relatively low (78%) (Figure 2.4).[23] However, another 10% does graduate after the age of 25 years and this is a trend that is also present in other Scandinavian countries, such as Iceland, Denmark and Finland. It should be noted that "graduation rate" is not the same as "completion": graduation rate is the sum of age-specific graduation rates (number of x-year-old-graduates divided by the total number of x-year-olds).[23]

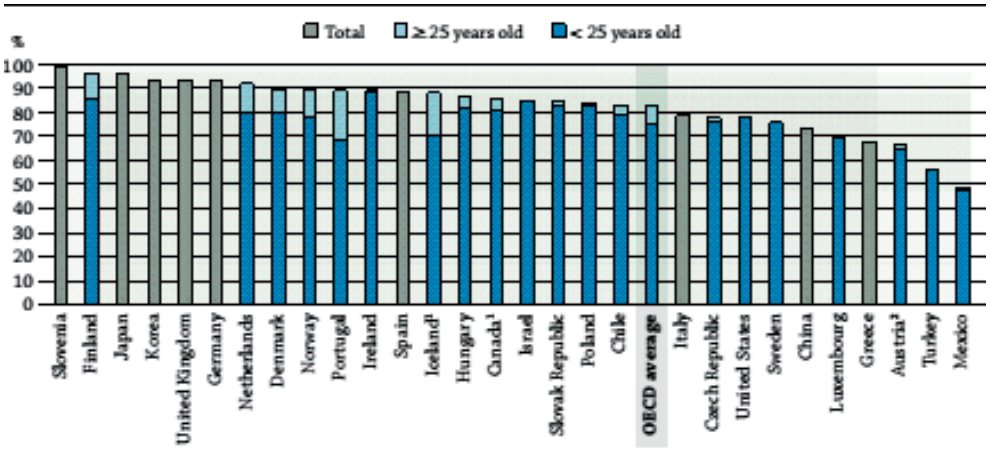


Figure 2.4 Upper secondary school graduation rate (2011). (Source: OECD, Education at a glance 2013, page 44[23])

2.3.4 Risk factors for high school dropout

High school dropout or completion is affected by different factors. In many studies, girls are more likely to complete high school.[23] The most frequent examined factor is socioeconomic status, measured as parental education, income, and occupation, which all have been shown to

be related with high school dropout.[36 58 59] Also the family living situation is a predictive factor for high school dropout.[60] Additionally, health problems and health behaviours during childhood and adolescence have been associated with educational attainment.[37 61 62] Both psychosocial factors and school-related factors have been associated with an increased risk for high school dropout.[36 38 63-67] The literature related to risk factors or predictors for school dropout is extensive and focuses mainly on factors related to demographic variables, individual behaviour, school engagement/academic achievement, family characteristics and peer relationships.[68] The literature related to health and school dropout is relatively new and growing (more detailed, please, see chapter 2.4.3).

2.3.5 High school dropout and labour market exclusion

There is an increase in prevalence of long-term sickness absence and disability pension with decreasing years of education.[14 69] In all OECD countries, people with disability have significantly more often an educational level lower than upper secondary education and are twice as likely to be unemployed, independent of financial times.[48] As shown in figure 2.1, individuals without an upper secondary education have a lower employment rate than people with upper secondary or tertiary education. On average, among OECD countries, men without an upper secondary education are almost twice likely to be unemployed as men with an upper secondary education, and almost three times likely to be unemployed as men with a tertiary education.[23] Also people who receive social insurance support have more often an educational level below upper secondary education.[70]

2.4 Adolescent health and vulnerability during the transition to adulthood

2.4.1 Disease, illness and sickness: ill health

The definition of health itself is difficult to operationalize, but the trilogy of "illness", "disease", and "sickness" has been used to capture different aspects of *ill health*.[71-73] Disease is defined on the condition that is diagnosed by a physician or other medical expert. In Young-HUNT, the self-reported somatic diseases based on a medical diagnosis by a physician or overweight/obesity measured by a nurse could be considered to classify under "disease". Illness is defined as the self reported mental and physical symptoms, which may be minor or temporary, but also severe, chronic and functional limiting. Most self reported symptoms in the Young-HUNT questionnaire are examples of illness. Finally, sickness is related to the social role of a person with an illness or disease, such as sickness absence from work or disease-related absence from school. These three concepts capture different aspects of ill health and there is some, but not complete, overlap between them.[74] When we use the term "adolescent health" in this thesis and papers, it refers mainly to the concept of ill health defined by "disease" and "illness".

2.4.2 The prevalence of chronic disease and illness in adolescence

Usually, it is a relatively easy part of a paper or manuscript to describe "prevalence" of a "condition". Remarkably, there is a shortage of age-specific epidemiologic data about this subject: a) many surveys or reports of chronic disease or illness fail to define adolescence because adolescents are either grouped with children (0-14 years) or with adults (15-24 years) or b) adolescence is recognized, but there is no consistency in the age limits.[75] Additionally, prevalence data can come from several sources which will affect the prevalence results: checklists of medical disorders or disease types, functional status assessments or defining

limitations in socially defined roles.[75] Finally, prevalence data will be influenced by the person who reports: the adolescent, the parents, or the medical doctor. The international information on adolescent health has major data gaps, and the presented data are therefore limited to Europe and USA who have best data availability.[76]

Most individual chronic conditions in adolescence are uncommon (with a few exceptions such as asthma, allergy and obesity), but the total burden is substantial (about 31% of all US children in 1988).[77] The number of US children and adolescents with reported limitation of activity due to a chronic health condition was 1.8% in 1960 and increased to 7% in 2004.[78] Most of the growth was related to the increase of a few high-prevalence conditions, such as obesity, asthma, and attention deficit and hyperkinetic disorder (ADHD).[78] Some of the growth might be due to changing social environments, such as more indoor time, less physical activity, increased energy intake, less energy and time for parenting and so on, but partly also because of changes in diagnostic practices and a social need for diagnoses.

In the international Health Behaviour in School-aged Children (HBSC) Study of 2009/2010, 23% of the 15 year old girls and 14% of the 15 year old boys reported fair or poor health, which was a slight decrease in prevalence compared to 2001/2002.[79] In a Norwegian study, youth´s self-reported health was associated with a broad range of health indicators (medical, psychological, social and health behaviours), reflected their overall sense of functioning and was a relative stable construct during adolescence.[80] In the same HBSC Survey, 44% of the 15 year old girls and 26% of the 15 year old boys reported multiple health complaints more than once a week (and the same prevalence as in 2001/2002).[79] The health complaints included both somatic (headache, backaches) and psychological (nervousness, difficulties in getting to sleep) symptoms.

A cross-sectional review across nations estimated the average rate of adolescent psychiatric disorder (12-19 years) about 22% and the most common diagnoses were drug abuse (12%), anxiety disorders (11%) and depressive disorders (6%).[81] From childhood to adulthood, there was an increase in panic disorder, agoraphobia and substance use disorder, while there was a decrease of separation anxiety disorder and ADHD.[81]

In conclusion, the total number of adolescents confronted with chronic disease and illnesses, with varying level of functional limitations, is substantial and will have a major impact on public expenditures for health and income support.

2.4.3 Adolescent health and school dropout

In an article of 2007 by Freudenberg concerning school dropout as a public health issue, the number of references related to adolescent health as a direct effect on school dropout was very limited and described health problems such as substance use, psychological, emotional, and behavioural problems.[17] The articles cited below have a prospective design, and if the study had a retrospective design (and a higher risk of recall bias), it will be mentioned.

In a British cohort born in 1958, men with chronic physical disorders had a higher risk to have no academic qualifications at age 23.[82] In the Add Health study (US), youth with self-reported (retrospective) epilepsy, diabetes, heart disease or cancer with onset before age 18 had less often graduated from high school.[83 84] However, in a review on students with asthma and school performance, the majority of studies showed no differences in levels of academic achievement (but none of the studies examined educational attainment).[85] Among the studies

showing difference, the difference in achievement was limited to students with severe and persistent asthma, or with asthma-related interruptions in sleep.

A Finnish longitudinal study covering the transition from adolescence to young adulthood showed that health selection, examined by psychosomatic symptoms, was particularly strong from adolescence to low education in early adulthood.[86]

A retrospective study (US) of 1995 showed for the first time the association between self-reported early-onset psychiatric disorders such as anxiety and mood disorders and failure to complete high school.[87] Although a (small) longitudinal study (US) in 2000 suggested that adolescents with a psychiatric disorder had a higher risk of failure to complete secondary school,[88] other studies suggested that there was no association between anxiety or depression and high school dropout or that an existing association was attributable to childhood disadvantages.[89-92] Newer longitudinal studies showed an association between school dropout and internalizing problems in adolescence, more specific depression, and especially for girls.[93-96]

A meta-analysis showed that problems with sleep quality, sleep duration, and sleepiness were associated with lower school performance.[97] None of the studies described specifically the relation with educational attainment.

A review described that children with ADHD, but also children with symptoms of ADHD but without formal diagnosis were far less likely to graduate from high school.[98] In a recent Canadian longitudinal study, inattention rather than hyperactivity was a predictor for not having a high school diploma at 22-23 years of age.[99] A recent longitudinal study (US) with

adjustment for IQ, learning problems and parental socio-economic status, participants with ADHD were more likely to drop out of high school.[100]

In several studies, poor health in childhood and adolescence, adjusted for parental socio-economic status, was associated with lower educational attainment.[36 38 39 62 101] A longitudinal study suggested that poor perceived health had more negative consequences on high school completion for the most socially advantaged racial group.[61]

A US study in New England Journal of Medicine in 1993 showed an association between adolescent overweight and educational attainment.[102] Three later longitudinal studies from UK, Finland and Danmark on adolescent obesity and socioeconomic outcomes were consistent with this result,[67 103 104] but a UK study on (self reported) *childhood* obesity found no association with high school dropout.[105]

2.4.4 Adolescent health and labour market exclusion

A large amount of literature has established that low birth-weight babies are more likely to suffer various deficits and have lower average educational attainment, but that their changes to become employed are not reduced (if not suffering serious disabilities).[106 107] Prior research has had less emphasis on how adolescent health affects labour market exclusion in young adulthood. While studies with outcomes such as long-term sickness and disability pension are mainly produced in Scandinavia, the majority of US and UK studies used the outcomes employment, public assistance and income.

Studies on men at conscript (18-20 years of age) in Norway and Sweden linked poor self-rated health, psychiatric and musculoskeletal diagnoses, and obesity with a higher risk of

early disability pension.[108-111] Other studies on adolescents also showed an association between somatic symptoms, benefits due to chronic disease (0-16 years of age), self-rated health, or ADHD and long-term sickness absence/disability pension.[13 15 112-114] Some of the studies presented attenuated associations (for benefits for chronic disease, mental health, self-rated health, BMI) after adjustment with educational level or ranking low on "IQ" test, while the association (for somatic symptoms) disappeared after adjustment for both educational level and cognitive ability in one study.[13 15 108-110 113]

Studies on employment and labour market integration used different outcomes such as "to be employed", "unemployed", "gainful activity", "adult weeks worked", and "public assistance", "income below poverty". Because of this spectrum of outcomes, the conclusions varied also. Children (0-16 years of age) who received benefits due to chronic disease had a higher risk for unemployment, while another study on children with chronic physical conditions showed higher risk for unemployment only for men.[15 82] Diabetes, cancer, epilepsy, and heart disease in childhood or adolescence are associated with a lower chance to be employed or a higher risk to receive public assistance, while benefits in childhood due to chronic disease reduced the change to be employed only if attained a low educational degree.[83 84 115] Several studies also suggest a higher risk for unemployment or lower chance to be employed for adolescents who had suffered of anxiety or depression.[88 90] A recent study on adolescents with depression confirmed this association with unemployment at age 26, but the association disappeared when adjusting for IQ, deviant peers and other covariates.[116] ADHD was associated with less paid employment and more social assistance.[117 118] In a recent study, the presence of ADHD reduced the employment rate with 10-14% and increased the use of social assistance with 15%, even when adjusted for "test score during high school".[119] Poor (retrospective) self-rated health in childhood (before age 16) was associated with less adult weeks worked in one

calendar year and lower income when adjusted for obtained educational level at that time.[120] Obesity in adolescence decreased the adult hourly earning in the 1958 British birth cohort with 7%, especially for women.[102] Other studies also showed that adolescent obesity was associated with a higher risk of unemployment, but only for women.[104 105] With adjustment for school performance, the association disappeared.[104]

2.4.5 Adolescent vulnerability during the transition to adulthood

Vulnerability refers to a dynamic process of negative adaption in the face of adversity.[19] As mentioned above (see chapter 2.3.4), many studies have defined individual risk factors for lower educational attainment or high school dropout in particular. A major part of the literature is dedicated to the social background during childhood and adolescence (see chapter 2.5). Several articles have addressed the negative effects of disadvantaged homes on school completion, the likelihood of becoming involved in risky behaviour and ending in poor jobs with large risks of health hazards.[41 121-124] Besides health problems, health-compromising behaviours, such as rarely participation in (un)organized exercise and especially smoking predicted low educational level in adulthood.[37] Adolescents with good self-esteem and subjective well-being also perform better at school and have better chance to obtain a high school degree.[36 65 125] In addition, social integration within the family, peer group and school is associated with educational trajectories: shorter education is more common among children with divorced parents and bullied adolescent were less likely to achieve the appropriate academic achievement benchmark for their age group.[64 126] Of the school factors, low educational achievement, attention and conduct disorders, and low educational aspirations appear to predict school dropout.[36 98 127 128] Finally, all above mentioned risk factors for low educational attainment are also associated with a higher risk of long-term sickness or disability in adulthood.[41 63 114 125 126 129 130]

However, there is a lack of knowledge on how adolescent vulnerability and subsequent school completion or dropout might interact in relation to health-related labour market exclusion in young adulthood.

2.5 Family background and context

A variety of mechanisms exists through which families and parents can influence the development of health and educational attainment in children – resource expenditure, role modeling, behavioral control, stimulation/nurturance, and heredity.

Socioeconomic position in adulthood has a profound association with adult health.[131] Because adolescents not yet have obtained their own socioeconomic position in terms of occupational class, educational level or income, SEP is usually defined by their parental SEP. Parental SEP disadvantage is not simply a coincidence. To some extent it reflects back on differences in human capacity (such as health and functioning) and life course opportunities which are carried forward through generations. Adolescents with lower parental SEP suffer from poorer physical and mental health outcomes compared to adolescents with higher parental SEP.[132-138] There is also evidence that participation in unhealthy behaviour follows the same social patterning, but not all studies are consistent: many studies for some important health-related behaviours, such as alcohol use, do not show social gradients.[139] This might relate to the role of peers in developing behaviours during adolescence.[139] Adolescents of parents with lower SEP have an increased risk for poor educational achievement and attainment.[36 140 141] In young adulthood, they also have a higher risk for unemployment and disability pension.[13 137]

The proportion of single-parents families has increased in the last few decades, many due to parental divorce.[142] A substantial literature exists on the effect of parental divorce on children and adolescents. Children with divorced parents are more at risk for lower measures of psychological well-being, academic achievement, self-concept, and social relations.[143 144] The negative effects also track into adulthood with shorter education, unemployment, more risky health behaviour, and negative life events.[126]

Families self-select into neighbourhoods and neighbourhoods may affect the values and social development of the parents and their children. A Norwegian study on the effect of family characteristics and residential location on educational attainment indicated that neighbourhoods (in Norway) might be of less importance than families.[145] The neighbourhood clustering of educational attainment in Norway was also substantially lower than in the United States.

When studying families, it always becomes the question to what degree the results are a consequence of nature (genetics) or nurture (environment). Genetic studies and epidemiological studies on twins are used to unravel this tangle. It has been shown that genes contribute substantially to the variance of many health conditions.[146-149] A US study found that, when IQ was low, shared environmental background of the family most heavily influenced educational attainment, but when IQ was high, genetic influences (primarily on characteristics other than IQ) influenced educational attainment.[150] A Swedish twin study found that the association between low education and disability pension disappeared when adjusted for familial factors that the twins shared.[151] In a Finnish and Swedish study, about one third of the variance in liability to disability pension irrespective of diagnosis was attributed to genetic effects and the effects seemed to be stronger at younger age.[6 152]

3 Aims

High school dropout is associated with higher morbidity and poorer work integration in later adult life. In a life course perspective, the transition from adolescence into adulthood is a critical period in life, in which the foundation for adult health and socioeconomic position might be laid. The first main aim of this study was to investigate the association between school dropout and labour market integration. Additionally, we wanted to study the association of adolescent health with the acquirement of a high school degree. We also wanted to investigate the extent to which adolescent vulnerability in general influenced the association between school dropout and labour market integration in young adulthood. Finally, we investigated the confounding role of the family, in specific the parental socioeconomic position, family living situation and in general the familial context shared by siblings.

More specifically, the main aims of this thesis were:

Paper I

- To study the association of high school dropout, self-reported adolescent health and long-term medical and non-medical benefits in young adulthood.

Paper II

- To study the association between several dimensions of self-reported health in adolescence and high school dropout, adjusted for observed confounding family variables such as parental socioeconomic background and family living conditions.
- To investigate whether the association remained after accounting for all shared, stable unobserved family characteristics.

Paper III

- To investigate the extent to which baseline differences between high school dropouts and completers (such as parental socioeconomic position, health measures, health

behaviours, psychosocial factors, and school-related problems) may explain the risk for long-term sickness absence and disability pension, both for the whole population and among siblings.

- To study if the more vulnerable adolescents with a high risk level for school dropout, in case of school dropout, have an even greater increase risk for long-term sickness and disability compared to adolescents with a low risk level.

4 Material and methods

4.1 Study design

The three papers are prospective population-based cohort studies. In 1995-97, 8950 of all junior high and high school attending adolescents in the county of Nord-Trøndelag (age 13-19 years) filled out the questionnaire of the Young-HUNT1 Survey. The response rate was 90%. The adolescent survey data was linked to data on the participants in the Norwegian population registers during the period 1998-2007 (paper I) or 1998-2008 (paper II and III) and to data on the parents in the HUNT2 Survey (paper II) and the Norwegian population registers (paper I, II and III) (figure 3.1). Linkages were obtained by the 11-digit personal identification number, which is unique to each resident in Norway.

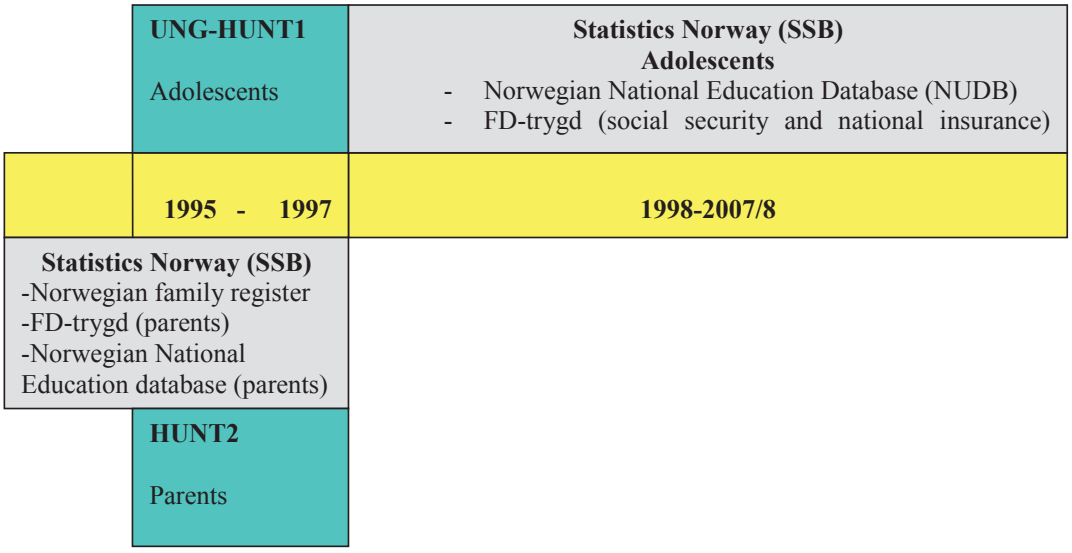


Figure 3.1 Schematic overview of the prospective design and linkages between the HUNT Study material and the Norwegian population registers.

This made it possible to link self-reported health information to register-based information on demography, education, and receipt of social insurance benefits, and to link parents and siblings (those who had the same mother). The main outcomes were high school dropout registered in the calendar year the participant turned 24 years old (paper II) and the receipt of social insurance benefits (paper I and III).

It should be noted that paper II and III are based on other data files than paper I because of an update of all files in 2011. This resulted in some differences in variable construction, which will be specified in the sections "4.3 Norwegian population registers" and "4.5 The study variables".

4.2 The Nord-Trøndelag Health Study (HUNT)

The HUNT Study (Helse undersøkelsen i Nord-Trøndelag) constitutes a large population database based on three health surveys of the adult population in Nord-Trøndelag County (HUNT1 in 1984-86, HUNT2 in 1995-97 and HUNT3 in 2006-8) and three health surveys of the adolescent population (Young-HUNT1 in 1995-97, Young-HUNT2 in 1999-2000 and Young-HUNT3 in 2006-08). At the time of HUNT2, adolescents aged 13-19 years were invited to the Young-HUNT1 Survey.

The Nord-Trøndelag County is situated in the middle of Norway and has a relative stable population of about 135.000 inhabitants. The population is homogenous, as less than 3% of the population is non-Caucasian. The county is considered to be fairly representative of Norway regarding geography, economy, industry, and sources of income, age distribution, morbidity and mortality.

A comprehensive description of the HUNT Study and the Young-HUNT Study is published elsewhere and gives more detailed information on the invitation procedures, participation and study variables.[153-155] The following sections will highlight the facts that are important for this doctoral thesis and adjunct papers.

4.2.1 The Young-HUNT1 Survey

In 1995-97, all adolescents (age 13-19 years) in the Nord-Trøndelag County were invited to fill out the Young-HUNT1 questionnaire and to attend the clinical part of the study. Because of low participation rate (7%) of the adolescents not attending school, the Young-HUNT data are not representative for this group. The study population focuses on *school attending* adolescents, more specifically the students on junior high (age 13-16) and high school (age 16-19). These students completed a questionnaire during a school hour in an exam-like situation and within a month, specially trained nurses visited all schools for interviews and measurements. A total of 8950 (90%) of the 9917 school attending adolescents completed the

Table 3.1 Attendance rate in the Young-HUNT1 Survey (1995-97). The study population is highlighted.

Young-HUNT1 Survey	Number invited	Response rate questionnaire		Response rate both questionnaire and clinical data	
	N	n	%	n	%
Junior high school	5004	4743	94.8	4598	91.9
High school	4913	4207	85.6	3810	77.5
Not in school or attending college university	285	34	11.9	31	10.5
Total	10202	8984	88.1	8439	82.7

.

questionnaire and 85% completed both the questionnaire and the clinical examination (Table 3.1).

Of the non-responders (10%), most were not in school when the study was conducted. From the invitation lists it is noted that non-responders compared to responders were older, more often boys and more often attended vocational than academic classes.

Self-reported information of the adolescents covering somatic and mental health, health behaviours, school problems, and psychosocial factors and clinical information such as weight and height from the Young-HUNT1 Survey is used in the papers.

4.2.2 The HUNT2 Survey

Simultaneously with the Young-HUNT1 Survey, the HUNT2 Survey was conducted. The HUNT2 covered adults aged 20 years and older. In total, 76% of women (n=35280) and 67% of men (n=30860) participated. Data was collected from several questionnaires, blood and urine samples and clinical measurements. Of all school attending adolescents in Young-HUNT1, respectively 94% (n=8373) and 86% (n=7666) could be linked to their biological mother and father in HUNT2.

Self-reported occupational class from mother and father in HUNT2 was used in paper II.

4.3 Norwegian population registers

Statistics Norway can supply various public register-based data collections on an individual level (person) such as data on education, social security and national insurance, and families. Linkages are obtained by the 11-digit personal identification number, but the datasets are delivered with de-identified reference numbers and are anonymous on the hands of the researcher.

4.3.1 Norwegian National Education Database (NUDB)

The National Education Database (NUDB) gathers all individually based statistics on education in Norway in annual files since 1970. Based on this annual files, course files are created and the three most important course files are the situation file with registered students (enrolments) by October 1st, the completed education file (graduates) from the previous school-/academic year and a file containing the population´s highest attained level of education (attainment). Additional, NUDB can extract fixed personal variables. More detailed information on the database can be found on Statistics Norway (http://www.ssb.no/a/english/mikrodata_en/).[156] For all papers, high school dropout was defined from the course files by using "highest attained level of education" and the exact date for attainment. To define parental education level, data from a course file was used in paper I, while fixed personal variables were used in paper II and III.

4.3.2 FD-Trygd (Forløpsdatabase Trygd)

FD-Trygd is a historical event database containing social insurance information for the whole population from 1992 and onwards. FD-Trygd consists of registrations of events in each

personal life span and can be put together to individual event histories. Basically, FD-Trygd is created by modeling and constructing cross-sectional data from the administrative registers to event data which reflect "change in circumstances/conditions". It implies that there is only a registration in the event database if an event has happened and as long nothing happens, nothing is registered. Consequently, we are forced to assume that the information gathered by Statistic Norway in FD-Trygd is complete. Statistics Norway has documented on its website the principles by which the data are processed and the consistency controls, and especially for national insurance (pension and benefits) cross-topic consistency controls have been performed. FD-Trygd contains information from NAV (the Norwegian Labour and Welfare Organisation, earlier called National Insurance Administration), Aetat (Employment directorate), Skattedirektoratet (Taxation) and other registers in Statistics Norway. The main and relevant topics used in the papers were pension and benefits, employment, social assistance, income and demography.

The statistical unit in FD-Trygd is the person, and it is possible to connect all people belonging to the same family by using the Family register. This results in nearly complete parental information on education level and income. More detailed information on FD-Trygd can be found on Statistics Norway (http://www.ssb.no/a/english/mikrodata_en/).[157]

4.4 Study population

The choice of excluding participants from the basic group "school attending adolescents" (N=8950) was dependent on the definition of the outcome. Similar for all articles is the exclusion of participants born after 1983 (4) and age-school level mismatch (4). Additionally,

participants with early disability pension or death during follow-up were also excluded, but the numbers differ in relation to the outcome in the articles (table 3.2).

In paper I, all participants with disability pension before age 20 or in process to disability pension before 1998 (start of follow-up period) were excluded. In paper II and III, we excluded participants with disability pension before age 22. In Norway, adolescents between age 16 and 21 are eligible to follow education at high school level, but disability pension before age 22 interferes with this opportunity (by law) to complete high school (outcome in paper II and the independent variable in paper III). Early disability (at the beginning of adult life) is related to severe health problems (often congenital), which was not the main focus of

Table 3.2 Summary of exclusion criteria in the published articles, total number of participants after exclusion and for complete cases.

	Exclusion	Outcome	N_{total} after exclusion	N_{total} Complete cases
All papers	Born after 1983 (4) Age-school level mismatch (4)			
Paper I	Death during follow-up (46) Disability pension at age 18 or 19 (66) Start of social insurance process before 1998 (35)	Benefits between age 19-28 (data from registers between 1998 to 2007)	8795	8339
Paper II	Death during follow-up (30) Disability pension within period 16-21 years (30) Missing educational data (8)	High school dropout at age 24	8873	varies from 8205 to 8696
Paper III	Death before age 24 (30) Migration before age 24 (57) Disability pension within period 16-21 years (30) Missing educational data (8)	Benefits between age 24-29 (data from registers between 1998 to 2008)	8816	6612

the papers. In paper III, participants who migrated before the start of the follow-up period were also excluded (57).

