Chronic Widespread Pain: A Three Year Followup of Pain Distribution and Risk Factors

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ABSTRACT. ***Objective.*** To describe the change of pain reports over time in 3 cohorts derived from the general population: (1) no chronic pain (NCP; n = 1156); (2) chronic regional pain (CRP; n = 502); and (3) chronic widespread pain (CWP; n = 242). To identify risk factors that predict the development or persistence of chronic widespread pain.

Methods. A 3-year followup from 1995 to 1998 with postal questionnaire to 2425 subjects of both sexes aged 20-74 years on the west coast of Sweden.

Results. At followup, a larger proportion of subjects with initial CRP compared to initial NCP reported CWP (16.4 and 2.2%, respectively; $p < 0.001$). The majority of subjects (56.9%) who primarily reported CWP remained in that group at followup, but 26.8% had changed status to CRP and 16.3% to NCP. The number of painful regions (7-12 vs 0 regions) reported at baseline was the strongest predictor for the development of CWP with an odds ratio (OR) of 12.13 (95% CI 4.47-32.88). The development of CWP was also predicted by higher age (OR = 3.13, 95% CI 1.47-6.69, age-group 59-74 years vs age-group 20-34 years), and a family history of chronic pain (OR = 1.87, 95% CI 1.14-3.07). A habit of drinking alcohol weekly (OR = 0.42, 95% CI 0.21-0.85) compared to the habit of never or seldom drinking alcohol was protective, as well as having personal social support (OR = 0.49, 95% CI 0.28-0.85). The persistence of CWP was predicted by the number of painful regions (13-18 vs 1-6 regions) at baseline (OR = 7.56, 95% CI 2.17-26.30), and being an immigrant (OR = 3.22, 95% CI 1.33-7.77).

Conclusion. Although the overall prevalence of CWP was stable over a 3-year period there was a considerable variation on an individual basis. This variability in expressing CWP was moderately predicted by a combination of risk factors, the most important being the number of painful regions at baseline. Future research will need to show how useful the identified factors are in clinical practice and whether intervention aimed at changing these factors will improve pain outcome. (J Rheumatol 2002;29:818–25)

Key Indexing Terms:
COHORT STUDIES MUSCULOSKELETAL SYSTEM PAIN
PREVALENCE RISK FACTORS

Chronic musculoskeletal pain is common in the general population, with a prevalence of 35-50%, according to several studies from Western Europe and the USA[1-5]. Chronic widespread pain (CWP) and especially the subgroup fibromyalgia (FM) have been a challenge regarding the understanding of its etiology, diagnosis, and treatment. Recently the focus in FM research has shifted from the peripheral muscles[6] to neurohormonal changes in the brain[7]. The initial mechanisms behind these changes are however still mostly unknown. It has been proposed that it is a consequence of longstanding stress and that there is an interaction of physical, psychological, social and neurobiological factors[8]. It has also been questioned if the subgroup FM is to be considered as a disease entity or if it is one end of a continuous scale of somatic expressions of distress[9,10]. Little is known about the initiation and longitudinal course of CWP and to what extent regional pain proceeds to chronic widespread pain[11].

In a prior cross-sectional study[12] we found that CWP was associated with female sex, older age, lower socio-economic group (according to occupation), being an immigrant, living in a socially compromised housing area, lower educational level, never or seldom drinking alcohol, not having personal support and a family history of chronic pain. Overall the factors studied were more frequently and strongly associated with CWP compared with chronic regional pain (CRP). This suggested different pathophysiology of CRP and CWP and that these factors could be operating as predictors either in the development or in the persistence of CWP.

This study is a 3 year followup of 3 cohorts derived from a general population sample: one cohort with no chronic pain (NCP), one with chronic regional pain (CRP), and one

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Submitted May 10, 2001; revision accepted September 26, 2001.

with chronic widespread pain (CWP). The aim was to study the longitudinal course of CRP and CWP and to what extent CRP proceeded to CWP. A second aim was to identify risk factors that predicted the development or persistence of CWP.

MATERIALS AND METHODS

Design. The study was designed as a 3 year followup with a postal questionnaire in May 1998 to 2425 subjects that initially responded to a cross sectional postal survey in May 1995.

