Early life determinants of musculoskeletal sickness absence in a cohort of Norwegians born in 1967–1976

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Abstract

In order to investigate the extent to which musculoskeletal sickness absence was influenced by a range of circumstances concerning family background and health in early life, we established a register-based cohort of all live-born in Norway between 1967 and 1976. Personal data on parental factors and health early in life were recorded prospectively from birth onward in the Medical Birth Registry of Norway, the National Insurance Administration, Statistics Norway, and the Central Population Register. We collected data in the National Insurance Administration on the first spell of medically certified long-term (> 16 days) musculoskeletal (International Classification of Primary Care group L) sickness absence in 2000–2003 among 378,356 participants who were considered to be at risk of sickness absence on January 1st, 2000. The 4-year musculoskeletal absence risk was 0.264 for women and 0.156 for men. Parental education level was associated with musculoskeletal sickness absence, with increasing adjusted relative risks by decreasing educational level for both genders. Associations with other early determinants (birth weight, childhood disease, parental survival, parental disability, parental income, and parental marital status) were all close to unity. Parental education level attributed 36% (95% confidence interval 33–38) to the population risk for women and 67% (64–70) for men. The parental education association was partly mediated through own educational attainment, which was strongly associated with musculoskeletal sickness absence in itself. Our data suggest that mechanisms acting early in life could influence later risk of musculoskeletal sickness absence.

Keywords: Norway; Educational status; Family characteristics; Occupational health; Socioeconomic factors; Musculoskeletal

Introduction

Work-related musculoskeletal disorders with sickness absence are major health and societal problems (Buckle, 2005; Henderson, Glozier, & Elliott, 2005). The current causal concept of musculoskeletal disorders is multifactorial: workplace or leisure time physical and psychosocial exposures, organisational factors at work, constitution, personality traits and coping strategies, and socioeconomic factors (Woods & Buckle, 2002). The same factors are also considered to be important determinants of sickness absence (Allebeck & Mastekaasa, 2004a; Kivimäki et al., 2003; Kristensen, 1991). Furthermore, sickness
absence is influenced by macro level factors such as municipal economy, social insurance systems, national economic policies, and international economic trends (Allebeck & Mastekaasa, 2004a; Kristensen, 1991; Virtanen, Kivimäki, Elovainio, Virtanen, & Vahtera, 2005). Consequently, socioeconomic inequalities in health are considered to involve both musculoskeletal disorders (Warren, Hoonakker, Carayon, & Brand, 2004; Woods & Buckle, 2002) and sickness absence (Fuhrer et al., 2002; North et al., 1993).

Social conditions early in life have been considered as covariates in studies addressing socioeconomic inequalities in health. Socioeconomic differences in sickness absence have been estimated in models adjusting for father’s occupational class in Whitehall II and the French Gazel cohort (Fuhrer et al., 2002; North et al., 1993). Warren et al. (2004) adjusted for a number of early life factors including parental education and income, and paternal occupation in a study on job characteristics, socioeconomic status and health outcomes including musculoskeletal disorders.

Results in several studies indicate that early life exposures influence adult health outcomes (Galobardes, Lynch, & Davey Smith, 2004; Poulton et al., 2002; Power & Matthews, 1997). The concept of life course epidemiology (Ben-Shlomo & Kuh, 2002; Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003) encompasses different models. These models have been categorised in two main groups: critical period models and accumulation of risk models (Ben-Shlomo & Kuh, 2002). The critical period model has had a focus on biological programming (Barker, 1998), but has also included theories on critical social conditions early in life, also termed social imprinting (Bäckman & Palme, 1998). The accumulation of risk model encompasses several potential mechanisms acting throughout life: factors could act independently, or mediate or modify the effects of other determinants in social, biological or psychological chains of risk (Kuh et al., 2003). The accumulation of risk model is close to the unhealthy life career hypothesis developed by Lundberg (1993). Lundberg (1993) suggested that unhealthy life careers could be the result of a triggering by adverse childhood social conditions and factors acting later in life, and furthermore, that social conditions early in life matter more than biologically induced susceptibility to disease introduced early in life. The evidence from life course studies indicates that some, but not all, adult health outcomes are influenced by circumstances in childhood (Galobardes et al., 2004; Poulton et al., 2002; Power & Matthews, 1997).