Finally, all main analyses are based on complete cases. The variable with highest number of missing data was BMI, conducted from the clinical examination of Young-HUNT1 (6.2% missing).

4.5 The study variables

4.5.1 High school dropout

The variable "high school dropout" was used both as independent variable (paper I and III) and as outcome (paper II). It was defined as either completion or dropout in the calendar year the participant turned 24 years old. Data were retrieved through linkage to the NUDB which coded level of education by NUS2000-standards, which implemented the international education standard ISCED97.[158] The NUS2000-codes consists of 6 numbers and the first number indicates the level of education. An education level of 13 years or more (level 4 to 8 by NUS2000) is coded as completed high school, while an education level of 12 years or less (level 0 to 3 by NUS2000) is coded as high school dropout. 8 participants had no information on education.

We chose to define the cohort dropout rate with a later point estimate (age 24) because firstly, we did not want to overestimate the dropout rate and secondly, it takes into account international varying educational systems and it will and can make international comparison easier.[53]

4.5.2 Social insurance benefits

Information on social insurance benefits was collected from FD-Trygd. The FD-Trygd-database is organized according to topics and contains one data-file for each topic or each scheme. In table 3.3, the topics, associated data-files and extracted data used in the papers are summarized.

Table 3.3 A schematic description of files in the Statistic Norway´s event database used to construct the outcome measures "long-term medical and non-medical benefits" (paper I and III).

Data-file (SSB)	Topic (SSB)	Type of benefit extracted (papers I and III)	Outcome measure (papers I and III)
F_UFP (1992-2008)	Disability pension	Disability pension (1998-2008*)	Long-term medical benefits (1998-2008*)
F_TU (2004-2008)	Time limited disability pension		
F_FUFOR (1992-2008)	Provisional disability pension		
F_SP (1992-2008)	Sickness benefits	Long-term sickness benefit (1998-2008*)	
TAB_REHAB (2002-2008)	Rehabilitation allowance	Rehabilitation allowance (2002-2008*)	
F_ATTF (1992-2001)	Rehabilitation allowance and vocational rehabilitation allowance	Rehabilitation and vocational rehabilitation allowance (1998-2001)	
F_ARSBOK (2001-2008)	Jobseekers	Vocational rehabilitation allowance (2002-2008*)	
F_ARBSOK_TOM2001 (1992-2001)	Job seekers	Unemployment benefit (financial support) (1998-2008*)	Long-term non-medical benefits (1998-2008*)
TAB_SHJ (1992-2008)	Receipt of social assistance	Social insurance support (financial) (1998-2008*)	

*For paper I the registration period ended in 2007.

.

In paper I, the outcome "long-term social insurance benefits" was constructed with three categories: long-term medical benefits, long-term non-medical benefits and no long-term benefits in the follow-up periods 19-28 years, 19-23 years and 24-28 years of age. In paper III, the outcome "long-term sickness absence and disability pension" was dichotomized as long-term medical benefits versus no long-term medical benefits in the follow-up period 24-29 years of age.

Long-term medical benefits were defined as permanent and temporary disability pension, medical and vocational rehabilitation, or sickness absence received at least 180 days during one calendar year in the follow-up period. Long-term non-medical benefits were defined as unemployment benefit received at least 180 days during one calendar year or social insurance support more than one month in a calendar year in the follow-up period.

We investigated the associations with *long-term* benefits, because the aim of the papers was to focus on labour market exclusion in early adulthood. There is no standard definition for long-term absence and different operationalizations have been suggested in the literature.[5 159] To avoid low sensitivity with a relative short absence (e.g. 2-8 weeks) and very low specificity with a longer absence because it rather detects social exclusion (e.g. one year), we arbitrary chose for a cut-off at more than 180 days per calendar year for sickness and unemployment benefits. Disability pension, medical and vocational rehabilitation are per se long-term benefits.

Local labour and welfare offices (NAV offices) in the municipality can support with financial social assistance ("social insurance support" in paper I) in case people cannot support

themselves economically. This means-tested benefit can be received even without prior labour market experience and are more often the only option for young adults without previous employment and income (on the contrary, receipt of sickness benefits and unemployment benefits requires employment with an annual income of a certain level). However, it also indicates economic hardship and repeated receipt of social assistance (more than once per calendar year) might be a direct indication of labour market exclusion.

Additionally, we were specifically interested in labour market exclusion *related to ill health*. Because of the health focus, we used in paper I a hierarchical principle: participants who received both long-term medical and non-medical benefits in the same calendar year were coded under "medical benefits".

The Young-HUNT1 cohort is a closed cohort with participants with varying ages (13 to 19 years) on varying inclusion years (1995 to 1997) and additionally, the participants entered the follow-up periods in different calendar years during the registration period in FD-Trygd from

Table 3.4 Examples for the follow-up period "19 to 28 years" in the registration period "1998-2007" in paper I to demonstrate the challenge related to "time" (age, registration years, time gap between inclusion and follow-up period).

Birth year	Year of Participation Young-HUNT1	Age at inclusion Young-HUNT1	Age at last registration year (2007) SSB	Registration year at age 19	Registration year at age 28	Follow-up time
1983	1997	Age 14	Age 24	2002	2011	6 years (02-07)
1979	1996	Age 17	Age 28	1998	2007	10 years (98-07)
1977	1995	Age 18	Age 30	1996	2005	8 years (98-05)

1998 to 2008. Because the *follow-up periods* were defined by age (e.g. 19 to 28 years), the participants had varying *follow-up times* during the follow-up periods. Follow-up time is the numbers of registration years within the registration period (1998-2007 or 2008) during the defined follow-up period (table 3.4). We adjusted for follow-up time in paper I and III.

4.5.3 Adolescent variables

Demographic variables and the health variable "self-rated health" are used in all papers. Health behaviours, psychosocial factors, school problems and the other health variables are used in paper II and III. Except for age, all the variables were obtained by the Young-HUNT1 Survey.

Demography

In all papers we adjusted for sex since there is a difference between boys and girls in school dropout and the receipt of long-term social insurance benefits.[23 48] Age was defined with year of birth in FD-Trygd and year of participation in Young-HUNT1 Survey and adjusted for in all papers.

Health

"Chronic somatic disease" or short "somatic disease" was defined as the presence of at least one self-reported chronic disease (asthma, diabetes, migraine, or epilepsy diagnosed by a doctor or any other illness that lasted longer than 3 months) versus none. There is evidence of good validity for questionnaire-based diagnosis of headache and migraine in the adult HUNT Survey,[160] although such validities of the Young-HUNT Survey have not been evaluated.

"Somatic symptoms" was based on the sum of self-reported presence (dichotomized into "never/seldom" and "sometimes/often") of eight symptoms (headache, neck or shoulder pain, joint or muscle pain, stomach pain, nausea, constipation, diarrhea, heart palpitations) during the last 12 months (Cronbach´s alpha=0.73). The variable was dichotomized into none or 1 symptom versus 2 or more symptoms (the highest tertile) (paper II). In paper III, we used the full range 0 (no symptoms) to 8 (8 symptoms). Both the terminology and the construct of the variable was a challenge because of lack of definition and uniformity in international literature. The terminology varies from "somatic symptoms", "symptom load" to "bodily complaints", "bodily distress" and even "well-being".[136 161-164] In order to keep it clear and avoid (not-validated) interpretations, we used "somatic symptoms" and "symptom load". Similarly, for the operational definition there is little consistency in the literature related to the number of symptoms included, the frequency, intensity and duration. With increased frequency and number of pain locations, the risk for subjective disability increased.[165] In our variables, we took into account the frequency (sometimes/often) and the load (2 or more; the full range).

"Psychological distress" was measured with the validated SCL-5 scale score – the Hopkins Symptom Checklist – a 4-integer 5 item short version of the original SCL-90 (Cronbach´s alpha=0.87).[166 167] The participants answered with a score from 1 (not at all) to 4 (very) if they had been constantly afraid and anxious, tense or uneasy, dejected or sad, felt hopelessness when they thought of the future or worried too much about various things in the last 14 days. The mean score of the sum of self-reported symptoms of anxiety and depression was calculated if they had filled out at least 3 items (range 1 to 4). In paper II, a score above 2 reflected high psychological distress, while the variable was continuous in paper III.[167]

"Insomnia" was defined as having difficulties in falling asleep the last month and dichotomized into "never/sometimes" versus "often/almost every night" (paper II and III). *"Concentration problems"* were self-reported difficulties with concentrating during class at the moment or earlier and dichotomized into "never/sometimes" versus "often/very often" (paper II and III).

"Self-rated health" was defined by using the question "How is your health at the moment?" and dichotomized into "good/very good" versus "poor/not so good" in all papers. Self-rated health in adolescence was a relative stable construct during adolescence and was related to medical, psychological, social and life-style factors.[80 168]

Trained nurses measured weight and height by following a standard protocol and using standardized meter bands and weight scales. Body Mass Index or *BMI* was computed as weight (in kilogram) divided by the squared value height (in meter). The classification in normal weight, overweight and obesity was defined by cut-offs for the appropriate age groups as proposed by Cole et al.[169] Overweight corresponded with an adult BMI from 25 to 30 and obesity a BMI of 30 and more.

In paper II we conducted additional analyses with insomnia, concentration problems and self-rated health as categorical variables (4 categories) and with somatic symptoms and psychological distress as continuous variables. All health variables are used as independent variables (exposures) in article II and as confounders in article I (self-rated health) and III.

Health behaviours

Lifestyle or health behaviours are associated with lower educational attainment and higher risk for receiving long-term social insurance benefits.[37 102 129 170] We adjusted for health behaviour in paper III not because we meant smoking or physical inactivity are a direct cause for high school dropout, but because they might be correlated to unknown factors that could confound the association between education and the receipt of benefits.

Because *BMI* as a chronic condition can reflect a health potential or risk, we defined it as a "health" variable in article II.[102] However, it can also reflect an underlying (not-specified) behaviour and has been defined as "health behaviour" in other studies, including our paper III.[136]

"Smoking" is dichotomized into smokers and non-smokers. Smokers are those who answered "Yes" to the question "Did you ever smoke?" combined with answering "Yes, I smoke daily" or "Yes, I smoke occasionally" to the question "Do you smoke now?". Non-smokers are those who had never smoked cigarettes or had stopped smoking.[171]

The variable *"physical activity"* differentiated between those who were active on more or less regular basis and those who had no regular physical activity. The inactive answered "Not every 14th day, but at least once a month", "Less than once a month" or "Never" on the question "Not during the average school day: How many DAYS a week do you play sports or exercise to the point where you breathe heavily and/or sweat?". The active answered "Everyday", "4-6 days a week", "2-3 days a week", "1 day a week" or "Not every week, but at least once every two weeks".

Psychosocial factors

In this category of variables we included variables that are related to the psychological development in, and in interaction with the social environment. We included beliefs and emotions from psychology (self-esteem, subjective well-being and loneliness) and family living situations which were associated with lower educational attainment and higher risk for receiving social insurance benefits.[36 65 66 125 126]

"Self-esteem" was measured with a short version of the Rosenberg Self-Esteem Scale consisting of 4 statements with a 4-point Likert scale (Cronbach´s alpha=0.74). We calculated a mean score within the range from 1 (low) to 4 (high self-esteem). This short version had a 0.95 correlation with the original Rosenberg Scale.[172]

"Subjective well-being" consisted of 3 questions with each a 7-point scale varying from "Very satisfied" to "Very dissatisfied"(Cronbach´s alpha=0.74).[173] The questions were related to feel usually satisfied/dissatisfied with life, strong/tired, cheerful/downhearted. We calculated a mean score within the range 1 (high) to 7 (low subjective well-being).

"Loneliness" was a categorical variable obtained from the question "Do you feel lonely" with a 5-point scale from 1 ("Very seldom or never") to 5 ("Very often").

The *"family living situation"* was defined by living in a traditional family (living together with both biological mother and father) or not.

School-related factors

School related factors such as reading and writing difficulties, academic problems, school related conduct, school related dissatisfaction, being bullied, educational aspirations, disease-related school absence have been associated with educational attainment and social insurance benefits.[61 63 64 98 114 127 128 130 174]

We defined *"Reading and writing difficulties"* as responding "Yes" to the question "Do you currently receive help for reading or writing problems?" or reporting major problems with either reading or writing the last 12 months.[63]

The Young-HUNT1 Survey had 14 statements concerning school-related factors that were generated for the purpose of the HUNT Study.[175] The responses to the statements were given on a 4-point Likert scale ranging from 1 "Never" to 4 "Very often". A principle component analysis with oblique rotation has been previously conducted and three factors were extracted.[176] The item "being bullied at school" did not load highly on any of these and was therefore treated as a measure with one item. *"Being bullied"* was a categorical variable based on the response on the statement "You are teased/harassed by other students" with a 4-point scale from 1 "Never" to 4 "Very often". *"Academic problems"* was computed as the mean score of five statements related to academic achievement such as "You understand what is being taught" and "Are you satisfied with your test results?". *"School related dissatisfaction"* was computed as the mean score of four statements related to dissatisfaction at school such as "You look forward to go to school" and "You think that gym or art is fun?". *"School-related conduct"* was computed as the mean score on four statements related to conduct problems at school such as "You argue with the teacher" and "You cannot manage to be calm/sit still during class?".

"Educational aspirations" was based on the question "What type of plans do you have regarding continued studies?" and categorized into three groups ("None/do not know", "High school" and "Higher education").

"*Disease-related absence from school"* was defined as reporting more than 2 weeks absence from school due to illness during the last 12 months.

4.5.4 Parental and familial variables

The actual resources of parental socioeconomic position can be measured by indicators such as education level, occupational class or income.[31] Parental socioeconomic position influences both educational attainment and the risk of receiving long-term social insurance benefits.[13 36] The main analyses in all papers were adjusted for maternal education level because there were only few missing data for this variable and because most adolescents (87%) were living with their mother. Additional analyses were carried out with adjustment for paternal education level (paper I and II), parental occupational class and income (paper II).

Parental education level (biological mother and father) was obtained through linkage to the coded level of education by NUS2000-standards in NUDB. The education level was divided into three categories: compulsory (primary and lower secondary education – level 1 and 2 by NUS2000), intermediate (upper secondary and post-secondary non-tertiary education – level 3 to 5 by NUS2000) and tertiary (undergraduate, graduate and post-graduate education – level 6 to 8 by NUS2000). For paper I, the education level was defined in calendar year 1995 and in paper II and III (because of different data files) it was registered at the time the participant was 16 years old.

To define the *parental occupational class,* the ten self-reported occupational classes of HUNT2 Survey were reorganized into the 7 occupational classes of the Erikson, Goldthorpe Portocarero social class scheme.[177]

Parental income was assessed by the mean annual income (Norwegian currency) in a two year period (1994 and 1995). The total income (including income from benefits) was used and defined by quintiles.

As described under psychosocial factors, also family living situation was taken into account in all papers.

4.6 Ethics

The Young-HUNT study was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Inspectorate. The current study was approved by the Regional Committee for Medical Research Ethics (reference 2010/1727-5), and was conducted according the Declaration of Helsinki. Participation in the Young-HUNT Study was voluntary. Each participant and also the parents/guardians of the participants younger than 16 years old gave their written consent to participate in the Young-HUNT Study, to use the Young-HUNT data for research and to link the Young-HUNT-data to other registers.

4.7 Statistical analyses

We presented in all papers the baseline characteristics of the investigated population. The statistical analyses in all papers were based on logistic regressions. The point estimates were presented with odds ratios (OR, all papers), risk differences (RD, all papers) and estimated 5- or 6-year risks (paper I and III) with 95% confidence intervals (CI). Predicted risk differences and 5- or 6-year risks were estimated from the logistic regression analyses with the covariates at their mean and follow-up time fixed at 5 or 6 years. Effect-measure modification was investigated with statistical interaction terms and reported when the p-value < 0.10. Data were analyzed with STATA V.11.1 (paper I) and STATA 12.1 (paper II and III) (StataCorp LP).

Paper I

We assessed the percentage of those receiving long-term medical and non-medical benefits in the follow-up period from 19 to 28 years old (mean follow-up time 8.2 years, range 6-10 years) for high school completers and dropouts separately. We also described the development over time of medical and non-medical benefit receipt in two successive 5-year periods (19-23 years and 24-28 years). Additionally, we estimated the percentage of the cohort who received long-term medical and non-medical benefits between ages 24-28 according to self-rated health and school dropout. Finally, we estimated the risk differences for medical or non-medical benefits compared to no benefits (period 24-28 years old) for complete cases (n=8339) with *multinomial regression analysis*. In the unadjusted models, high school dropout and follow-up time were included. The adjusted models included also self-rated health, sex and the mother´s education level. We tested for statistical interaction between school dropout and sex, and school dropout and self-rated health.

Multinomial logistic regression was used since the outcome variable had 3 nominal categories: no benefits (reference, base category), medical benefits and non-medical benefits.[178] The multinomial logistic regression assumes that the data are case specific (each independent variable has a single value for each case), that the dependent variable cannot be perfectly predicted from the independent variables for each case and there is no need for the independent variables to be statistically independent from each other. Basically, the point estimates of the mlogit command in STATA are reported in RRR (relative risk ratio): the risk ratio for an outcome category relative to the risk ratio of the base category:

$$RRR_j(a,b) = \frac{P(Y=j|x=a)/\ P(Y=0|x=a)}{P(Y=j|x=b)/\ P(Y=0|x=b)}$$

with the relative risk ratio for outcome Y=j versus base outcome Y=0 for a binary independent variable with values of x=a versus x=b.

To make it more straightforward to understand, we used *post-estimation commands* in STATA 11.1 to assess the risk differences ("mfx, predict") and the 5-year risk ("prvalue"). The prvalue-command in STATA measures the predicted probability of an outcome. The mfx-command in STATA estimates a marginal effect, or a partial effect, which measures how the probability for an outcome changes as a categorical variable (e. g. school completion versus dropout) changes from 0 to 1 with the other variables at their means or fixed (as defined by the predict-command):

Marginal effect of "$X_{\text{school dropout}}$"=$Pr(Y=j|X, X_{\text{school dropout}}=1) - Pr(Y=j|X, X_{\text{school dropout}}=0)$

with outcome Y=j

The difference in predicted probability of an outcome (in the paper described as "risk difference") can be expressed between the range of 0 to 1, or in percentages (0% to 100%).

Paper II

With sex- and age-adjusted *logistic regressions* we investigated the association for each health variable with high school dropout. The analyses were conducted on complete datasets for each health model defined by the health variable, with the N varying from 8205 to 8669 according to the investigated model. We added maternal education level and family living situation to adjust for possible socioeconomic confounders. We performed additional analyses using various socioeconomic variables (maternal, paternal and highest parental education, income and occupation separately and combined). We tested for statistical interaction between the health variables and sex and between the health variables and maternal education level. A sensitivity analysis by use of complete case-only was performed (n=7730). The predicted risk differences were estimated with the post-estimation command "margins" in STATA 12.1.

In multivariable *sibling fixed-effect (conditional logit) models* we estimated the association for each health variable with high school dropout while we took into account the heterogeneity at the family level.[179] These models were adjusted for sex, age and family living situation.

With sex- and age adjusted *multilevel logistic regression* for complete cases (n=7730) we investigated to what degree high school dropout was determined by the family of origin.[180] We included consecutively the individual characteristics (health variables) and the family variables (maternal education level and family living situation) to investigate the extent to which family level differences were explained by these individual and contextual characteristics.

The logistic regression models conditional on sharing the same biological mother ("clogit" command in STATA) or the *siblings fixed-effect logistic regression* models ("xtlogit, fe"

command in STATA) are equivalent.[181] These analyses with siblings discordant by their high school graduation status assessed the role of the familial factors (such as 50% shared genes and shared environment) on the relationship between each health variable and school dropout. It is important to notice that the conditional (fixed-effect) logistic regression only controls for *stable* (observed and unobserved) family characteristics (e. g. maternal education level) *shared* by the siblings. The observed stable variables are omitted from the analyses. If the shared stable family factors cause both the health problem and school dropout, one would expect the siblings-fixed effect analyses to show no relationship between exposure and outcome. The odds ratios of sibling fixed-effect models have a cluster-specific interpretation.

In the *multilevel logistic regression* analyses we estimated a conditional intraclass correlation coefficient (ICC) and the median odds ratio (MOR).[182] The ICC for a logistic regression was estimated with the linear threshold method because the scale of the individual level residual variance V_i (probability scale, variance for the underlying unobserved variable "individual propensity to dropout" or the variance within the families of origin) and the family level residual variance V_f (logistic scale, the variance between the families of origin) were not directly comparable. The underlying individual unobserved variable can be converted into a logistic scale and the individual level residual variance for this logistic distribution is equal to $\pi^2/3$ (=3.29). The ICC can be calculated as:

$$ICC = V_f / (V_f + 3.29)$$

The ICC expresses the propensity to drop out of high school that can be attributed to the family.

The median odd ratio (MOR) translates the variance between the families of origin (V_f) in the widely used odds ratio scale. The MOR depends directly on the variance between families and can be computed with the following formula:

$$MOR = \exp[\sqrt{(2 \times V_f)} \times 0.6745]$$
$$\approx \exp(0.95\sqrt{V_f})$$

The MOR quantifies the variation between clusters (families) by comparing two persons with the same covariates when randomly chosen from two different families. The MOR is defined as the median odds ratio between the person of higher propensity and the person of lower propensity. If the MOR is one, there is no variation between families.

Paper III

We investigated the association between high school dropout and long-term sickness absence and disability pension between ages 24 and 29 with sex-, age- and follow-up time adjusted logistic regression on complete datasets (N=6651). We consecutively added maternal education level, health measures, health behaviour, psychosocial factors, and school-related factors to adjust for possible confounders. We tested statistical interaction between high school dropout and sex and between high school dropout and maternal education level.

In sex-, age- and follow-up time adjusted *conditional logistic regression* we estimated long-term sickness and disability among siblings with and without high school dropout while controlling for all stable family characteristics that siblings share (number of siblings=316).[181] Consecutively, we added health measures, health behavior, psychosocial factors, and school related factors.

To investigate the overall vulnerability among adolescents, we developed a *propensity score* (from 0 to 1) which calculated the probability of a subject being in a specific group (high school dropout), conditional on that subject´s values on variables that influence group membership.[183] To compute this conditional vulnerability for dropout, we used a logistic regression with the dependent variable being high school dropout and the independent variables being sex, age, maternal education level, health measures, health behaviour, psychosocial factors, and school related factors. We computed the quintiles of the estimated propensity score with the first quintile representing the lowest probability to drop out of high school and the fifth quintile representing the highest probability. We tested the ability of the propensity score to differentiate those with high school completion from those with dropout (c-index), whether there was sufficient overlap between the two groups on the propensity score (box-plot) and whether the propensity score created groups comparable on baseline characteristics (comparing dropouts versus completers in the five propensity strata on important covariates). We carried out a logistic regression analysis with a statistical interaction between high school dropout and the propensity score stratified by quintiles. Predicted risk differences and 6-year risk estimates were calculated with the post-estimation command "margins" in STATA 12.1. As sensitivity analyses, we calculated a weighted estimate of the pooled odds ratio across the propensity score strata and we used propensity score matched methods to estimate the average treatment effect on the treated (ATT; in our case "the average dropout effect on the dropouts") based on the propensity score. Matching was performed with the technique radius matching with a propensity score radius of 0.1.[184]

About a quarter of the study population had missing data on the self-reported questionnaire, which might cause bias and/or inefficient estimates. As a sensitivity analysis we performed

multiple imputation with chained equations (MICE) procedures to obtain 20 imputed datasets by following the recommendations in the guidelines ("mi impute chained" command in STATA 12).[185] The multiple imputation uses the distribution of the observed data: it estimates a set of plausible values for the missing data drawn from the posterior predictive distribution of the missing data conditional on the observed data and it will sequentially also include imputed variables of x_i. The 20 created datasets were individually but identically analyzed. The estimates were combined to obtain overall estimates and confidence intervals. The used technique assumes that the missing data are missing at random. Many variables that are associated with non-participation in surveys were included in the dataset and it reduces the probability that data being missing does depends on unobserved data, conditional on the observed data.

All the relevant variables from the main multivariable analyses except "academic problems" (because of collinearity with "concentration problems") were used in the multiple imputation models. Additional to the outcome long-term sickness and disability, we included also unemployment and social insurance support. All variables were included as binary variables and imputed using logistic regression. Those variables without specific cut-off were dichotomized into highest tertile versus the two lowest tertiles. We repeated the main multivariable logistic regressions with these binary variables and compared it with the results obtained by the analyses of the imputed datasets.

5 Results

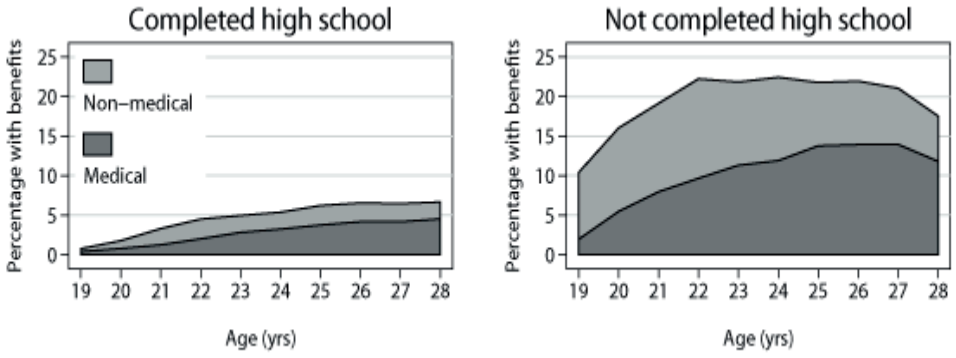
5.1 Paper I

School dropout – a major public health challenge: A 10-year prospective study on medical and non-medical social insurance benefits in young adulthood, The Young-HUNT 1 study (Norway)

Information on education and social insurance benefits from 8795 was used to describe the percentage of those who were receiving benefits. The multinomial logistic regression analyses were performed on complete cases (n=8339).

A total of 17% was registered as being high school dropouts at age 24. There was a 27% school dropout rate in adolescents who reported poor health compared with 16% in those who reported good health.

We observed a large difference in benefits receipt between high school dropouts and completers (Figure 4.1).



Figur 4.1 Percentage receiving long-term medical and non-medical social insurance benefits at different ages in the follow-up period for (A) persons who completed high school at age 24 and (B) persons who did not complete high school at age 24.

Most participants who received medical benefits in the early period (age 19-23) also did so in the late period (age 24-28) (66%, 344). On the other hand, most participants who received non-medical benefits in the early period did not receive any benefits in the late period (62%, 474).

The predicted 5-year risk of receiving benefits between ages 24-28 was 21% (95% CI 20-23). High school dropouts had a 5-year risk of receiving benefits of 44% (CI 41-48), compared with 16% (CI 15-17) in those who completed high school (adjusted for self-rated health, parental education and sex). The predicted 5-year risk of receiving any long-term social insurance benefits in adolescents who reported poor health was 33% (CI 30-37) compared with 20% (CI 19-21) in those who reported good health. The risk increase between good and poor health was substantial and of the same size for both medical and non-medical benefits, independently of high school completion (Table 4.1).

There was no evidence for statistical interaction between high school dropout and sex ($p>0.1$) and between high school dropout and self-rated health ($p>0.1$).

Table 4.1 Predicted risk difference (RD, in %, with 95% confidence intervals (CI)) of receiving long-term medical and non-medical benefits between the age of 24 and 28 from multinomial logistic regression models*.

