Prevalence of musculoskeletal pain in the general Swedish population from 1968 to 2002: Age, period, and cohort patterns

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ABSTRACT

We examined age, period, and cohort patterns in musculoskeletal pain prevalence between 1968 and 2002 in the Swedish population. A repeated nationally representative survey allowed cross-sectional comparisons of ages 18–75 (5 waves n = 5000), and ages 77+ at later waves (2 waves n = 500). Cross-sectional 10-year age group differences in 5 waves, time-lag differences between waves (shifts across time) for age groups, and within-cohort differences between waves for 10-year birth cohorts followed over time were analyzed using graphs and ordered logistic regressions. The outcome scale was based on the three items measuring slight or severe pain in back, shoulder, and joints during the past 12 months. Adjusted for the age-related increase, the cohorts followed over time did not show significant period change, except for cohorts born during 1940s. Beginning with the 1940s' cohorts pain prevalence increased over the period, and after baseline later cohorts also entered adulthood and the study with a higher pain prevalence. The prevalence of pain in the adult population thus increased with the passage through age and time of the 1940s cohorts. While there were no pronounced cohort differences at baseline in 1968, results demonstrated strong age effects in pain. The results indicate that the prevalence of musculoskeletal pain among the oldest age groups may increase in the future, when more baby-boomers are entering their oldest ages.

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1. Introduction

Estimates of musculoskeletal pain prevalence vary [11,47,48]. Many cross-sectional studies show how pain increases with age [5–7,9,10,12,13,18,20,21,23,27,39,46]. Several longitudinal studies have evaluated changes when individuals age [8,18,35,52]. Individuals' pain experiences often vary over time, and while within individuals pain patterns are not always straightforwardly interpreted in terms of an age pattern, longitudinal results confirm pain's age relatedness.

Some studies have reported of increases in specific age groups' pain prevalence over time [20,24,28,29], but it remains unclear whether there are differences between birth cohorts.

A birth cohort refers to a group of people born at about the same time. Cohorts tend to share similar social and cultural circumstances potentially increasing homogeneity compared to other birth cohorts [42]. Differences observed between cohorts may be due to historical differences, e.g., in the social or physical environment during childhood or young adulthood, but they may also be due to differences in the structure or size of different birth cohorts, e.g., baby-boomers may differ in attitudes and behavior from previous and later cohorts partly due to their greater numbers.

Although cohorts may differ historically, differences exist in part because of the different factors that influence them during a study period, i.e., between baseline and follow-up. Period effects reflect the specific events that have had the potential to change the cohorts during a specific time period, i.e., the studied period. This can concern sudden events and changes in the physical and social environments, e.g., policy changes, but may also include more subtle changes such as shifting attitudes.

Age effects are due to biological, psychological, and behavioral processes connected with maturing and becoming older [14]. Not least are age effects important because they may identify similarities across different generations, countries, and cultures.

There is a fundamental difficulty in assessing developmental trends in pain experience in available research. Specifically, it is not possible to assess the effects of age, period, or cohort unambiguously. Current designs consistently confound two effects [22,37]. Cross-sectional designs, which measure age or cohort differences at one time point, confound age and cohort effects. Longitudinal designs, which follow cohorts over age or time, similarly confound age and period effects. Likewise, repeated cross-sectional designs...
whereby age-specific prevalence changes over time or across cohorts are estimated, so called time-lag analyses [44], confound period and cohort effects. To identify the effects, a simultaneous modelling of age, period, and cohorts is usually necessary.

This study examines age, period, and cohort patterns in musculoskeletal pain prevalence.

2. Material and methods

2.1. Material

In the analyses, data from two surveys were combined, the Level of Living Surveys (in Swedish: LevnadsNivåUndersökningen, LNU) [19] and the Swedish Panel Study of living conditions of the oldest old (SWEOLD) [33]. The LNU sample covered ages 15–75, and SWEOLD covered ages 77–98.

The LNU was conducted in 1968, 1974, 1981, 1991, and 2000. All waves were nationally representative cross-sectional samples of the adult population at the time of the survey. In 1991 the lower age limit was raised from 15 to 18 but the ceiling of 75 years was maintained. Between 1968 and 2000 the response rate fell from 90.6% to 76.6%. The number of respondents decreased from 5654 to 5126. The surveys were carried out in collaboration with Statistics Sweden, and professional interviewers conducted the interviews. In 1968 the sample consisted of approximately six thousand randomly selected persons between the ages of 15 and 75. When the survey was repeated in 1974, everyone from the initial sampling in 1968 who was living in Sweden and less than 76 years old were addressed again. In 1974 and at each successive wave, a sample of younger persons (who had been below the lower age limit in the earlier wave but who had now passed it) and newly arrived immigrants was added to maintain the samples' cross-sectional representativeness of the Swedish population. No other additions were made to the samples.

