Introduction

The Black Report \(^1\) became a turning point in understanding social inequalities in health. Subsequent research has led to further development of concepts and considerable empirical evidence.\(^2\) This research has added to our understanding of social causation and the influence of the environment (life-style, material conditions, psycho-social factors and social capital).\(^3,4\) One major contribution is the concept of life course influence—how biological and social factors operating at different stages of life contribute to inequalities in adult health.\(^3,4\) Empirical evidence suggests that health selection is not an important cause of later socio-economic position (SEP).\(^5\) However, the life course concept has led to renewed interest in indirect selection.\(^6,7\) Indirect selection is a mechanism involving early factors that have a potential to affect social inequalities in health because they are simultaneously involved in status attainment processes and in disease development.\(^5,8\) These factors are not involved in direct causal relations between health and SEP, but third variables responsible for their co-variation: personality, cognitive ability, and bodily and mental fitness.\(^6\) In a life course perspective, there could be a continuous feedback between such third factors and SEP, rendering disentangling of selection and causal mechanisms difficult.\(^6,7,9\)

The ‘chain of risk’ is one causal model that has been proposed to elucidate the effects of life course factors on adult SEP and later health.\(^3\) This model could be applied in the study of indirect selection and social inequalities in health. In its simplest form, early life factors could influence health in adult life by mediation via adult SEP or through other paths. The adult SEP gradient in health would then be influenced by the strength of associations between early factors (e.g. social class of origin or cognitive ability) and adult SEP, and the strength of associations between early factors and subsequent health. Interpreting social health gradients as only due to social causation would then be incorrect because these early factors would act as confounders.

Social inequalities in mental health have been documented for a number of outcomes including suicide,\(^10–14\) major psychiatric morbidity\(^15–19\) and common mental disorders.\(^19–24\) Also early life factors including parental SEP, personality
factors and intellectual capacity have been associated with psychiatric outcomes \cite{21,22,23,24,25} but results are not consistent \cite{26,27}.

In contrast, research investigating influences of early social and biological factors on adult social gradients in health is scarce. One example relating to mental health is reported in the British National Child Development Study; prevalences of psychological distress at age 33 years showed clear social gradients, ranging from 7\% (occupational classes I and II) to 19\% (IV and V) for women and from 4\% to 12\% for men \cite{28}. In both genders, inequalities were substantially reduced when early factors were included in the regression models: school qualifications, cognitive ability, school behaviour, parental aspirations and interest, and social class at birth \cite{29}. This suggests that social inequalities in adult psychological distress are partly accounted for by these early factors.

We have established a register-based cohort of all live born in Norway 1967–76, which includes repeated measures of social and biological circumstances and health recorded chronologically throughout life \cite{30,31}. Associations between life course factors and subsequent suicide \cite{32}, educational attainment \cite{33} and disability pension \cite{34} have been reported. In the present study, we followed up men born in 1967–71. The study was restricted to men because we included conscript data that were not available for women. The study objectives were to examine social gradients in selected mental health outcomes in young adulthood and to estimate to what degree these gradients were dependent on parental and individual characteristics in early life.

### Methods

#### Participants

The study population included all 170 678 male live births registered in the Medical Birth Registry of Norway, 1967–71. The national identification numbers of child and parents allowed linkage with national registers: the Central Population Register, Cause of Death Register and Education Register of Statistics Norway; the Armed Forces Personnel Data Base; the benefit, income and sickness absence registries of the Norwegian Labour and Welfare Administration.

Inclusion criteria varied for the different study outcomes (table 1). For suicide all 160 914 residents with education level data and who were alive at the end of the calendar year of their 28th birthday participated. In the disability part, we excluded in addition all who received disability pension before start of follow-up, leaving 158 484 for analysis. For sickness absence, we included 126 124 men who were considered to be at risk on 1st January 2000 (exclusion categories provided in table 1).

#### Study variables

Linkages provided data that mostly were updated regularly, e.g. vital status, residence, marital status, education, pension benefit, income and sickness absence.

Study outcomes were derived from data in the Cause of Death Register (suicide), and the National Insurance Administration (disability pension and sickness absence). Suicide was defined as death with ICD-10 diagnosis X60–X84 between the calendar year of the 29th birthday and 2004. Psychiatric disability was based on ICD-10 diagnosis F00–F99 between the calendar year of the 29th birthday and 2003. Disability subgroups were schizophrenia (F20–F29), and neurosis and personality disorders (F40–F49, F60–F69). Psychiatric sickness absence was registered if the first medically certified spell between 2000 and 2003 with duration of >16 days was a group P diagnosis (International Classification of Primary Care).

