Rheumatology 2007;46:666–671
Advance Access publication 4 November 2006
doi:10.1093/rheumatology/kel363

The role of psychosocial factors in predicting the onset of chronic widespread pain: results from a prospective population-based study

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***Objective*. Chronic widespread pain (CWP) is strongly associated with psychosocial distress both in a clinical setting and in the community. The aim of this study was to determine the contribution of measures of psychosocial distress, health-seeking behaviour, sleep problems and traumatic life events to the development of new cases of CWP in the community.**

***Methods*. In a population-based prospective study, 3171 adults aged 25–65 yrs free of CWP were followed-up 15 months later to identify those with new CWP. Baseline data were available on their scores from a number of psychological scales including Illness Attitude Scales (IAS), Somatic Symptom Checklist (SSC), Hospital Anxiety & Depression Scale, Sleep Problems Scale, and Life Events Inventory.**

***Results*. 324 subjects [10%, 95% confidence interval (CI) 9.2, 11.3] developed new CWP at follow-up. After adjustment for age and sex, three factors independently predicted the development of CWP: scoring three or more on the SSC [odds ratio (OR) 1.8, 95% CI 1.1, 3.1], scoring eight or more on the Illness Behaviour subscale of the IAS (OR 3.3, 95% CI 2.3, 4.8), and nine or more on the Sleep Problem Scale (OR 2.7, 95% CI 1.6, 3.2). Subjects exposed to all three factors were at 12 times the odds of new CWP than those with low scores on all scales.**

Conclusion. Subjects are at substantial increased odds of developing CWP if they display features of somatization, health-seeking behaviour and poor sleep. Psychosocial distress has a strong aetiological influence on CWP.

KEY WORDS: Pain, Psychosocial, Risk factors, Prospective.

Introduction

The relationship between fibromyalgia and psychosocial distress is well-established. Studies show a high prevalence of current or lifetime depression [1], anxiety and somatization disorder [2, 3] in clinical patients. Psychosocial factors were also strongly related to chronic widespread pain (CWP), the cardinal symptom of fibromyalgia, in community-based subjects unselected for their consultation behaviour [4, 5]. However, these studies were cross-sectional and cannot determine the temporal relationship between psychosocial distress and the onset of CWP. Psychosocial distress may be a consequence of chronic pain, rather than the cause of it.

As far as we are aware, there has been only one prospective study examining the relationship between psychosocial distress and the onset of CWP. In 2001, we reported from the results of a population-based prospective study (The Altrincham Pain Study) of 1658 adult subjects free of CWP [6]. We observed an increased odds of developing new CWP 12 months later, among subjects who at baseline displayed aspects of the process of somatization, as measured by the Somatic Symptom Checklist (SSC), and health seeking behaviour, as measured by the Illness Behaviour subscale of the Illness Attitude Scales (IAS) [6]. Although robust predictors of symptom onset, the proportion of all new cases we were able to predict was relatively modest; among subjects in the highest odds groups, the prevalence of new CWP was 21%, while the majority of subjects did not develop CWP. Of all the new cases identified, only 6% were in the highest odds groups suggesting that other factors, psychosocial or otherwise, contribute to the development of CWP.

There are a number of other psychosocial factors that may be important in identifying those at risk of new pain. Thus, anxiety and depression are well-known correlates of fibromyalgia and CWP [4, 7]. Disturbances of sleep pattern are very common in fibromyalgia [8, 9], with patients often complaining of unrefreshing sleep, repeated arousals at night and morning fatigue [10, 11]. Stressful life events have also been reported to precede fibromyalgia in various retrospective studies [12, 13].

The aim of the current study was to attempt a more robust evaluation of the contribution of psychological distress (anxiety, depression and somatization), health seeking behaviour, sleep disturbances and traumatic life events to the onset of CWP.

Subjects and methods

Study design

This was a population-based prospective study in which participants were contacted by postal survey. At baseline, the presence

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Submitted 29 March 2006; revised version accepted 21 September 2006.

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or absence of CWP was recorded in addition to the assessment of a large number of psychosocial factors. Fifteen months after the baseline survey, subjects free of CWP at baseline were followed-up, and new cases of CWP were ascertained.

Study subjects

A random sample of 11 000 subjects aged 25–65 yrs was selected from three population-based primary care registers covering a socio-demographically mixed urban area. Subjects were recruited from three disparate socio-demographic areas, with widely varying levels of employment and income, with the aim that the population would be broadly representative of a typical suburban population. The project had ethics committee approval from South Manchester Local Research Ethics Committee and South Cheshire Local Research Ethics Committee. All subjects gave written informed consent.