The SWEOLD study originated from the LNU. It consists of two cross-sectionally nationally representative surveys of people aged 77–98 who were living in Sweden. They were interviewed in 1992 and 2002 and response rates were 95.4% and 88.5%, respectively [38]. The two waves for this study contain all those persons aged 77 and older who were originally eligible for the LNU. Interviews were conducted in participant's homes or institutions. The participants who could not be interviewed directly (11.9% and 12.8%) were interviewed by proxy, i.e., with a spouse, or caretaking personnel.

Musculoskeletal pain was measured with a scale based on three questions. These items were part of a longer list of diseases and symptoms. The list was introduced with the question: “Have you had any of the following diseases or disorders during the last 12 months?” The particular items concerning musculoskeletal pain in this list were: (a) pain or ache in hands, elbows, legs or knees; (b) pain or ache in back or hips, ischias; and (c) ache in shoulders. Each item could be answered with no, mild or severe. Reports of mild pain added a point to the scale, while severe pain added two. The musculoskeletal pain scale thus went from 0 to 6 points. In the graphs – to facilitate presentation – this scale was dichotomized, no pain was coded 0 and mild or severe pain at any location 1.

The questions about pain do not follow the International Association for the Study of Pain (IASP) classification of pain syndromes. This was because LNU is a sociologic multipurpose interview survey that began already in 1968, before the IASP classification was created. In the interest of consistency the questions have remained identical throughout the surveys, not least, to improve the ability to estimate trends over time. Pain in the back, hips, shoulders, and the extremities is assumed to be musculoskeletal in origin. A chi-square score test was used to confirm that adding the different items was reasonable, i.e., that the different levels as well as the different locations of musculoskeletal pain displayed the same age–period–cohort pattern. Graphs, logistic regressions models and chi-square tests were also examined separately for the different items in order to find potentially deviating patterns.


2.2. Analysis

The data are analyzed in two steps. First, the data are presented graphically to explore cohort, period, and age trends. Six graphical arrangements are presented: (a) cohorts by period, (b) cohorts by age, (c) period by cohorts, (d) period by age, (e) age by cohort, and (f) age by period. Each arrangement demonstrates unique (but not independent) information about pain patterns. Second, ordered logistic regression models were applied to analyze the degree to which age, period, and cohorts predicted the likelihood of having pain.

In the graphical representations of the data this study examines (1) cross-sectional 10-year age group differences at five points in time: 1968, 1974, 1981, 1991/1992, and 2000/2002, (2) time-lag differences between waves (shifts across time) for 10-year age groups and (3) longitudinal within-cohort changes between waves for 10-year birth cohort panels followed over age and time. A total of six different graphic descriptions are possible, as change in all three designs can be described in two alternative ways, e.g., longitudinal changes may be described either over calendar time or over age.

Sometimes it is possible to infer the presence of age, period, and cohort effects by examining the three kinds of designs (the cross-sectional, the time-lag analysis and the longitudinal) separately [37]. No effects would result in no measurable changes in any design. Second, when there is only one kind of effect, two of the designs will show a corresponding pattern and the third design will indicate no change. Thus, if a phenomenon is affected by age but not by period or cohort, then cross-sectional and longitudinal designs will indicate a similar age-related change, while a time-lag analysis will indicate negligible changes. When only period effects are present, a time-lag analysis and the longitudinal design will indicate a corresponding pattern of change over the time period, while the cross-sectional design will indicate no differences. Similarly, when only cohort effects are present, the cross-sectional design and a time-lag analysis will show a corresponding pattern of cohort differences, while the longitudinal design will indicate no change [2].

When all the three designs indicate change, two or three effects are present. In order to separate the effects, so called age–period–cohort regression models are called for [25,40,45]. The age limits together with the irregular intervals between waves truncated some cohorts in some waves; these data were disregarded in the graphs (for the 1965–1974 cohort in the 1981 and 1991 waves, the 1955–1964 cohort in 1974, the 1945–1954 cohort in 1968, and the 1925–1934 cohort in 2002). Similarly, data on the oldest 10-year cohorts in the later waves was disregarded because of the upper age limit and the high mortality in the oldest ages (the 1895–1904 cohort in 1974 and 2002, and the 1892–1894 cohort in 1974 and later).

The irregular intervals also made it necessary to approximate most of the data points for the graphs of the cohort differences by age. (That is, when the graphs were following cohorts there were no observations for some the cohorts when they were in the particular age we wanted them to be in, while there were
observations from both an earlier age and a later age.) These approximations were based on the curves describing the age differences in the longitudinal design, i.e., estimates for the specific ages were taken from the Excel spreadsheet used for the longitudinal age curves (i.e., e) in Fig. 1.