	Medical benefits				Non-medical benefits			
	Crude†		Adjusted‡		Crude†		Adjusted‡	
	RD	CI	RD	CI	RD	CI	RD	CI
High school dropout	15.1	(12.5 to 17.6)	15.3	(12.5 to 18.1)	15.2	(12.5 to 17.9)	13.2	(10.4 to 16.0)
versus completion	ref.		ref.		ref.		ref.	
Poor self-rated health	7.0	(4.2 to 9.7)	4.8	(2.3 to 7.4)	6.2	(3.4 to 9.1)	4.6	(2.0 to 7.3)
versus good health	ref.		ref.		ref.		ref.	
Male	-3.4	(-4.9 to -2.0)	-4.3	(-5.8 to -2.8)	-0.1	(-1.6 to 1.4)	-1.8	(-3.3 to -0.3)
versus female	ref.		ref.		ref.		ref.	
Maternal education								
Primary	ref.		ref.		ref.		ref.	
Intermediate	-4.0	(-6.0 to -2.0)	-2.6	(-4.6 to -0.7)	-3.0	(-5.0 to -1.0)	-1.9	(-3.8 to 0.01)
Tertiary	-6.5	(-8.2 to -4.7)	-4.3	(-6.2 to -2.3)	-7.4	(-9.0 to -5.8)	-5.8	(-7.5 to -4.0)

*Estimated risk difference in the 5-year risk to receive medical and non-medical benefits relative to no benefits.
†Crude model with the follow-up time at 5 years.
‡Adjusted model with the follow-up time at 5 years and the covariates at their mean.

5.2 Paper II

Adolescent health and high school dropout: a prospective cohort study of 9000 Norwegian adolescents (The Young-HUNT).

Based on the 8750 participants who scored their health in the Young-HUNT1, the total N in the whole population analyses for each health variable varied from 8205 to 8696, and in the sibling fixed-effect models from 581 to 649.

All explored health dimensions were strongly associated with high school. In models adjusted for maternal education level, the risk differences of high school dropout according to health exposures varied between 3.6% (95% CI 1.7 to 5.5) for having ≥1 somatic disease versus none and 11.7% (6.3 to 17.0) for being obese versus normal weight (Table 4.2). Adjustment

for other parental socioeconomics measures did not alter the results (parental educational level, income and occupational class). Complete cases analyses (n=7730) showed the same associations between the health variables and school dropout. For all health variables, there was no evidence for effect measure modification by sex or maternal education (p-value for interaction >0.1).

Table 4.2 Odds ratio for high school dropout according to indicators of adolescent health in the whole population (crude and adjusted models) and within the families (sibling fixed-effect models). Values in parentheses are 95% confidence intervals (CI).

	Crude[a]		Adjusted[b]	Within family effect[c]	
	N dropout	Odds ratio (CI)	Odds ratio (CI)	N dropout	Odds ratio (CI)
Somatic disease					
None	1070	1.00	1.00	244	1.00
1 or more	358	1.39 (1.22 to 1.59)	1.32 (1.15 to 1.51)	78	1.06 (0.70 to 1.60)
Somatic symptoms					
None or 1	803	1.00	1.00	169	1.00
2 or more	566	1.51 (1.34 to 1.71)	1.42 (1.25 to 1.62)	125	1.29 (0.87 to 1.90)
Psychological distress					
Low	1202	1.00	1.00	298	1.00
High	180	1.69 (1.41 to 2.03)	1.56 (1.30 to 1.88)	37	1.07 (0.64 to 1.78)
Insomnia					
Never/seldom	1201	1.00	1.00	272	1.00
Often/every night	193	1.67 (1.40 to 1.99)	1.66 (1.39 to 1.99)	39	1.27 (0.75 to 2.15)
Concentration difficulties					
Never/seldom	881	1.00	1.00	197	1.00
Often/very often	497	2.13 (1.88 to 2.43)	1.98 (1.74 to 2.26)	108	1.69 (1.12 to 2.53)
Self-rated health					
Very good/good	1163	1.00	1.00	261	1.00
Not so good/bad	245	2.07 (1.77 to 2.43)	1.81 (1.53 to 2.13)	48	1.44 (0.87 to 2.39)
BMI					
Normal weight	975	1.00	1.00	133	1.00
Overweight	231	1.47 (1.25 to 1.73)	1.34 (1.14 to 1.58)	39	0.93 (0.55 to 1.56)
Obese	71	2.39 (1.80 to 3.18)	2.20 (1.64 to 2.95)	14	4.18 (1.11 to 15.7)

[a] Crude models adjusted for sex and age.

[b] Adjusted for sex, age, maternal education level and family living situation.

[c] Sibling fixed-effect models are adjusted for sex, age and family living situation

Total N varies for each health variable in the total population from 8205 to 8696, and in the sibling fixed-effect models from 581 to 649

The results from the analyses comparing differentially exposed siblings, confirmed these results with the exception of weaker associations for somatic diseases and psychological distress (Table 4.2).

School dropout was strongly clustered within families (family level conditional intraclass correlation 0.42). When individual health variables and contextual variables (maternal education level and family living situation) were included, the unexplained cluster heterogeneity decreased substantially, yielding an ICC of 0.29.

5.3 Paper III

High school dropout and long-term sickness and disability in young adulthood: a prospective propensity score stratified cohort study (the Young-HUNT study)

We performed the main analyses on complete cases (n=6612).

The crude 6-year risk difference for long-term sickness absence or disability pension for a school dropout compared to a completer was 0.21 or 21% points (95% confidence interval (CI), 17 to 24). The risk difference gradually decreased to 15% points (95% CI, 12 to 19) with the successive adjustment for maternal education level, health measures, health behavior, psychosocial factors, and school-related factors (Table 4.3). There was no evidence for statistical interaction between school dropout and sex and between school dropout and maternal education level ($p > 0.1$).

A sensitivity analysis with multiple imputation by chained equation procedures showed that the magnitude and direction of the differences in long-term sickness or disability in young adulthood were in accordance to the main analysis of complete data.

The sibling fixed-effect analysis confirmed the results from the total population, but the odds ratios were substantially lower (Table 4.3).

Table 4.3 Risk difference* and odds ratio for long-term sickness or disability between age 24 to 29 years for high school dropouts versus school completers in the whole population and within the families.

	Model 0	Model 1	Model 2	Model 3	Model 4	Model 5
Dropout *versus* completion(ref.)						
Whole population						
Risk difference	20.8 (17.0 to 24.7)	18.7 (15.0 to 22.4)	17.4 (13.8 to 21.1)	16.6 (13.0 to 20.4)	15.8 (12.2 to 19.5)	15.3 (11.7 to 19.0)
Odds ratio	3.92 (3.28 to 4.68)	3.53 (2.95 to 4.24)	3.34 (2.8 to 4.0)	3.20 (2.65 to 3.86)	3.07 (2.54 to 3.71)	2.96 (2.44 to 3.60)
Within family[1]						
Odds ratio	1.89 (0.96 to 3.74)	–	2.03 (1.01 to 4.08)	2.53 (1.15 to 5.54)	2.48 (1.13 to 5.49)	2.39 (1.04 to 5.47)

*Estimated risk difference in the 6-year risk for long-term sickness and disability with the covariates at their mean.
Risk difference (in %, with 95% CI) and odds ratio (with 95% CI) in the whole population with logistic regression models (N=6612) and within the families with sibling fixed-effect models (N=316).
Model 0: adjusted for sex, age, and follow-up time.
Model 1: model 0 +adjusted for maternal education level.
Model 2: model 1 + adjusted for somatic disease, symptom load, psychological distress, concentration problems, insomnia, and self-rated health.
Model 3: model 2 + adjusted for overweight, smoking, and physical activity.
Model 4: model 3 + adjusted for self-esteem, subjective well-being, loneliness, and family living situation.
Model 5: model 4 + adjusted for reading and writing difficulties, bullying, disease-related school absence, educational aspirations, academic problems, school dissatisfaction, and school-related conduct.
[1] In the Within-family models the covariate maternal education level is omitted.

Overall, high school dropout increased the risk for sickness or disability regardless of the risk factor level present for high school dropout. In figure 4.2 this is visualized by the predicted 6-year risks for long-term sickness and disability for each stratum of the propensity score of the school dropouts and completers. The pooled odds ratio across the propensity score strata was

2.95 (95% CI, 2.44 to 3.57), which resulted in an estimated risk difference between school dropouts and completers of 16.7% points (95% CI, 12.2 to 21.3). This was similar to the estimated ATT of 0.165 (95% CI, 0.136 to 0.194) in the radius matched propensity score analyses.

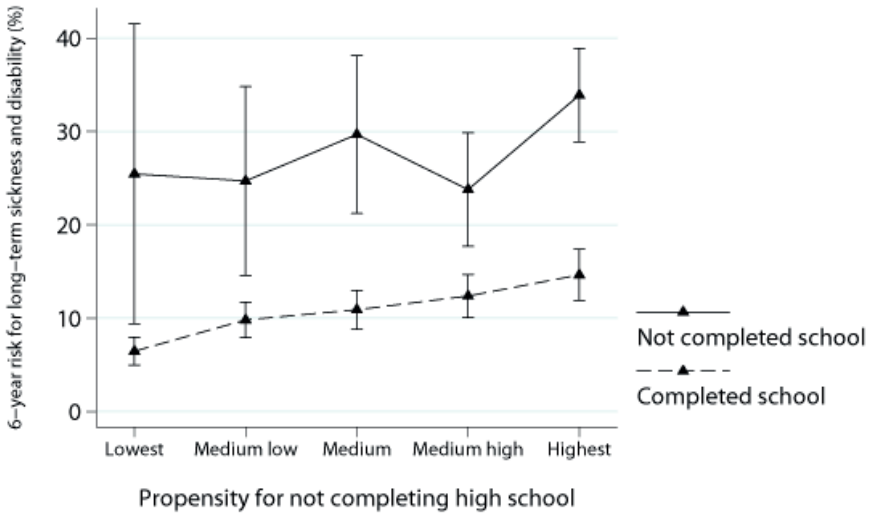


Figure 4.2 Estimated 6-year risk for long-term sickness or disability at age 24-29 according to the adolescents´ propensity for not completing high school and high school graduation status (N=6651).

A high school dropout with lowest risk factor level (stratum one) had a higher risk for long-term sickness and disability than a high school completer with the highest risk factor level (stratum five). We had no evidence of effect measure modification between the propensity score and dropout ($p > 0.1$).

6 Discussion

6.1 Main findings

The overall aim was to investigate health related factors in adolescence, accomplishment of secondary school, and labour market integration in young adulthood, and to explore the role of the family. Principal findings can be summarized as follows:

- There was a strong association between poor self-rated health, high school dropout and the risk of receiving medical and non-medical benefits in young adulthood.
- We found an increased risk of high school dropout for all explored dimensions of ill health in adolescence. This was also true when comparing siblings, with the exception of psychological distress and chronic somatic disease.
- High school dropout was strongly associated with parental socioeconomic position and strongly clustered at the family level, but the impact of ill health on school dropout seemed to exist in all families and across all social classes.
- The strong association between high school dropout and long-term sickness and disability in young adulthood remained even when adjusted for adolescence vulnerability (defined by parental socioeconomic position, adolescent health, health-related risk behaviours, psychosocial risk factors, and school problems). This was also true when comparing siblings.
- A high school dropout had systematically a higher risk for long-term sickness and disability independent of the level of risk factors to drop out of school.

6.2 Strengths and limitations

The goal of epidemiologic studies is to obtain an accurate estimate, i. e. a valid and precise estimate of the frequency of an outcome or of the effect of an exposure on the occurrence of an outcome in the source population of the study. A further goal is to obtain an estimate that is generalizable to the relevant target populations.

6.2.1 Internal validity (lack of systematic error)

Another word for systematic errors in estimates is bias. The opposite of bias is validity: an estimate with little systematic error can be described as valid.[186] This chapter describes the internal validity or the validity of the estimates as they relate to the source population. Systematic error leads to nonrandom deviation of the estimates from the true values and is independent of the study size.[186] We can classify bias into three broad categories: selection bias, information bias, and confounding.[187]

6.2.1.1 Selection bias

Selection bias is a systemic error that originates from factors that influence study participation and from procedures used to select subjects.[186] Basically, the relation between exposure and outcome might be different for those who participated and those who should have been theoretically eligible for the study, including those who did not participate.

The participation rate in the Young-HUNT1 Survey was high: 90% of all school attending adolescents filled out the questionnaire, and 85% completed both the questionnaire and the clinical exam. However, we cannot rule out the possibility of selection bias. Firstly, we did

not include adolescents who were not enrolled at school at baseline, and this increases the risk for selection bias due to school dropout among the senior high school students. Secondly, as school dropout is the result of a long-term process which is often characterized by absenteeism prior to dropout, there might also be more school dropouts among the non-respondents. If present, such bias would likely lead to underestimation of the associations between the exposures and the outcomes in the present studies.

In paper I, we excluded 81 participants (<1%) who died during follow-up or received disability pension during follow-up (when the social insurance process was started before 1998). In fact, we selected based on the outcome and it could potentially bias our outcome. However, the group was small and did not affect our results. We included these participants in paper III.

Loss to follow-up is unlikely to play an important role in our studies as we could rely on national register-based information that was nearly complete for our outcomes. Data on emigration provided by the Statistics Norway allowed us to censor for people who emigrated from Norway (paper III). The percentage of emigrants was small (<1%) and did not affect our estimates.

We presented the main analyses in all papers based on complete case analyses. This implies for paper III that we excluded about 24.6% of the participants because of 1 or more missing data in the Young-HUNT1 Survey material. Further investigation on the pattern of missingness taught us that especially future school dropouts had more often missing data. Based on this pattern, our estimates are probably underestimated. Comparison of the basic models (model 0 and 1) based on the whole population and based on the complete cases,

confirmed a pattern of underestimation of 1 to 1.5% points in the predictions of risk differences.

As a sensitivity analysis, we also performed the analyses after multiple imputation and they did not differ substantially from the analyses of the complete cases. Multiple imputation requires that the missing data are missing at random (MAR), which means that the probability of data being missing does not depend on the unobserved data, conditional on the observed data. Using the rich information in the Young-HUNT study to impute missing data, we assumed that missing data were missing at random. In fact, a large majority of participants (83%) lacked information on just 1 or 2 variables. Many variables that are associated with non-participation in surveys were included in the dataset, which reduces the probability that data missing does depend on unobserved data, conditional on the observed data.

6.2.1.2 Information bias

Information bias can be caused when the information collected about or from the study subjects is erroneous.[186] As the variables in the papers mainly are discrete variables, the measurement error is usually called classification error or misclassification. Misclassification for either exposure or outcome can be differential or nondifferential. Misclassification of exposure is called nondifferential if it is not related to the occurrence or presence of the outcome. If the misclassification of exposure is different for those with and without a certain outcome, it is differential. Similarly, misclassification of the outcome is nondifferential if it is unrelated to the exposure; otherwise, it is differential.[186] Differential misclassification can either exaggerate or underestimate an effect. Nondifferential misclassification tends to produce estimates that are biased toward the null.[186]

Based on the prospective design, registration of the exposures are likely to be unrelated to the outcomes (school dropout or benefits). However, as school dropout is the endpoint of a long-term process, there might be a theoretical possibility of differential misclassification of the self-reported health variable in paper I. It could be possible that participants who experienced school-related difficulties, more often reported poor self-rated health because adolescents conceptualize health as a construct related to medical, psychological, social, and lifestyle factors.[80] If so, our estimates could be overestimated, especially for the older adolescents. Because of the prospective design and the use of national registers to collect and define our outcomes, it is unlikely that we have a differential misclassification of our outcomes.

Potential information bias is more likely to be related to the self-reported health exposures. In paper I and II, the exposure "self-rated health" with four answer categories was dichotomized. Similarly, we dichotomized most of the exposures in paper II. The psychological distress measure (SCL-5) was dichotomized with a cut off at 2 as suggested by Strand et al.[167] This cut-off is validated only for a population above 15 years of age. The other dichotomized measurements have not been validated. Potential misclassification due to dichotomization was considered to be nondifferential or independent of the other variables and could give a bias towards null. Therefore, we performed also sensitivity analyses in paper II using variables with all existing categories or as continuous variables and these analyses showed dose-response relationship between all the health variables and the risk for high school dropout. Because weight and height (BMI) were collected by trained nurses using a standard protocol and classified as proposed by the International Obesity Task Force (IOFT), we assume that misclassification of BMI is less likely.

As self-reported information is collected from the participant, the sibling designs might be more sensitive to information bias: if siblings tend to respond in a more similar way on e. g. questions related to mental health because of family attitudes to that subject, it might cause a bias towards null. As we did not find an association between psychological distress and school dropout in the siblings fixed designs, we could not define whether this was due to clustering of psychological distress in families, to other unknown factors that siblings shared or simply the consequence of information bias.

Our outcome measures were collected from FD-Trygd and National Education Database (NUDB) by the Statistics Norway.[156 157] It is difficult to judge the quality of these large databases, but Statistics Norway has documented on its website the principles by which the data are processed and the consistency controls. Especially for national insurance (pension and benefits), cross-topic consistency controls have been performed. When we controlled the quality of the variable "maternal education level" by using different data sources from Statistics Norway, we could confirm a very good consistency over the different data files. As easy it was to construct a "school dropout" variable based on one data file, as challenging it was to construct "long-term benefits" variables. First, the construction of "benefit" variables is based on several data files and on several benefit structures, which both could change over time (e.g. files on rehabilitation benefits or unemployment before and after 2001, introduction of time limited disability pension in 2004) and be potential sources of errors.[157] Second, the construct "180 days on benefits during one calendar year" may result in underestimation of the long-term benefits receipt as participants receiving 40 days of benefits at the end of calendar year 1 and 160 days in calendar year 2 will be registered as "no long-term benefits". Third, another source of underestimation of "non-medical long-term benefits" is the definition "unemployment" by registering only those who received financial support. We did not include

non-financial unemployment measures. Potential misclassification is assumed to be nondifferential and if present, it would result in underestimated estimates.

6.2.1.3 Confounding

Confounding can be thought of as mixing of effects. A confounder must be associated with the outcome (as a cause or a proxy for a cause for the outcome) and be associated with the exposure (but not be an effect of the exposure).[186] There are several ways to control for confounding: common methods in the study design are restriction, stratification or matching, and with multivariable analyses it is possible to control for several confounders at once.

Also confounders can be misclassified: nondifferential misclassification can result in residual confounding and might distort the effect-measure modification, while differential misclassification might result in a complete distortion of the effect estimates.[186] In all papers, we adjusted for familial socio-economic position with maternal education level as this variable was most complete and least exposed to misclassification. Also other familial SEP indicators have been thoroughly examined in paper II, but they were not used in the other papers as there was a higher risk for misclassification (occupational class was dependent on self-reported information of the parents and income could be defined in many ways). In paper III, we adjusted for many confounders which were mainly based on the self-reported information of the participants. Some of these confounders, e. g. school-related problems, are more at risk for differential misclassification (reporting of school-related problems might be dependent of the process of school dropout). Therefore, we performed additional analyses with other study designs: we used a propensity score for dropout (stratified and matched) and these analyses resulted in estimates of the same size and direction.

To assess which confounders should be included, we used causal diagrams or directed acyclic graphs.[188] Controlling for variables that are mediators (intermediate variables between exposure and outcome) might introduce bias.[186] Other publications with the same research focus as in paper II (examining adolescent health and educational attainment) often also adjust for educational achievement in high school. We chose not to adjust for school problems as in our hypothesis ("Does poor health increase the risk for school dropout?") it should be a mediator, although we realize that there might be a reciprocal effect between health and school problems. If we should have adjusted for the mediator "school problems" and there was a potential confounder between school problems and dropout, such as IQ, than it could result in an underestimation of the direct effect of poor health on dropout and an overestimation of the indirect (school-problem mediated) effect.

The Young-HUNT1 Survey had comprehensive psychosocial, demographic and clinical data on each participant, and allowed us to control for many confounding factors. We cannot exclude the possibility of uncontrolled confounding, i. e. factors for which we had no information could have a confounding effect. The siblings fixed analyses in paper II and paper III suggested that stable family characteristics shared by the siblings are important in understanding the examined associations, and we might not have captured all the necessary characteristics related to the family, such as coping behaviours, familial health, and genetics.[152 189-191] Nevertheless, it appears unlikely that there was a remaining confounder in paper III that (alone) could be potentially able to influence our results considerably, as it would need to be strongly associated with both exposure and outcome, and be unrelated to the other factors that were included in the analyses. Finally, there might be residual confounding due to the fact that we used only information on the adolescents at one point (Young-HUNT1)

in time and eventually changes in physical and psychological health over time could not be taken into account.

6.2.2 Precision (lack of random error)

Random error is the variability in the data that we cannot readily explain: it is a fluctuation in the data caused by any factors that randomly affect the result of the measurement.[186] The opposite of random error is precision and an estimate with little random error is precise. In our studies, the random error around the estimate is indicated with a level of confidence of 95%.

The precision depends on the size of the study, the prevalence of the exposure and the prevalence of the outcome. In general, the main analyses in all papers are based on the "whole population" with a large sample size which results in estimates with high precision (and small confidence intervals). Paper II and III have analyses on a subpopulation ("siblings disconcordant on outcome") with substantially lower N, and of course, lower precision. Similarly, sensitivity analysis with propensity score matching were based on the radius technique (with m:1) matching, in stead of e.g. nearest neighbour matching (1:1) as radius matching produced the same estimates, but with higher precision. All study exposures were common for the adolescents in the cohort, although dividing overweight into overweight and obesity groups reduced the precision in the subgroup "obesity", especially when analyzing siblings (paper II). Finally, the outcomes (school dropout and benefits) were common in the cohort. Because we studied a cohort in young adulthood when long-term medical benefits might be less frequently present, we studied the proportion of benefits over a longer time period (5 or 6 years period).

6.2.3 External validity (generalizability)

External validity refers to whether, and the degree to which, our results apply to people outside the studied population.[186] As the Young-HUNT1 Survey was designed as a population based cohort of adolescents in Nord-Trøndelag in 1995-97 and had a response rate of 90% for school-attending adolescents, it is reasonable to state that our results are generalizable for the county of Nord-Trøndelag. To a large extent they are also generalizable to Norway, as the Norwegian population is relatively homogenous in relation to health, education, labour and welfare. Exceptions might be ethnic groups (which are underrepresented in Young-HUNT1) and inhabitants of large cities (with more heterogeneous groups). We studied adolescents between 13-19 years of age and our results can not be generalized to younger age groups.

Our social measurements, such as school dropout and labour market integration, are strongly dependent of the Norwegian organization of the school system, labour market and welfare systems. Comparative descriptive statistics from OECD (see 2.1.2 Labour market exclusion) suggests that school dropout and labour market integration are a common challenge for all countries. However, there might be national differences in prevalence because of different organizational structures, e.g. obligatory school attendance until age 16 (Norway) or 18 (e.g. Belgium), financial support in case of unemployment in maximum 24 months (Norway) or indefinitely (e.g. Belgium). Similarly, the HBSC Study described that health problems in adolescence occurred in all participating countries, although the prevalence may differ (please see 2.4.2 “The prevalence of chronic disease and illness in adolescence”). Hence, we can not generalize the size of our effect estimates to other nations because of the differences in prevalence, but the presence of such associations are highly likely to exist in other countries also.

6.3 Interpretation of the main findings

6.3.1 Adolescence as a turning point during the life course

Social causation theory

As described earlier under "Pathways to health-related labour market integration" (Background), socioeconomic conditions have been accepted as a major explanation for social inequalities in health (social causation theory). In paper I and III, we confirmed as such that (own) socioeconomic position, defined by high school completion or not, was associated with an at least three times increased risk for both medical and non-medical benefits. The novelty in paper I was that these differences were dramatic and present from the day the adolescents entered the labour market as very young adults, and persistent throughout whole the examined period in young adulthood. Gravseth et al.[13] described an increased risk for early disability pension among men and women with low education. At an age of 30 years, 2% of the low educated persons had a disability pension compared to less than 0.5% among the high educated group. Bjerkedal et al.[115] and Kristensen et al.[15] described that men with low educational level were more often unemployed or registered as inactive on the labour market in young adulthood.

Adolescence as a turning point

In this era characterized by a proliferation of educational and training schemes, these findings related to the youth-adult transition are far from trivial.[192] In paper I, the strong association between dropout and subsequent benefits remained unchanged when adjusting for confounding factors such as parental socioeconomic position and self-rated health in adolescence. Gravseth et al.[13] found also the great importance of education. The overall population attributable risk (PAR) for disability pension before age 36 years for a persons' educational level was twice as high as the PAR for all four childhood factors (birth weight,

childhood disease benefit, maternal marital status and parental disability). Also a Finnish study showed an unchanged strong association between educational attainment and disability pension in middle-aged adults when adjusting for family-related childhood adversities.[193] Despite the strong associations, we were still cautious to draw conclusions related to causality in paper I and stated "We do not know whether high school dropout itself is the cause of long-term medical and non-medical benefits in young adulthood or whether high school dropout and long-term social insurance benefits are joint consequences of other individual or contextual factors, like health, family or school (confounding)". Indirectly, the latter referred to the main life course causal model based on an accumulation model with risk clustering. Therefore, we extended our model (with main focus on long-term sickness absence and disability pension) in paper III by taking into account the available information in Young-HUNT1 on the adolescent vulnerability such as self-reported health, health-related risk behaviour, psychosocial risk factors, school related problems, and parental socioeconomic position. In this case, we could explain about one fourth of the association between dropout and medical benefits. Paper III contributes to the international literature by combining many of the known risk factors for school dropout into one score, the propensity score for dropping out of high school. By comparing participants within the same strata of the propensity score, or even by matching them to another person with a similar score, dropping out of school increased the risk for long-term sickness absence or disability pension systematically with approximately 16%. The graph in paper III visualized also that an adolescent with a high level of risk factors present, but succeeding at high school had a lower risk for being out of the labour force because of sickness or disability than an adolescent with nearly no risk factors present, but who did not complete high school. As such, the educational achievements during adolescence, confirmed with a high school degree, can be a marked change of direction or a turning point during the life course.[19 194]

Chains of risk with additive effect

In paper I, self-rated health in adolescence increased the risk for both medical and non-medical benefits in young adulthood with nearly 5% points, even while adjusting for parental socioeconomic position and high school completion. In paper III, the risk for long-term sickness and disability increased with about 7% points for a person with highest propensity to dropout compared to a person with lowest propensity, also independent of high school completion or dropout. In the study of Harkonmäki et al.,[193] the sum of family-related childhood adversities increased the risk for disability pension partly through, but also additive to low educational level. Two Scandinavian studies on men showed that IQ influenced educational attainment, but that IQ and educational level also independently were associated with disability pension.[110 111] Our findings suggested that the association between adolescent vulnerability and long-term sickness and disability not only was mediated through educational attainment, but that there also was an independent effect of adolescent vulnerability on medical benefits in young adulthood. Our results are therefore supportive for a life course model based on an accumulation of risks with chains of risk with additive effects. One Swedish study on sickness absence not only found additive effects of serious domestic problems in childhood and own adult socioeconomic position, but also a statistical interaction (for men only).[41] However, we did not find such gender-specific differences (data not shown).