Education level at the age of 28 years served as indicator of adult SEP. We used three measures on basis of the nine-level variable in the Education Register: \cite{35} number of years of education (between 0 and 20) as a continuous variable according to the Statistics Norway standard \cite{36} and ordered variables with five or three levels, the latter used when needed to achieve robust results.

We considered two sets of early factors: parental characteristics and individual characteristics. Register data for parents were based on their identity as indicated in the birth record (father’s identity missing for 4.5\% of the participants). Parental education level was an exception, because data were retrieved from the participant’s record in the Education Register. Accordingly, missing data on father’s education were considerably fewer than for other paternal characteristics. Mother’s and father’s education levels at participant age 16 years were collapsed into five levels as for the participants. Parental education level in three categories, classified according to the parent with highest educational attainment, was used in some analyses. Mother’s and father’s income levels at index age 16 years were divided into four categories. Four categories of mother’s marital status were used according to her status at the participant’s birth and age 18 years: married or unmarried on both occasions, divorced or separated on either occasion or other. Mother’s total number of children was recorded. Mother and father were classified as disabled or dead if the event occurred before the son was 18 years of age. Individual characteristics included birth-weight, chronic childhood disease and conscript variables. Birth-weight in singletons was grouped into four categories based on Z-scores; multiple births were allocated a separate category. Childhood disease was defined as receiving basic and/or attendance benefit because of chronic disease before the age

### Table 1: Study populations followed up for suicide, psychiatric disability, and psychiatric sickness absence, among men born in Norway in 1967–71

<table>
<thead>
<tr>
<th>Category (person-years)</th>
<th>Follow-up period</th>
<th>Numbers</th>
<th>Excluded (total percentage)</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suicide follow-up (1 113 758 years)</td>
<td>1996/2000-04*</td>
<td>170 678</td>
<td>9764 (5.7%)</td>
<td>Missing education data at age 28, death or emigration before the end of the year of the 28th birthday</td>
</tr>
<tr>
<td>Disability follow-up (935 194 person-years)</td>
<td>1996/2000-03*</td>
<td>158 484</td>
<td>2430 (1.4%)</td>
<td>Receiving disability pension before the end of the year of the 28th birthday</td>
</tr>
<tr>
<td>Sickness absence follow-up (425 443 person-years)</td>
<td>2000-03</td>
<td>126 124</td>
<td>32 360 (19.0%)</td>
<td>Death or emigration before 2000; under education, receiving disability pension, or having an absence spell on 1st January 2000; no income(^a) in 2000</td>
</tr>
</tbody>
</table>

\(^a\): Start of follow-up in the calendar year of the 29th birthday

\(^b\): Pensionable income below the limit that entitles to sickness allowance
of 17 years. The Armed Forces Personnel Data Base provided several conscript data. Men drafted for compulsory military service are obliged to complete several examinations at age 18 years, including a test of general ability. General ability is highly correlated with the Wechsler Adult Intelligence Scale and is recorded as stanine scores, i.e. single digits from 1 (low) to 9 (high). Scores are normally distributed with mean 5 and SD 2 in the general population. Mental and physical functions were assessed by the conscription board physician and classified into ordered categories. Body mass index defined as weight divided by height (kilogram per squared metres) and body height were categorized. Military duty was classified as completed, drafted but not completed and not drafted. Four background factors were included in the regression models: year of birth (five categories), mother’s age at the son’s birth (five categories), birth order (five categories) and type of municipality at participant age 16 years (seven categories). Details of the independent variables and their categories are provided in the Supplementary table 1.

### Statistical analysis

We used Stata/SE 10.1 software. Men were followed up from 1st January of the year of their 29th birthday (suicide, disability) or 1st January 2000 (sickness absence). Follow-up continued through 2004 (suicide) or 2003 (disability, sickness absence), or date of death, emigration or study outcome, whichever came first. Suicide, disability and sickness absence were estimated as mortality or incidence rates per 100 000 person-years. We computed mean educational years as well as health outcome rates within strata of the independent variables. Social inequalities in health were based on time until the outcome event and estimation of hazard ratio (HR) in Cox proportional hazard regression. Adult SEP was included in the analysis as a continuous variable (years of education) resulting in HR estimates associated with a 1-year increment in education. We applied serial regression models as suggested by Baron and Kenny in order to estimate the impact of parental and individual characteristics on social inequalities in health. Variance estimates of the regression coefficients were corrected for dependencies between brothers by using the cluster option in STATA with the mother’s identity as cluster variable. Ninety-five percent confidence intervals (CI) were computed for all associations. Throughout, missing values were included in the regression models as separate categories. The Cox regression results were assessed for proportionally with Stata’s stphplot option.