Contrasting this interest in life course factors as determinants of chronic disease, the evidence of effects from early life exposures on the risks of musculoskeletal disorders and sickness absence later in life is rather scarce. Extensive reviews on risk factors for musculoskeletal disorders (Woods & Buckle, 2002) and sickness absence (Allebeck & Mastekaasa, 2004b) include scarcely any emphasis of factors early in life. There are a few studies examining influences from childhood or adolescence on later musculoskeletal disorders or sickness absence. The results are not consistent, which could be due to differences in early life factors considered. Bäckman and Palme (1998) investigated a wide range of social and health conditions throughout the life course in a prospective study of individuals born in Stockholm in 1953. Other studies have addressed more restricted factors such as school results in adolescence (Khatun, Ahlgren, & Hammarström, 2004), parental smoking habits (Eriksen, 2004), father’s social class (Power & Matthews, 1997), and the memory of an unhappy childhood (Taylor, 1968). It should also be recognised that data on early life conditions were collected retrospectively in some of these studies (Eriksen, 2004; Taylor, 1968).

We have established a register-based cohort of all persons born in Norway between 1967 and 1976. Data include repeated measures of social circumstances and health recorded prospectively throughout life (Kristensen, Bjerkedal, & Irgens, 2004). The purpose of the present study was to quantify the relations between a range of health and social circumstances experienced throughout childhood and the 4-year risk of medically ascertained musculoskeletal absence from work in young adult age. In addition, we intended to study to which degree any factors acting later in life influenced the associations between early factors and later musculoskeletal absence. Our aim was to throw light on the potential life course chains of risk (Kuh et al., 2003) for this particular outcome.

Methods

Participants

The study population included all 626,928 live births who were registered in the Medical Birth
Registry of Norway in between 1967 and 1976. The national identification number of the child and the parents allowed linkage with benefit, income and sickness absence registers in the National Insurance Administration, the education register in Statistics Norway, and data on residence, marital status, and childbirths in the Central Population Register. Persons who were considered not to be at risk of sickness absence on January 1st, 2000, were excluded from analysis. Persons excluded \( (N = 248\,572, 39.6\% \text{ of the total}) \) included (in categories not mutually exclusive) persons who had died (2.5%) or emigrated (2.1%) before the start of follow-up as well as persons with pensionable income in 2000 below the limit that entitles to sickness allowance (18.2%). Only 9.6% of the total had lack of income as the only exclusion criterion. Also persons assumed to have a marginal relation to paid work either because they were under education (24.5%) or disability pensioners (1.8%) at the start of follow-up were excluded, regardless if they had income above the limit. Persons who were registered with sickness absence on January 1st, 2000 (3.2%) were excluded because they were not at risk at start of follow-up. After exclusions, 378,356 participants (index persons) remained for analysis.

**Study outcome**

The study outcome was first spell of musculoskeletal sickness absence recorded between 2000 and 2003. General practitioners in Norway are obliged to label all sickness absence forms with an International Classification of Primary Care (ICPC) diagnosis to get them accepted by the health authorities. Medically certified spells, usually with duration of more than 16 days were registered with a diagnosis based on the doctor’s notification and coded according to the ICPC (http://www.globalfamilydoctor.com/wicc/sensi.html). We considered spells with ICPC group L (musculoskeletal) diagnoses as musculoskeletal sickness absences.

**Early determinants**

Linkage provided longitudinal data for the index persons and their parents, including annual updates on education, pensionable income, pension benefits, deaths, emigrations, marital status, and childbirths.