Subjects. The target population was all 70,704 inhabitants aged 20-74 in Halmstad and Laholm, 2 municipalities and healthcare districts on the west coast of Sweden. Halmstad is a middle-sized town (83,488 inhabitants December 31, 1995) with rural surroundings. Occupational opportunities are dominated by commerce, sea trading, light industry, military and civilian education and health services. Laholm is a smaller town (23,120 inhabitants Dec 31, 1995) where occupations within light industry and agriculture dominate.

In 1995 a sample of 3928 subjects aged 20-74 years and representative for the target population was selected from the official computerized population register. This register is sorted by day of birth. In each of the 2 municipalities every 18th man and woman were selected. There were 2425 subjects (62%) who responded to the initial questionnaire in May 1995 and they received a new postal questionnaire in May 1998.

Questionnaire. Identical questionnaires, with the exception of questions concerning other health problems and socio-demographic data, were used in 1995 and in 1998. The questionnaire was divided in 2 parts (the first being the well established Medical Outcome Survey Short Form-36) and this study was based on results from the second part, which consisted of 52 questions with focus on the experience and location of chronic pain in the musculoskeletal system. The questionnaire was piloted and validated in a group of 50 subjects, 25 subjects from an out-patient rheumatic clinic and 25 subjects attending a primary care center. Several questions and the method of recording pain distribution had also been used in prior studies[5,13].

There was an overall key question on chronic musculoskeletal pain: Have you experienced pain lasting more than 3 months during the last 12 months? It was explained in an introduction to the question that the pain should be persistent or regularly recurrent in the musculoskeletal system. Location and distribution of pain was reported by a drawing of the body with predefined regions (Figure 1). Each region was also described by name, and pain was reported by ticking appropriate boxes next to the descriptions.

There were also questions on socio-demographic background factors. Educational level was dichotomized in 2 groups, more than 2 years of education after comprehensive school or not. Based on their occupation the responders were classified according to Swedish socio-economic classification, SEI[14]. The 18 basic socio-economic classes were merged to 4 groups: manual workers, assistant non-manual employees, intermediate/higher non-manual employees including upper-level executives, and others. The others included self-employed, farmers, housewives and students. The self-employed subgroup was heterogeneous and included subjects with both manual and non-manual work. Immigration status (immigrant or native-born Swede) was evaluated from the question: Have you or your parents moved to Sweden from another country? The country could also be specified. Personal social support was evaluated from the question: Do you feel that you have one or more persons that can give you a thorough personal support to cope with distress and problems in life? Smoking habit was reported by 3 multiple-choice alternatives to the question: Do you smoke? The 3 alternatives were: I have never smoked, I have stopped, and yes. Alcohol habits were reported by 5 multiple-choice alternatives to the question: How often do you drink alcohol including beer, wine and spirits? The alternatives were: never, very seldom, monthly, weekly, and daily. In the analyses and presentation these