Potential danger for social exclusion

We described adolescence as a turning point in life, which refers to a life event that produces a lasting shift in the life course event.[195] In fact, the majority of adolescents in high school pass through the system in a steady stream. Many of them will make the transition to a tertiary education student, while others choose to go directly into the labour force. The life event to cease formal schooling is a rather stressful event which can be a point in the life course that

represents a substantial change in direction, such as a pathway to marginalization and social exclusion.[195] It may be a turning point for one individual, but not necessarily for another. Similarly, completing high school as a "vulnerable" adolescent can be the turning point to social integration and affluence.[194] In paper I, it appeared that once people in early adulthood had received medical benefits, the majority received medical benefits later on in adult life also. About two third of the receivers of non-medical benefits in the early twenties returned to work or school. However, they still had a larger risk for receiving benefits than young people who had not received benefits in the early twenties. These findings are consistent with international literature on social exclusion. A British study described that young people have a higher risk to remain NEET (not in education, employment, or training) mainly due to lack of qualifications, and because of poor labour market experiences.[196] This effect was stronger the longer they had been NEET. A Norwegian study also described a greater risk of social exclusion for young people out of work for more than one year, and if participation was the norm, the risk increased with even shorter breaks.[5] In our study, we could not differentiate for marginalization and social exclusion, but high school dropouts had in general a much higher risk to be out of the work force for (a) longer period(s) than high school completers. During a five year period (24-28 years of age), about 50% of the school dropouts had been on benefits for at least 6 months during one calendar year or with other words, about half of this group was in potential danger for marginalization and social exclusion.

Possible mechanisms

In paper III, we observed that a high school had systematically a higher risk for long-term sickness and benefits, independent of the level of adolescence disadvantages. With obtained level of education in young adulthood, the young adult creates now his own SEP and his future opportunities in the labour market. The poor odds for high school dropouts for health-

related exclusion from labour market could not simply be explained by previous health status, health-related behaviour, parental SEP, or other risk factors in adolescence. Why do school dropouts face so poor odds? Are the possible mechanisms situated at a societal level, an individual level, or a reciprocal effect between these levels?

In all industrialized countries, high school dropouts are forced into certain social circumstances: reduced work prospects and jobs with a higher risk for increased job strain and more physical demands.[23] One could argument that e.g. low decision latitude is the "cause" for later long-term sickness absence, but the strong associations disappeared with adjustment for educational attainment and childhood IQ.[8] In this longitudinal study, low decision latitude seemed to be rather a mediator, than a direct cause. Ultimately, when ill health is present, there might be an increased risk for medicalization during the social process of dropout and subsequent reduced work integration, as with job loss and unemployment.[197]

Might there be skills and qualifications on the individual level that adolescents may develop in school or that are important to complete high school? First, it is not likely that only "one factor" could be able to explain the association between dropout and long-term sickness, because the association is very strong and the factor usually will be associated with one or more of the variables included in paper III. Second, there will be, with a high probability, reciprocal effects between such factors and the social context, such as school and work environments. To succeed at school, cognitive ability is important, but no absolute guarantee for success. One should also be willing to exert a considerable effort and self-regulation has been suggested to be the most essential asset.[191] Self-regulation has been defined and examined in many ways, and goal level, persistence, effort, and self-efficacy had the strongest effect on learning.[191] Learning is no longer only related to school-context, but work has also

become more complex and knowledge-centric, requiring employees to adapt to changing job demands. The ability to adapt is one thing, the perception of their ability to change themselves and their environment might be another ting. This perception might be influenced by personality and coping strategies.[198 199] A stress moderator, sense of coherence, has been associated with ill health and sickness absence in previous studies.[200] According to Antonovsky, people with a high sense of coherence use more effective coping strategies to handle stress.[201] However, the more resistance resources an individual possesses, such as education, wealth, work-related factors, and social support, the better are the chances for a strong sense of coherence. These education and work experiences are associated with the three key areas of sense of coherence (comprehensibility, manageability, meaningfulness), and thus give rise or reinforce sense of coherence. Sense of coherence is associated with school- and work stress, but the extent to which it is influenced by school- and work related stress, or the extent to which it predicts such stress (and subsequent ill health) remains questionable.[202] If the first mechanism is the most prominent, than we are back to "start": in that case, a higher risk for long-term sickness and disability is inherent to the status of school dropout in modern society, unless there would be societal changes that creates better work perspectives and opportunities for this group.

6.3.2 Health selection during adolescence

Health selection

In paper I, we touched the subject "health selection" as we examined and found associations between self-rated health, school dropout and benefits. We went more in depth in paper II, especially in relation to educational attainment. As described in chapter 2.4.3 "Adolescent health and school dropout", we could find evidence in the literature for an association between each of the examined health problems and school dropout (eventually low

educational attainment or achievement), although there were also some studies which did not found such association. All of these studies had adjusted for parental SEP (education, social class and/or income), but only a few tested the confounding effect of parental SEP.[67 84] In paper II, we found a solid association between chronic somatic disease, high symptom load, psychological distress, insomnia, concentration problems, poor self-rated health, overweight, and obesity in adolescence and school dropout. The associations were slightly attenuated with adjustment for parental SEP and family living situation. In concordance with other studies, ill health in adolescence had an effect on high school completion, independent of parental SEP.[39 61 127] To formulate it differently: the relationship between ill health was not less important for higher social classes. As such, our study suggests the existence of a health selection process during the educational trajectory in adolescence. Additionally, in paper I, the results suggested also that health selection continues during the work attainment process in young adulthood in concordance with a study of Haas[203], although it is not that strong as the social causation effect based on own socioeconomic position. All together, social causation and health selection mechanisms are not mutually exclusive and besides paper I and III, paper II is also additional evidence for the "chains of risk with additive effects"-model in the life course perspective.

The within-family analyses

When controlling for stable shared family background characteristics in paper II, the impact of ill health in adolescence on school completion remained in the case of high symptom load, insomnia, concentration difficulties, poor self-rated health and obesity. These results are in concordance with other studies on poor self-rated health in adolescence and educational attainment.[39 62] For concentration problems, our result is inconsistent with the study of Fletcher et al.[204] The adolescents with ADHD in the Add Health study did have a higher risk

for school dropout, but when comparing siblings, the sibs with ADHD did not have a higher risk for dropout than their sibs without ADHD, which Fletcher et al. contributes to the negative spill over effect: the presence of a sibling with ADHD is negative for the other siblings educational outcomes. The regression as presented by Fletcher et al. was tested out on our material, and showed a similar negative effect of siblings´ concentration problems (data not shown) on own educational outcome. Therefore, the spillover effect is maybe not a good enough explanation why our result of the siblings fixed effects analyses is inconsistent. Of course, a screening set for ADHD symptoms is not the same as self-reported concentration problems. Additionally, the Canadian study suggested that inattention predicts low long-term educational attainment, while hyperactivity was no longer a predictor when inattention was taken into account.[99] As far as we know, our findings that siblings with high symptom load, insomnia, or obesity have a higher risk to dropout of school compared to their healthy siblings are a novelty. These results strengthen the health selection hypothesis in adolescence and the findings are not irrelevant as today, obesity is an "epidemic", the sleep quality of youth is in danger because of computer use, and sickness benefits for minor mental disorders such as insomnia increased (see 2.2.4).

For somatic disease, psychological distress, and overweight, the impact on school dropout was completely attenuated for siblings differentially exposed and this may suggest that shared family background characteristics are essential in the association between health and school dropout. However, the results are not consistent with two other studies which found that siblings with diabetes and depression had a higher risk for dropout compared to their healthy siblings.[84 205] It might be because of the fact that psychological distress is clustered within families,[146] and that our measurement of symptoms of mental illness did not differentiate good enough psychiatric pathology between siblings. Similarly, it would have been favorable

to differentiate in different types of somatic disease, but our sample size was too small to reproduce the same analyses with only diabetes or epilepsy. Finally, in similarity with the concentration variable, we tested out the spillover effect between siblings for psychological distress: in fact, the presence of reported high psychological distress in a sibling had more effect on dropout than one´s own reported distress (data not shown). But, if it would be so that the presence of psychological distress in a sibling reduces the parental investments of parents with the other children, or if there should be a direct negative influence of living with a sibling with psychological distress, the question still remains why we did not found this effect in case of "concentration problems".

Possible mechanisms

There are several mechanisms which may explain the effect of ill health in adolescence on educational attainment: directly through impairment of cognitive development (e.g. epilepsy) or by health-related absenteeism from school with subsequently poorer school achievement.[206] Additionally to poorer performance, they may develop more weaken relationships to peers and teachers, which could have secondary effects on their educational attainment.[61] In worst case, they may be stigmatized and subsequently discriminated, which may affect the adolescents´ motivation and willingness to attend school.[32 206 207] There may also be other subjective limitations: adolescents struggling with illness (and their parents, teachers) may reduce their educational expectations by believing that they are limited by their health, by trying to keep themselves healthy rather than pushing themselves academically, or by believing that the expected benefits from education (employment) is rather low.[61]

6.3.3 The key role of the family

High school dropout

We described that terminating the adolescent educational trajectory without a high school degree might be a turning point in life in regard to subsequent labour market integration in young adulthood. The finding in paper II that about 42% of the propensity for school dropout could be attributed to the family level is extremely high, because in general the ICC in most observational studies is not higher than 20%.[180] One might doubt the correctness of such result, but it is comparable with the results from a study examining years of educational attainment.[208] Although we did not spend much attention to parental SEP or family living situation and their association with school dropout, they are without any doubt strong predictors. These variables also explained a large part of the clustering of school dropout on the family level. In this perspective, the facts that the associations between the health problems and school dropout remained nearly unchanged by adjusting for parental SEP and family living situation, and that many of the associations still existed when comparing siblings, suggest a robust health selection process in the attainment of education. But at the same time, it also underlines the key role of the family as a social context in this process of school dropout.

Social insurance benefits

As described above, the family is still important in regard to (health-related) labour market integration, although own SEP has a considerable higher impact as shown in paper I and III. This result is in concordance with earlier described studies of Gravseth et al.[13 110] Compared with high school dropout, the attribution of the family level to the propensity to receive long term sickness benefits and disability pension has been reduced to 21% (unadjusted, analysis not shown) which on itself was still a fairly high percentage. We are not aware of any study

reporting the variance at family level in relation to sickness absence or disability pension. When adjusted for stable family factors that siblings shared, the association between dropout and benefits was attenuated, but remained strong. This is not in concordance with the study of Samuelsson et al.[151] as they found that shared family factors explained the association of DP with education completely. However, they collapsed school dropouts with high school completers, and examined a middle aged population with disability pension.

Possible mechanisms

Based on our family level conditional approach and the multilevel analyses, the results of our studies do not allow us to make any conclusions related to the nature-nurture debate. Parental SEP, measured by education level, could explain about one fourth of the clustering of school dropout on the family level and presumably this results from a mixture of the heritable transmission of intelligence and a variety of social-psychological mechanisms operating within the family. The relative importance of genetics versus environment might even fluctuate in the diversiform presence of the individual traits as suggested by Johnson et al.[150] However, three thirds of the clustering on the family level was not yet explained by either parental SEP and individual health, as well as we have no explanation why shared family factors could explain the association between school dropout and psychological distress, but not the association with concentration problems. A Norwegian twin study suggested the correlation between education and anxiety to a large extent was caused by common genes and not much by shared common environment.[209] A Dutch twin study described that bipolar twin pairs (both the affected and the unaffected sibling) displayed underperformance at school compared to control twins, and the heritability of the underperformance was estimated to be as high as 85%.[210] Besides the importance of hereditary of IQ and other personal characteristics,[150] parental health has also been suggested to explain some of the between-

family variability in educational attainment.[208] Additionally, family health vulnerability could reduce the association between parent and offspring receipt of medical benefits more than parental SEP, suggesting the importance of both genetic and environmental health exposures for health-related labour market integration.[190] The variance in liability to disability pension due to genetic effects is estimated to be as much as one third and even more at younger age.[6 152]

Is the family role reduced to the genetic contribution in the offspring? First, it should be noted that estimates of shared environmental family variance from twin studies often are reported as negligible because of non-significant results, but the studies often lack precision due to low sample size.[211] Second, not only family capacity (such as health and functioning), but also family processes (such as family functioning and parenting practices) might affect educational attainment.[208] Especially family functioning, defined by problem solving, communication, roles, affective responsiveness, affective involvement, and behavioural control, exhibited independent associations with educational attainment.[208] Although also family-related environmental variables are under significant genetic influence, studies on the gene-environmental correlation of internalizing problems and temperamental traits of children suggested that these characteristics evoked environmental reactions in parents that exacerbated these characteristics.[212-214] Studies on IQ and educational attainment, or on the development of psychopathology suggested that environment is much more important in the presence of vulnerability (genetic predisposition for low IQ or psychopathology), which underlines the importance of the family beyond the genetics.[150 215]

6.4 Implications

6.4.1 On research

Our papers systematically suggest that poor health in adolescence may jeopardize both academic and employment prospects. At the same time, the literature showed that individual health in adolescence has not been recognized and studied seriously as a possible selective mechanism during the transition into adulthood until the last 5 years. There is absolutely a shortage of research that explores the reciprocal relations between health and school achievement. We also do not know the extent to which health-related mechanisms for school dropout are applicable for (health related) labour market exclusion. The development of a research agenda with focus on health-related determinants of school dropout and subsequent labour market integration in young adulthood should be a priority.

It is not possible to reduce health disparities in adult life without reducing disparities in educational achievement. Educational research showed that young people are more likely to graduate if schools foster student engagement and if young people do feel connected to at least one adult in their school.[216 217] A potential research avenue might be studies on health interventions that engage young people in their schools and that connect them to a caring adult. However, evaluation studies that assess impact of (coordinated) health programs on school dropout are rare because such health programs are seldom coordinated, and if existing, they do not target reducing school dropout as an outcome.[17]

6.4.2 On policy

The individual life time consequences and socio-economic costs of dropout are substantial, irrespective of the presence or absence of good prospects during adolescence. A major

implication on policy should be the reduction of high school dropout to a minimum. However, effectuation of this simple conclusion is of another caliber.

First, what is the minimum? According to the Europe 2020 target for educational attainment, it should be below 10% within 2020. Is this a feasible target or will it create new problems? Might it be possible, if Europe reaches the target, that the minimum educational level over time will be shifted to a bachelor degree (and create even larger health inequalities)? Might it be that a unilateral focus on "academic" requirements will reinforce health selection mechanisms during the transition into adulthood? It also raises the ethical question whether reducing the dropout rate de facto is the only solution or whether it is the definition of a solution by a society (based on knowledge and technocracy) who fails to include a large group of young people that (for whatever reason) no longer prioritizes their "academic" career, but maybe could benefit of alternative pathways into adult life and labour market?

Second, there are three alternative approaches to reduce dropout rates: targeted (focused on students at risk), comprehensive (attempt to change school environments to improve outcome for all students), and systematic (changes to the entire educational system).[216] All approaches have limited record of success, and even when success has been recorded in a specific project and setting, it has been difficult to copy the success on a larger scale.[216 218] One of the major conclusions until today is that the causes of dropout are complex, and so must be the solutions. As it often is not only the result of what takes place in school, the solution must involve more than schools, in particular it should also provide adequate support to families.[216] Although we did not unravel the mechanisms of school dropout on the family level in our papers, our results support the importance of the family as pointed out by Rumberger.

Reframing school dropout as a public health issue has the potential to encourage policymakers to see the problem of dropout as central to community health and as a possible long-term solution contributory to improve population health.[17] Additionally, it might introduce new partners into the strategies to reduce school dropout: parents, health services, and young people.[17] It is naïve to believe that school health services on its own will reduce school dropout. But youngsters who did not feel connected to school reported more often poor health, more smoking and visited more often school nurses.[217 219] Our research also underpins the presence of health selection mechanisms during the adolescent educational trajectory. As such, school health providers might have a role in identifying adolescents in need of assistance. Where necessary, school health providers can be helpful to provide an effective combination of personalized treatment that includes pharmacologic and environmental (family-based) interventions. Healthier students learn better, but there is also evidence that restructuring schools by just adding dropout-prevention services rather than changing teaching and learning does not reduce dropout rates.[218 220] Because of shortage in evidence on the reciprocal connections between health and academic achievement, we advocate for the *development* of evidence-based multidisciplinary approach in which coordinated health program interventions to reduce dropout, preferably with cooperation of schools and families, should be one of the pillars.

As stated in the first paragraph, one of the major goals should be to reduce dropout to a minimum. This implies also the recognition of the fact that a high school degree is probably not obtainable by everybody. There is some evidence that students graduating with alternative vocational pathways can do better in the transition to the labour market, in terms of avoiding unemployment at least, than students without any high school qualifications.[221] The key for more successful approaches seems to be providing rigorous and meaningful pathways

that combine career and technical education. Such alternatives help to re-integrate dropouts into the education system while providing them with occupational skills and experience that can assist in the transition from education to work.

7 Conclusions

In this thesis we studied the associations between health related factors in adolescence, high school dropout, and labour market exclusion in young adulthood, with specific attention to the family context.

Our studies described that high school dropout and problematic work integration are substantive problems. Although high school dropout was strongly clustered at the family level, poor health in all its dimensions compromised the opportunity to complete high school for adolescents of all social classes. High school dropout increased systematically the risk for problematic work integration due to impaired health, independent of the disadvantage or risk level for dropout observed in adolescence. All these observations were also true when comparing siblings, except for the associations between chronic somatic diseases, psychological distress, and high school dropout.

Future research on educational attainment and labour market exclusion in young adulthood should also pay attention to health-related mechanisms. As the problems are complex, it will be necessary to combine emphasis on health-related mechanisms with other dimensions of vulnerability, contexts (such as family and schools) and a life course framework (e.g. to study reciprocal effects, define critical periods). Because of the complexity, other research methods such as qualitative research or intervention studies might be valuable alternatives.

For policy, our findings support the importance of (early) prevention of school dropout where possible, in combination with increased attention to labour market integration and support for those who fail to complete high school. Reframing school dropout as a public health challenge may encourage policymakers to see school dropout as central for community health, and it may introduce new partners into dropout prevention strategies, such as school health care services, the youngsters and their families.

8 References

1. Bjørngaard JH, Krokstad S, Johnsen R, Karlsen AO, Pape K, Støver M, et al. Epidemiological research on disability benefits in the Nordic countries. Norwegian Journal of Epidemiology 2009; 19: 103-14.
2. Lindbøl M. Utvikling i uførediagnoser per 31. desember 2011. Oslo: NAV, 2011.
3. Gjesdal S, Bratberg E. Diagnosis and duration of sickness absence as predictors for disability pension: results from a three-year, multi-register based* and prospective study. Scand J Public Health 2003; 31: 246-54.
4. Labriola M, Lund T. Self-reported sickness absence as a risk marker of future disability pension. Prospective findings from the DWECS/DREAM study 1990-2004. Int J Med Sci 2007; 4: 153-8.
5. Raaum O, Rogstad J, Røed K, Westlie L. Young and out: An application of a prospects-based concept of social exclusion. Journal of Socio-Economics 2009; 38: 173-87.
6. Harkonmaki K, Silventoinen K, Levalahti E, Pitkaniemi J, Huunan-Seppala A, Klaukka T, et al. The genetic liability to disability retirement: a 30-year follow-up study of 24,000 Finnish twins. PLoS One 2008; 3: e3402.
7. Reime LJ, Claussen B. Municipal unemployment and municipal typologies as predictors of disability pensioning in Norway: a multilevel analysis. Scand J Public Health 2013; 41: 158-65.
8. Henderson M, Clark C, Stansfeld S, Hotopf M. A lifecourse approach to long-term sickness absence-a cohort study. PloS ONE 2012; 7: e36645.
9. Blum RW, Bastos FI, Kabiru CW, Le LC. Adolescent health in the 21st century. Lancet 2012; 379: 1567-8.
10. Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. Int J Epidemiol 2002; 31: 285-93.
11. Bronfenbrenner U. Toward an experimental ecology of human-development. Am Psychol 1977; 32: 513-31.
12. CSDH. Closing the gap in a generation: health equity through action on the social determinants of health. Final report of the Commision on Social Determinants of Health. Geneva: World Health Organization, 2008.
13. Gravseth HM, Bjerkedal T, Irgens LM, Aalen OO, Selmer R, Kristensen P. Life course determinants for early disability pension: a follow-up of Norwegian men and women born 1967-1976. Eur J Epidemiol 2007; 22: 533-43.
14. Bruusgaard D, Smeby L, Claussen B. Education and disability pension: a stronger association than previously found. Scand J Public Health 2010; 38: 686-90.
15. Kristensen P, Bjerkedal T. [Employment trends--the impact of chronic disease in childhood and low educational level]. Tidsskr Nor Laegeforen 2004; 124: 2879-83.
16. Eikemo TA, Huisman M, Bambra C, Kunst AE. Health inequalities according to educational level in different welfare regimes: a comparison of 23 European countries. Sociol Health Illn 2008; 30: 565-82.
17. Freudenberg N, Ruglis J. Reframing school dropout as a public health issue. Prev Chronic Dis 2007; 4: A107.
18. WHO. The second decade: improving adolescent health and development. Geneva: World Health Organization, 2001.
19. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. J Epidemiol Community Health 2003; 57: 778-83.

20. Sawyer SM, Afifi RA, Bearinger LH, Blakemore SJ, Dick B, Ezeh AC, et al. Adolescence: a foundation for future health. Lancet 2012; 379: 1630-40.
21. Patton GC, Viner R. Pubertal transitions in health. Lancet 2007; 369: 1130-9.
22. Viner RM, Ozer EM, Denny S, Marmot M, Resnick M, Fatusi A, et al. Adolescence and the social determinants of health. Lancet 2012; 379: 1641-52.
23. OECD. Education at a glance 2013: OECD indicators. Paris: OECD Publishing, 2013.
24. Krieger N. A glossary for social epidemiology. J Epidemiol Community Health 2001; 55: 693-700.
25. Atkinson AB, Hills J. Social exclusion, poverty and unemployment. Exclusion, employment and opportunity. London: Center for Analysis of Social Exclusion, 1998.
26. Marmot MG. Understanding social inequalities in health. Perspect Biol Med 2003; 46: 9-23.
27. Graham H, Power C. Childhood disadvantage and health inequalities: a framework for policy based on lifecourse research. Child Care Health Dev 2004; 30: 671-8.
28. Kawachi I, Subramanian SV, Almeida-Filho N. A glossary for health inequalities. J Epidemiol Community Health 2002; 56: 647-52.
29. Regidor E. Measures of health inequalities: part 1. J Epidemiol Community Health 2004; 58: 858-61.
30. Regidor E. Social determinants of health: a veil that hides socioeconomic position and its relation with health. J Epidemiol Community Health 2006; 60: 896-901.
31. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. Annu Rev Public Health 1997; 18: 341-78.
32. West P. Rethinking the health selection explanation for health inequalities. Soc Sci Med 1991; 32: 373-84.
33. Lundberg O. Childhood living conditions, health status, and social mobility: A contribution to the health selection debate. European Sociological Review 1991; 7: 149-62.
34. Manor O, Matthews S, Power C. Health selection: the role of inter- and intra-generational mobility on social inequalities in health. Soc Sci Med 2003; 57: 2217-27.
35. Power C, Matthews S, Manor O. Inequalities in self rated health in the 1958 birth cohort: lifetime social circumstances or social mobility? BMJ 1996; 313: 449-53.
36. Huurre T, Aro H, Rahkonen O, Komulainen E. Health, lifestyle, family and school factors in adolescence: Predicting adult educational level. Educational Research 2006; 48: 41-53.
37. Koivusilta L, Arja R, Andres V. Health behaviours and health in adolescence as predictors of educational level in adulthood: a follow-up study from Finland. Soc Sci Med 2003; 57: 577-93.
38. Case A, Fertig A, Paxson C. The lasting impact of childhood health and circumstance. J Health Econ 2005; 24: 365-89.
39. Haas SA, Fosse NE. Health and the educational attainment of adolescents: evidence from the NLSY97. J Health Soc Behav 2008; 49: 178-92.
40. Hertzman C. The biological embedding of early experience and its effects on health in adulthood. Ann N Y Acad Sci 1999; 896: 85-95.
41. Bäckman O, Palme J. Social background and sickness absence: A study of a Stockholm cohort. Acta Sociologica 1998; 41: 349-62.
42. Mæland JG. Velferdsystemen i Norge. In: Mæland JG, Haug K, Høyer G, Krokstad S, eds. Sosialmedisin. Oslo: Gyldendal Akademisk, 2011.
43. Esping-Andersen G. The three worlds of welfare capitalism. Cambridge: Polity Press, 1990.

44. Navarro V, Muntaner C, Borrell C, Benach J, Quiroga A, Rodriguez-Sanz M, et al. Politics and health outcomes. Lancet 2006; 368: 1033-7.
45. Lundberg O, Yngwe MA, Stjarne MK, Elstad JI, Ferrarini T, Kangas O, et al. The role of welfare state principles and generosity in social policy programmes for public health: an international comparative study. Lancet 2008; 372: 1633-40.
46. Westin S. Welfare for all—or only for the needy? The Lancet 2008; 372: 1609-10.
47. van der Wel KA, Dahl E, Thielen K. Social inequalities in "sickness": does welfare state regime type make a difference? A multilevel analysis of men and women in 26 European countries. Int J Health Serv 2012; 42: 235-55.
48. OECD. Sickness, Disability and Work: breaking the barriers. A synthesis of findings across OECD countries. Paris: OECD Publishing, 2010.
49. Bratsberg B, Fevang E, Roed K. Disability in the welfare state: An unemployment problem in disguise? IZA Discussion Paper No 4897. Bonn: Institute for the Study of Labour, 2010.
50. OECD. Jobs for youth: Norway Paris: OECD Publishing, 2008.
51. NAV. Statistikkportal/Jobb og helse. Secondary Statistikkportal/Jobb og helse. http://www.nav.no/Om+NAV/Tall+og+analyse/Jobb+og+helse.
52. Brage S, Nossen JP, Kann IC, Thune O. Sykefravær med diagnose innen psykiske lidelser 2000-2011. Arbeid of Velferd 2012; 3: 24-37.
53. Lamb S, Markussen E, Teese R, Polesel J, Sandberg N, editors. School dropout and completion: International comparative studies in theory and policy. Dordrecht: Springer Science+Business Media B.V., 2011.
54. Markussen E, Sandberg N, Lødding B, Frøseth MW. Bortvalg og kompetanse: gjennomføring, bortvalg og kompetanseoppnåelse i videregående opplæring blant 9749 ungdommer som gikk ut av grunnskolen på Østlandet våren 2002 : hovedfunn, konklusjoner og implikasjoner fem år etter. Oslo: NIFU STEP, 2008.
55. Fakta om utdanning 2013 - nøkkeltall fra 2011. Oslo: Statistisk Sentralbyrå, 2012.
56. Fakta om utdanning 2005 - nøkkeltall. Oslo: Statistisk Sentralbyrå, 2004.
57. Statistisk årbok 2012. Oslo - Kongsvinger: Statistisk Sentralbyrå, 2012.
58. Kristensen P, Gravseth HM, Bjerkedal T. Educational attainment of Norwegian men: Influence of parental and early individual characteristics. J Biosoc Sci 2009; 41: 799-814.
59. Rumberger RW. Dropping out of high school - the influence of race, sex, and family background. American Educational Research Journal 1983; 20: 199-220.
60. Wojtkiewicz RA. Simplicity and complexity in the effects of parental structure on high school graduation. Demography 1993; 30: 701-17.
61. Jackson MI. Understanding links between adolescent health and educational attainment. Demography 2009; 46: 671-94.
62. Lê F, Diez Roux A, Morgenstern H. Effects of child and adolescent health on educational progress. Soc Sci Med 2013; 76: 57-66.
63. Pape K, Bjorngaard JH, Westin S, Holmen TL, Krokstad S. Reading and writing difficulties in adolescence and later risk of welfare dependence. A ten year follow-up, the HUNT Study, Norway. BMC Public Health 2011; 11: 718.
64. Rothon C, Head J, Klineberg E, Stansfeld S. Can social support protect bullied adolescents from adverse outcomes? A prospective study on the effects of bullying on the educational achievement and mental health of adolescents at secondary schools in East London. J Adolesc 2011; 34: 579-88.
65. de Araujo P, Lagos S. Self-esteem, education, and wages revisited. Journal of Economic Psychology 2013; 34: 120-32.