Classification of parental education was based on the Norwegian standard NUS2000 (Statistics Norway, 2003). Associations between maternal and paternal educational attainment and musculoskeletal sickness absence were of similar strength, so we collapsed maternal and paternal education level into one variable with five categories depending on the parent with highest level: graduate tertiary or higher; undergraduate tertiary; upper secondary, final year or post-secondary/non-tertiary; upper secondary, basic; and lower secondary or less. Maternal marital status was categorised as married, unmarried, and previously married both at index person birth and at index person age 16 years. All other parental characteristics were classified according to the parent (maternal, paternal) and the index person’s attained age (0–6 years; 7–15 years). Maternal and paternal factors considered were vital status (deceased or not), disability (pension or not), and income level (mean income below the sickness absence compensation limit or above).

In the study population, we have earlier reported that both childhood disease and birth weight were associated with subsequent lack of work participation (Kristensen et al., 2004). We used the same two independent variables in the present analysis. Childhood disease was defined as either insurance benefit from chronic disease before age 7 years, or birth injury, or congenital malformation. Birth weight in singletons was standardised for gender and separated into four categories; multiple births were allocated a separate category.

The characteristics and distribution of the early determinants are more fully described elsewhere (Kristensen, Bjerkedal, Irgens, Gravseth, & Brevik, 2005).

**Later determinants**

In order to examine associations of early determinants mediated through factors acting later in life, we included data on the index persons’ educational attainment, income level, and family pattern. The most recent coding of the index person’s education provided educational attainment, applying the same categories as for the parents. Income level in 1999 was categorised into gender-specific quintiles. We used December 1999 data on childbirths and marital status to establish a family pattern variable. Index persons were classified as unmarried, married, or previously married, each divided into persons with and without children. The highest education level, the highest income quintile, and being unmarried without
Statistical analysis

We used Stata/SE 9.2 software (www.stata.com). Based on the individual occurrence of incident musculoskeletal sickness absence during the years 2000–2003, we computed 4-year risk estimates in groups. Since musculoskeletal sickness absence was prevalent, we estimated associations as relative risks in Poisson regression rather than the more conventional odds ratios in logistic regression (Greenland, 2004). Poisson regression of risk data produces too wide confidence interval estimates (Greenland, 2004). Consequently, we used the robust variance option in Stata providing crude and adjusted relative risk (RR) estimates with corresponding 95% confidence intervals (CI). The study population included index persons from the same family (the total of index persons’ mothers numbered 280,268). Applying robust variance estimation for cluster-correlated data solved this violation of the independent observation assumption (Williams, 2000). We used the cluster command and specified maternal identity as the cluster variable. All analyses were performed separately for women and men.

The multivariable modelling was based on the temporal relation between considered factors (Kuh et al., 2003). The relation between parental education level, own education level, and musculoskeletal sickness absence could serve as an example of asymmetry between factors: own education level could influence sickness absence but not parental education level. Parental education level could influence both own education level and sickness absence, and the influence on absence could act through own education as an intermediate. In this model, the relation between parental education level and musculoskeletal sickness absence might be mediated through but not confounded by own education level whereas the association with own education level might be confounded by but not mediated through parental education level (Greenland, Pearl, & Robins, 1999).

We applied a targeted, sequential analytic strategy of a priori formulated models, distinguishing between potential mediators and confounders (Lamont et al., 2000; Victora, Huttly, Fuchs, & Olinto, 1997). One main goal was to estimate associations with early determinants (parental factors, birth weight, and childhood disease) after controlling for potential confounders. Furthermore, we wanted to estimate the degree to which early factor associations with absence were mediated through factors later in life.

Three models were applied. Model 1 included the parental variables, birth weight, and childhood disease. Model 2 included model 1 factors plus the index person’s educational attainment. Model 3 included, in addition, the index person’s income in 1999 and family pattern in 1999.

We also considered other variables that were correlated with early determinants and were associated with musculoskeletal sickness absence in univariate analysis. In all three models, we included year of birth (10 categories), birth order (5 categories), maternal and paternal age at birth (5 categories each, missing data on paternal age for 5.7%), and place of residence at age 16 years (six regions, missing data for 0.2%). Throughout, missing values were included in the models as separate categories.