### Ethical Approval

The Regional Committee for Medical Research Ethics approved the study [reference number S-06028].

### Results

During follow-up, we registered 243 suicides (rate 21.8), 1363 psychiatric disabilities (rate 145.7) with subgroups schizophrenia (326 cases, rate 34.9) and neurosis and personality disorders (342 cases, rate 58.0). Psychiatric sickness absence counted 4955 first spells (rate 1164.7).

All the early factors under study were associated with educational attainment at the age of 28 years as well as with the psychiatric outcomes (Supplementary table 1). Most of the relations were moderate except for both parents’ education level and general ability. The early factors were more strongly related to all psychiatric disability, neurosis and personality disability and sickness absence, than with suicide and schizophrenia disability rates.

Social inequalities in health are presented as crude suicide, disability and absence rates across levels of educational attainment in table 2. There were consistent and strong gradients with increasing rates for lower education levels. The rates in the lowest compared with the highest levels were >5-fold increased for suicide and sickness absence, and were considerably steeper for the disability categories. The largest rate differences were found between basic upper secondary level (11 years) and completed upper secondary level (12–13 years) for all outcomes.

The impact of early factors on SEP gradients in health is presented in the four regression models in table 3. Early factors together (Model 4) explained >40% of the social gradients in all psychiatric disability and the neurosis/personality subgroup, with individual factors counting more than parental factors. The impact of early factors was more moderate for suicide, schizophrenia disability and sickness absence, individual factors having largest impact. In fact, the Model 2 estimates including parental characteristics were further away from the null than Model 1 for both suicide and schizophrenia disability.

### Table 2 Social inequalities in health: crude incidence rates of suicide, psychiatric disability and psychiatric sickness absence according to education level at the age of 28 years, among men born in Norway in 1967–71

<table>
<thead>
<tr>
<th>Socio-economic position (education level in years)</th>
<th>Number</th>
<th>Percent</th>
<th>Rates per 100 000 person-years (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tertiary, long (18–20 years)</td>
<td>10833</td>
<td>6.7</td>
<td>6.8 (0.9–12.8) 6.3 (3.3–9.4) 4.8 (2.1–7.4) 0.0 (0.0–3.7) 335.8 (242.9–428.6)</td>
</tr>
<tr>
<td>Tertiary, short (15 years)</td>
<td>35190</td>
<td>21.9</td>
<td>12.1 (8.9–15.3) 52.7 (45.3–60.1) 19.5 (14.3–24.7) 16.6 (12.7–20.5) 623.6 (546.6–700.7)</td>
</tr>
<tr>
<td>Upper secondary, complete (12–13 years)</td>
<td>64052</td>
<td>39.8</td>
<td>19.4 (16.2–22.6) 82.8 (75.9–89.6) 26.8 (22.7–30.9) 29.4 (25.5–33.4) 1102.0 (1024.1–1179.9)</td>
</tr>
<tr>
<td>Upper secondary, basic (11 years)</td>
<td>37574</td>
<td>23.4</td>
<td>33.3 (28.1–38.5) 305.4 (286.7–324.1) 62.0 (53.8–70.7) 127.7 (116.0–139.5) 1870.6 (1707.7–2033.6)</td>
</tr>
<tr>
<td>Lower secondary or less (0–9 years)</td>
<td>13265</td>
<td>8.2</td>
<td>37.7 (27.0–48.4) 375.3 (342.4–408.2) 64.7 (51.5–78.0) 162.5 (143.0–182.1) 2160.0 (1859.7–2460.2)</td>
</tr>
</tbody>
</table>
Table 3 HR of suicide, psychiatric disability and psychiatric sickness absence, among men born in Norway in 1967–71, in association with education level (years of education) at age 28