66. Suldo SM, Shaffer EJ. Looking beyond psychopathology: The dual-factor model of mental health in youth. School Psychology Review 2008; 37: 52-68.
67. Winding TN, Nohr EA, Labriola M, Biering K, Andersen JH. Personal predictors of educational attainment after compulsory school: Influence of measures of vulnerability, health, and school performance. Scand J Public Health 2012; 41: 92-101.
68. Battin-Pearson S, Newcomb MD, Abbott RD, Hill KG, Catalano RF, Hawkins JD. Predictors of early high school dropout: A test of five theories. J Educ Psychol 2000; 92: 568-82.
69. Lund T, Labriola M, Villadsen E. Who is at risk for long-term sickness absence? A prospective cohort study of Danish employees. Work 2007; 28: 225-30.
70. Dahl G, Enes AW, Epland J, Jorgensen T, Trewin C. Langtidsmottakere av sosialhjelp. Økonomiske analyser 3/2006. Oslo: Statistics Sentralbyrå, 2006.
71. Huber M, Knottnerus JA, Green L, Horst Hvd, Jadad AR, Kromhout D, et al. How should we define health? BMJ 2011; 343: d4163.
72. Boorse C. Health as a theoretical concept. Philosophy of Science 1977; 44: 542-73.
73. Susser M. Disease, illness, sickness; impairment, disability and handicap. Psychol Med 1990; 20: 471-73.
74. Wikman A, Marklund S, Alexanderson K. Illness, disease, and sickness absence: an empirical test of differences between concepts of ill health. J Epidemiol Community Health 2005; 59: 450-54.
75. Sawyer SM, Drew S, Yeo MS, Britto MT. Adolescents with a chronic condition: challenges living, challenges treating. Lancet 2007; 369: 1481-9.
76. Patton GC, Coffey C, Cappa C, Currie D, Riley L, Gore F, et al. Adolescent Health 4 Health of the world's adolescents: a synthesis of internationally comparable data. Lancet 2012; 379: 1665-75.
77. Newacheck PW, Stoddard JJ. Prevalence and impact of multiple childhood chronic illnesses. J Pediatr 1994; 124: 40-48.
78. Perrin JM, Bloom SR, Gortmaker SL. The increase of childhood chronic conditions in the United States. JAMA 2007; 297: 2755-9.
79. Currie C, Zanotti C, Morgan A, de Looze M, Roberts C, Samdal O, et al. Social determinants of health and well-being among young people. Health Behaviour in School-aged Children (HBSC) study: international report from the 2009/2010 survey. Health Policy for Children and Adolescents. Copenhagen: WHO Regional Office for Europe, 2012.
80. Breidablik HJ, Meland E, Lydersen S. Self-rated health in adolescence: a multifactorial composite. Scand J Public Health 2008; 36: 12-20.
81. Costello EJ, Copeland W, Angold A. Trends in psychopathology across the adolescent years: what changes when children become adolescents, and when adolescents become adults? J Child Psychol Psychiatry 2011; 52: 1015-25.
82. Pless IB, Power C, Peckham CS. Long-term psychosocial sequelae of chronic physical disorders in childhood. Pediatrics 1993; 91: 1131-36.
83. Maslow GR, Haydon A, McRee A-L, Ford CA, Halpern CT. Growing up with a chronic illness: Social success, educational/vocational distress. J Adolesc Health 2011; 49: 206-12.
84. Fletcher JM, Richards MR. Diabetes's 'health shock' to schooling and earnings: Increased dropout rates and lower wages and employment in young adults. Health Aff (Millwood) 2012; 31: 27-34.
85. Taras H, Potts-Datema W. Childhood asthma and student performance at school. J Sch Health 2005; 75: 296-312.

86. Huurre T, Rahkonen O, Komulainen E, Aro H. Socioeconomic status as a cause and consequence of psychosomatic symptoms from adolescence to adulthood. Soc Psychiatry Psychiatr Epidemiol 2005; 40: 580-7.
87. Kessler RC, Foster CL, Saunders WB, Stang PE. Social consequences of psychiatric disorders, I: Educational attainment. Am J Psychiatry 1995; 152: 1026-32.
88. Stoep AV, Beresford SAA, Weiss NS, McKnight B, Cauce AM, Cohen P. Community-based study of the transition to adulthood for adolescents with psychiatric disorder. Am J Epidemiol 2000; 152: 352-62.
89. Johnson JG, Cohen P, Dohrenwend BP, Link BG, Brook JS. A longitudinal investigation of social causation and social selection processes involved in the association between socioeconomic status and psychiatric disorders. J Abnorm Psychol 1999; 108: 490-9.
90. Fergusson DM, Woodward LJ. Mental health, educational, and social role outcomes of adolescents with depression. Arch Gen Psychiatry 2002; 59: 225-31.
91. Miech RA, Caspi A, Moffitt TE, Wright BRE, Silva PA. Low socioeconomic status and mental disorders: A longitudinal study of selection and causation during young adulthood. Am J Sociol 1999; 104: 1096-131.
92. Breslau J, Lane M, Sampson N, Kessler RC. Mental disorders and subsequent educational attainment in a US national sample. J Psychiatr Res 2008; 42: 708-16.
93. McLeod JD, Fettes DL. Trajectories of failure: The educational careers of children with mental health problems. AJS 2007; 113: 653-701.
94. McLeod JD, Uemura R, Rohrman S. Adolescent mental health, behavior problems, and academic achievement. J Health Soc Behav 2012; 53: 482-97.
95. Needham BL. Adolescent depressive symptomatology and young adult educational attainment: An examination of gender differences. J Adolesc Health 2009; 45: 179-86.
96. Fletcher JM. Adolescent depression: diagnosis, treatment, and educational attainment. Health Econ 2008; 17: 1215-35.
97. Dewald JF, Meijer AM, Oort FJ, Kerkhof GA, Bögels SM. The influence of sleep quality, sleep duration and sleepiness on school performance in children and adolescents: A meta-analytic review. Sleep Med Rev 2010; 14: 179-89.
98. Loe IM, Feldman HM. Academic and educational outcomes of children with ADHD. J Pediatr Psychol 2007; 32: 643-54.
99. Pingault JB, Tremblay RE, Vitaro F, Carbonneau R, Genolini C, Falissard B, et al. Childhood trajectories of inattention and hyperactivity and prediction of educational attainment in early adulthood: a 16-year longitudinal population-based study. Am J Psychiatry 2011; 168: 1164-70.
100. Fried R, Petty C, Faraone SV, Hyder LL, Day H, Biederman J. Is ADHD a risk factor for high school dropout? A controlled study. J Att Disord 2013. doi: 10.1177/1087054712473180. Feb 4 (Epub ahead of print)
101. Haas SA. Health selection and the process of social stratification: the effect of childhood health on socioeconomic attainment. J Health Soc Behav 2006; 47: 339-54.
102. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med 1993; 329: 1008-12.
103. Sargent JD, Blanchflower DG. Obesity and stature in adolescence and earnings in young adulthood: Analysis of a british birth cohort. Arch Pediatr Adolesc Med 1994; 148: 681-87.
104. Laitinen J, Power C, Ek E, Sovio U, Jarvelin MR. Unemployment and obesity among young adults in a northern Finland 1966 birth cohort. Int J Obes Relat Metab Disord 2002; 26: 1329-38.

105. Viner RM, Cole TJ. Adult socioeconomic, educational, social, and psychological outcomes of childhood obesity: a national birth cohort study. BMJ 2005; 330: 1354.
106. Hack M. Young adult outcomes of very-low-birth-weight children. Sem Fetal Neonatal Med 2006; 11: 127-37.
107. Dong Y, Chen SJ, Yu JL. A systematic review and meta-analysis of long-term development of early term infants. Neonatology 2012; 102: 212-21.
108. Upmark M, Lundberg I, Sadigh J, Allebeck P, Bigert C. Psychosocial characteristics in young men as predictors of early disability pension with a psychiatric diagnosis. Soc Psychiatry Psychiatr Epidemiol 1999; 34: 533-40.
109. Upmark M, Lundberg I, Sadigh J, Bigert C. Conditions during childhood and adolescence as explanations of social class differences in disability pension among young men. Scand J Public Health 2001; 29: 96-103.
110. Gravseth HM, Bjerkedal T, Irgens LM, Aalen OO, Selmer R, Kristensen P. Influence of physical, mental and intellectual development on disability in young Norwegian men. Eur J Public Health 2008; 18: 650-5.
111. Johansson E, Leijon O, Falkstedt D, Farah A, Hemmingsson T. Educational differences in disability pension among Swedish middle-aged men: role of factors in late adolescence and work characteristics in adulthood. J Epidemiol Community Health 2012; 66: 901-07.
112. Upmark M, Thundal K. An explorative, population-based study of female disability pensioners: the role of childhood conditions and alcohol abuse/dependence. Scand J Public Health 2002; 30: 191 - 99.
113. Henderson M, Stansfeld S, Hotopf M. Self-rated health and later receipt of work-related benefits: evidence from the 1970 British Cohort Study. Psychol Med 2013; 43: 1755-62.
114. Mordre M, Groholt B, Sandstad B, Myhre AM. The impact of ADHD symptoms and global impairment in childhood on working disability in mid-adulthood: a 28-year follow-up study using official disability pension records in a high-risk in-patient population. BMC Psychiatry 2012; 12: 174.
115. Bjerkedal T, Kristensen P, Skjeret GA, Brevik JI. [A follow up of persons who received basic and/or supplemental benefits in childhood]. Tidsskr Nor Laegeforen 2006; 126: 436-9.
116. Naicker K, Galambos NL, Zeng Y, Senthilselvan A, Colman I. Social, demographic, and health autcomes in the 10 years following adolescent depression. J Adolesc Health 2013; 52: 533-38.
117. Currie J, Stabile M, Manivong P, Roos LL. Child health and young adult outcomes. J Hum Resour 2010; 45: 517-48.
118. Kupper T, Haavik J, Drexler H, Ramos-Quiroga JA, Wermelskirchen D, Prutz C, et al. The negative impact of attention-deficit/hyperactivity disorder on occupational health in adults and adolescents. Int Arch Occup Environ Health 2012; 85: 837-47.
119. Fletcher JM. The effects of childhood ADHD on adult labor market outcomes. Health Econ 2013. doi: 10.1002/hec.2907. Feb 21 (Epub ahead of print)
120. Smith JP. The impact of childhood health on adult labor marktet outcomes. Rev Econ Stat 2009; 91: 478-89.
121. Lundberg O. Childhood conditions, sense of coherence, social class and adult ill health: exploring their theoretical and empirical relations. Soc Sci Med 1997; 44: 821-31.
122. Lundberg O. The impact of childhood living conditions on illness and mortality in adulthood. Soc Sci Med 1993; 36: 1047-52.

123. Vågerö D, Illsley R. Explaining Health Inequalities: Beyond Black and Barker: A discussion of some issues emerging in the decade following the Black Report. European Sociological Review 1995; 11: 219-41.
124. Bäckman O, Nilsson A. Pathways to social exclusion—A life-course study. European Sociological Review 2011; 27: 107-23.
125. Waddell GR. Labor-market consequences of poor attitude and low self-esteem in youth. Econ Inq 2006; 44: 69-97.
126. Huurre T, Junkkari H, Aro H. Long-term psychosocial effects of parental divorce: a follow-up study from adolescence to adulthood. Eur Arch Psychiatry Clin Neurosci 2006; 256: 256-63.
127. Currie J, Stabile M. Child mental health and human capital accumulation: the case of ADHD. J Health Econ 2006; 25: 1094-118.
128. Rothon C, Arephin M, Klineberg E, Cattell V, Stansfeld S. Structural and socio-psychological influences on adolescents' educational aspirations and subsequent academic achievement. Social Psychol Educl 2011; 14: 209-31.
129. Biering-Sorensen F, Lund J, Hoydalsmo OJ, Darre EM, Deis A, Kryger P, et al. Risk indicators of disability pension. A 15 year follow-up study. Dan Med Bull 1999; 46: 258-62.
130. Mittendorfer-Rutz E, Hensing G, Westerlund H, Backheden M, Hammarstrom A. Determinants in adolescence for adult sickness absence in women and men: a 26-year follow-up of a prospective population based cohort (Northern Swedish cohort). BMC Public Health 2013; 13: 75.
131. Marmot MG, Kogevinas M, Elston MA. Social/economic status and disease. Annu Rev Public Health 1987; 8: 111-35.
132. Chen E, Martin AD, Matthews KA. Socioeconomic status and health: do gradients differ within childhood and adolescence? Soc Sci Med 2006; 62: 2161-70.
133. Due P, Lynch J, Holstein B, Modvig J. Socioeconomic health inequalities among a nationally representative sample of Danish adolescents: the role of different types of social relations. J Epidemiol Community Health 2003; 57: 692-8.
134. Morgen CS, Mortensen LH, Rasmussen M, Andersen AM, Sorensen TI, Due P. Parental socioeconomic position and development of overweight in adolescence: longitudinal study of Danish adolescents. BMC Public Health 2010; 10: 520.
135. Sagiv SK, Epstein JN, Bellinger DC, Korrick SA. Pre- and postnatal risk factors for ADHD in a nonclinical pediatric population. J Att Disord 2013; 17: 47-57.
136. Huurre T, Aro H, Rahkonen O. Well-being and health behaviour by parental socioeconomic status: a follow-up study of adolescents aged 16 until age 32 years. Soc Psychiatry Psychiatr Epidemiol 2003; 38: 249-55.
137. Power C, Matthews S. Origins of health inequalities in a national population sample. Lancet 1997; 350: 1584-9.
138. Nilsen SM, Krokstad S, Holmen TL, Westin S. Adolescents' health-related dietary patterns by parental socio-economic position, The Nord-Trøndelag Health Study (HUNT). The European Journal of Public Health 2010; 20: 299-305.
139. Hanson MD, Chen E. Socioeconomic status and health behaviors in adolescence: a review of the literature. J Behav Med 2007; 30: 263-85.
140. McLoyd VC. Socioeconomic disadvantage and child development. Am Psychol 1998; 53: 185-204.
141. Schoon I, Bynner J, Joshi H, Parsons S, Wiggins RD, Sacker A. The influence of context, timing, and duration of risk experiences for the passage from childhood to midadulthood. Child Dev 2002; 73: 1486-504.

142. Child Trends. World family map 2013: Mapping family change and child well-being outcomes. Washington: Child Trends, 2013.
143. Amato PR. Children of divorce in the 1990s: an update of the Amato and Keith (1991) meta-analysis. J Fam Psychol 2001; 15: 355-70.
144. Størksen I, Røysamb E, Moum T, Tambs K. Adolescents with a childhood experience of parental divorce: a longitudinal study of mental health and adjustment. J Adolesc 2005; 28: 725-39.
145. Raaum O, Salvanes KG, Sørensen EØ. The neighbourhood is not what it used to be. The Economic Journal 2006; 116: 200-22.
146. Kendler KS. Twin studies of psychiatric illness: an update. Arch Gen Psychiatry 2001; 58: 1005-14.
147. Naukkarinen J, Rissanen A, Kaprio J, Pietilainen KH. Causes and consequences of obesity: the contribution of recent twin studies. Int J Obes 2012; 36: 1017-24.
148. Ferreira PH, Beckenkamp P, Maher CG, Hopper JL, Ferreira ML. Nature or nurture in low back pain? Results of a systematic review of studies based on twin samples. Eur J Pain 2013; 17: 957-71.
149. Pinto LA, Stein RT, Kabesch M. Impact of genetics in childhood asthma. J Pediatr (Rio J) 2008; 84: S68-75.
150. Johnson W, Deary IJ, Iacono WG. Genetic and environmental transactions underlying educational attainment. Intelligence 2009; 37: 466-78.
151. Samuelsson A, Alexanderson K, Ropponen A, Lichtenstein P, Svedberg P. Incidence of disability pension and associations with socio-demographic factors in a Swedish twin cohort. Soc Psychiatry Psychiatr Epidemiol 2012; 47: 1999-2009.
152. Narusyte J, Ropponen A, Silventoinen K, Alexanderson K, Kaprio J, Samuelsson A, et al. Genetic liability to disability pension in women and men: a prospective population-based twin study. PLoS One 2011; 6: e23143.
153. Krokstad S, Langhammer A, Hveem K, Holmen T, Midthjell K, Stene T, et al. Cohort Profile: The HUNT Study, Norway. Int J Epidemiol 2013; 42: 968-77.
154. Holmen TL, Bratberg G, Krokstad S, Langhammer A, Hveem K, Midthjell K, et al. Cohort profile of the Young-HUNT Study, Norway: A population-based study of adolescents. Int J Epidemiol 2013. doi: 10.1093/ije/dys232. Feb 4 (Epub ahead of print)
155. Holmen J, Midthjell K, Krüger Ø, Langhammer A, Holmen TL, Bratberg GH, et al. The Nord-Trøndelag Health Study 1995-97 (HUNT 2): objectives, contents, methods and participation. Norsk epidemiologi 2003; 13: 19-32.
156. Vangen T. Nasjonal utdanningsdatabase NUDB. Dokumentasjonsrapport: datavarehus for utdanningsdata, 1970-2006. Oslo: Statistisk sentralbyrå, 2007.
157. Akselsen A, Lien S, Sivertstøl Ø. FD-Trygd. List of variables. Oslo: Statistics Norway, 2007.
158. Rognan A, Barrabes N. NUS2000, Dokumentasjonsrapport. Oslo: Statistisk sentralbyrå, 2001:36.
159. Kivimaki M, Ferrie JE, Hagberg J, Head J, Westerlund H, Vahtera J, et al. Diagnosis-specific sick leave as a risk marker for disability pension in a Swedish population. J Epidemiol Community Health 2007; 61: 915-20.
160. Hagen K, Zwart JA, Aamodt AH, Nilsen KB, Brathen G, Helde G, et al. The validity of questionnaire-based diagnoses: the third Nord-Trondelag Health Study 2006-2008. J Headache Pain 2010; 11: 67-73.
161. Simon GE, VonKorff M, Piccinelli M, Fullerton C, Ormel J. An international study of the relation between somatic symptoms and depression. N Engl J Med 1999; 341: 1329-35.

162. Ravens-Sieberer U, Torsheim T, Hetland J, Vollebergh W, Cavallo F, Jericek H, et al. Subjective health, symptom load and quality of life of children and adolescents in Europe. Int J Public Health 2009; 54: 151-9.
163. Henningsen P, Zipfel S, Herzog W. Management of functional somatic syndromes. The Lancet 2007; 369: 946-55.
164. Fink P, Schröder A. One single diagnosis, bodily distress syndrome, succeeded to capture 10 diagnostic categories of functional somatic syndromes and somatoform disorders. J Psychosom Res 2010; 68: 415-26.
165. Hoftun GB, Romundstad PR, Zwart J-A, Rygg M. Chronic idiopathic pain in adolescence – high prevalence and disability: The young HUNT study 2008. Pain 2011; 152: 2259-66.
166. Tambs K, Moum T. How well can a few questionnaire items indicate anxiety and depression? Acta Psychiatr Scand 1993; 87: 364-7.
167. Strand BH, Dalgard OS, Tambs K, Rognerud M. Measuring the mental health status of the Norwegian population: a comparison of the instruments SCL-25, SCL-10, SCL-5 and MHI-5 (SF-36). Nord J Psychiatry 2003; 57: 113-8.
168. Breidablik HJ, Meland E, Lydersen S. Self-rated health during adolescence: stability and predictors of change (Young-HUNT study, Norway). Eur J Public Health 2009; 19: 73-8.
169. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 2000; 320: 1240-3.
170. Mansson NO, Eriksson KF, Israelsson B, Ranstam J, Melander A, Rastam L. Body mass index and disability pension in middle-aged men--non-linear relations. Int J Epidemiol 1996; 25: 80-5.
171. Bratberg G, Nilsen T, Holmen T, Vatten L. Sexual maturation in early adolescence and alcohol drinking and cigarette smoking in late adolescence: a prospective study of 2,129 Norwegian girls and boys. Eur J Pediatr 2005; 164: 621-25.
172. Ystgaard M. Vulnerable adolescents and social support. An approach to prevent psychological distress and suicide. Oslo: Center of Social Network, 1993.
173. Moum T, Naess S, Sorensen T, Tambs K, Holmen J. Hypertension labelling, life events and psychological well-being. Psychol Med 1990; 20: 635-46.
174. Sourander A, Jensen P, Rönning JA, Niemelä S, Helenius H, Sillanmäki L, et al. What is the early adulthood outcome of boys who bully or are bullied in childhood? The Finnish "From a Boy to a Man" study. Pediatrics 2007; 120: 397-404.
175. Tambs E. A study of sexual abuse of children. Oslo: Norwegian Institute of Public Health, 1994.
176. Myklestad I, Roysamb E, Tambs K. Risk and protective factors for psychological distress among adolescents: a family study in the Nord-Trondelag Health Study. Soc Psychiatry Psychiatr Epidemiol 2012; 47: 771-82.
177. Krokstad S, Ringdal K, Westin S. Classifying people by social class in population based health surveys: Two methods compared. Norsk epidemiologi 2002; 12: 19-25.
178. Hosmer DW, Lemeshow S. The multinomial logistic regression model. Applied logistic regression, second edition. 2nd ed. New York: John Wiley & Sons, Inc, 2000:260-87.
179. Donovan SJ, Susser E. Commentary: Advent of sibling designs. Int J Epidemiol 2011; 40: 345-9.
180. Twisk JWR. Applied multilevel analysis. Cambridge: Cambridge University Press, 2006.
181. Allison PD. Fixed effects logistic models. In: Allison PD, ed. Fixed effects regression models. United States of America: SAGE publications, Inc., 2009:28-48.
182. Merlo J, Chaix B, Ohlsson H, Beckman A, Johnell K, Hjerpe P, et al. A brief conceptual tutorial of multilevel analysis in social epidemiology: using measures of clustering in

multilevel logistic regression to investigate contextual phenomena. J Epidemiol Community Health 2006; 60: 290-7.
183. Katz MH. Methods for adjusting for baseline differences between treatment groups. In: Katz MH, ed. Evaluating clinical and public health interventions: A practical guide to study design and statistics.1ed. Cambridge: Cambridge University Press, 2010:101-15.
184. Becker SO, Ichino A. Estimation of average treatment effects based on propensity scores. The Stata Journal 2002; 2: 358-77.
185. White IR, Royston P, Wood AM. Multiple imputation using chained equations: Issues and guidance for practice. Stat Med 2011; 30: 377-99.
186. Rothman KJ, Greenland S, Lash TL. Modern epidemiology. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008.
187. Delgado-Rodriguez M, Llorca J. Bias. J Epidemiol Community Health 2004; 58: 635-41.
188. Hernán MA, Hernández-Díaz S, Werler MM, Mitchell AA. Causal knowledge as a prerequisite for confounding evaluation: An application to birth defects epidemiology. Am J Epidemiol 2002; 155: 176-84.
189. Suominen S, Gould R, Ahvenainen J, Vahtera J, Uutela A, Koskenvuo M. Sense of coherence and disability pensions. A nationwide, register based prospective population study of 2196 adult Finns. J Epidemiol Community Health 2005; 59: 455-9.
190. Pape K, Bjørngaard JH, De Ridder KAA, Westin S, Holmen TL, Krokstad S. Medical benefits in young Norwegians and their parents, and the contribution of family health and socioeconomic status. The HUNT Study, Norway. Scand J Public Health 2013; 41: 455-62.
191. Sitzmann T, Ely K. A meta-analysis of self-regulated learning in work-related training and educational attainment: what we know and where we need to go. Psychol Bull 2011; 137: 421-42.
192. Bynner J. Education and family components of identity in the transition from school to work. International Journal of Behavioral Development 1998; 22: 29-53.
193. Harkonmaki K, Korkeila K, Vahtera J, Kivimaki M, Suominen S, Sillanmaki L, et al. Childhood adversities as a predictor of disability retirement. J Epidemiol Community Health 2007; 61: 479-84.
194. Bynner J. Rethinking the youth phase of the life-course: The case for emerging adulthood? Journal of Youth Studies 2005; 8: 367-84.
195. Hutchison ED. A life course perspective. In: Graves K, ed. Dimensions of human behaviour: The changing life course. 4th ed. United States of America: SAGE Publications, Inc., 2011:1-39.
196. Bynner J, Parsons S. Social exclusion and the transition from school to work: The case of young people not in education, employment, or training (NEET). J Vocat Behav 2002; 60: 289-309.
197. Westin S, Schlesselman JJ, Korper M. Long-term effects of a factory closure: Unemployment and disability during ten years' follow-up. J Clin Epidemiol 1989; 42: 435-41.
198. Ackerman PL, Chamorro-Premuzic T, Furnham A. Trait complexes and academic achievement: Old and new ways of examining personality in educational contexts. Br J Educ Psychol 2011; 81: 27-40.
199. Christensen U, Schmidt L, Kriegbaum M, Hougaard CØ, Holstein BE. Coping with unemployment: Does educational attainment make any difference? Scand J Public Health 2006; 34: 363-70.
200. Volanen S-M, Lahelma E, Silventoinen K, Suominen S. Factors contributing to sense of coherence among men and women. Eur J Public Health 2004; 14: 322-30.

201. Antonovsky H, Sagy S. The development of a sense of coherence and its impact on responses to stress situations. J Soc Psychol 1986; 126: 213-25.
202. Torsheim T, Aaroe LE, Wold B. Sense of coherence and school-related stress as predictors of subjective health complaints in early adolescence: interactive, indirect or direct relationships? Soc Sci Med 2001; 53: 603-14.
203. Haas SA, Glymour MM, Berkman LF. Childhood health and labor market inequality over the life course. J Health Soc Behav 2011; 52: 298-313.
204. Fletcher J, Wolfe B. Child mental health and human capital accumulation: the case of ADHD revisited. J Health Econ 2008; 27: 794-800.
205. Fletcher JM. Adolescent depression and educational attainment: results using sibling fixed effects. Health Econ 2010; 19: 855-71.
206. Needham BL, Crosnoe R, Muller C. Academic failure in secondary school: The inter-related role of health problems and educational context. Soc Probl 2004; 51: 569-86.
207. Crosnoe R, Muller C. Body Mass Index, Academic Achievement, and School Context: Examining the Educational Experiences of Adolescents at Risk of Obesity. J Health Soc Behav 2004; 45: 393-407.
208. Boyle MH, Georgiades K, Racine Y, Mustard C. Neighborhood and family influences on educational attainment: results from the Ontario Child Health Study follow-up 2001. Child Dev 2007; 78: 168-89.
209. Tambs K, Kendler KS, Reichborn-Kjennerud T, Aggen SH, Harris JR, Neale MC, et al. Genetic and environmental contributions to the relationship between education and anxiety disorders – a twin study. Acta Psychiatr Scand 2012; 125: 203-12.
210. Vonk R, van der Schot AC, van Baal GCM, van Oel CJ, Nolen WA, Kahn RS. Premorbid school performance in twins concordant and discordant for bipolar disorder. J Affect Disord 2012; 136: 294-303.
211. Smith GD. Epidemiology, epigenetics and the 'Gloomy Prospect': embracing randomness in population health research and practice. Int J Epidemiol 2011; 40: 537-62.
212. Kendler KS, Baker JH. Genetic influences on measures of the environment: a systematic review. Psychol Med 2007; 37: 615-26.
213. Narusyte J, Neiderhiser JM, D'Onofrio BM, Reiss D, Spotts EL, Ganiban J, et al. Testing different types of genotype-environment correlation: an extended children-of-twins model. Dev Psychol 2008; 44: 1591-603.
214. Kendler KS. Parenting: a genetic-epidemiologic perspective. Am J Psychiatry 1996; 153: 11-20.
215. Franić S, Middeldorp CM, Dolan CV, Ligthart L, Boomsma DI. Childhood and adolescent anxiety and depression: Beyond heritability. J Am Acad Child Adolesc Psychiatry 2010; 49: 820-29.
216. Rumberger RW. Dropping Out. Why students dropout of high school and what can be done about it. Havard: Havard University Press, 2011.
217. Bonny AE, Britto MT, Klostermann BK, Hornung RW, Slap GB. School disconnectedness: identifying adolescents at risk. Pediatrics 2000; 106: 1017-21.
218. Dynarski M, Gleason P, Rangarajan A, Wood R. Impacts of dropout prevention programs. Final report. Washington: Mathematica Policy Research, Inc., 1998.
219. Homlong L, Rosvold EO, Haavet OR. Can use of healthcare services among 15-16-year-olds predict an increased level of high school dropout? A longitudinal community study. BMJ Open 2013; 3: e003125.
220. Basch CE. Healthier students are better learners: A missing link in school reforms to close the achievement gap. J Sch Health 2011; 81: 593-98.