Population attributable risk (PAR) is a function of the population prevalence of a risk factor and the strength of an association and can be interpreted as the proportional reduction in population risk that would occur in the hypothetical case that the whole population experience the risk of the (unexposed) reference category (Greenland, 1998). Accordingly, a sickness absence PAR of 30% for a risk factor means that the risk of sickness absence in the study population would have been 30% lower than what was actually observed in the event that all had experienced the absence risk of the reference category. PARs for different factors were calculated in the AFLOGIT procedure in Stata after including the factor in the regression model as a dichotomous variable where values except for the reference value were collapsed into one category (excluding missing values). This provided adjusted PAR estimates with 95% CIs in the three regression models. We interpreted early determinant RRs and PARs in the first model as measures of strength, respectively impact of those factors on musculoskeletal sickness absence. The degree of changes in early determinant RRs and PARs in subsequent models was interpreted as measures of the mediating strength of the later determinants that were introduced.

Ethics approval

The Regional Committee for Medical Research Ethics has approved the study.
Results

Spells of musculoskeletal sickness absence were recorded in 43,605 out of 165,419 women (risk 0.264) and 33,231 out of 212,937 men (risk 0.156). A total of 4,428 (1.2%) index persons died or emigrated, 3,490 of whom were lost before any absence spell, and 1,168 (0.3%) became disability pensioners during the four year follow-up.

Table 1 provides distributions of parental educational attainment and corresponding associations with musculoskeletal sickness absence in model 1. Parental educational attainment was associated with musculoskeletal sickness absence: the lower the education level the higher the risks and RRs. This gradient was evident for both genders but strongest for the men. Associations between other early determinants and absence were close to unity. Slight associations with adjusted RRs between 1.10 and 1.20 were found for maternal and paternal disability, and unmarried/divorced status of the mother (data not shown). The other parental factors, childhood disease, and birth weight had practically no influence (data not shown).

Model 2 results in Table 1 show that the index person’s educational attainment was strongly associated with musculoskeletal sickness absence. The pattern is consistent: low education level was strongly associated with high musculoskeletal sickness absence risk. Low income, having children, and being previously married in 1999 showed associations close to unity (data not shown).

PAR estimates for variables in the three models are presented in Table 2. Parental education was the only early determinant that had large impact (model 1), with adjusted PAR estimates of 36% for women and 67% for men. Other parental factors, birth weight, and childhood disease had little or no impact (data not shown). PAR estimates in model 2 show that own educational attainment had a dominant impact on musculoskeletal sickness absence. Furthermore, the change in the parental education association with absence between model 1 and model 2 indicates that the index person’s education level was a major mediating factor. Further inclusion of income and family pattern (model 3) demonstrated a moderate impact of both factors, but none influenced the PAR of parental education level.

We examined the role of pregnancies and childbirths among index women during follow-up in order to explore the gender difference in absence risk and its association with parental education level. Based on data on date of childbirth between 2000 and 2004 we divided the women into those without childbirth ($N = 86,079$) and those with one or more childbirths ($N = 78,961$). Furthermore, we assumed that a first musculoskeletal absence among women took place during pregnancy if the first date of absence was less than 250 days before delivery. Other absences were assumed not to start during pregnancy. Women with no childbirth between 2000 and 2004 had a musculoskeletal absence risk of 0.243 and a parental education PAR of 43% (95% CI 39–47). Women with childbirths had an absence risk of 0.286 and a corresponding PAR of 31% (95% CI 28–35). The lower PAR in the latter group was mainly restricted to first musculoskeletal absences during pregnancy (risk 0.174, PAR 27%, 95% CI 21–31). Women in the childbirth category had an absence risk of 0.112 outside pregnancy and a parental education level PAR of 39% (95% CI 33–45), which was close to the PAR for women with no childbirths during follow-up.