<table>
<thead>
<tr>
<th>Category</th>
<th>Model 1&lt;sup&gt;a&lt;/sup&gt; HR (95% CI)</th>
<th>Model 2&lt;sup&gt;b&lt;/sup&gt; HR (95% CI)</th>
<th>Model 3&lt;sup&gt;c&lt;/sup&gt; HR (95% CI)</th>
<th>Model 4&lt;sup&gt;d&lt;/sup&gt; HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suicide</td>
<td>0.815 (0.760–0.873)</td>
<td>0.804 (0.745–0.867)</td>
<td>0.855 (0.791–0.925)</td>
<td>0.847 (0.780–0.920)</td>
</tr>
<tr>
<td>Percent change from Model 1</td>
<td>+5.9%</td>
<td>+22.0%</td>
<td>-17.4%</td>
<td></td>
</tr>
<tr>
<td>All psychiatric disability</td>
<td>0.668 (0.648–0.690)</td>
<td>0.692 (0.670–0.715)</td>
<td>0.803 (0.777–0.831)</td>
<td>0.808 (0.781–0.836)</td>
</tr>
<tr>
<td>Percent change from Model 1</td>
<td>-7.2%</td>
<td>-40.7%</td>
<td>-42.1%</td>
<td></td>
</tr>
<tr>
<td>Disability, schizophrenia</td>
<td>0.768 (0.724–0.814)</td>
<td>0.741 (0.697–0.788)</td>
<td>0.845 (0.789–0.905)</td>
<td>0.816 (0.761–0.874)</td>
</tr>
<tr>
<td>Percent change from Model 1</td>
<td>+11.6%</td>
<td>-33.2%</td>
<td>-20.7%</td>
<td></td>
</tr>
<tr>
<td>Psychiatric sickness absence</td>
<td>0.632 (0.601–0.664)</td>
<td>0.679 (0.644–0.715)</td>
<td>0.769 (0.729–0.811)</td>
<td>0.794 (0.751–0.840)</td>
</tr>
<tr>
<td>Percent change from Model 1</td>
<td>-12.7%</td>
<td>-37.2%</td>
<td>-44.1%</td>
<td></td>
</tr>
<tr>
<td>All psychiatric disability</td>
<td>0.805 (0.792–0.817)</td>
<td>0.822 (0.809–0.836)</td>
<td>0.843 (0.829–0.858)</td>
<td>0.855 (0.840–0.870)</td>
</tr>
<tr>
<td>Percent change from Model 1</td>
<td>-9.1%</td>
<td>-19.6%</td>
<td>-25.6%</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Model 1 adjusted for background factors (year of birth, mother’s age at birth, birth order, type of municipality)
<sup>b</sup> Model 2 adjusted for background factors and parental characteristics (mother’s and father’s education, mother’s and father’s income, mother’s marital status, mother’s number of children, mother’s and father’s disability, mother’s and father’s vital status)
<sup>c</sup> Model 3 adjusted for background factors and individual characteristics (birth-weight, chronic childhood disease, conscript measures (general ability, mental function, physical function, body mass index, height), military duty completion)
<sup>d</sup> Model 4 adjusted for background factors, parental characteristics and individual characteristics

Table 4 HR of suicide and disability from schizophrenia, among men born in Norway in 1967–71, according to parental and own education level

<table>
<thead>
<tr>
<th>Education level&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Suicide Cases</th>
<th>Rate&lt;sup&gt;b&lt;/sup&gt;</th>
<th>HR&lt;sup&gt;c&lt;/sup&gt; (95% CI)</th>
<th>Disability, schizophrenia Cases</th>
<th>Rate&lt;sup&gt;b&lt;/sup&gt;</th>
<th>HR&lt;sup&gt;c&lt;/sup&gt; (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parental high, own high</td>
<td>12</td>
<td>8.7</td>
<td>1 (Reference)</td>
<td>26</td>
<td>22.2</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>Parental medium, own high</td>
<td>10</td>
<td>16.5</td>
<td>2.3 (1.0–5.2)</td>
<td>7</td>
<td>13.5</td>
<td>0.6 (0.3–1.3)</td>
</tr>
<tr>
<td>Parental low, own high</td>
<td>12</td>
<td>10.5</td>
<td>1.5 (0.7–3.3)</td>
<td>10</td>
<td>10.2</td>
<td>0.4 (0.2–0.9)</td>
</tr>
<tr>
<td>Parental high, own medium</td>
<td>15</td>
<td>21.3</td>
<td>2.8 (1.3–6.1)</td>
<td>21</td>
<td>35.1</td>
<td>1.4 (0.7–2.5)</td>
</tr>
<tr>
<td>Parental medium, own medium</td>
<td>20</td>
<td>22.8</td>
<td>3.2 (1.5–6.7)</td>
<td>23</td>
<td>30.8</td>
<td>1.1 (0.6–2.0)</td>
</tr>
<tr>
<td>Parental low, own medium</td>
<td>51</td>
<td>17.9</td>
<td>2.5 (1.3–5.0)</td>
<td>57</td>
<td>23.6</td>
<td>0.8 (0.5–1.3)</td>
</tr>
<tr>
<td>Parental high, own low</td>
<td>19</td>
<td>64.1</td>
<td>6.3 (3.0–13.4)</td>
<td>36</td>
<td>153.3</td>
<td>4.0 (2.3–7.1)</td>
</tr>
<tr>
<td>Parental medium, own low</td>
<td>17</td>
<td>33.2</td>
<td>3.4 (1.5–7.6)</td>
<td>34</td>
<td>82.1</td>
<td>1.9 (1.1–3.5)</td>
</tr>
<tr>
<td>Parental low, own low</td>
<td>87</td>
<td>31.6</td>
<td>3.2 (1.6–6.5)</td>
<td>112</td>
<td>49.9</td>
<td>1.1 (0.6–1.9)</td>
</tr>
</tbody>
</table>