221. Lamb S. Alternative pathways to high school graduation: An international comparison. Santa Barbara: UC Santa Barbara/Gevirtz Graduate School of Education, 2008.

Paper I

Is not included due to copyright

Paper II

OPEN ACCESS Freely available online

PLOS ONE

Adolescent Health and High School Dropout: A Prospective Cohort Study of 9000 Norwegian Adolescents (The Young-HUNT)

Karin A. A. De Ridder[1,2]*, Kristine Pape[1], Roar Johnsen[1], Turid Lingaas Holmen[3], Steinar Westin[1], Johan Håkon Bjørngaard[1,4]

1 Department of Public Health and General Practice, Norwegian University of Science and Technology, Trondheim, Norway, **2** Department of Physical Medicine and Rehabilitation, Levanger Hospital, Nord-Trøndelag Health Trust, Levanger, Norway, **3** HUNT Research Center, Department of Public Health and General Practice, Norwegian University of Science and Technology, Levanger, Norway, **4** Forensic Department and Research Centre Bröset, St. Olavs Hospital, Trondheim University Hospital, Trondheim, Norway

Abstract

Background: High school dropout is of major concern in the western world. Our aims were to estimate the risk of school dropout in adolescents following chronic somatic disease, somatic symptoms, psychological distress, concentration difficulties, insomnia or overweight and to assess to which extent the family contributes to the association between health and school dropout.

Methods: A population of 8950 school-attending adolescents (13–21 years) rated their health in the Young-HUNT 1 Study (90% response rate) in 1995–1997. High school dropout or completion, was defined with the Norwegian National Education Database in the calendar year the participant turned 24 years old. Parental socioeconomic status was defined by using linkages to the National Education Database, the National Insurance Administration and the HUNT2 Survey. We used logistic regression to estimate odds ratios and risk differences of high school dropout, both in the whole population and among siblings within families differentially exposed to health problems.

Results: All explored health dimensions were strongly associated with high school dropout. In models adjusted for parental socioeconomic status, the risk differences of school dropout according to health exposures varied between 3.6% (95% CI 1.7 to 5.5) for having ≥1 somatic disease versus none and 11.7% (6.3 to 17.0) for being obese versus normal weight. The results from the analyses comparing differentially exposed siblings, confirmed these results with the exception of weaker associations for somatic diseases and psychological distress. School dropout was strongly clustered within families (family level conditional intraclass correlation 0.42).

Conclusions: Adolescent health problems are markers for high school dropout, independent of parental socioeconomic status. Although school dropout it strongly related to family-level factors, also siblings with poor health have reduced opportunity to complete high school compared to healthy siblings. Public health policy should focus on ensuring young people with poor health the best attainable education.

Citation: De Ridder KAA, Pape K, Johnsen R, Holmen TL, Westin S, et al. (2013) Adolescent Health and High School Dropout: A Prospective Cohort Study of 9000 Norwegian Adolescents (The Young-HUNT). PLoS ONE 8(9): e74954. doi:10.1371/journal.pone.0074954

Editor: Jennifer Beam Dowd, Hunter College, City University of New York (CUNY), CUNY School of Public Health, United States of America

Received May 31, 2013; **Accepted** August 6, 2013; **Published** September 23, 2013

Funding: KDR and KP are financed by the Norwegian University of Science and Technology. JHB is financed by the by the Research Council of Norway and the Liaison Committee between the Central Norway Regional Health Authority and the Norwegian University of Science and Technology. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

* E-mail: karin.de.ridder@ntnu.no

Introduction

High school dropout is a major concern in most Western countries because it is associated with lower employment rate and poor health [1,2]. Already in the first decade of adulthood, school dropout is associated with a substantially higher probability of receiving medical and non-medical social insurance benefits, suggesting that mechanisms in adolescence are at the basis of these adversities [3].

Adult health is strongly related to educational attainment. 4While prior research has mainly considered poor health a consequence of low education, recent twin studies have suggested that, in some cases, the relation can be the result of health selection – poor health causing lower education [4,5]. There is evidence that suggests an association between poor health in adolescence and low educational achievement, as self-rated health in adolescence is associated with adult educational level [6,7]. Other studies have indicated that chronic physical conditions or disabilities [8], mental or psychosomatic symptoms [9], attention problems [10], and sleep problems [11] are associated with poor educational attainment. In addition, height and weight, which reflects a latent

health potential, results in differences in educational attainment [12].

However, confounding from familial genetic, environmental and socioeconomic factors could also influence these associations [13], yet it is still unclear to what extent family factors can be attributed to the association between health and school dropout.

We studied the associations between several dimensions of self-reported health in adolescence and high school dropout, adjusting for parental socioeconomic background and family living situation. Additionally, by comparing siblings, we tested if the associations remained after accounting for all shared, stable unobserved family characteristics.

Methods

Participants

The Young-Hunt study is the adolescent part of the HUNT study (The Nord-Trøndelag Health Study, http://www.ntnu.no/hunt) in the county Nord-Trøndelag, Norway [14]. All school-attending students in the middle and secondary school were invited to fill in a comprehensive questionnaire during a class hour, and 8949 completed the questionnaire (90% response rate). This population based survey was carried out between autumn 1995 and spring 1997. Participants were linked to their biological parents through the National Identity Number. Adolescents and their parents were linked to the Norwegian National Education Database (http://www.ssb.no/mikrodata). Parental information was also obtained by linkage to the National Insurance Administration (income) and the HUNT2 study (occupational class). Siblings (having the same biological mother) were identified through the National Register Code in the family register. We excluded 76 individuals because of missing educational data (8), age-school mismatch (4), born after 1983 (4), died during follow-up (30) and disability pension within the period (16–21 years) when they were eligible for high school education (30).

The Regional Committee for Medical Research Ethics approved the present study (reference 2010/1527-5, in accordance with the Helsinki declaration). Each participant and the parents/legal guardians of the participants younger than 16 years old gave their written consent to participate in the Young-Hunt Study.

School dropout

In Norway, basic education is compulsory up to the start of senior high school (upper secondary education) at age 16. Every 15- to 16-year-old has a statutory right to 3 years of senior high school which consists of both general and vocational tracks. In the follow-up period (1998-2008), we registered the outcome high school for all participants as either completion or dropout in the calendar year the participant turned 24 years old. We accomplished this using the linkage to the Norwegian National Education Database.

Health measures

We identified several health dimensions based on the self-reported health information provided by the study participants. We defined somatic disease as having asthma, diabetes, migraine, or epilepsy diagnosed by a doctor or having any other illness that lasted longer than 3 months. Subjective health problems are common in adolescence, tend to occur in a cluster and symptom load scores have been considered as measuring a latent trait of psychosomatic complaints [15]. Somatic symptom scores were based on the sum of self-reported presence of eight symptoms (headache, neck or shoulder pain, joint or muscle pain, stomach pain, nausea, constipation, diarrhea, heart palpitations; each one dichotomized into "never/seldom" and "sometimes/often") during the last 12 months (Cronbachs alpha 0.73). This symptom score was dichotomized into the two lowest tertiles (none or one symptom) versus the highest tertile (two or more symptoms). Psychological distress was measured with the SCL-5 scale score – a validated 4-integer 5 item short version of the original SCL-90 (Hopkins Symptom Checklist) [16]. The variable was dichotomized with a cut-off point at 2.0 [17]. Insomnia was defined by having difficulties falling asleep in the last month and dichotomized into "never/sometimes" versus "often/almost every night". Concentration difficulties were defined as having difficulties concentrating during class and dichotomized into "never/sometimes" versus "often/very often". We measured self-rated health using the question "How is your health at the moment?" and dichotomized the four response alternatives into "good/very good" versus "poor/not so good".

Trained nurses measured height and weight following a standard protocol using standardized meter bands and weight scales. Body mass index (BMI) was defined by cut-offs for the appropriate age groups as proposed by the International Obesity Task Force (IOFT) described by Cole et al. [18] Overweight corresponded with the adult BMI from 25 to 30 and obesity a BMI of 30 and more.

Parental socio-economic position

Parental education level was registered at the time the participant was 16 years old and divided into three categories: compulsory (primary and lower secondary education), intermediate (upper secondary and post-secondary non-tertiary education) and tertiary (under-graduate, graduate and post-graduate education). Parental income was assessed by the mean annual income (Norwegian currency) in a two year period (1994 and 1995). The total income (including income from benefits) was used and defined by quintiles. Parental occupational class was defined by Erikson Goldthorpe Portocarero (EGP) social class scheme in HUNT2 [19]. The family living situation was defined by living in a "traditional family" (with both the biological mother and father) or not.

Statistical analysis

Primary analysis investigated the association for each health variable with high school dropout. Sex- and age-adjusted logistic regression analyses were conducted on complete datasets for each model defined by the health variable, with the total N varying for each model. The percentage of missing data varied from 2.0% to 7.5%. To adjust for possible socioeconomic confounders, maternal education level and family living situation were added to the model. Maternal education level was chosen because this measure of parental socioeconomic status (SES) had little missing data (0.5%) compared with the other measures, and 87% of the adolescents were living with their mother. We performed additional analyses using various socioeconomic variables (maternal, paternal and highest parental education, income and occupation separately and combined). We carried out tests for statistical interaction between our health variables and sex and between health variables and parental socioeconomic status. We also performed sensitivity analysis by use of complete case-only (n = 7730), which restricted the analysis to participants with complete data for all exposures, outcomes and confounder variables.

Secondary analysis estimated multivariable sibling fixed-effect (conditional logit) models in order to account for unobserved heterogeneity at the family level (number of siblings = 698). Sibling fixed-effect logistic regression models or logistic regression models conditional on sharing the same biological mother are equivalent

[20]. The model attends to the family of origin and focuses on the siblings discordant on high school graduation status. It compares health among siblings within the same families, thereby controlling for all family background characteristics (observed and unobserved) that the siblings share. These sibling fixed-effect models were adjusted for sex, age and family living situation.

Finally, we investigated to what degree high school dropout was determined by the family of origin with sex- and age-adjusted multilevel logistic regression for complete cases (n = 7730). Thereafter, we included the individual characteristics (health variables) and the family variables (maternal education level and family living situation) to investigate the extent to which family level differences were explained by these individual and contextual characteristics. We estimated a conditional intraclass correlation coefficient (ICC) with linear threshold method and the median odds ratio (MOR) [21]. The ICC expresses the propensity to dropout of school that can be attributed to the family. The MOR quantifies the variation between clusters (families) by comparing two persons with the same covariates when randomly chosen from two different families. The MOR is defined as the median odds ratio between the person of higher propensity and the person of lower propensity. If the MOR is one, there is no variation between families.

Point estimates obtained from logistic regression analyses are presented as odds ratios (OR) and risk differences (RD) with 95%

Table 1. Characteristics of the total cohort, all the siblings within the cohort and the siblings with different outcome (school completion/dropout) within the sibling cohort.

	Total cohort	All siblings	Siblings with different outcome
High school dropout	1488 (17)	516 (16)	346 (50)
Mean (SD) Age (years)	16.0 (1.94)	16.1 (2.0)	16.1 (2.1)
Male	4463 (50)	1628 (50)	330 (53)
Individual health factors			
Somatic disease			
1 or more	1813 (20)	676 (21)	160 (22)
Missing	(0)	(0)	(0)
Somatic symptoms			
2 or more	3094 (35)	1118 (35)	272 (39)
Missing	(4)	(3)	(5)
Psychological distress			
High	879 (10)	324 (10)	88 (13)
Missing	(2)	(2)	(4)
Insomnia			
Often/every night	887 (10)	316 (10)	81 (12)
Missing	(1)	(1)	(2)
Concentration difficulties			
Often/very often	2101 (24)	771 (24)	215 (31)
Missing	(2)	(2)	(4)
Self-rated health			
Not so good/bad	951 (11)	332 (10)	97 (14)
Missing	(2)	(2)	(3)
BMI			
Overweight	1184 (13)	412 (13)	92 (13)
Obese	251 (3)	84 (3)	22 (3)
Missing	(6)	(6)	(7)
Family factors			
Maternal education level			
Primary	2405 (27)	835 (25.5)	275 (39)
Intermediate	4404 (49.5)	1558 (48)	319 (46)
Tertiary	2023 (23)	848 (26)	98 (14)
Missing	(0.5)	(0.5)	(1)
Family living situation			
Traditional family	6418 (74)	2483 (76)	446 (64)
Missing	(2)	(2)	(2)
Observations	**8873**	**3256**	**698**

Figures are numbers (percentages), unless stated otherwise.
doi:10.1371/journal.pone.0074954.t001

Table 2. Odds ratio for high school dropout according to indicators of adolescent health in the whole population (crude and adjusted models) and within the families (sibling fixed-effect models).

	Crude[a]		Adjusted[b]	Within family effect[c]	
	N dropout	Odds ratio (CI)	Odds ratio (CI)	N dropout	Odds ratio (CI)
Somatic disease					
None	1070	1.00	1.00	244	1.00
1 or more	358	1.39 (1.22 to 1.59)	1.32 (1.15 to 1.51)	78	1.06 (0.70 to 1.60)
Somatic symptoms					
None or 1	803	1.00	1.00	169	1.00
2 or more	566	1.51 (1.34 to 1.71)	1.42 (1.25 to 1.62)	125	1.29 (0.87 to 1.90)
Psychological distress					
Low	1202	1.00	1.00	298	1.00
High	180	1.69 (1.41 to 2.03)	1.56 (1.30 to 1.88)	37	1.07 (0.64 to 1.78)
Insomnia					
Never/seldom	1201	1.00	1.00	272	1.00
Often/every night	193	1.67 (1.40 to 1.99)	1.66 (1.39 to 1.99)	39	1.27 (0.75 to 2.15)
Concentration difficulties					
Never/seldom	881	1.00	1.00	197	1.00
Often/very often	497	2.13 (1.88 to 2.43)	1.98 (1.74 to 2.26)	108	1.69 (1.12 to 2.53)
Self-rated health					
Very good/good	1163	1.00	1.00	261	1.00
Not so good/bad	245	2.07 (1.77 to 2.43)	1.81 (1.53 to 2.13)	48	1.44 (0.87 to 2.39)
BMI					
Normal weight	975	1.00	1.00	133	1.00
Overweight	231	1.47 (1.25 to 1.73)	1.34 (1.14 to 1.58)	39	0.93 (0.55 to 1.56)
Obese	71	2.39 (1.80 to 3.18)	2.20 (1.64 to 2.95)	14	4.18 (1.11 to 15.7)

Values in parentheses are 95% confidence intervals (CI).
[a]Crude models adjusted for sex and age.
[b]Adjusted for sex, age, maternal education level and family living situation.
[c]Sibling fixed-effect models are adjusted for sex, age and family living situation
Total N varies for each health variable in the total population from 8205 to 8696, and in the sibling fixed-effect models from 581 to 649.
doi:10.1371/journal.pone.0074954.t002

confidence intervals (CI). A risk difference describes how 1 unit change in an independent variable (eg, somatic disease or not) alters the absolute risk of a current outcome (eg, high school dropout). Risk differences were estimated from the logistic regression models with the covariates at their mean. Data were analyzed with STATA 12.1 (StataCorp LP).

Results

Description of participants

The characteristics of the whole population and the siblings are presented in table 1. The mean follow-up time was 8.0 years (range 3 to 12 years). The baseline mean age of the participants was 16 years (range 13 to 21 years). At the age of 24, 1488 (17%) had not completed high school, more boys (20%) than girls (14%). Compared with the whole sibling sample, the sample of siblings discordant on graduation status (n = 698) was characterized by more mothers with only primary education, fewer traditional families, more psychological distress, poorer self-reported health and more concentration problems.

Whole study population analyses

There were crude associations between all health variables and a subsequent risk of high school dropout (table 2). The associations were attenuated with adjustment for maternal education level and family living situation. Adjustment for other parental socioeconomic measures (educational level of both parents, parental income and parental occupational class) – separately and combined – did not alter the results (data not shown). Parental education level was the most important socioeconomic measure and was strongly associated with high school dropout. The absolute increase in the risk of high school dropout according to the different health measures varied between 3.6% (95% CI 1.7 to 5.5) for having 1 or more somatic disease and 11.7% (6.3 to 17.0) for being obese corresponding to the adjusted models in table 3. The risk differences for all variables for the whole population analyses in table 2 are shown in table 3. We performed additional analyses with insomnia, concentration problems and self-rated health as categorical measures (using all 4 categories) and with symptom load and psychological distress as continuous measures. We found indications of a dose-response relationship between all the health variables and the risk for high school dropout. Complete case analyses of only participants with complete data (n = 7730) showed the same associations between the health variables and school dropout. For all health variables, there was no evidence for effect measure modification by sex or maternal education.

Table 3. Risk difference (RD) of school dropout from logistic regression models[a].

	Crude[b]	Adjusted[c]
	Risk difference (CI)	**Risk difference (CI)**
1 or more somatic disease	4.8 (2.8 to 6.9)	3.6 (1.7 to 5.5)
Versus none	ref.	ref.
2 or more somatic symptoms	5.7 (4.0 to 7.5)	4.5 (2.8 to 6.2)
Versus none or 1	ref.	ref.
High psychological distress	8.0 (4.9 to 11.1)	6.2 (3.3 to 9.0)
Versus low	ref.	ref.
Often/every night insomnia	7.8 (4.8 to 10.8)	7.2 (4.3 to 10.0)
Versus Never/seldom	ref.	ref.
Often/very often concentration difficulties	11.3 (9.2 to 13.4)	9.3 (7.4 to 11.3)
Versus never/seldom	ref.	ref.
Not so good/bad self-rated health	11.7 (8.7 to 14.7)	8.5 (5.8 to 11.3)
Versus very good/good	ref.	ref.
BMI		
Normal weight	ref.	ref.
Overweight	5.4 (3.0 to 7.8)	3.7 (1.5 to 5.9)
Obese	14.1 (8.4 to 19.7)	11.7 (6.3 to 17.0)

Figures are percentages with 95% confidence interval (CI).
[a]Estimated risk difference in the risk to drop out of high school relative to complete high school.
[b]Crude models with the covariates sex and age at mean.
[c]Adjusted models with the covariates sex, age, maternal education level and family living situation at mean
Total N varies for each health variable from 8205 to 8696.
doi:10.1371/journal.pone.0074954.t003

Within family analyses

The sibling fixed-effect analysis confirmed the results from the total population, except for somatic diseases and psychological distress; although the precision was reduced due to reduced statistical power in the within-family models (table 2).

Clustering by family analyses

High school drop-out was substantially clustered in families (table 4). About 42% of the adolescents' propensity to drop out of high school could be attributed to the family. Likewise, the median of the odds ratios (MOR) between the person with a high propensity and the person with a low propensity is estimated to be 4.3. When individual health variables and contextual factors (maternal education level and family living situation) were included, the unexplained cluster heterogeneity decreased substantially, yielding a MOR of 2.98. However, a large proportion of the clustering by family still remained unexplained.

Table 4. Clustering of high school dropout on the family level for complete cases (n = 7730).

	Crude[a]	Adjusted[b] for health	Adjusted[c] for health and family characteristics
ICC	41.8%	36.4%	28.7%
MOR	4.30	3.68	2.98

Figures are intraclass coefficients (ICC%) and median odds ratios (MOR).
[a]Crude model adjusted for sex and age.
[b]Model adjusted for sex, age, somatic disease, somatic symptoms, psychological distress, insomnia, concentration difficulties, self-rated health and BMI.
[c]Model adjusted for sex, age, somatic disease, somatic symptoms, psychological distress, insomnia, concentration difficulties, self-rated health, BMI, maternal education level and family living situation.
doi:10.1371/journal.pone.0074954.t004

Discussion

In this large prospective population based study over 11 years, we found an increased risk of high school dropout for all explored dimensions of adolescent ill health. With the exception of psychological distress and somatic disease, this was also true when comparing siblings. Although high school dropout was strongly associated with parental socioeconomic class and strongly clustered at the family level, the negative impact of ill health on school dropout seemed to exist in all families and across all social classes.

Strengths and limitations of the study

The results were based on a large number of participants, and outcome measures were attained from nearly complete and accurate register-based information. Furthermore, we were able to control for several confounding variables, and our sibling design made it possible to control for any known and unknown family factors shared by siblings. Although the participation rate was high (90%), we cannot rule out the possibility of selection bias: firstly, it is reasonable to assume higher dropout rates among the non-responders and secondly, we included only the adolescents enrolled in school at baseline, which may have excluded especially older adolescents who had already dropped out from school. Selection bias might affect the results with attenuated associations between adolescent health problems and school dropout. We relied mainly on self-reported health measurements, and it is

noteworthy that structured clinical assessments of the participants' health status could have given more valid and reliable baseline information. However, such an approach would not have been feasible in a study of this size. The precision of the sibling fixed-effect analyses was reduced due to the lower number of siblings compared to the whole population.

Comparison with other studies

The literature related to adolescent health and educational attainment is limited, and studies with a prospective design have been sparse. On the population level, we found dose-response associations between psychological distress, somatic disease, symptom load, insomnia, concentration difficulties, self-rated health and overweight/obesity and school dropout, which is consistent with other studies [8,11,22–32]. The effect of obesity on school dropout is greater than other health problems, which is consistent with Gortmaker et al. [31].

In cases where there was a high symptom load, insomnia, concentration difficulties, poor self-rated health and obesity, the impact of poor adolescent health remained even when controlling for stable family background characteristics. In the case of self-rated health, this finding is consistent with other studies [29,33], but for concentration difficulties, this finding is inconsistent with the study of Fletcher et al. [28] in that they did not find any effect of ADHD symptoms on risk for high school dropout within the family. However, a self-report of concentration difficulties is not the same as a screening set for ADHD symptoms, and there is some evidence that inattention rather than hyperactivity predicts low long-term educational attainment [34]. We are not aware of other studies that have compared siblings with different levels of symptom load, insomnia, or weight and the risk of subsequent school dropout. Our results strengthen the hypothesis that health problems in adolescence could have adverse causal effect on future socioeconomic position.

The results of the within-family models differ from the whole-population models for somatic disease, psychological distress and overweight. Their impact on school dropout was completely attenuated when comparing siblings differentially exposed. This may reflect the confounding effect of shared family background characteristics and suggests that such shared factors are essential in the association between health and school dropout. However, psychological distress is also clustered within families [35], and an on-off measure of symptoms of mental illness may not be enough to differentiate psychiatric pathology between siblings. Fletcher et al. [36] found that siblings with depression had a higher risk for dropout compared to their siblings without depression. Also, Fletcher and Richards [26] found lower educational attainment for adolescents with diabetes. Our variable on somatic disease included diabetes, but we could not reproduce the same analyses with only diabetes because of lack of power.

As in many other studies, adolescents from lower socioeconomic classes had substantial higher risk for not completing high school [37]. Our study showed that *all* examined health dimensions increased the risk for school dropout independent of, and additive to, socioeconomic group defined by parental education, income or occupation, which is in concordance with other studies [29,30,38]. The relationship between poor health and school dropout was not less important for higher social classes. Previous research suggested that socioeconomic inequalities in health during adult life were to a large extent due to social causation, and health selection was only slightly involved [39,40]. However, most of this research was on adult populations. Our study suggests a robust health selection process in the attainment of education during adolescence. About 42% of the propensity for school dropout could be attributed to the family level, and is comparable with the results from studies examining years of educational attainment [41]. This underlines the importance of the family as a social context in the process of school dropout and stresses the importance of investigating how health is, or can become, an independent risk factor for school dropout.

Possible mechanisms

Adolescent health could influence educational attainment through several mechanisms [30]. Poor health could impair cognitive development or affect educational participation because of absenteeism from school, resulting in poorer school achievement [42]. It is also possible that poor health could weaken peer relationships, which could have secondary effects on educational attainment. Additionally, adolescents themselves, parents and teachers could have reduced educational expectations of an adolescent who is limited by poor health. Reduced encouragement and investment in education could also occur if the expected benefits from education (employment) were regarded as low. Youth may also be stigmatized and subsequently discriminated by peers and teachers for some health problems like obesity, which can affect youths motivation and willingness to attend school [31,43,44].

Conclusion and policy implication

Poor health in all its dimensions compromises the opportunity to complete high school for adolescents of all social classes, and the educational gradient that develops with poor health in the picture reduces future work prospects and adult health. Public health policies should ensure that young people with poor health are provided with the best attainable education, thereby preventing them from having their future opportunities substantially reduced. There is still a gap in our information about the mechanisms at work in the relationship between adolescent health and educational attainment. Further research will need to focus on the family perspective, but also a life course perspective in order to better understand adolescents' social integration process through education. With more knowledge on this topic, additional preventive measures at an early stage may reduce the number of young people living on the fringe of society with poor health and poor prospects for working life.

Acknowledgments

The Nord-Trøndelag Health Study (The HUNT Study) is a collaboration between HUNT Research Centre (Faculty of Medicine, Norwegian University of Science and Technology NTNU), Nord-Trøndelag County Council, Central Norway Health Authority and the Norwegian Institute of Public Health.

Author Contributions

Conceived and designed the experiments: KDR KP RJ TLH JHB. Analyzed the data: KDR KP JHB. Wrote the paper: KDR KP RJ TLH SW JHB. Responsible for the Young-HUNT data collection: TLH.

References

1. OECD (2012) Equity and quality in education: supporting dissadvantaged students and schools. Paris.
2. Marmot MG (2003) Understanding social inequalities in health. Perspect Biol Med 46: 9–23.