We performed stratified analyses in order to examine the relation between parental education level, index person education levels, and musculoskeletal absence closer (Table 3). Parental education level had an impact on musculoskeletal sickness absence in all subset levels of own education and for both genders. PAR levels were heterogeneous across own education levels, being highest for participants with lower categories of education.

The impact of parental educational attainment was also evident when we examined specific educational groups. One example is women with nursing and caring education: nurses (undergraduate tertiary level, $N = 9,461$, musculoskeletal sickness absence risk 0.264) had a parental education level PAR of 13% (95% CI 4–22); the corresponding PAR for auxiliary nurses (upper secondary levels, $N = 6,370$, absence risk 0.389) was 35% (95% CI 9–53).

Discussion

We accomplished a complete follow-up of nearly 380,000 persons for a duration of 27–36 years, starting at birth, in this nation-wide cohort. Parental education had large impact on musculoskeletal sickness absence. Young Norwegian women would have had a musculoskeletal sickness absence risk 36% lower than what was actually observed had all experienced the same risk as young women whose mother or father had a graduate tertiary education.
The corresponding reduction in the male part of this population would have been 67%. Further, the results indicate that own education contributes strongly to the risk of musculoskeletal sickness absence. Judged from the change in parental education PAR between model 1 and model 2, a substantial part of the association between parental education and absence is mediated through own education. This mediating effect is not the full explanation, however, as parental education had considerable impact on musculoskeletal sickness absence within separate levels of own education.
The risk of musculoskeletal sickness absence was higher for women than for men. However, associations and impacts of parental and own educational attainment as well as own income level were stronger for men than for women. The explanation could well be that the men had a lower reference absence risk. Crude risks presented in Table 1 indicate that risk differences associated with the study factors were quite similar for both genders, and estimation of adjusted risk differences instead of risk ratios confirmed this (data not shown). The gender difference in musculoskeletal sickness absence may depend on an increased occurrence during pregnancy. This has been confirmed for back pain absence in Sweden (Sydsjö, Alexanderson, Dastserri, & Sydsjö, 2003). The influence of pregnancy during follow-up on the relation between parental education level and absence is interesting, and can partly explain the gender differences in our study. However, a further pursuit of this issue would benefit from some rearrangements of the data and analytical changes.

Comparisons with results in other studies addressing the relation between conditions early in life and musculoskeletal sickness absence are difficult. Musculoskeletal sickness absence is influenced by social determinants that may change across countries and over time. Norway has a rather high occurrence of

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### Table 2

<table>
<thead>
<tr>
<th>Category</th>
<th>Model 1&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Model 2&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Model 3&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PAR (95% CI)</td>
<td>PAR (95% CI)</td>
<td>PAR (95% CI)</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parental educational attainment</td>
<td>36% (33–38)</td>
<td>18% (15–21)</td>
<td>17% (14–20)</td>
</tr>
<tr>
<td>Index person’s educational attainment</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Index person’s income 1999</td>
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<tr>
<td>Index person’s family structure 1999</td>
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<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parental educational attainment</td>
<td>67% (64–70)</td>
<td>38% (33–43)</td>
<td>37% (32–41)</td>
</tr>
<tr>
<td>Index person’s educational attainment</td>
<td></td>
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<td></td>
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<tr>
<td>Index person’s income 1999</td>
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<tr>
<td>Index person’s family structure 1999</td>
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</table>

*Note: PAR = population attributable risk, CI = confidence interval.*

<sup>a</sup>Model 1 including year of birth, parity, geographical region, maternal and paternal age, birth weight, childhood disease, and maternal and paternal factors in different periods (vital status, disability, income, marital status), and parental educational attainment.

<sup>b</sup>Model 2 including variables in Model 1, and index person’s educational attainment.

<sup>c</sup>Model 3 including variables in Model 2, index person’s income 1999, and index person’s family structure 1999.