<sup>a</sup> High level, tertiary; medium level, complete upper secondary; low level, basic upper secondary or lower
<sup>b</sup> Per 100 000 person-years
<sup>c</sup> Adjusted for background factors (year of birth, mother’s age at birth, birth order, type of municipality), parental characteristics (mother’s and father’s income, mother’s marital status, mother’s number of children, mother’s and father’s disability, mother’s and father’s vital status) and individual characteristics (birth-weight, chronic childhood disease, conscript measures (general ability, mental function, physical function, body mass index, height), military duty completion)

We also examined the role of early factors on associations between education and outcomes in table 3 by excluding the factors one by one. The HR changes away from the null in Model 2 for suicide and schizophrenia were solely due to the effect of parental education. General ability was the single factor with strongest impact on the gradients. Excluding general ability from Model 4 led to markedly reduced estimates of early factor influences on the education—schizophrenia association, from −20.7% to −2.4%. Omitting general ability from Model 4 reduced also the early factor influence for all psychiatric disability (from −42.1% to −26.1%), neurosis/personality disability (from −44.1% to −33.2%) and sickness absence (from −25.6% to −16.5%). For suicide, completed military duty had largest impact on Model 4.

The combined pattern of parental and own education level in association with suicide and schizophrenia hazard was examined closer (table 4). For both outcomes, distinctly higher rates and adjusted HRs were found for high parental education combined with low own education level.

Discussion

In this population, comprising all men born in Norway in 1967–71, we found strong and consistent social gradients in suicide, psychiatric disability and psychiatric sickness absence. Parental and individual characteristics, general ability at age 18 years being most influential, seemed to contribute to the SEP gradients for all study outcomes. The early factor influence was strongest for neurosis or personality disorder disability.

Validity

The study was based on linkage between national registers and contains individual-level data gathered consecutively. Linkage is considered to be complete with nationwide coverage. These strengths make selection bias an unlikely problem, and lack of power was a minor problem. Selective loss could be a particular problem for sickness absence with a large excluded fraction. We examined this closer by performing analyses including men under education (n = 19 180). This generated sickness absence results close to those presented (data not shown).

There are other limitations with a potential for information bias or confounding that may be more apparent. These limitations are related to analytical model specifications, lack of quality in study variables and lack of potentially important variables.

Social inequalities in health are influenced by complex social and biological processes. Model specifications and causal...
models are complex, but evidently much cruder than the truth. Modelling could be a particular problem when addressing processes that evolve over extended periods, where feedback between selection processes and social causation could develop. Differentiation between health selection and social causation could be a particular problem for schizophrenia. We recorded the incidence of schizophrenia disability, not disease debut. Our outcome should therefore be interpreted as schizophrenia disability subsequent to established adult SEP. Education level at the age of 28 years could be a consequence of schizophrenia with an earlier debut but such reverse causality would be less plausible for the strong negative association with general ability 10 years earlier (Supplementary table 1). This is in agreement with negative IQ associations among Swedish conscripts and Danish youths tested before the schizophrenia diagnosis. Data in national registers are crude and often collected for other purposes than research. Data quality problems with proxy variables can influence results. Suicide registration could be incomplete, and differential misclassification according to SEP could result in bias. It is reassuring that suicide completeness in the years of follow-up is considered to be high in Norway. It is important to recognize that data were collected independently in different registers, and we consider it unlikely that errors were differential. Therefore, such errors should tend to attenuate true associations rather than create false associations. Another obvious shortcoming in our study is lack of important variables. Missing childhood indicators could be important. Examples are school qualifications, behaviour, personality, and indicators of own effort or parental aspiration and interest. The most plausible net effect of limitations in variable quality and lack of variables is that the reported influences of early factors on the social health gradients are underestimated. This would for example be the effect of omitting a personality trait that was associated with an improvement in educational attainment and a beneficial effect on mental health.