Next, ordered logistic regression models using the SAS statistical software were used to evaluate the changes [43]. This is an extension of the binary response model in logistic regression, which allows for an ordered multi-categorical outcome [15]. Significant chi-square score tests supported our proportional odds’ assumptions, i.e., the independent variables’ ORs were the same over the different levels of pain in the scale.

All observations (n = 28,685) from the seven waves were collapsed into one data set. The first model included period and age

Fig. 1. Percent with at least some kind of musculoskeletal pain. Cross-sectional, time-lag and longitudinal analysis of age, period, and cohort patterns.

differences, the second model included period and cohort differences, and the third model included age, period, and cohort differences.

To better match the graphical layout the models included interaction terms. The first model included the interaction terms between age and period, i.e., between the age group and the survey year. The second and the third models included the interaction terms between cohort and period, i.e., between the 10-year birth cohort and the survey year.

These interaction terms were modelled using effect-coded dummy variables. This made the models estimate the period changes in average across the different age groups and cohorts. The effect-coded variables make it possible to compare each category against all categories’ average, instead of using one particular category as a reference category.

To facilitate interpretation the contrasts of the dummies have been made within each survey year. They do not consequently represent the formally relevant contrasts of the statistical interactions between cohort and period (or between age and period). In the presented table it is, however, possible to conclude a significant statistical interaction indirectly by comparing relevant ORs, such as, when comparing an OR significantly larger than 1.0 for one year with an OR that is smaller than 1.0 for another year concerning an age group or cohort.

Controls for truncated cohorts (dummy variables for the cohorts in the waves enumerated above) and the lower age limit in earlier survey waves (dummies for the ages up to 17 years, and ages up to 18 years in the 1968, 1974, 1981, and 1991 wave) were used in models.

We treated all the observations as independent, even if some persons reappeared in later waves. If there is less variation among the observations from the same persons than among the other observations, this design might lead to erroneously low standard errors. To check this, i.e., to ensure that our assumption about independent observations was reasonable, we estimated some additional models. We used the Huber/White sandwich estimate of variance, with correction for clustering of observations [30]. The original estimator by Huber [26] (1967) and White [49], however, do not deal with clustering. The robust estimator used here, also called the cluster-correlated robust estimate of variance, is a straightforward generalization of the original estimator. William [50] gives a short proof of the validity of the estimator. We used this analytical approach only to ensure that error terms of the independent observations did not deviate from individuals with single measurements. The results indicated that our initial assumption about independent observations was reasonable.

3. Results

3.1. Figures

Fig. 1 shows the percent of people with musculoskeletal pain using the dichotomized scale. Differences over cohorts based on the cross-sectional design and time-lag analysis are presented in the top panel, differences over the time period based on the longitudinal design and the time-lag analysis in the middle panel, and differences over age based on the longitudinal and cross-sectional designs in the bottom panel. Headings show the design and the effects that the curve confounds.

Fig. 1(a) shows the cohort differences by period, indicating cohort or age effects. The figure plots the cross-sectional prevalence by birth cohort in each survey wave. For example, the dark blue curve for 1968 shows that the prevalence of pain decreases gradually for each later born cohort from a prevalence above 60% for the 1892–1894 cohort to about 30% for the 1975–1981 cohort. The curves in general show that pain prevalence becomes successively lower for later born cohorts. There are some exceptions to this tendency; between the 1895–1904 and 1905–1914 cohorts in the 2000 wave; between the 1915–1924 and 1925–1934 cohorts in the 1991 wave; and between the 1925–1934 and 1935–1944 and the 1945–1954 and 1955–1964 cohorts in the 2000 wave; here tendencies to increases in pain prevalence are indicated instead. But overall the survey waves suggest a cross-sectional cohort dependency in which later born cohorts display successively lower pain prevalence. Due to the inherent confounding in cross-sectional data it is, however, not possible to know whether this decrease with later born cohorts is due to cohort or age effects.

The cohort differences by age Fig. 1(b) (indicating cohort or period effects) plots the prevalence by cohort at each age. For example, the light blue curve for age 45–54 indicates that the prevalence of pain in this age gradually increases with later born cohorts, from slightly less than 50% for the 1915–1924 cohort to 60% for the 1945–1954 cohort. In general, the curves indicate that in corresponding ages pain prevalence was higher for later born cohorts than for earlier born. Exceptions to this tendency is found between the 1895–1904, 1905–1914, and 1915–1924 cohorts in ages 65–74, and between the 1905–1914, and 1915–1924 cohorts in ages 55–64; where stable rates are indicated. But due to the inherent confounding in time-lag analysis it is not possible to know whether this general increase seen with later born cohorts is due to cohort or period effects. But the graphs’ indicated increase is inconsistent with the decreasing trend in the surveys shown in Fig. 1(a). This inconsistency indicates that the differences between cohorts are not due to any obvious cohort effect. That is, if there were no age or period effect present, Fig. 1(a) and (b) would have indicated a similar pattern.