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### Table 3

<table>
<thead>
<tr>
<th>Index person’s educational attainment</th>
<th>Women</th>
<th></th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Number&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Risk of absence</td>
<td>PAR&lt;sup&gt;b&lt;/sup&gt; (95% CI)</td>
<td>Number&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Risk of absence</td>
<td>PAR&lt;sup&gt;b&lt;/sup&gt; (95% CI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Graduate tertiary</td>
<td>8647</td>
<td>0.114</td>
<td>6% (−3 to +14)</td>
<td>12571</td>
<td>0.013</td>
<td>7% (−23 to +29)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Undergraduate tertiary</td>
<td>50643</td>
<td>0.197</td>
<td>18% (13–22)</td>
<td>38026</td>
<td>0.038</td>
<td>23% (8–36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Final upper secondary</td>
<td>61415</td>
<td>0.273</td>
<td>20% (13–26)</td>
<td>101961</td>
<td>0.158</td>
<td>44% (37–50)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basic upper secondary</td>
<td>35569</td>
<td>0.349</td>
<td>22% (11–31)</td>
<td>45158</td>
<td>0.251</td>
<td>34% (22–44)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower secondary or less</td>
<td>8593</td>
<td>0.378</td>
<td>34% (16–48)</td>
<td>14207</td>
<td>0.273</td>
<td>50% (35–62)</td>
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</tr>
</tbody>
</table>

*Note: PAR = population attributable risk, CI = confidence interval.*

<sup>a</sup>Persons with missing information on parental or own educational attainment excluded (552 women, 1014 men).

<sup>b</sup>Adjusted for year of birth, birth order, geographical region, maternal and paternal age, birth weight, childhood disease, and maternal and paternal factors in different periods (vital status, disability, income, marital status).
sickness absence, and our results might not apply in countries with lower absence levels. We faced another comparison problem, as the early life factors under study were different across studies. We also recognise that disentangling life course processes is inherently difficult, making interpretations of empirical data more or less dependent on a priori mechanistic knowledge (Bosma, 2006, Hallqvist, Lynch, Bartley, Lang, & Blane, 2004).

In the metropolitan Stockholm study, several early life factors related to health and social conditions were associated with sickness absence at age 27 in both genders (Bäckman & Palme, 1998). The early life factors that had impact on sickness absence in young adult age included father’s socioeconomic status, sickness absence from school, family receiving social welfare benefits, and registration in childhood by the Child and Youth Welfare Committee (Bäckman & Palme, 1998). The impact of those early factors were considered to be partly mediated through own education and socioeconomic status in young adult age, and Bäckman and Palme (1998) interpret their findings as a support of a combination of the unfavourable life career hypothesis and the social imprint hypothesis. Our results concerning the combination of parental and own education could be interpreted likewise, but we should keep in mind that the early life factors in our study were different from those of Bäckman and Palme (1998). Furthermore, like Bäckman and Palme (1998), our results provide no support to effects on musculoskeletal sickness absence from biological programming related to prenatal or early postnatal growth and development (Barker, 1998; Forsdahl, 1977).

Results in some life course studies have indicated that factors early in life may have different impact on different chronic diseases (Galobardes et al., 2004; Poulton et al., 2002; Power & Matthews, 1997). Our study is in support of such heterogeneity: we have earlier found that failure in achieving a work career at all seems to be closer related to birth weight, childhood health, and material disadvantage (Kristensen et al., 2004, 2005). This is different from musculoskeletal sickness absence, which seems to be closer linked to parents’ educational level, but not to early life health and only to a limited degree to material conditions in childhood.

Several limitations are apparent in our study. The most obvious shortcoming is that we did not have data on occupation or work environment. The study was aimed at exploring the role of early life factors, and we would have wanted to examine the interrelation between such early factors and occupational factors, particularly physical strain and psychosocial factors. We tried to solve the lack of occupational data by analysing distinct strata of own educational attainment as proxies for occupation (e.g., educational categories of nursing and caring). Besides lack of occupational data, our study did not cover factors relating to school performance (Bäckman & Palme, 1998; Khatun et al., 2004), personality (Kristensen, 1991; Peter & Siegrist, 1997), or life-style factors (Power et al., 2005). Clearly, all these factors might influence the role and explain the mechanism of associations between parental education level and musculoskeletal sickness absence.