3. De Ridder KAA, Pape K, Johnsen R, Westin S, Holmen TL, et al. (2012) School dropout: a major public health challenge: a 10-year prospective study on medical and non-medical social insurance benefits in young adulthood, the Young-HUNT 1 Study (Norway). J Epidemiol Community Health 66: 995–1000.
4. Osler M, McGue M, Christensen K (2007) Socioeconomic position and twins' health: a life-course analysis of 1266 pairs of middle-aged Danish twins. Int J Epidemiol 36: 77-83.
5. Fujiwara T, Kawachi I (2009) Is education causally related to better health? A twin fixed-effect study in the USA. Int J Epidemiol 38: 1310–1322.
6. Huurre T, Aro H, Rahkonen O, Komulainen E (2006) Health, lifestyle, family and school factors in adolescence: Predicting adult educational level. Educational Research 48: 41–53.
7. Koivusilta L, Arja R, Andres V (2003) Health behaviours and health in adolescence as predictors of educational level in adulthood: a follow-up study from Finland. Soc Sci Med 57: 577–593.
8. Pless IB, Power C, Peckham CS (1993) Long-term psychosocial sequelae of chronic physical disorders in childhood. Pediatrics 91: 1131–1136.
9. Breslau J, Lane M, Sampson N, Kessler RC (2008) Mental disorders and subsequent educational attainment in a US national sample. J Psychiatr Res 42: 708–716.
10. Polderman TJC, Boomsma DI, Bartels M, Verhulst FC, Huizink AC (2010) A systematic review of prospective studies on attention problems and academic achievement. Acta Psychiatr Scand 122: 271–284.
11. Dewald JF, Meijer AM, Oort FJ, Kerkhof GA, Bögels SM (2010) The influence of sleep quality, sleep duration and sleepiness on school performance in children and adolescents: A meta-analytic review. Sleep Medicine Reviews 14: 179–189.
12. Puhl RM, Heuer CA (2009) The stigma of obesity: A review and update. Obesity 17: 941–964.
13. Viner RM, Ozer EM, Denny S, Marmot M, Resnick M, et al. (2012) Adolescence and the social determinants of health. Lancet 379: 1641–1652.
14. Holmen TL, Bratberg G, Krokstad S, Langhammer A, Hveem K, et al. (2013) Cohort profile of the Young-HUNT Study, Norway: A population-based study of adolescents. Int J Epidemiol doi:10.1093/ije/dys232.
15. Ravens-Sieberer U, Torsheim T, Hetland J, Vollebergh W, Cavallo F, et al. (2009) Subjective health, symptom load and quality of life of children and adolescents in Europe. Int J Public Health 54 Suppl 2: 151–159.
16. Tambs K, Moum T (1993) How well can a few questionnaire items indicate anxiety and depression? Acta Psychiatr Scand 87: 364–367.
17. Strand BH, Dalgard OS, Tambs K, Rognerud M (2003) Measuring the mental health status of the Norwegian population: a comparison of the instruments SCL-25, SCL-10, SCL-5 and MHI-5 (SF-36). Nord J Psychiatry 57: 113–118.
18. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH (2000) Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 320: 1240–1243.
19. Krokstad S, Ringdal K, Westin S (2002) Classifying people by social class in population based health surveys: Two methods compared. Norsk epidemiologi 12: 19–25.
20. Allison PD (2009) Fixed effects logistic models. In: Allison PD, editor. Fixed effects regression models. United States of America: SAGE publications, Inc. pp. 28–48.
21. Merlo J, Chaix B, Ohlsson H, Beckman A, Johnell K, et al. (2006) A brief conceptual tutorial of multilevel analysis in social epidemiology: using measures of clustering in multilevel logistic regression to investigate contextual phenomena. J Epidemiol Community Health 60: 290–297.
22. Kessler RC, Foster CL, Saunders WB, Stang PE (1995) Social consequences of psychiatric disorders, I: Educational attainment. Am J Psychiatry 152: 1026–1032.
23. McLeod JD, Fettes DL (2007) Trajectories of failure: The educational careers of children with mental health problems. AJS 113: 653–701.
24. Fletcher JM (2008) Adolescent depression: diagnosis, treatment, and educational attainment. Health Econ 17: 1215–1235.
25. Maslow GR, Haydon A, McRee A-L, Ford CA, Halpern CT (2011) Growing up with a chronic illness: Social success, educational/vocational distress. J Adolesc Health 49: 206–212.
26. Fletcher JM, Richards MR (2012) Diabetes's 'health shock' to schooling and earnings: Increased dropout rates and lower wages and employment in young adults. Health Aff 31: 27–34.
27. Huurre T, Rahkonen O, Komulainen E, Aro H (2005) Socioeconomic status as a cause and consequence of psychosomatic symptoms from adolescence to adulthood. Soc Psychiatry Psychiatr Epidemiol 40: 580–587.
28. Fletcher J, Wolfe B (2008) Child mental health and human capital accumulation: the case of ADHD revisited. J Health Econ 27: 794–800.
29. Haas SA, Fosse NE (2008) Health and the educational attainment of adolescents: evidence from the NLSY97. J Health Soc Behav 49: 178–192.
30. Jackson MI (2009) Understanding links between adolescent health and educational attainment. Demography 46: 671–694.
31. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH (1993) Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med 329: 1008–1012.
32. Needham BL (2009) Adolescent depressive symptomatology and young adult educational attainment: An examination of gender differences. J Adolesc Health 45: 179–186.
33. Lê F, Diez Roux A, Morgenstern H (2013) Effects of child and adolescent health on educational progress. Soc Sci Med 76: 57–66.
34. Pingault JB, Tremblay RE, Vitaro F, Carbonneau R, Genolini C, et al. (2011) Childhood trajectories of inattention and hyperactivity and prediction of educational attainment in early adulthood: a 16-year longitudinal population-based study. Am J Psychiatry 168: 1164–1170.
35. Kendler KS (2001) Twin studies of psychiatric illness: an update. Arch Gen Psychiatry 58: 1005–1014.
36. Fletcher JM (2010) Adolescent depression and educational attainment: results using sibling fixed effects. Health Econ 19: 855–871.
37. Rouse CE, Barrow L (2006) U.S. elementary and secondary schools: Equalizing opportunity or replicating the status quo? Future Child 16: 99–123.
38. Currie J, Stabile M (2006) Child mental health and human capital accumulation: the case of ADHD. J Health Econ 25: 1094–1118.
39. Power C, Matthews S, Manor O (1996) Inequalities in self rated health in the 1958 birth cohort: lifetime social circumstances or social mobility? BMJ 313: 449–453.
40. Elstad JI, Krokstad S (2003) Social causation, health-selective mobility, and the reproduction of socioeconomic health inequalities over time: panel study of adult men. Soc Sci Med 57: 1475–1489.
41. Boyle MH, Georgiades K, Racine Y, Mustard C (2007) Neighborhood and family influences on educational attainment: results from the Ontario Child Health Study follow-up 2001. Child Dev 78: 168–189.
42. Needham BL, Crosnoe R, Muller C (2004) Academic failure in secondary school: The inter-related role of health problems and educational context. Soc Probl 51: 569–586.
43. Crosnoe R, Muller C (2004) Body mass index, academic achievement, and school context: Examining the educational experiences of adolescents at risk of obesity. J Health Soc Behav 45: 393–407.
44. West P (1991) Rethinking the health selection explanation for health inequalities. Soc Sci Med 32: 373–384.

Paper III

De Ridder et al. BMC Public Health 2013, 13:941
http://www.biomedcentral.com/1471-2458/13/941

BMC Public Health

RESEARCH ARTICLE Open Access

High school dropout and long-term sickness and disability in young adulthood: a prospective propensity score stratified cohort study (the Young-HUNT study)

Karin A A De Ridder[1,2*], Kristine Pape[1], Koenraad Cuypers[3], Roar Johnsen[1], Turid Lingaas Holmen[4], Steinar Westin[1] and Johan Håkon Bjørngaard[1,5]

Abstract

Background: High school dropout and long-term sickness absence/disability pension in young adulthood are strongly associated. We investigated whether common risk factors in adolescence may confound this association.

Methods: Data from 6612 school-attending adolescents (13–20 years old) participating in the Norwegian Young-HUNT1 Survey (1995–1997) was linked to long-term sickness absence or disability pension from age 24–29 years old, recorded in the Norwegian Labour and Welfare Organisation registers (1998–2008). We used logistic regression to estimate risk differences of sickness or disability for school dropouts versus completers, adjusting for health, health-related behaviours, psychosocial factors, school problems, and parental socioeconomic position. In addition, we stratified the regression models of sickness and disability following dropout across the quintiles of the propensity score for high school dropout.

Results: The crude absolute risk difference for long-term sickness or disability for a school dropout compared to a completer was 0.21% or 21% points (95% confidence interval (CI), 17 to 24). The adjusted risk difference was reduced to 15% points (95% CI, 12 to 19). Overall, high school dropout increased the risk for sickness or disability regardless of the risk factor level present for high school dropout.

Conclusion: High school dropouts have a strongly increased risk for sickness and disability in young adulthood across all quintiles of the propensity score for dropout, i.e. independent of own health, family and socioeconomic factors in adolescence. These findings reveal the importance of early prevention of dropout where possible, combined with increased attention to labour market integration and targeted support for those who fail to complete school.

Keywords: School dropouts, Sick leave, Disability insurance, Risk factors, Adolescent, Adult, Prospective cohort study, Propensity score, HUNT study

* Correspondence: karin.de.ridder@ntnu.no
[1]Department of Public Health and General Practice, Norwegian University of Science and Technology, 7491 Trondheim, Norway
[2]Youth Health Care, Department of Public Health and Primary Care, KU Leuven, 3000 Leuven, Belgium
Full list of author information is available at the end of the article

Background

Young people dropping out from school, never being included in or leaving the labour market due to health problems or disability represent an individual hazard and a society challenge [1,2]. Prospective studies of health and social functioning in young adulthood among dropouts are rare, although there is evidence to suggest a substantially higher risk of sickness and disability among high school dropouts compared to school completers [3,4]. Hence, a better understanding of the complex role of adolescent health and socioeconomic factors underlying the association between school dropout and subsequent sickness and disability may provide important information for social welfare strategies and for public health policy.

The association between school dropout and subsequent sickness and disability could be confounded by the co-occurrence of lower childhood socioeconomic position (SEP), adolescent ill health and other risk factors [5-16]. In a life-course framework, the accumulation of risks may be clustered and often be related to the family's socioeconomic position in society [17]. Hence, baseline differences in risk profiles between high school dropouts and completers, to a large extent, may explain their further trajectories in adulthood and their risk for long-term sickness and disability [18,19]. Another life-course framework model is the chain of risk model, which resembles what has been described as a "pathways model" [17], where each exposure increases the risk of a subsequent exposure, but in addition to an independent effect on the outcome irrespective of the later exposure.

In a large prospective study of about 6612 Norwegians, we investigated the role of adolescent health, health-related behaviours, psychosocial factors, school problems and parental socioeconomic position in the association between high school dropout and long-term sickness absence or disability pension in young adulthood. We hypothesized that the more vulnerable adolescents with a high risk level for school dropout would, in case of school dropout, have an even greater increased risk for long-term sickness absence or disability pension compared to the adolescents with a low risk level for school dropout.

Methods

Participants

Young-HUNT is the adolescent part of the HUNT Study (The Nord-Trøndelag Health Study, www.ntnu.no/hunt) in the county of Nord-Trøndelag, Norway [20]. All school attending students of middle and secondary school in 1995–97 were invited to participate in the Young-HUNT1 Survey, and 8949 adolescents (90% response rate) completed a comprehensive questionnaire during a class hour. Data from Young-HUNT1 were linked to information about social insurance benefits from the Norwegian Labour and Welfare Organisation registers (FD-trygd) in the period 1998–2008. Adolescents and their parents were linked to the Norwegian National Education Database (http://www.ssb.no/mikrodata). Parents and siblings (those with the same biological mother) were identified through the national identity number in the Norwegian national family register.

We excluded 2333 adolescents from this study. Causes for exclusion were disability pension collected within the period (16–21 years old) when they were eligible for high school education (30), missing educational data (8), death before age 24 (30), migration before age 24 (57), born after 1983 (4) or age-school level mismatch (4). Because of complete cases analyses, 2204 individuals were excluded due to missing data on the questionnaire or the physical examination (BMI).

The present study was approved by The Regional Committee for Medical Research Ethics (reference 2010/1527-5), and was conducted according the Declaration of Helsinki. Each participant and the parents/legal guardians of the participants younger than 16 years old gave their written consent to participate in the Young-HUNT Study.

Long-term sickness absence or disability pension

The outcome was long-term sickness absence or disability pension defined as medical benefits for permanent and temporary disability pension, medical, and vocational rehabilitation or sickness benefits received at least 180 days in one calendar year. This was based on annual registrations from the National Insurance Administration in the period 1998 to 2008 and defined as at least one episode of long-term medical benefits in a calendar year during the six-year follow-up period between age 24 and 29 years.

School dropout

Basic education in Norway is compulsory up to the start of senior high school (upper secondary education) at age 16. Every 15- to 16-year-old has a statutory right to 3 years of senior high school which consists of both general and vocational tracks. In the follow-up period (1998–2008), we registered the outcome high school for all participants as either having obtained (completion) or having *not* obtained (dropout) a certificate of senior high school (general or vocational track) in the calendar year the participant turned 24 years old. We chose to measure dropout at a later point estimate to avoid overestimation of the dropout rates because of the flexibility in study options and to make international comparison easier, because it is less dependent of the national school structure [2]. Data were retrieved through linkage to the Norwegian National Education Database which coded level of education by NUS2000-standards, which implemented the international education standard ISCED97.

Covariates

We defined the characteristics of the participants according to demographic data (age and sex), follow-up time, health, health behavior, psychosocial factors, school-related factors, and maternal education level. Follow-up time was the number of years from age 24 to end of follow-up or maximum age 29 in the period 1998–2008 when alive or not migrated. Maternal education level was registered at the time the participant was 16 years old and divided into three categories: compulsory (primary and lower secondary education), intermediate (upper secondary and post-secondary non-tertiary education) and tertiary (under-graduate, graduate and post-graduate education). Assessments of health and health behavior were based on the self-reported information from the participants in the Young-HUNT1 Survey (1995–1997): somatic disease (asthma, diabetes, migraine, epilepsy, or other longstanding illness), somatic symptom load, psychological distress, concentration difficulties, insomnia, self-rated health, smoking, and physical activity level. Trained nurses measured height and weight following a standard protocol. Body mass index (BMI) was defined by cutoffs for the appropriate age groups as proposed by Cole et al. [21]. Psychosocial factors included self-esteem, subjective well-being, loneliness, and family living situation. School-related factors included self-reported reading and writing difficulties, bullying, disease-related school absence, educational aspirations, academic problems, school dissatisfaction, and school-related conduct. (see Additional file 1: Table A for operational definition of the covariates).

Statistical methods

We presented baseline characteristics of participants who completed or dropped out of high school. Primary analysis investigated the association between high school dropout and long-term sickness or disability between ages 24 and 29. We used sex-, age- and follow-up time adjusted logistic regression on complete datasets (N=6651). Logistic regression was preferred above Cox regression analyses because we were mainly interested in estimating the *absolute* risk difference (and the effect of known confounders on this risk difference), rather than assessing the relative risk of receiving benefits for a person at risk per unit time. To adjust for possible confounders, we successively added maternal education level, health measures, health behavior, psychosocial factors, and school-related factors. We carried out tests for statistical interaction between high school dropout and sex and between high school dropout and maternal education level. Since a quarter of the study population had missing data at baseline, we also performed a sensitivity analysis with multiple imputations by chained equations (MICE) procedures to obtain 20 imputed datasets, which included most of the participants who had missing data (N=8805) (see Additional file 1: Table C for details about the imputation modeling procedure) [22]. Using the rich information in the Young-HUNT study to impute missing data, we assumed that missing data were missing at random. Many variables that are associated with non-participation in surveys were included in the dataset, which reduces the probability that data missing does depend on unobserved data, conditional on the observed data (see Additional file 1: Table B for description of missing data). The multiple imputation analyses are not presented as the main analyses as it was technically impossible to perform an imputation without comprehensive manipulation of the data, such as redefinition of the continuous variables into binary or ordinary variables and exclusion of the variable "academic problems" (important to calculate the propensity score) because of collinearity.

We also estimated multivariable conditional logistic regression models in order to control for factors that are shared within families (Number of siblings=316). By conditioning on the family of origin, these models compare long-term sickness or disability among sibships with and without high school dropout while controlling for all family background characteristics (observed and unobserved) that the siblings share [23]. These models were adjusted for sex, age, and follow-up time. Successively, we added health measures, health behavior, social factors, and school-related factors.

To investigate conditional vulnerability of dropout, we computed the propensity score (from 0 to 1) by using logistic regression; the dependent variable was high school dropout and the independent variables (covariates) were sex, age, maternal education level, health and health behavior measures, psychosocial factors, and school-related factors. The propensity score is a calculation of the probability to drop out of high school for a participant with specific predictive factors (regardless of whether they dropped out of high school or not). We computed the quintiles of the estimated propensity score with the first quintile representing the lowest probability to drop out of high school and the fifth quintile representing the highest probability. Within these strata, the covariates in the groups with high school dropout and completers are similarly distributed [24]. We carried out a logistic regression analysis with a statistical interaction between high school dropout and the propensity score stratified by quintiles.

As a sensitivity analysis, we also obtained a weighted estimate of the pooled odds ratio across the propensity score strata. Furthermore, we used propensity score matched methods in STATA to estimate the average treatment effect on the treated (ATT), or in our case "the average dropout effect on the dropouts", based on the propensity score. We used the technique radius matching with a propensity score radius of 0.1 [25].

Data were analyzed with STATA 12.1 (StataCorp LP). Odds ratios (OR) and risk differences (RD) were presented with 95% confidence intervals (CI). Risk differences were estimated from the logistic regression analyses with the covariates at their mean and follow-up time (from age 24 to 29) at 6 years.

Results

The study cohort with complete datasets (N=6612) consisted of 3375 girls (51%) and 3237 boys (49%). The baseline mean age of the participants was 16.1 years old (range 13 to 20 years). The mean follow-up time from age 24 to 29 was 4.5 years (range 1 to 6 years). During the follow-up period between the ages 24 and 29, 739 (11%) had long-term sickness or disability, more girls (13%) than boys (9%).

Overall, at the age of 24, 910 (14%) had not completed high school. High school dropouts were more likely than completers to be male, to have a mother with low education and less likely to live in a traditional family. In addition, they were more likely to have health problems, to smoke, to be physically inactive, to be lonely or bullied, and to have reported lower self-esteem and school related problems (Table 1).

The regression analyses displayed in Table 2 show the associations between high school dropout and long-term sickness or disability between ages 24 and 29. In the crude model, the risk difference for long-term sickness or disability for high school dropouts compared with high school completers was 0.21 or 21% points (95% CI 17 to 25). With the successive adjustment for maternal education level, health measures, health behavior, psychosocial factors, and school-related factors, the risk difference gradually decreased to 15% points (95% CI, 12 to 19). There was no evidence for effect measure modification by sex or maternal education level (p-value for interactions > 0.1). The magnitude and direction of the differences in long-term sickness or disability in young adulthood based on the main analyses of complete data and the sensitivity analysis of multiple imputations were in accordance to those presented in Table 2 (see Additional file 1: Table C).

The sibling analysis confirmed the results from the total population, but the odds ratios were substantially lower (Table 2). The precision was reduced due to reduced statistical power in the within-family models. Table D (see Additional file 1) presents the variables that were included in the propensity score analysis, along with the regression coefficients and standard errors. The c-index for the propensity score was 0.76, and figure A (see Additional file 1) visualizes the overlap between the two groups (high school dropouts and completers) on the propensity score. Table 3 presents the risk differences and odds ratios for long-term sickness or disability for high school dropouts compared to high school completers for each stratum of the propensity score. Overall, a high school dropout had a higher risk for long-term sickness or disability in each stratum. The pooled odds ratio across the propensity score strata was 2.95 (95% CI, 2.44 to 3.57), which results in an estimated risk difference between school dropouts and completers of 16.7% points (95% CI, 12.2 to 21.3). This is similar to the estimated ATT of 0.165 (95% CI, 0.136 to 0.194) in the radius matched propensity score analyses (see Additional file 1: Table E). A high school *completer* in stratum 1 (lowest risk) had a 7% (95% CI, 5 to 8) risk for long-term sickness or disability, while a high school *dropout* in stratum 5 (highest risk) had a 34% (95% CI, 29 to 39) risk (Figure 1). Compared to a participant in stratum 1, a person in stratum 5 had 7% points (95% CI, 4 to 10) higher risk for long-term sickness and disability. We found weak evidence of effect measure modification between the propensity score and dropout (p-value for interaction > 0.1).

Discussion

In this large prospective study, we found a strong association between high school dropout and long-term sickness or disability in young adulthood even after adjustment for parental socioeconomic position, health in adolescence, health-related risk behaviours, psychosocial risk factors, and school problems. Not only did a high school dropout systematically have a higher risk for long-term sickness and disability independent of propensity to drop out, but also a high school completer with the highest predicted tendency to drop out (high risk factor level present) had a lower risk for medical benefits than a school dropout with the lowest predicted tendency to dropout (low risk factor level present).

Strengths and limitations

The strengths of the study are the high number of participants, the prospective longitudinal design stratified by propensity score, and the robust associations. The main exposures (high school dropout and parental SEP) and outcome were based on nearly complete and high-quality national registers. The study population was school attending adolescents, and there was a high participation rate (90%). There might be more school dropouts among the non-responders and this might have led to some underestimation of the examined associations. The risk factors in adolescence relied on a self-reported questionnaire with missing data for a quarter of our study population, which might have caused bias; however sensitivity analyses with multiple imputed data produced comparable results. The number of sibling groups with different outcome status was

Table 1 Baseline characteristics of high school dropouts and high school completers (N= 6612)

	School dropouts (n=910)		School completers (n=5702)	
Demographics				
Age, mean, yr	16.09	(15.86-16.11)	16.10	(16.04-16.14)
Male	58.57	(55.36-61.77)	47.42	(46.13-49.72)
Maternal education level				
Primary	41.09	(37.90-44.30)	23.41	(22.31-24.51)
Intermediate	47.14	(43.90-50.39)	50.84	(49.54-52.14)
Tertiary	11.79	(10.68-13.85)	25.75	(24.61-26.88)
Health				
1 or more somatic disease	24.73	(21.92-27.53)	19.66	(18.63-20.69)
Symptom load, mean	1.60	(1.49-1.70)	1.31	(1.26-1.35)
High psychological distress, mean	1.52	(1.48-1.56)	1.45	(1.43-1.46)
Concentration problems	36.59	(33.46-39.72)	21.76	(20.70-22.84)
Insomnia	14.07	(11.81-16.33)	9.53	(8.76-10.28)
Poor self-rated health	17.14	(14.69-19.59)	9.27	(8.52-10.03)
Health behavior				
BMI				
Overweight	18.46	(15.94-20.98)	13.47	(12.58-14.36)
Obese	5.49	(4.01-6.98)	2.59	(2.18-3.01)
Smoking	34.62	(31.52-37.71)	18.82	(17.80-19.83)
No physical activity	19.67	(17.09-22.25)	10.93	(10.12-11.74)
Psychosocial factors				
Self-esteem, mean	2.95	(2.91-2.98)	3.05	(3.03-3.06)
Subjective well-being, mean	2.86	(2.80-2.93)	2.68	(2.65-2.70)
Loneliness, mean	2.10	(2.03-2.16)	2.01	(1.97-2.03)
Traditional family	59.12	(55.92-62.32)	77.50	(76.41-78.58)
School-related factors				
Reading and writing difficulties	15.60	(13.24-17.96)	6.42	(5.78-7.05)
Being bullied, mean	1.23	(1.19-1.26)	1.16	(1.15-1.17)
Disease-related school absence	9.67	(7.75-11.59)	3.95	(3.44-4.45)
Aspiration for higher education	38.57	(35.41-41.91)	46.62	(45.32-47.91)
Academic problems, mean	2.15	(2.12-2.18)	1.87	(1.86-1.89)
School-related dissatisfaction, mean	2.40	(2.36-2.44)	2.26	(2.25-2.28)
School-related conduct, mean	1.59	(1.56-1.62)	1.45	(1.44-1.46)

The numbers are proportions (in %), unless stated otherwise, with 95% confidence intervals between parentheses.

low, and therefore these results, from the sibling comparison, should be interpreted with care. Because we measured the risk factors in adolescence only once at baseline, there could be some residual confounding. It is however unlikely that this could explain the strong association that remained after full adjustment. Other variables on personal characteristics, like self-regulation, coping behaviour, or intellectual performance, or on general interpretations, like social capital or social cohesion, might have been relevant.

Previous literature

A few previous studies have investigated potential explanatory factors in adolescence for the association between educational level in general and long-term sickness or disability [4,19,26]. A Norwegian population based study found a higher risk for disability pension for high school dropouts when adjusted for parental position, low birth weight, and childhood disease benefits [4]. Two Scandinavian studies suggested that both educational level and IQ independently were associated

Table 2 Risk difference* and odds ratio with 95% confidence intervals for long-term sickness or disability between age 24 to 29 years for high school dropouts versus school completers in the whole population and within the families

	Model 0	Model 1	Model 2	Model 3	Model 4	Model 5
Dropout *versus* completion (ref.)						
Whole population						
Risk difference	20.8 (17.0 to 24.7)	18.7 (15.0 to 22.4)	17.4 (13.8 to 21.1)	16.6 (13.0 to 20.4)	15.8 (12.2 to 19.5)	15.3 (11.7 to 19.0)
Odds ratio	3.92 (3.28 to 4.68)	3.53 (2.95 to 4.24)	3.34 (2.8 to 4.0)	3.20 (2.65 to 3.86)	3.07 (2.54 to 3.71)	2.96 (2.44 to 3.60)
Within family[1]						
Odds ratio	1.89 (0.96 to 3.74)	–	2.03 (1.01 to 4.08)	2.53 (1.15 to 5.54)	2.48 (1.13 to 5.49)	2.39 (1.04 to 5.47)

*Estimated risk difference in the 6-year risk for long-term sickness and disability with the covariates at their mean.
Risk difference (in %, with 95% CI) and odds ratio (with 95% CI) in the whole population (logistic regression models, N=6612) and within the families (sibling fixed-effect models, N=316).
Model 0: adjusted for sex, age, and follow-up time.
Model 1: model 0 +adjusted for maternal education level.
Model 2: model 1 + adjusted for somatic disease, symptom load, psychological distress, concentration problems, insomnia, and self-rated health.
Model 3: model 2 + adjusted for overweight, smoking, and physical activity.
Model 4: model 3 + adjusted for self-esteem, subjective well-being, loneliness, and family living situation.
Model 5: model 4 + adjusted for reading and writing difficulties, bullying, disease-related school absence, educational aspirations, academic problems, school dissatisfaction, and school-related conduct.
[1]In the Within-family models the covariate maternal education level is omitted.

with the risk of receiving disability pension [19,26]. We also found that the association between high school dropout and long-term sickness or disability pension remained strong, even when controlling for a larger variety of adolescent characteristics than in previous studies.

The associations between high school dropout and long-term sickness or disability attenuated, but remained strong when controlling for characteristics shared by the family. A Swedish twin study indicated that the association between educational level and disability pension could be attributed to childhood factors and genetic make-up [27]. However, they combined high school dropouts and completers in the same educational group, although dropouts have substantially higher risks than completers [3,4,19]. Nevertheless, some familial confounding might play an important role in understanding the causes of long-term medical benefits, and we might not have captured all the necessary characteristics related to the family, such as coping behaviours, familial health, and genetics [28-30].

Finally, we are not aware of any study which examines the risk of long-term sickness and disability considering the propensity to drop out of high school based on known risk factors and actual high school graduation status.