We therefore continue and examine the corresponding patterns distributed by period on the x-axis. Fig. 1(c) shows the period differences by cohort, indicating period or age effects; i.e., the figure plots the prevalence by time period (calendar time) in each 10-year cohort. For example, the yellow curve for the 1905–1914 cohort shows that the prevalence of pain in this cohort increases over time from about 56% in year 1968 to above 80% in year 2002. The curves in general indicate that pain prevalence increases between 1968 and 2002. Exceptions are between 1968 and 1992 for the 1895–1904 cohort and between 1991 and 2001 for the 1925–1934 cohort. At the same time, the inherent confounding in the longitudinal analysis makes it impossible to know whether these increases in pain prevalence over the time period between 1968 and 2002 were due to period or age effects.

Fig. 1(d) shows period differences by age, indicating period or cohort effects. The figure plots the prevalence by period for each age group. In Fig. 1(d) the prevalence of pain for each age group is followed over time instead of that for cohorts in Fig. 1(c). For example, the light blue curve for age group 45–54 indicate that the prevalence of pain in this age group increases from slightly below 50% in year 1968 to slightly above 60% in year 2000. The curves indicate increases in pain prevalence between 1968 and 2002 for all age groups (except between last waves for age group 18–24, where tendency to a decrease is indicated). Due to the inherent confounding in time-lag analysis it is not possible to know whether these increases in pain prevalence were due to period or cohort effects. But at large, the trend in the time-lag analysis shows a correspondence to the longitudinal trend in Fig. 1(c). While Fig. 1(c) confounds period with age effects, Fig. 1(d) confounds period with cohort effects. To the extent that a similar trend may be extracted from these two figures this also suggests a common factor, i.e., the period effect.

Next we examine the corresponding patterns distributed by age on the x-axis. Fig. 1(e) shows age differences by birth cohort, indi-
cating age or period effects. The figure plots the prevalence by age in each cohort. The curves in Fig. 1(e) are similar to those in Fig. 1(c), except that differences have been distributed over age instead of time. The curves indicate that as people get older they more often report of pain, i.e., all cohorts showed increasing rates of pain with increasing age. The consistent increase with age seems to drop at pension age, after which it starts to increase again. (The 1895–1904 cohort, however, does not indicate this, as it only indicates decrease in the oldest ages.) Moreover, due to the inherent confounding in longitudinal data it is not possible to know whether this longitudinal age-related increase is due to age or period effects.

Fig. 1(f) shows age differences by period, indicating age or cohort effects. The figure plots the prevalence by age in each survey. The curves in Fig. 1(f) are similar to those in Fig. 1(a), but reversed and distributed over age instead of cohorts. The curves show that pain was age-dependent in all survey years; rates are higher in older age groups. (Rates in the oldest age groups in the 1991 wave and in 1992 SWEOLD are exceptions and here the oldest age groups possibly have a lower rate than the second oldest age groups.) Due to the inherent confounding in cross-sectional data it is not possible to know whether this cross-sectional age-related increase seen in all waves is due to age or cohort effects. But at large, the cross-sectional age trend in Fig. 1(f) also shows a correspondence to the longitudinal age trend in Fig. 1(e).

Age, period, and cohort effects cannot be observed directly but effects may be inferred. In the figures each graph is composed of two of the effects. When a single effect dominates, the two of the descriptions of the change over cohort, period, or age (in the top, middle, or bottom panel) will correspond. The correspondence between longitudinal Fig. 1(c) and time-lag Fig. 1(d) descriptions of the period differences in middle panel suggests that period effects are present. But in the bottom panel the longitudinal Fig. 1(e) age and cross-sectional Fig. 1(f) age differences also show correspondence, indicating an additional presence of age effects.

The figures thus suggest the presence of both period and age effects, but no obvious cohort effect. This result indicates the necessity to use regression models to partition the effects.

3.2. Models

To evaluate the effects closer regression models were therefore applied. Whereas, the graphs present dichotomised results and the models estimate odds for the scale. In Table 1, Model I includes period and age comparisons, Model II includes period and cohort comparisons, while Model III includes age, period, and cohort comparisons. The first row in each model shows the period changes, which are modelled in average across the different age groups and cohorts.