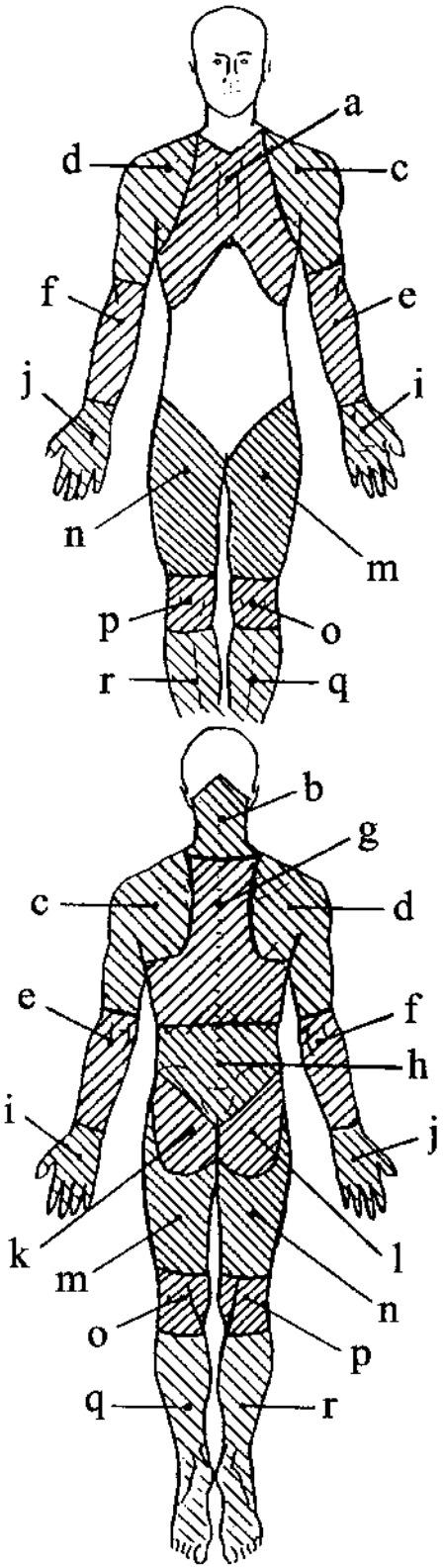

Figure 1. Pain drawing with predefined body regions used in the questionnaire. Letters refer to corresponding explanations given in the questionnaire.

were merged into 3 groups (never/seldom, monthly and weekly/daily) due to small number of subjects in especially the daily group (n = 15). The amount of alcohol intake was not recorded. Family history of chronic pain was as assessed with one question: Do you have someone close to you (parents or siblings) with chronic pain?

Definitions of chronic pain. In 1990 the American College of Rheumatology (ACR) presented criteria for FM[15]. These include criteria for chronic widespread pain that could be adapted for use in a postal survey. In addition to CWP, the diagnostic criteria of FM also include an abnormal number of "tender points." This diagnosis would thus require physical examination and the prevalence of FM could therefore not be estimated in the present study.

Pain was considered to be chronic if it had been persistent or recurrent for more than 3 months during the last 12 months. CWP was evaluated from the drawing in Figure 1. According to the 1990 ACR criteria, pain was considered widespread when present in both the left and right side of the body and also above and below the waist. Shoulders and buttocks were considered separately for each side. In addition axial skeletal pain (i.e. in the cervical spine, the anterior chest, the thoracic spine or the lower back) should be present. When chronic pain was present but criteria for a widespread condition were not met, the subject was regarded as having chronic regional pain.

Ethics. The study was approved by the Ethics Research Committee, Faculty of Medicine, University of Lund, Sweden. The computerized registration was approved by the Swedish Data Inspection Board.

Statistics. The statistical analysis was done with the statistical package SPSS. Statistical comparison of prevalence was done by 2-sided chi-squared test. Age-adjusted and age-sex-adjusted prevalence rates were adjusted by the direct method using the Swedish census population of 1997 as a standard. Multivariable logistic regression analyses with computation of odds ratios (OR) were performed with simple contrast to a reference group for each of the variables. The analyses were checked for interactions between gender and all the other variables. In the multivariable analyses only those with non-missing data on all variables were included. Choosing the "don't know" alternative for family history of chronic pain was not considered a missing value.

RESULTS

There were 1922 subjects responding to the 3-year followup questionnaire in 1998, which constituted 79% of the 2425 that responded to the first survey in 1995. The respondents were grouped in 4 categories according to their response (Table 1): no chronic pain, chronic regional pain, chronic widespread pain, and a group with subjects that could not be categorized (Unknown). There were no significant ($p < 0.01$) changes in the prevalence rates of CRP or CWP over the 3 years.

The transition of subjects between the 3 main categories from 1995 to 1998 is shown in Figure 2. Seventy subjects classified as Unknown in 1995 or 1998 were excluded in this analysis, giving 1852 subjects that could be evaluated. At followup, a significantly larger proportion of subjects with initial CRP compared to subjects with initial NCP (16.4 vs 2.2%; $p < 0.001$) had developed CWP. The majority of subjects (56.9%) who primarily reported CWP remained in that group at followup, but 26.8% had moved to CRP and 16.3% to NCP.

The initial number of regions with pain in 1995 predicted the outcome in 1998 (Table 2). The risk to present CWP in 1998 showed a highly significant trend ($p < 0.001$) with increasing number of painful regions at baseline in 1995. There was an inverse but equally strong trend ($p < 0.001$) concerning NCP.