Possible interpretations

A high school dropout had systematically a substantial higher risk for long-term sickness and disability, independent of the disadvantage or risk level for dropout that was observed in adolescence. Young adulthood is a stage of the life cycle were people acquire social roles, such as the work role, and school dropout is the first formal registration of own SEP and one's future opportunities in the labour market. Whatever life course history, a school dropout is confronted with reduced work prospects and higher risk for increased job strain, more physical demands, lower self-esteem, and lower sense of coherence [31]. According to the present study's results, the risk of health related exclusion following high school dropout cannot simply be identified by health-related behaviours, parental socioeconomic position, or other risk factors in adolescence. In a life-course approach study, low decision latitude as a young adult was strongly associated with later long term sickness absence, but the effect disappeared when educational attainment and childhood IQ were included in the analyses [32]. One possibility is that school dropouts face an increased risk in a "no exit" situation and are forced into social circumstances

Table 3 Risk difference and odds ratios for long-term sickness or disability with 95% confidence intervals between the ages 24 and 29 years for school dropouts compared with school completers within each stratum of propensity score for dropping out of high school (N=6612)

	N Medical benefits	RD (CI)	OR (CI)
Lowest propensity	79	18.1 (2.3 to 33.9)	4.76 (2.00 to 11.37)
Medium low	120	16.4 (5.8 to 27.1)	3.16 (1.78 to 5.63)
Medium propensity	142	18.7 (10.3 to 27.2)	3.43 (2.20 to 5.33)
Medium high	159	11.0 (4.7 to 17.3)	2.20 (1.50 to 3.23)
Highest propensity	239	19.5 (14.1 to 24.8)	3.11 (2.31 to 4.18)

Risk difference (RD, in %, with 95% CI) and odds ratios (OR, with 95% CI).

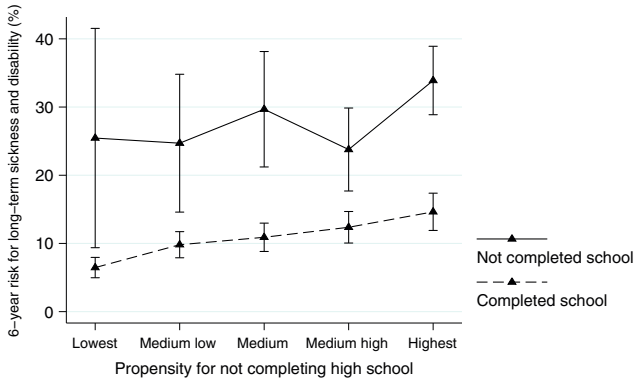


Figure 1 Estimated 6-year risk for long-term sickness or disability with 95% confidence intervals at age 24–29 according to the adolescents' propensity for not completing high school and high school graduation status (N=6612).

that offer no alternative choices. It might also be that they are less able to adapt successfully when they become ill because they lack qualifications and skills which their peers might develop at school or which are necessarily to maintain schooling. For a successful learning process, not only cognitive ability is important. Self-regulation has been shown the most essential asset for the willing to exert considerable effort to learn [33]. In the self-regulation construct, goal level, persistence, effort, and self-efficacy had the strongest effect on learning. Additionally, they might perceive their ability to change their environment and themselves in this environment differently. Personality and coping strategies might affect this perception, and subsequent schooling and labour market integration [34,35]. Finally, in the presence of ill health, there might be an increased risk for medicalization during the social process of school dropout and the possible subsequent reduced work integration, as with job loss and unemployment [36].

Our multivariable adjustments could explain about a quarter of the strong association in the adjusted analyses. Additionally, those with a high propensity to dropout had a higher risk for sickness and disability independent of completing high school or not, which may support the chain of risk model with additive effects [17]. Also the siblings fixed effect analyses showed that there might be some "general susceptibility" related to shared familial factors. Nevertheless, the robust and strong association that remained in all analyses suggests that the mechanisms involved in school dropout and young people's subsequent integration in the labour market should be investigated and focused on in preventive strategies.

Implications

High school dropout is a major public health challenge because it concerns many young people who are in danger of marginalization and social exclusion. Avoiding the main cause and preventing dropout based on a multidisciplinary approach so that children with disadvantages may succeed, should be a public health priority. However, it may be unrealistic to believe that a high school degree is obtainable by everybody. Nonetheless, there should be greater effort towards better integration in high school and in the labour market, including alternative school tracks in cooperation with the labour market and on the job competence-enhancing possibilities. Preferably, these should not be merely B-tracks, but socially accepted and valued alternatives based on learning by doing for those who strive to complete high school.

Conclusions

Even for those born into and raised with good prospects, high school dropout strongly contributes to a problematic or failing of work integration due to impaired health. Future research and preventive measures should pay attention to school and work integration beyond the individual perspective, and include contextual factors in schools and families. It will demand a collaboration of school policies, labour market, public health policies, and research to find sustainable and socially accepted and valued alternatives.

Additional file

Additional file 1: Table A-E and Figure A related to operationalisation of the covariates, description of missing variables, multiple imputation analyses, creation of the propensity score, and matched propensity score analyses.

Abbreviations

BMI: Body mass index; MICE: Multiple imputation by chained equations procedures; SEP: Socioeconomic position; ATT: Average treatment effect on the treated.

Competing interests
The authors declare that they have no competing interests.

Authors' contributions
All authors were involved in the design, contributed to the interpretation of the results, and approved the final version of the article. TLH is PI of the Young-HUNT Study and has been responsible for the Young-HUNT data collection. KDR did the scientific literature review and extracted the data. KDR, KP, and JHB did the statistical analyses, reviewed the results, wrote the manuscript, and revised it following critical review by all authors. All authors take responsibility for the integrity and accuracy of the data analysis and the decision to submit this paper for publication. All authors read and approved the final manuscript.

Acknowledgements
The Nord-Trøndelag Health Study (The HUNT Study) is a collaboration between HUNT Research Centre (Faculty of Medicine, Norwegian University of Science and Technology NTNU), Nord-Trøndelag County Council, Central Norway Health Authority and the Norwegian Institute of Public Health.

Author details
[1]Department of Public Health and General Practice, Norwegian University of Science and Technology, 7491 Trondheim, Norway. [2]Youth Health Care, Department of Public Health and Primary Care, KU Leuven, 3000 Leuven, Belgium. [3]Department of Public Health and Surveillance, Scientific Institute of Public Health, WIV-ISP (Site Elsene), 1050 Brussels, Belgium. [4]Department of Public Health and General Practice, HUNT Research Center, Norwegian University of Science and Technology, 7600 Levanger, Norway. [5]Forensic Department and Research Centre Bröset, St. Olav's University Hospital Trondheim, 7491 Trondheim, Norway.

Received: 30 April 2013 Accepted: 4 October 2013
Published: 9 October 2013

References

1. OECD: *Sickness, disability and work: breaking the barriers. A synthesis of findings across OECD countries.* Paris: OECD Publishing; 2010.
2. Lamb S, Markussen E, Teese R, Polesel J, Sandberg N (Eds): *School dropout and completion: International comparative studies in theory and policy.* Dordrecht: Springer Science+Business Media B.V; 2011.
3. De Ridder KA, Pape K, Johnsen R, Westin S, Holmen TL, Bjorngaard JH: **School dropout: a major public health challenge: a 10-year prospective study on medical and non-medical social insurance benefits in young adulthood, the Young-HUNT 1 Study (Norway).** *J Epidemiol Community Health* 2012, **66**(11):995–1000.
4. Gravseth HM, Bjerkedal T, Irgens LM, Aalen OO, Selmer R, Kristensen P: **Life course determinants for early disability pension: a follow-up of Norwegian men and women born 1967–1976.** *Eur J Epidemiol* 2007, **22**(8):533–543.
5. Graham H, Power C: **Childhood disadvantage and health inequalities: a framework for policy based on lifecourse research.** *Child Care Health Dev* 2004, **30**(6):671–678.
6. Koivusilta LK, Rimpela AH, Rimpela M, Vikat A: **Health behavior-based selection into educational tracks starts in early adolescence.** *Health Educ Res* 2001, **16**(2):201–214.
7. Huurre T, Aro H, Rahkonen O, Komulainen E: **Health, lifestyle, family and school factors in adolescence: predicting adult educational level.** *Educ Res* 2006, **48**(1):41–53.
8. Rothon C, Head J, Klineberg E, Stansfeld S: **Can social support protect bullied adolescents from adverse outcomes? A prospective study on the effects of bullying on the educational achievement and mental health of adolescents at secondary schools in East London.** *J Adolesc* 2011, **34**(3):579–588.
9. Winding TN, Nohr EA, Labriola M, Biering K, Andersen JH: **Personal predictors of educational attainment after compulsory school: influence of measures of vulnerability, health, and school performance.** *Scand J Public Health* 2013, **41**(1):92–101.
10. Power C, Matthews S: **Origins of health inequalities in a national population sample.** *Lancet* 1997, **350**(9091):1584–1589.
11. Power C, Atherton K, Strachan DP, Shepherd P, Fuller E, Davis A, Gibb I, Kumari M, Lowe G, Macfarlane GJ, *et al*: **Life-course influences on health in British adults: effects of socio-economic position in childhood and adulthood.** *Int J Epidemiol* 2007, **36**(3):532–539.
12. Henderson M, Stansfeld S, Hotopf M: **Self-rated health and later receipt of work-related benefits: evidence from the 1970 British Cohort Study.** *Psychol Med* 2013, **43**(8):1755–1762.
13. Pape K, Bjorngaard JH, Westin S, Holmen TL, Krokstad S: **Reading and writing difficulties in adolescence and later risk of welfare dependence. A ten year follow-up, the HUNT Study, Norway.** *BMC Public Health* 2011, **11**:718.
14. Pape K, Bjørngaard JH, Holmen TL, Krokstad S: **The welfare burden of adolescent anxiety and depression: a prospective study of 7500 young Norwegians and their families: the HUNT study.** *BMJ Open* 2012, **2**(6):e001942.
15. Neovius K, Neovius M, Rasmussen F: **The combined effects of overweight and smoking in late adolescence on subsequent disability pension: a nationwide cohort study.** *Int J Obes* 2009, **34**(1):75–82.
16. De Ridder KAA, Pape K, Johnsen R, Holmen TL, Westin S, Bjørngaard JH: **Adolescent health and high school dropout: a prospective cohort study of 9000 Norwegian adolescents (The Young-HUNT).** *PLoS One.* **8**(9):e74954.
17. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C: **Life course epidemiology.** *J Epidemiol Community Health* 2003, **57**(10):778–783.
18. Najman JM: **Commentary: general or cause-specific factors in explanations of class inequalities in health.** *Int J Epidemiol* 2001, **30**(2):296–297.
19. Johansson E, Leijon O, Falkstedt D, Farah A, Hemmingsson T: **Educational differences in disability pension among Swedish middle-aged men: role of factors in late adolescence and work characteristics in adulthood.** *J Epidemiol Community Health* 2012, **66**(10):901–907.
20. Holmen TL, Bratberg G, Krokstad S, Langhammer A, Hveem K, Midthjell K, Heggland J, Holmen J: **Cohort profile of the young-HUNT study, norway: a population-based study of adolescents.** *Int J Epidemiol* 2013. doi:10.1093/ije/dys232. Feb 4. (Epub ahead of print).
21. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH: **Establishing a standard definition for child overweight and obesity worldwide: international survey.** *BMJ* 2000, **320**(7244):1240–1243.
22. White IR, Royston P, Wood AM: **Multiple imputation using chained equations: issues and guidance for practice.** *Stat Med* 2011, **30**(4):377–399.
23. Donovan SJ, Susser E: **Commentary: advent of sibling designs.** *Int J Epidemiol* 2011, **40**(2):345–349.
24. Katz MH: **Methods for adjusting for baseline differences between treatment groups**. In *Evaluating clinical and public health interventions: a practical guide to study design and statistics.* 1st edition. Edited by Katz MH. Cambridge: Cambridge University Press; 2010:101–115.
25. Becker SO, Ichino A: **Estimation of average treatment effects based on propensity scores.** *Stata J* 2002, **2**(4):358–377.
26. Gravseth HM, Bjerkedal T, Irgens LM, Aalen OO, Selmer R, Kristensen P: **Influence of physical, mental and intellectual development on disability in young Norwegian men.** *Eur J Public Health* 2008, **18**(6):650–655.
27. Samuelsson A, Alexanderson K, Ropponen A, Lichtenstein P, Svedberg P: **Incidence of disability pension and associations with socio-demographic factors in a Swedish twin cohort.** *Soc Psychiatry Psychiatr Epidemiol* 2012, **47**(12):1999–2009.
28. Suominen S, Gould R, Ahvenainen J, Vahtera J, Uutela A, Koskenvuo M: **Sense of coherence and disability pensions. A nationwide, register based prospective population study of 2196 adult Finns.** *J Epidemiol Community Health* 2005, **59**(6):455–459.
29. Narusyte J, Ropponen A, Silventoinen K, Alexanderson K, Kaprio J, Samuelsson A, Svedberg P: **Genetic liability to disability pension in women and men: a prospective population-based twin study.** *PLoS One* 2011, **6**(8):e23143.
30. Pape K, Bjørngaard JH, De Ridder KAA, Westin S, Holmen TL, Krokstad S: **Medical benefits in young Norwegians and their parents, and the contribution of family health and socioeconomic status. The HUNT Study, Norway.** *Scand J Public Health* 2013, **41**(5):455–462.
31. Volanen S-M, Lahelma E, Silventoinen K, Suominen S: **Factors contributing to sense of coherence among men and women.** *Eur J Public Health* 2004, **14**(3):322–330.
32. Henderson M, Richards M, Stansfeld S, Hotopf M: **The association between childhood cognitive ability and adult long-term sickness absence in three British birth cohorts: a cohort study.** *BMJ Open* 2012, **2**(2):e000777.

33. Sitzmann T, Ely K: **A meta-analysis of self-regulated learning in work-related training and educational attainment: what we know and where we need to go.** *Psychol Bull* 2011, **137**(3):421–442.
34. Christensen U, Schmidt L, Kriegbaum M, Hougaard CØ, Holstein BE: **Coping with unemployment: does educational attainment make any difference?** *Scand J Public Health* 2006, **34**(4):363–370.
35. Ackerman PL, Chamorro-Premuzic T, Furnham A: **Trait complexes and academic achievement: Old and new ways of examining personality in educational contexts.** *Br J Educ Psychol* 2011, **81**(1):27–40.
36. Westin S, Schlesselman JJ, Korper M: **Long-term effects of a factory closure: unemployment and disability during ten years' follow-up.** *J Clin Epidemiol* 1989, **42**(5):435–441.

doi:10.1186/1471-2458-13-941
Cite this article as: De Ridder *et al.*: High school dropout and long-term sickness and disability in young adulthood: a prospective propensity score stratified cohort study (the Young-HUNT study). *BMC Public Health* 2013 **13**:941.

Additional file provided with Paper III

Additional file 1: 133 KB

Table A-E and Figure A related to operationalisation of the covariates, description of missing variables, multiple imputation analyses, creation of the propensity score, and matched propensity score analyses.

http://www.biomedcentral.com/1471-2458/13/941/additional

Appendix

Table A. Operationalisation of the covariates

Chronic somatic disease	Self-reported asthma, diabetes, migraine, or epilepsy diagnosed by a doctor or any other illness that lasted longer than 3 months. Dichotomized into "yes" or "no".
Symptom load	The sum of self-reported presence (dichotomized into "never/seldom" and "sometimes/often") of eight symptoms (headache, neck or shoulder pain, joint or muscle pain, stomach pain, nausea, constipation, diarrhea, heart palpitations) during the last 12 months. Range 0 (no symptoms) to 8 (8 symptoms)
Psychological distress	A mean score based on self-reported symptoms of anxiety and depression measured by SCL-5 scale score – a validated 4-integer 5 item short version of the original SCL-90*. Continuous variable with range 1 (low) to 4 (high psychological distress). (*Hopkins symptom checklist)
Concentration problems	Self-reported difficulties concentrating during class and dichotomized into "never/sometimes" versus "often/ very often".
Self-rated health	Self-rated health using the question "How is your health at the moment?" and dichotomized into "good/very good" versus "poor/not so good".
Insomnia	Difficulties falling asleep in the last month and dichotomized into "never/sometimes" versus "often/ almost every night"
BMI	Overweight corresponded with the adult BMI from 25 to 30 and obesity a BMI of 30 and more.
Smoking	Smokers are those who answered "Yes" to the question about ever smoking combined with answering "Yes, I smoke daily" or "Yes, I smoke occasionally" to the question "Do you smoke now?" Non- smokers are those who had never smoked cigarettes or had stopped smoking.
Physical activity	Self-reported activity about if they exercise until they get out of breath or sweat outside school hours was dichotomized into inactive (less than every 14^{th} day) or active (more than every 14^{th} day).
Self-esteem	A mean score based on a short version of the Rosenberg Self-Esteem Scale consisting of 4-integer 4 item version. Range 1 (low) to 4 (high self-esteem).
Subjective well-being	A mean score based on 3 questions with each a 7-point scale from very satisfied to very dissatisfied. Range 1 (high) to 7 (low subjective well-being).
Loneliness	The question "Do you feel lonely?" with a 5-point scale from "very often" to "very seldom or never".
Family living situation	Living in a traditional family (with both the biological mother and father) or not.
Reading and writing difficulties	Defined as responding "yes" to the question "Do you currently receive help for reading or writing problems?" or reporting major problems with either reading or writing the last 12 months.
Being bullied	Response on the statement "You are teased/harassed by other students" with a 4-point scale from "never" to "very often".
Educational aspirations	Based on the question "What type of plans do you have regarding continued studies?" categorized into 3 groups ("none/do not know", "high school" and "higher education")
Disease-related absence from school	Reporting more than 2 weeks absence from school due to illness during the last 12 months.
Academic problems	A mean score based on self-reported problems related to academic achievement based on 5 questions with a 4-point scale. Range 1 (never) to 4 (very often).
School-related dissatisfaction	A mean score based on self-reported problems related to dissatisfaction at school based on 4 questions with a 4-point scale. Range 1 (never) to 5 (very often).
School-related conduct	A mean score based on self-reported school-related conduct problems based on 4 questions with a 4-point scale. Range 1 (never) to 5 (very often).

APPENDIX

Table B. Number of missing variables (% in parenthesis) in the dataset (N=8816) for the whole population and stratified by the variable "school dropout".

	Total n=8816	School dropouts n=1474	School completers n=7342
Demographics			
Age, mean	0 (0)	0 (0)	0 (0)
Male	0 (0)	0 (0)	0 (0)
Maternal education level	39 (0.4)	20 (1.4)	19 (0.3)
Health			
1 or more somatic disease	0 (0)	0 (0)	0 (0)
Symptom load, mean	314 (3.6)	80 (5.4)	234 (3.2)
High psychological distress	169 (1.9)	68 (4.6)	101 (1.4)
Concentration problems	180 (2.0)	69 (4.6)	111 (1.5)
Insomnia	132 (1.5)	52 (3.5)	80 (1.1)
Poor self-rated health	148 (1.7)	35 (2.4)	113 (1.5)
Health behavior			
BMI	545 (6.2)	168 (11.4)	377 (5.1)
Smoking	208 (2.4)	50 (3.4)	158 (2.2)
No physical activity	116 (1.3)	37 (2.6)	79 (1.1)
Psychosocial factors			
Self-esteem	219 (2.5)	81 (5.5)	138 (1.9)
Subjective well-being	133 (1.5)	50 (3.4)	83 (1.1)
Loneliness	137 (1.5)	54 (3.7)	83 (1.1)
Traditional family	128 (1.5)	34 (2.3)	94 (1.3)
School-related factors			
Reading and writing difficulties	307 (3.5)	103 (7.0)	202 (2.8)
Being bullied	224 (2.5)	83 (5.6)	141 (1.9)
Disease-related school absence	522 (5.9)	180 (12.2)	342 (4.7)
Aspiration for higher education	306 (3.5)	75 (5.1)	231 (3.1)
Academic problems	360 (4.1)	117 (7.9)	243 (3.3)
School-related dissatisfaction	454 (5.1)	141 (9.6)	313 (4.3)
School-related conduct	325 (3.7)	102 (7.0)	223 (3.0)

APPENDIX

Table C. Odds ratio of receiving long-term medical benefits between age 24 to 29 years for high school dropouts compared to school completers in the whole population for complete cases analysis (logistic regression models, N=6607) and analysis based on multiple imputations (logistic regression models, N=8805).

	Model 0	Model 1	Model 2	Model 3	Model 4	Model 5
dropout *versus* completion (ref.)						
Complete cases						
Odds ratio	3.93	3.54	3.36	3.21	3.08	2.96
	(3.29 to 4.70)	(2.95 to 4.25)	(2.80 to 4.05)	(2.66 to 3.87)	(2.55 to 3.73)	(2.44 to 3.59)
Multiple imputation						
Odds ratio	3.88	3.57	3.35	3.12	2.94	2.74
	(3.37 to 4.47)	(3.09 to 4.13)	(2.90 to 3.88)	(2.67 to 3.65)	(2.52 to 3.45)	(2.32 to 3.22)

*Estimated risk difference in the 6-year risk to receive long-term medical benefits with the covariates at their mean.

Model 0: adjusted for sex, age and follow-up time

Model 1: model 0 +adjusted for maternal education level.

Model 2: model 1 + adjusted for somatic disease, symptom load, psychological distress, concentration problems, insomnia, and self-rated health.

Model 3: model 2 + adjusted for overweight, smoking, and physical activity.

Model 4: model 3 + adjusted for self-esteem, subjective well-being, loneliness, and family living situation.

Model 5: model 4 + adjusted for reading and writing difficulties, bullying, disease-related school absence, educational aspirations, school dissatisfaction, and school-related conduct.

Multiple imputation procedure:

Assumption: missing at random

Basically, the fraction of missing data for each variable was low and varied from 0% to 6.2%. 83% of the participants with missing data had missing data for only 1 or 2 variables.
The variable with most missing data was “BMI”, which was based on the measurement of height and weight by nurses. 388 of the participants with missing information on BMI (71%) had a full completed questionnaire (no other missing data) . It is unlikely that they could not meet up or be measured because of their BMI itself. It could be possible that they were less willing to meet up because of a psychological reluctancy, but we included several psychological measurements in the dataset that can predict the probability of missingness (psychological distress, self-esteem, subjective well-being) and the incomplete variable. It could also be possible that a group of individuals are less dutyful, and in that case the variable “school dropout” might (as a proxy) predict the probability of missingness and the incomplete variable. Finally, some participants were maybe not able to meet up, completely at random: e.g. had to catch the buss, had to be in another class/examination, and so on.

Variable selection and imputation model

Multiple imputation with chained equations with

- *Imputing binary variables with logistic regression (logit):*

Symptom load, psychological distress, insomnia, concentration problems, self-reported health, smoking, physical activity, self-esteem, subjective well-being, lonileness, family living situation, reading and writing difficulties, being bullied, school-related dissatisfaction, school-related conduct problems, disease-related school absence

- *Imputing ordered categorical variables with ordinal logistic regression (ologit):*

Maternal education level, educational aspiration, BMI

- *Using outcome, auxiliary outcomes and other not imputed variables:*

Medical benefits between age 24-29, unemployment between age 24-29, social assistance between age 24-29, age, sex, somatic disease, follow-up time, linkage between siblings

- *Variable of complete case analysis not included in multiple imputation:*

Variable "academic problems" was omitted because of too much collinearity with "concentration problems".

Note 1: No interactions were included because the complete case analyses showed no statistical significant interactions.
Note 2: Because of practical difficulties, all imputed variables were included as either binary or ordinal categorical variables.

Number of imputations and estimates: The 20 created datasets were individually but identically analyzed. The estimates were combined to obtain overall estimates and confidence intervals.

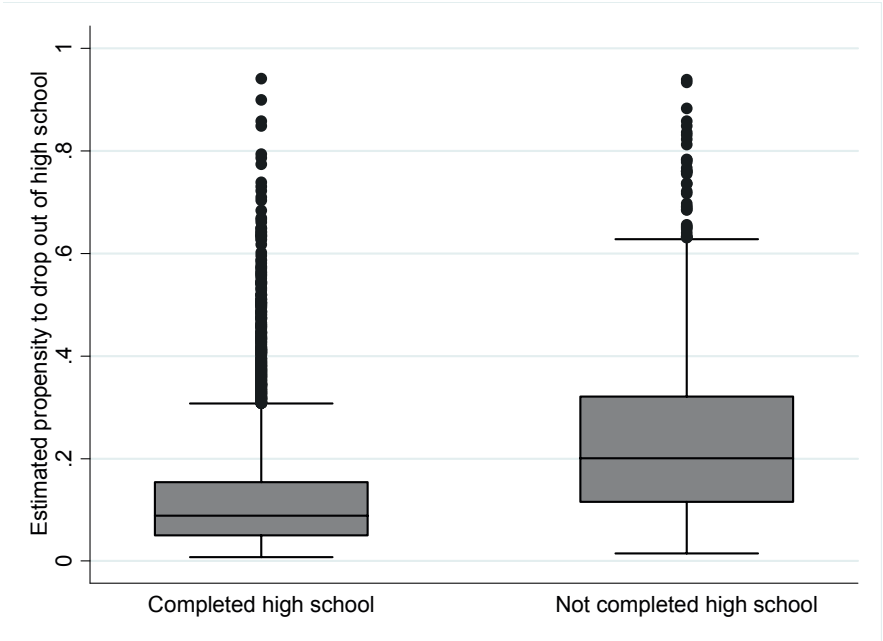
APPENDIX

Table D. Log odds ratios of high school dropout and standard errors for the variables included in the propensity analysis. (N=6612)

	Coefficient	Standard error	95% confidence interval		P-value
			Lower	Upper	
Sex	0.573	0.086	0.404	0.742	<0.0001
Age	-0.136	0.024	-0.183	-0.089	<0.0001
Maternal education level					
Intermediate	-0.474	0.084	-0.639	-0.310	<0.0001
Tertiary	-1.114	0.123	-1.356	-0.874	<0.0001
Chronic somatic disease	0.104	0.092	-0.077	0.280	0.26
Symptom load	0.028	0.029	-0.028	0.085	0.32
Psychological distress	-0.119	0.103	-0.321	0.083	0.25
Concentration problems	-0.182	0.109	-0.396	0.032	0.09
Insomnia	0.202	0.123	-0.403	0.445	0.10
Self-rated health	0.035	0.124	-0.202	0.271	0.77
BMI					
Overweight	0.291	0.102	0.090	0.491	0.005
Obese	0.621	0.188	0.253	0.989	0.001
Smoking	0.490	0.092	0.310	0.670	<0.0001
Physical activity	0.418	0.106	0.209	0.626	<0.0001
Self-esteem	-0.102	0.090	-0.278	0.075	0.26
Subjective wellbeing	-0.076	0.057	-0.188	0.037	0.18
Loneliness	-0.043	0.050	-0.140	0.054	0.38
Family living situation	-0.645	0.081	-0.805	-0.486	<0.0001
Reading and writing difficulties	0.697	0.117	0.466	0.928	<0.0001
Being bullied	0.050	0.081	-0.110	0.209	0.54
Educational aspirations	-0.090	0.047	-0.183	0.003	0.06
Disease-related absence from school	0.600	0.152	0.301	0.898	<0.0001
Academic problems	1.356	0.139	1.082	1.630	<0.0001
School-related dissatisfaction	-0.127	0.089	-0.302	0.048	0.16
School-related conduct	-0.073	0.099	-0.267	0.120	0.45
Constant	-0.967	0.551	-2.047	0.112	0.08

APPENDIX

Figure A. Comparison of box plots of propensity score for likelihood of dropping out of school.



APPENDIX

Table E. Average effects of treatment on treated (ATT) with and without restriction of the region of common support, including bootstrapped propensity scores and standard error (SE) with 100 replications of the ATT.

Matching method	School dropouts, n	School completers, n	ATT	SE	95% CI[a] for ATT
Region of common support: propensity score 0.0163-0.9392 (n=6550)					
Radius (0.1)	910	5640	0.165	0.015	0.136-0.194
Region of common support not imposed: propensity score 0.0081-0.9414 (n=6612)					
Radius (0.1)	910	5702	0.165	0.015	0.136-0.194

[a]Confidence interval