In Table 1 Model I could be said to correspond to Fig. 1(f) as it contrasts the different age groups with each other within each survey wave. The first row for period shows the relative odds for the earlier waves in comparison with the reference category, the 2000/2002 wave. The estimate (OR = 0.53) shows that the odds for pain were almost half the size in 1968 in comparison with those in 2000 (for the population aged 18–76). Larger ORs for later waves indicate that odds for pain increased over time. Since Model I includes estimates of age changes, these estimates of period change were adjusted for age. At the same time, these “period” comparisons correspond to a time-lag analysis, which confounds period with cohort effects.

In Model I, the first column for age shows the different age groups’ relative odds in 1968, the second column in 1974, the third in 1981, etc. The odds for the different 10-year age groups are compared against the age groups’ average odds (in each wave) – instead of being compared against a reference category. In 1968 the ORs indicate that older age groups have higher odds, while younger age groups have odds lower than the average. The ORs indicate that odds increase gradually with increasing age. The second column indicates a similar pattern for 1974, and the other columns for later waves show similar patterns.

The estimated values for the oldest age groups 77–84 and 85+ in 1992 and 2002 have been set within parentheses. This is to show that the values for these age groups have been excluded from the estimations of reference value, i.e., the average odds of all age groups in ages 18–76 in each wave. This ensures that the same age ranges are compared in all years in row one for the ORs of period. At the same time, these age groups’ estimated ORs are also relative to the average odds of ages 18–76 in each wave. It can therefore be read similarly, e.g., OR 2.29 for age group 77–84 in 2002 suggests 2.29 times higher odds than in average for persons aged 18–76 this year.

Because the lower age limit was raised in later surveys, Model I also includes controls for age groups 18 years and below in 1968, 1974, 1981, and 1991 (for which estimates are shown as controls). This ensures that the same age range (19–76) is compared in all years in row one for the ORs of period. In order not to truncate the first observations of the youngest cohorts included in following models the controls were included (instead of excluding data) to make Model I compatible to Model III. (The significant estimate among these controls indicates that in 1981 persons aged 15–17 in 1981 had odds for pain below the average in the age group 15–24.)

Since Model I includes estimates of period change, the estimates of age changes were adjusted for period change. At the same time, these comparisons between age groups correspond to a cross-sectional analysis which confounds age with cohort effects. Both kinds of comparisons included in Model I thus lack the critical adjustment for cohort effects.

In Model II the first row shows ORs for period. The odds for pain in 1968 were about a fourth the size of those in 2000/2002 – for the four cohorts that could be followed over the whole time period from 1968 to 2000/2002, the 1905–1914, 1915–1924, 1925–1934, and 1935–1944 cohorts. Larger ORs for later waves indicate that odds for pain increased with time. Since Model II includes the comparisons between cohorts these estimates of period change were adjusted for cohort effects. At the same time, these “period” comparisons correspond to a longitudinal analysis confounding period with age effects.

The estimated period change varies considerably from Model I to Model II. While the period change was adjusted for age in Model I, the period change is adjusted for cohort in Model II. The period changes included in Model I confound period and cohort effects, but the period changes in Model II confound period and age effects instead.

In Model II, the first column shows the different cohorts’ relative odds in 1968. The odds for the different 10-year cohorts are compared against the cohorts’ average odds. Earlier born cohorts had higher odds than average, while later born cohorts had odds lower than average. There is a suggestion of an increase, with later born cohorts having successively lower odds. In column 2, for 1974 the ORs indicate a similar pattern, and the other columns for later waves also indicate fairly similar patterns (least so for 1991/1992 wave where fewer of the cohorts deviated significantly from the average).

The estimated values for the cohorts – other than the four cohorts followed over the whole period from 1968 to 2000/2002 – have been set within parentheses. This is to show that the values for these cohorts have been excluded from the estimations of reference value, i.e., the average odds for the four (1905–1914, 1915–1924, 1925–1934, and 1935–1944) cohorts in each wave. This ensures that the same cohorts are compared in all years in...

Table 1

Models of odds for musculoskeletal pain using ordered logistic regressions, with age, period, and cohort as independent variables.