Sociodemographic factors that in 1995 were found to be associated with either CRP or CWP were older age, female sex, lower socio-economic group, being an immigrant, living in a socially compromised housing area, lower educational level, smoking habit, alcohol consumption, personal support, and having a family history (parents or siblings) of chronic pain[12]. These factors were now introduced in 2 multivariable logistic regression analyses.

Developing CWP from NCP or CRP (Table 3) was significantly ($p < 0.05$) predicted by the oldest age-group and having a family history of chronic pain independent of other variables. A weekly/daily habit of drinking alcohol and having a personal social support were protective. The highest OR was noted for the presence of more than 6 painful regions at baseline 1995. There were no significant interactions between sex and the other evaluated variables. The protective effect of alcohol was also tested in a model that included the use of analgesics with similar effect (data not shown). The positive predictive value of this model was 17%, which could be compared to the proportion developing CWP of those having CRP or NCP at baseline (6%). The persistence of CWP (Table 4) was significantly ($p < 0.05$) and independently predicted by being an immigrant and having more than 12 regions with pain at baseline 1995. There was a significant interaction between sex and family history of chronic pain, where family history was predictive in women (OR = 3.1, 95% CI 1.3-7.4) but not in men (OR = 0.4, 95% CI 0.1-1.9). The positive predictive value of this model was 71%, which could be compared to the proportion with persistence of CWP (57%).

DISCUSSION

This study was a 3-year followup of 3 cohorts derived from a general population sample: no chronic pain, chronic

Table 1. The distribution of subjects with age- and sex-adjusted prevalence (95%CI) in 1995 and 1998 in the 3 studied groups and in the group with subjects who could not be categorized (Unknown).

	Total	No Chronic Pain		Chronic Regional Pain		Chronic Widespread Pain		Unknown	
Year	n	n	% (95% CI)	n	% (95% CI)	n	% (95% CI)	n	% (95% CI)
1995	2425	1466	61.7 (58.5–65.0)	588	23.9 (22.0–25.9)	303	11.4 (10.1–12.6)	68	3.0 (2.2–3.7)
1998	1922	1156	62.3 (58.5–66.0)	502	25.3 (23.0–27.6)	242	11.2 (9.8–12.7)	22	1.2 (0.07–1.7)

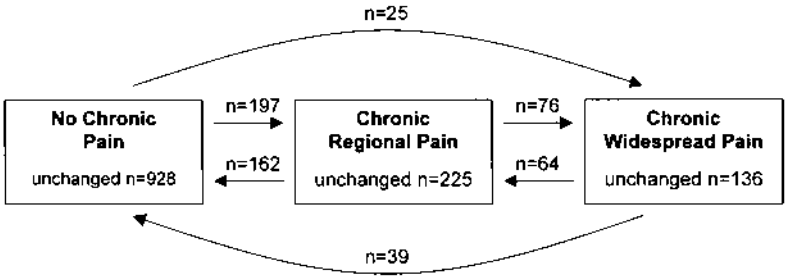

Figure 2. The 3 pain groups with transition of subjects to and from the different groups between 1995 and 1998. Seventy subjects classified as Unknown in 1995 or 1998 were excluded (n = 1852).

Table 2. The pain group outcome at 3-year followup in relation to initial number of regions with pain.

No. of Regions with Pain 1995	n	Pain Group Outcome in 1998 NCP %	CRP %	CWP %
0	1150	80.7	17.1	2.2
1–3	275	43.3	48.4	8.4
4–6	227	23.3	44.9	31.7
7–9	105	17.1	34.3	48.6
10–12	61	14.8	24.6	60.7
13–15	15	6.7	6.7	86.7
16–18	18	0.0	11.1	88.9
Total	1851	60.9	26.3	12.8

NCP = no chronic pain; CRP = chronic regional pain; CWP = chronic widespread pain.

regional pain, and chronic widespread pain. There were no changes in the prevalence rates of CRP or CWP over the 3 years but a substantial proportion (30.4%) of the subjects moved between the 3 categories. The development of CWP from NCP or CRP during a 3-year period was predicted by higher age, a family history of chronic pain, and the number of painful regions at baseline. A weekly/daily habit of drinking alcohol and having personal social support was protective. The persistence of CWP was predicted by being an immigrant and the number of painful regions.