Comparison with other studies and inferences

We found stronger social health gradients than in earlier reports. This was the case for suicide as well as for morbidity studies based on in-patient or self-reported diagnoses. Differences could be due to the narrow age span of the men under study; also, we applied a more detailed SEP classification than in most other studies. Education could possibly be a better indicator of SEP than occupation class or income in young adult age. Steeper social gradients in morbidity in the present study is also likely because disability and sickness absence could be more socially influenced than in-patient and self-report diagnoses. However, gradients were also stronger than psychiatric disability and sickness absence in other reports. We found largest rate differences between basic upper secondary and completed upper secondary education for all outcomes. This could have some implications for prevention because completed upper secondary level is a prerequisite for college or university studies, and it is also the limit for completed vocational training.

Our aim was not to provide full explanations for inequality, but rather to elucidate the role and impact of factors acting early in life. In general, we found that early factors partly accounted for adult health inequalities because they were determinants of both adult social position and the psychiatric outcomes under study. The results provide documentation that indirect selection matters in explaining social inequalities in mental health. We found only few other studies addressing the influence of early life factors on adult health gradients. It is interesting to note that our results are not far from effects of early factors on social gradients in psychological distress at age 33 years in the National Child Development Study. Ability score at age seven was one of the most influential factors, reducing the social gradient with 26% for males whereas social class at birth had a more modest effect with a gradient reduction of 8%. Childhood SEP had also little impact on adult social gradients in depression and anxiety in the same cohort.

We found that both suicide and schizophrenia disability rates were highest for the combination of high parental and low own education level. This is in agreement with an inverse parental SEP gradient for suicide as well as for schizophrenia in several studies but not all studies. This particular pattern could have several interpretations. One possibility is that a mismatch between parental aspirations and expectations and own ability could have adverse effects on mental health. It has also been suggested that schizophrenia could be diagnosed earlier for patients with parents in high social positions. We should recognize that distinguishing between direct (‘drift’) and indirect selection for schizophrenia could be extremely difficult because the disease often is preceded by adverse social events long before diagnosis.

The generalizability of this study is probably limited and extending the interpretations to other populations and other time periods should be done with caution. This is particularly true for outcomes based on national regulations such as disability and sickness absence. SEP gradients in health is also dependent on age and gender. The causes of health inequalities show many of the same features for women and men, but women could be more strongly influenced by parenthood.

The results of this study indicates that public health policies to reduce social inequalities in health should be directed to measures that include the whole life span and act across generations. European countries suggest that early life measures should include family and school issues. Conclusions

Early factors and particularly general ability contributed substantially in accounting for inequalities in neurosis and personality disorder disability. Early factors played a more modest role for the other outcome entities. We suspect that the role of indirect selection from early factors could be underestimated in our study because we lack data on personality factors that could be important antecedents, particularly for schizophrenia or suicide. General ability was the only early factor having substantial influence on inequalities, although it does not qualify as the ‘fundamental cause’. Supplementary data

Supplementary data are available at EURPUB online Funding

Research Council of Norway (Grant no. 161321/V50).

Conflicts of interest: None declared.

Key points

- Social gradients were steep for suicide, psychiatric disability and psychiatric sickness absence, and indirect selection from early life factors could explain a substantial part of these social inequalities. This
implies that public health policies to reduce social inequalities in health should be directed towards measures that include the whole life span and act across generations.

- Differences in study outcome rates were largest between men with basic upper secondary education (11 years) and men with complete upper secondary education (12–13 years). This could indicate that preventive intervention should be directed to educational policies because complete upper secondary level is a prerequisite for college or university studies, and it is also the limit for completed vocational training.

- Suicide and schizophrenia disability were associated with a combination of high parental and low own education level, but the inference of this finding is unclear.

References


15. Agerbo E, Byrne M, Eaton WW, Mortensen PB. Marital and labor market status in the long run in schizophrenia. Arch Gen Psychiatry 2004;61:28–33.