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<td>1892–1894</td>
<td>2.92***</td>
<td>7.24***</td>
<td>(2.10***</td>
<td>(0.97, –)</td>
<td>(–, –)</td>
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<td>(–, –)</td>
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<td>(2.10)</td>
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<td>(0.32)</td>
<td>(0.20)</td>
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<td>1905–1914</td>
<td>1.02</td>
<td>0.93</td>
<td>0.73***</td>
<td>0.64***</td>
<td>0.78</td>
</tr>
<tr>
<td>1915–1924</td>
<td>0.94</td>
<td>1.04</td>
<td>1.03</td>
<td>0.74***</td>
<td>0.96</td>
</tr>
<tr>
<td>1925–1934</td>
<td>0.99</td>
<td>0.96</td>
<td>1.16</td>
<td>1.35***</td>
<td>0.93</td>
</tr>
<tr>
<td>1935–1944</td>
<td>1.05</td>
<td>1.08</td>
<td>1.14</td>
<td>1.56***</td>
<td>1.43***</td>
</tr>
<tr>
<td>1945–1954</td>
<td>(0.91)</td>
<td>(1.01)</td>
<td>(1.37)</td>
<td>(1.57)</td>
<td>(1.57)</td>
</tr>
<tr>
<td>1955–1964</td>
<td>(1.36)</td>
<td>(1.54)</td>
<td>(2.16)</td>
<td>(2.34)</td>
<td></td>
</tr>
<tr>
<td>1965–1974</td>
<td>(1.15)</td>
<td>(2.31)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1975–1981</td>
<td>(2.21)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controls (a, b)</td>
<td>(0.97, 1.13)</td>
<td>(0.89, 0.91)</td>
<td>(0.63, 1.52)</td>
<td>(1.15, –)</td>
<td>(–, –)</td>
</tr>
</tbody>
</table>

*p < .05.
**p < .01.
***p < .001.

The 2000/2002 wave is the reference category (OR = 1.0). The ORs for earlier waves show the period estimate in average over the different age groups.

The ORs estimate the odds for each age group in comparison with the age groups’ average odds (in each wave).

Controls, i.e., values within the parentheses are excluded from the estimations of the average odds, which are compared over survey years and presented as a period effect. These controls ensure that the same age range is compared in all years.

The model also includes controls for age groups 18 and below (a), and 17 years and below (b). In Model I these controls of the lower age limits in earlier survey waves ensure that the same age range is compared in all waves. To be stringent these controls were also kept in Model II and Model III.

Period estimates in average over the different cohorts.

The ORs estimate the odds for each cohort in comparison with the cohorts’ average odds (in each wave).

Controls, i.e., all values within the parentheses are excluded from the estimations of the average odds, which are compared over survey years and presented as period effects. They ensure that the average against which the cohorts are compared was only based on the four cohorts followed in all waves.

Controls, odds for the truncated cohorts, i.e., because of the irregular waves these estimates are only based on some of the birth years which should have been included in the 10-year birth span of the cohorts.

The linear estimate shows the relative odds for an increase of 10 years in age.

row one for the ORs of period. At the same time, ORs for the other cohorts are also relative to these average odds (of the four cohorts) in each wave. It can therefore be read similarly, e.g., OR 2.92 for 1892–1894 cohort in 1968 suggests 2.92 times higher odds than in average for the 1905–1944 cohorts in the first survey year. The model also includes some cohort estimates that were truncated by the irregular waves. These estimates were only based on some of the birth years within the 10-year birth span of the cohorts and they represent controls.

Since Model II includes estimates of period change, the estimates of cohort ORs were adjusted for period change. At the same time, these comparisons between cohorts correspond to a cross-sectional analysis which confounds cohort with age effects. Both kinds of comparisons included in Model II thus lack the critical adjustment for age effects.

In the final Model III the age changes have been included in addition to the cohort and period changes in Model II. It thus represents a full age–period–cohort model. Model I indicates that it is reasonable to include a linear estimate of the increase in odds with increasing age. This is indicated by each of the survey years. The last model, Model III, shows the same variable estimates as Model II but adjusted for the increase of pain that were due to aging, i.e.,

the average linear age-related increase that was common to all observations. The estimated age effect (OR = 1.47) suggests that the odds in average increased one and half times for each decade older people became.

The estimated period effect, i.e., the increase of the odds (OR = 0.96) between 1968 and 2000/2002 – for the four cohorts followed longitudinally – indicates that the odds for pain were not in average higher in 2000/2002 than they were in 1968. The period ORs are considerably different in Model III in comparison with those in Model I or Model II. While the period ORs were adjusted for either age as in Model I or cohort as in Model II, the period ORs are adjusted for both cohort and age in Model III. While the “period” comparisons in Model II confound period and age effects, the period comparisons included in Model III have adjusted for age in addition representing the pure period effect.