It has been questioned to what extent chronic regional pain leads to chronic widespread pain[11]. The longitudinal studies are few and small. In one study of 141 subjects assessed twice 27 months apart, 19% of those with CRP at baseline reported CWP at followup[16]. Andersson, *et al* reported that 10% of subjects with local neck-shoulder pain had developed generalized pain in a 24-month followup of 165 subjects[17]. In this study the major pathway to CWP was from the group with CRP 3 years before. Only a small proportion of subjects with NCP developed CWP. It is not possible from this study to evaluate if the pathway directly from NCP to CWP exists or if these subjects have passed through a period of CRP. The number of reported painful regions at baseline was the most important predictive factor both for the development of CWP from CRP and for the persistence of CWP. These findings indicate the presence of a pain spectrum where the division of subgroups such as CRP and CWP could be artificial. This is also in agreement with what has been reported from other studies[10]. Accordingly, it could be questioned if a pain syndrome such as FM should be regarded as a clinical entity[9].

The large proportion (42.8%) that no longer had CWP at followup was in concordance with a study from Manchester recently reported by McBeth, *et al*[18]. In a 12-month followup of 225 subjects with CWP (ACR 1990 definition), they found that 56% still reported CWP, 33% reported other pain, and 11% reported no pain. These figures are close to ours, although the followup time was different. The findings in our study and in the Manchester study indicated that the group with CWP was heterogeneous and that also subjects in this group move within the spectrum of pain severity. It has been reported that 25% of subjects with CWP at screening did not meet the criteria at followup some weeks or months later[19]. It may also indicate that the method used for the classification is sensitive but not specific for the more severe pain syndromes. This was proposed by our group in a previous study where only 34% of subjects that reported CWP in a postal questionnaire met the criteria of CWP at a clinical followup one year later[20]. An improved classification of CWP for epidemiological studies has been proposed from a Manchester group[21]. Results from their studies indicate that it is possible to identify individuals with more truly widespread pain than is the case by the ACR 1990 definition.

Although a spectrum of chronic pain could be identified, the results from this study and a previous work[12] suggest that there are aspects other than the quantity of pain to consider in the development of CWP. The association between sociodemographic variables and the development as well as the persistence of CWP reported in this study is an illustration of that. The association between chronic musculoskeletal pain and older age is well known from several studies[5,22,23]. In a previous study the prevalence of CWP was about twice as high in women as in men. In the present study female sex non-significantly tended to predict

Table 3. Subjects developing chronic widespread pain between 1995 and 1998 compared to those still having no chronic pain or chronic regional pain.

	No	Crude[a] Analysis (n = 1551) OR (95% CI)	p	Multivariable[b] Analysis (n = 1551) OR (95% CI)	p
Sex					
Men	740	1.00		1.00	
Women	811	1.48 (0.97–2.25)	0.066	1.33 (0.83–2.14)	0.234
Age, yrs					
20–33	392	1.00		1.00	
34–46	383	1.56 (0.78–2.85)	0.205	1.46 (0.68–3.16)	0.331
47–58	403	1.90 (0.98–3.70)	0.059	1.87 (0.88–3.99)	0.102
59–74	373	3.12 (1.66–5.86)	< 0.001	3.13 (1.47–6.69)	0.003
Socio-economic group[c]					
Group A	447	1.00		1.00	
Group B	221	1.34 (0.66–2.73)	0.413	1.40 (0.61–3.22)	0.433
Group C	685	1.88 (1.10–3.21)	0.022	1.63 (0.77–3.44)	0.205
Others	198	1.63 (0.78–3.41)	0.193	1.58 (0.66–3.80)	0.302
Immigrant status					
Swede	1408	1.00		1.00	
Immigrant	143	2.49 (1.41–4.40)	0.002	1.84 (0.96–3.54)	0.067
Housing area[d]					
All other	1518	1.00		1.00	
Area A	33	1.69 (0.50–5.72)	0.402	0.85 (0.20–3.52)	0.823
Educational level					
High	555	1.00		1.00	
Low	881	1.14 (0.72–1.82)	0.577	0.64 (0.33–1.24)	0.190
Other	115	0.44 (0.13–1.46)	0.178	0.25 (0.06–0.93)	0.040
Smoking habit					
Never	828	1.00		1.00	
Former smoker	410	1.40 (0.85–2.31)	0.190	1.44 (0.84–2.47)	0.189
Current smoker	313	1.88 (1.14–3.12)	0.013	1.65 (0.95–2.88)	0.075
Alcohol habit					
Never/seldom	498	1.00		1.00	
Monthly	671	0.63 (0.40–1.00)	0.050	0.81 (0.49–1.35)	0.417
Weekly/daily	382	0.35 (0.18–0.67)	0.001	0.42 (0.21–0.85)	0.017
Personal support					
No	177	1.00		1.00	
Yes	1374	0.36 (0.22–0.60)	< 0.001	0.49 (0.28–0.85)	0.011
Family history of chronic pain					
No	1061	1.00		1.00	
Yes	351	2.74 (1.73–4.33)	< 0.001	1.87 (1.14–3.07)	0.013
Don't know	139	1.81 (0.93–3.52)	0.081	1.10 (0.53–2.27)	0.803
Regions with pain 1995					
0	1101	1.00		1.00	
1–6	424	7.91 (4.87–12.85)	< 0.001	6.91 (4.20–11.37)	< 0.001
7–12	26	18.07 (7.05–46.30)	< 0.001	12.13 (4.47–32.88)	< 0.001