A second problem concerns interpretation of the results. We applied complex analytical models in a non-randomised study using data inevitably hampered with error. We should therefore be cautious in interpreting our RR and PAR estimates as representations of causality or as potentials for prevention. Model specifications are based on choices, which will necessarily have implications for interpretations of the results. However, parental and index person educational attainment represented the two dominating factors, and other measured childhood determinants seemed to play a minor role in explaining the absence risk. Another fact in support of this main finding is that analyses using alternative outcome choices that were more extreme (e.g., the risk of more than one musculoskeletal absence spell) showed stronger associations with parental or own education level than the ones presented (data not shown).

A third problem concerns misclassification of subjects at risk. The at-risk status for an individual might change during the four years of follow-up. We had only limited possibilities to follow such changes. Part-time workers might be at risk only during limited periods, which could explain the low musculoskeletal sickness absence risk in the lowest income quintile (Table 1). The same would probably apply for new disability pensioners. It is reassuring, however, that analyses under different assumptions (not shown) produced results nearly identical to the reported ones. This was the case for analyses restricted to those with higher incomes, or when we excluded those who died, emigrated or became disability pensioners during follow-up. We also performed Cox regression analyses with censoring for death, emigration, incident disability, and
non-musculoskeletal sickness absence. Cox regression produced hazard ratios slightly in excess of the RRs reported, but was abandoned because PAR estimation was not kept as an option.

Finally, we might have a problem by excluding persons receiving disability pension or being under education, who fulfilled the income criterion that entitles to sickness absence benefit. Excluding this large group (21.4% of the total population) could potentially introduce biased results. Supposedly, this group had mainly periodic work. Their risk of musculoskeletal sickness absence was somewhat lower and the parental education level distribution slightly more favourable compared to those included in the study. The associations between parental education and absence were almost identical to those reported in Table 1 when we included this group in the analysis (data not shown). This suggests that this exclusion did not introduce bias in our results.

In our opinion, a direct effect of parental education on musculoskeletal absence is implausible. The results suggest that parental education has an additional effect besides being mediated through own educational attainment, but we have no data to explore such mechanisms. This makes inferences speculative. In addition to mediation through own education level, the parental education level could act through occupation and work environment. Our data do not support that this is the entire explanation, however, because the parental education association was apparent within specific educational entities such as nurses and auxiliary nurses. Another possibility is that parental education level was a determinant of individual characteristics established early in life, which could influence later musculoskeletal absence risk. Parental education level could be an indicator of how coping and control beliefs are rooted and shaped in childhood (Bosma, 2006). Sickness absence has been associated with individual coping strategies (Kristensen, 1991; Peter & Siegrist, 1997). Peter and Siegrist (1997) point to interaction between individual passive coping and insufficient reward at work as a potential mechanism for sickness absence proneness. This explanation could fit with our results, also because the parental education level association was strongest for subjects with low education level themselves (indicating jobs with less reward). Another possibility could be that parental education level had an influence on physical activity and ability during childhood and adolescence, given higher educated parents were more likely to encourage their children to physical activity (Raudsepp, 2006).

The results of the present study suggest that early life experience affects later musculoskeletal sickness absence risk. Our concept of musculoskeletal sickness absence should not be restricted to currently acting causal factors. This should have implications for directions of research and the need to uncover the key mechanisms that could explain why parental education level had such high impact. In particular, it would be interesting to explore whether these mechanisms involve individual characteristics established early in life. Furthermore, explanations for the distinct gender difference should be explored closer. Our knowledge may not be sufficient to recommend specific preventive approaches, but should neither be disregarded. This means that we should not expect that short-term efforts to prevent musculoskeletal sickness absence have lasting effects. We should rethink and include life-course perspectives in research and preventive programs.

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