Similarly, since Model III includes adjustments for period and age the ORs for cohorts now captures the pure cohort effect. In Model III, the first column shows the different cohorts’ odds in comparison with the average odds in 1968. Since no ORs are significant the model indicates no significant differences between cohorts at baseline in 1968. This is a considerable change from Model II where older cohorts had higher odds than younger cohorts in 1968, indicating that ORs for cohort in Model II captured age and not cohort effects. In later waves and in 2000/2002 especially the direction even becomes the opposite. Whilst the later born cohorts had lower odds than earlier born in Model II, the age adjustment in Model III shows that later born cohorts in fact had higher odds than earlier born in later surveys.

Since our modelling includes interaction effects between periods and cohorts this also means that we have allowed the period effects to vary across cohorts having differing effects on different cohorts, relaxing the otherwise quite strict assumption about a period effect – that it has the same effect on all. Indirectly, the odds for the 1935–1944 cohort shows a significant period effect between 1968 and 2000, as odds were significantly higher than average in 1991 and 2000 (whereas in 1968 odds were about average, OR = 1.05). Similarly, for 1925–1934 cohort odds were significantly higher in 1981 and 1991, but then in 2002 odds were significantly lower than in average. After entering the study in 1974 with about average odds, the 1945–1954 cohort had odds above average in later waves. Even later born cohorts also entered the study with odds that were significantly higher than in average.

The comparison of the period changes in Model I encompassed a larger segment of the sample than in Model II and Model III, where the average OR estimate for period change was limited to the four cohorts that could be followed the whole time period from 1968 to 2000/2002. The analysis also shows that the younger cohorts are essential to understand the development over the time period between 1968 and 2000/2002. Model III indicates that the period change captured by the OR 0.53 for 1968 in comparison with 2000 in Model I can be ascribed to a period change occurring after 1974 in the 1935–1944 and 1945–1954 cohorts, as well as to the later born cohorts who entered the study in age 18 with a higher prevalence of pain than earlier cohorts had had in corresponding age.

We also examined whether increase in odds for age as was curvilinear in Model III. This is a pattern that has been suggested by some studies [7,10,23], and it is indicated by some of the survey waves, i.e., the 1991 and possibly the 2000 wave in Fig. 1(f). This pattern was also indicated longitudinally by several cohorts shown in Fig. 1(e). A quadratic and a cubic term of age were therefore included in the last model. Results were not significant (with a p = 0.06 for the cubic term). The other estimates were not much altered by allowing the age effect to be curvilinear. Since there was no significant support of a curvilinear age effect, this model was not presented in the table.

Lastly, we examined whether the pattern in Model III differed by gender. Though women had significantly higher odds for pain than men (OR = 1.39, p < 0.05), patterns were similar. Since patterns were similar separate models for men and women are not presented.

4. Discussion

The results suggest that, at large, age effects dominate musculoskeletal pain patterns. The odds to have pain became about one and a half times higher for each decade older people became.

In the age–period–cohort model, there were no pronounced cohort differences at baseline in 1968. Thus, the results do not suggest cohort effects in pain (given that cohort effects are interpreted as differences deriving from a time point preceding baseline).

While analyses also identified changes in the pain prevalence between 1968 and 2000 in the adult population aged 18–76, for the cohorts followed longitudinally there was no period effect (in average), as the whole increase in rates could be ascribed to the age effect.

Instead, the results suggest that the increase in pain prevalence (in the adult population) was largely due to the new cohorts that had entered the study after baseline – with higher rates of pain than earlier cohorts had had in corresponding age. Yet, some of the cohorts followed longitudinally, i.e., foremost the 1935–1944 cohort, did show a significant period effect, demonstrating an increase in pain rates over time that was above the increase of pain that was to be expected by age alone. After entering the study in 1974 with about average odds, the 1945–1954 cohort later also showed an increase in pain, i.e., a period effect.

The above-mentioned pattern can be interpreted as an interaction between cohort and period effects. With the 1940s cohorts – for which pain increased over the period – there also came general increase in pain prevalence as later cohorts entered the study with higher initial levels of pain.

In separate analyses, the different pain sites presented similar patterns. Significant chi-square score tests also suggest that the pattern was same over the scale’s different levels of pain.

The analysis confirmed the presence of strong age effects in pain. Increasing pain could be a natural consequence of aging of the organism, possibly due to osteoarthrosis, reflecting the wear and tear of the musculoskeletal apparatus. But differences in pain reporting may be a result of differences in attitudes and reporting behavior, as well as changes in the presence of pain.

Studies that have examined musculoskeletal pain prevalence in the population have found increases [20,24,28,29]. Our results show that pain reporting increased with the passing of the 1940s cohorts through the 1970s and 1980s. This pattern corresponds to what in public debate often is referred to as cohort effects – the larger (societal) change that occurs with the passage of a particular birth cohort through age and time [31]. Similar to the US “baby-boomer” generation, in Sweden people born in the 1940s have been identified as a generation that has changed society with its passage through age and time [31]. Possibly, the social pressure to maintain stoicism decreased with the passing of this generation through age and time.