[a] Univariate except that sex is age-adjusted, age is sex-adjusted, and all other are age-sex-adjusted.[b] Adjusted for all other variables in the table. [c] Group A: Intermediate/higher non-manual employees and upper-level executives; Group B: Assistant non-manual employees; Group C: Manual workers; [d] Area A: Socially compromised area.

both the development and persistence of CWP. This could indicate vulnerability related to female sex. Another explanation could be that factors promoting CWP are more common among females[24]. Having a history of chronic pain in the family (parents or siblings) was noted to predict development of CWP. This could be due to inheritance but it could also be an effect of learned strategies for coping with problems in life. A Finnish study of widespread musculoskeletal pain in twin pairs concluded that environmental familial factors were much more important than genetic factors[25]. Associations between lack of social support and musculoskeletal pain have been described[26-28]. In this study a personal social support was protective for the development of CWP. Immigrants to Sweden are known to have more health problems than native-born Swedes[29,30]. In the present study being an immigrant was a strong predictor for the persistence of CWP over a 3-year period. With our broad definition of being an immigrant and without knowledge of

Table 4. Subjects with persistence of CWP between 1995 and 1998 compared with those with improvement to NCP and CRP.

	No	Crude[a] Analysis (n = 231) OR (95% CI)	p	Multivariable Analysis [b](n = 231) OR (95% CI)	p
Sex					
Men	71	1.00		1.00	
Women	160	1.43 (0.81–2.54)	0.22	1.39 (0.71–2.69)	0.34
Age, yrs					
20–33	17	1.00		1.00	
34–46	48	1.22 (0.40–3.71)	0.72	0.99 (0.28–3.46)	0.99
47–58	78	1.58 (0.54–4.48)	0.41	1.46 (0.44–4.86)	0.54
59–74	88	1.90 (0.66–5.45)	0.23	2.06 (0.61–6.96)	0.24
Socio-economic group[c]					
Group A	27	1.00		1.00	
Group B	40	1.05 (0.39–2.83)	0.92	0.86 (0.26–2.88)	0.81
Group C	139	1.33 (0.58–3.07)	0.50	1.36 (0.43–4.29)	0.60
Others	25	1.08 (0.36–3.28)	0.89	1.28 (0.34–4.82)	0.72
Immigrant status					
Swede	190	1.00		1.00	
Immigrant	41	3.00 (1.37–6.58)	0.006	3.22 (1.33–7.77)	0.009
Housing area[d]					
All other	220	1.00		1.00	
Area A	11	1.50 (0.41–5.46)	0.53	1.08 (0.26–4.62)	0.91
Educational level					
High	34	1.00		1.00	
Low	178	1.02 (0.47–2.20)	0.96	0.93 (0.32–2.67)	0.89
Other	19	0.52 (0.16–1.65)	0.27	0.45 (0.11–1.79)	0.26
Smoking habit					
Never	116	1.00		1.00	
Former smoker	71	1.60 (0.86–2.98)	0.14	1.43 (0.69–2.95)	0.34
Current smoker	44	1.82 (0.88–3.81)	0.11	1.51 (0.67–3.39)	0.32
Alcohol habit					
Never/seldom	129	1.00		1.00	
Monthly	66	1.04 (0.57–1.92)	0.89	1.32 (0.65–2.66)	0.45
Weekly/daily	36	0.89 (0.42–1.90)	0.77	1.10 (0.44–2.77)	0.84
Personal support					
No	56	1.00		1.00	
Yes	175	0.76 (0.41–1.43)	0.39	0.88 (0.43–1.79)	0.71
Family history of chronic pain					
No	106	1.00		1.00	
Yes	86	2.24 (1.22–4.11)	0.01	1.73 (0.89–3.38)	0.11
Don't know	39	1.49 (0.70–3.17)	0.30	1.35 (0.58–3.12)	0.48
Regions with pain 1995					
1–6	63	1.00		1.00	
7–12	136	1.80 (0.96–3.38)	0.07	1.40 (0.71–2.76)	0.33
13–18	32	9.29 (2.87–30.04)	< 0.001	7.56 (2.17–26.30)	0.001

[a]Univariate except that sex is age-adjusted, age is sex-adjusted and all other are age-sex-adjusted
[b]Adjusted for all other variables in the table
[c]Group A: Intermediate/higher non-manual employees and upper-level executives
Group B: Assistant non-manual employees
Group C: Manual workers
[d]Area A: Socially compromised area (see text)

the reason for migration (labor migration or refugee) or which cultural differences that may exist, it's not possible in this study to conclude the cause of this association.

The negative association between a more frequent use of alcohol and the development of CWP is problematic to interpret. It could indicate that there is a protective effect of alcohol but it could also be confounded by for example that these subjects had health problems at baseline that made them refrain from alcohol. One possible theory for a biological effect is that alcohol is an antagonist to the NMDA receptor in the spinal cord[31,32]. Activation of NMDA receptors in the spinal cord has been shown to result in central sensitization, increasing the neurons' response to pain stimuli[33].

We have not assessed or evaluated any psychological factors in this study. In a recent population-based study[34],

CWP was reported to be associated to mental disorders. The direction of cause and effect was not possible to establish in that study. McBeth, *et al* found that subjects who one year earlier displayed features of somatization were more likely to have persisting CWP at followup. Psychological assessment could thus be an important issue for future longitudinal studies on potential risk factors for CWP.

A possible problem with this study was a selection bias due to non-response especially in the first cross-sectional survey. In an analysis of the non-responders in our prior work, we found that people with chronic pain were more prone to respond than people without chronic pain, giving a higher estimate of the prevalence. In this followup of the previously defined cohorts the response-rate was 79%. The response-rate differed between sexes ($p = 0.03$), with women having a response-rate of 81% and men 77%. The respondents' mean age was 3.4 years higher ($p < 0.001$) than that of non-respondents. Respondents and non-respondents showed no difference in the distribution of subjects between the 3 studied subgroups. The response-rate was not supposed to significantly bias the results from the longitudinal analysis.

Another possible problem was misclassification of subjects with respect to pain sub-groups or sociodemographic factors. The material was thoroughly checked for errors, and subjects that could not be classified were excluded from the analyses. Misclassification of different exposure was likely to be non-differential between the 3 cohorts. One exception might be family history of pain. A recall bias could be expected with a high reported frequency among subjects presently experiencing chronic pain.

Another problem could be the validity of reports on frequency of alcohol consumption. It is not unlikely that subjects with high alcohol-consumption report a lower figure. This was also a reason for not including questions on the amount of alcohol intake in the questionnaire. The problem was likely to be non-differential between the 3 cohorts. There was also a validity problem with the alternative, No I have stopped, to the question on smoking habits, as this didn't measure the time of smoking cessation. This alternative was treated separately in the analyses.

To conclude, the overall prevalence of CWP was stable over a 3-year period, although there was a considerable variation in the individual status. Although the extent of pain was the most powerful risk factor, a number of other predictors were also identified. Future research will need to show how useful the identified factors are in clinical practice to identify individuals at risk and whether intervention aimed at changing these factors will improve pain outcome.

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