On the other hand period change is often attributed to societal changes, such as the structural change that the society went through the second half of the 20th century. For example, the composition of the work force and the work content has changed and it may have affected generations differently. Pain prevalence has been shown to vary with occupation and social class [34,36,51]. There was also a suggestion of an age-related decrease at pension age, even if a curvilinear age effect could not be significantly

supported empirically. This drop of pain levels at pension age could be related to leaving working life. The normal retirement age in Sweden is 65 years, although many people retire before this. It is possible that the decrease in pain may thus be explained by improvement of those individuals with work-related aches and pain.

In the oldest ages musculoskeletal pain is closely associated with functional limitations [1]. In an earlier study based on the same material we showed that the rate of mobility limitations decreased between 1968 and 1992, which indicates that pain and limitations display different overtime trends in the population [2]. Even so the increase in pain over time is likely to be related to trends in other interrelated conditions or risk factors, such as in obesity [17,32,41].

We did not find any evidence of a differing age–cohort–period pattern for the genders, but we replicated what most studies find – higher levels of pain among women. These results could be due to different ways of talking about or reporting of pain or they could reflect real differences in the occurrence of pain. However, these results correspond to the clinical experience that women more often than men seek medical treatment for pain problems.

Our description of the generational patterns in pain indicates that regular epidemiological analyses of the overtime trends may need to be complemented with age–period–cohort analysis of patterns. In this case, regular analysis (i.e., cross-sectional and repeated cross-sectional designs) only shows that pain is strongly age related and that it increased over time – most noticeable for younger age groups [20]. Besides confirming the strong age effect, our analysis elaborates these results. Our analysis indicates that it was with the passing of the 1940s generation through age and time that the increase in pain reporting occurred (later cohorts entering adulthood and the study with higher initial levels of pain). With the arrival of the 1940s generation to the oldest age groups our results indicate that pain is likely to increase in these ages. Yet, in regular repeated cross-sectional analysis only the increase of pain reporting in younger ages is apparent, suggesting a different prognosis.

The composition of the population being studied is always changing in ways that may not be well understood. For example, immigration may increase or decrease the population’s risk for certain diseases. Mortality steadily changes the risk composition of the population, because those who die (and are thus removed from consideration) may have been at greater or lower risk of the diseases of interest than the surviving population. Different cohorts not only have different life histories and exposures to risk factors, prevention programs, and treatment methods, but even those exposures which they share with other cohorts may have been at different ages in the different cohorts.

The present study minimizes some of the above concerns by being done in Sweden, where the population pyramid has been more stable than in many countries, and both immigration and emigration have been relatively modest and stable.

The present study shares most of the technical properties with two earlier studies based on the same material. In these we found other patterns, with cohort effects dominating dental health and care, and age effects dominating mobility [3,4]. The differing results indicate that the present result is not a result of survey technique, i.e., any general flaw in the study’s technical properties. But these properties still represent possible sources of bias, e.g., measurement problems, non-response, and representativity.

Pain is defined and rated in a quite general way and the period (12 months) of perceived pain symptoms is quite long. Thus, the prevalence becomes quite high and the clinical relevance of these symptoms may seem questionable. These symptoms may include various symptoms which do not necessarily have much impact on an individual’s function or perceived health status. The prevalence reported in this study could therefore refer to different thresholds to perceive and report of this kind of symptoms rather than to the clinical impact of musculoskeletal disorders. Notably different measurement instruments are likely to have different sensitivity among other properties.

A reasonable assumption is otherwise that possible measurement error is randomly distributed among the aggregated individuals, making the prevalence estimates unbiased. But even if prevalence estimates would be biased, the over time comparisons could be unbiased. If measurement error remains the same over time, this would be the case. That measurement error is equally distributed over time or age are straightforward and usual model assumptions.

Inconveniently, it is not possible to disentangle age, period and cohort by empirical analysis alone [16,22]. Our results could also be due to another constellation of the effects, in which some cancelled each other out, partly or wholly. For example, our estimated age effect could in fact be the result of an even stronger age effect that period effects successfully countered throughout the examined period. Unfortunately, such alternative solutions cannot be ruled out. It represents the so-called identification problem, common to all approaches that try to identify age, period, and cohort effects. But a better understanding of conditions and effects across countries and cultures will enable us to evaluate changes more accurately.

The results demonstrated a strong age effect in musculoskeletal pain. With the passage through age and time of the 1940s generation, pain prevalence increased generally for this and later born cohorts.

Conflict of interest

The authors declare that there are no financial or other relationships.

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