Baseline assessment of CWP

Subjects were sent a questionnaire by mail, which inquired whether they had experienced any pain during the last month that had persisted for at least 24 h, and, if so, whether the pain had been present for more than 3 months. Four manikin drawings of the body were included (front, back and sides), on which subjects were asked to indicate the sites of pain. These methods, which have been extensively studied [14, 15], were used to determine the location and duration of pain. On the basis of this information, a trained observer using standard manikin data capture forms for the presence or absence of CWP categorized subjects. CWP was defined using the American College of Rheumatology (ACR) criteria for fibromyalgia [16]. To satisfy these criteria, subjects must have pain present in two contralateral quadrants of the body, above and below the waist, and in the axial skeleton, and this pain must have been present for at least 3 months.

Baseline assessment of psychosocial factors

This comprised a number of different scales derived from subject-completed questionnaires.

The Somatic Symptom Checklist (SSC). The SSC was devised as a screening test for somatization disorder [17]. The scale includes six basic items: trouble breathing, frequent vomiting (when not pregnant), loss of voice for more than 30 min, being unable to remember what you have been doing for hours or days (without the influence of alcohol or drugs), difficulty swallowing and frequent pain in the fingers or toes. A seventh item, frequent trouble with menstrual cramps, is included for female respondents. These symptoms are included in the American Psychiatric Association's criteria for somatization disorder [18]. In the study from which these criteria were derived, a threshold between three and four resulted in a sensitivity of 73% and specificity of 94% for identifying cases of somatization disorder. To avoid spurious associations with new cases of CWP, only the 'non-pain' somatic symptoms were examined (i.e. frequent trouble with menstrual cramps and frequent pain in the fingers or toes were excluded). The total score was, therefore, between zero and five for both males and females. The SSC modified in this way predicted the development of CWP in the community [6].

Illness Attitude Scales (IAS). The IAS [19] assesses attitudes and concerns about illness and health. Each scale includes three items, each scored from zero to four, providing a total score between 0 and 12. Individual scales assess worry about health, concern about pain, health habits, hypochondriacal beliefs, thanatophobia (fear of death), disease phobia, bodily preoccupation, treatment experience and effect of symptoms. A study based on a principal components analysis demonstrated that of the 27 items which make up the IAS, 17 measure two dimensions reflecting 'health anxiety' and 'illness behaviour' [20]. The 'health anxiety' subscale consists of 11 items (such as 'Are you worried that you may get a serious illness in the future?') and has a total score between 0 and 44, with a general population mean score of 9.1 (S.D. 6.9). The 'illness behaviour' subscale consists of six items (such as 'Do your bodily symptoms stop you from working?') and has a total score between 0 and 24, with a general population mean score of 4.7 (S.D. = 4.2).

Hospital Anxiety and Depression Scale (HAD). The HAD was developed to identify 'caseness' (possible and probable) of anxiety disorders and depression among patients with physical health complaints in non-psychiatric hospital units [21]. The scale was divided into an Anxiety subscale (HAD-A) and a Depression subscale (HAD-D), both containing seven intermingled items. Each of the 14 items asks about symptoms of anxiety or depression in the last week on a 0–3 scale. Scores on each subscale of 10–11 gives a high probability of an anxiety or a depressive episode being present.

Sleep Problem Scale. This validated four-item Sleep Problem Scale [22] asks about problems with sleep within the past month. Responses are scored in the range of 0–5, giving a total score of between 0 and 20. The scale has been used extensively in previous studies on fibromyalgia and CWP [15, 23, 24].

Life Events Inventory. Information on adverse life events associated with a significant marked or moderate long-term threat in the previous 6 months was obtained using the 12-item 'List of Threatening Experiences' [25], modified from a 67-item life events inventory [26]. The categories cover personal relationships, employment, illness and financial and legal problems.

Follow-up

Subjects free of CWP at baseline were mailed an identical questionnaire after 15 months. Methods for categorizing pain were identical to those used in the baseline survey. The observer, categorizing pain status from the follow-up pain manikins, was blinded to subjects' baseline status, measures of psychosocial distress, and all other information included in the baseline questionnaire. New CWP at follow-up was defined using the ACR definition as aforediscussed.

Statistical analysis

Those subjects who provided complete data at baseline and follow-up were included in the analysis. The distributions of baseline psychosocial scale scores were not Gaussian. The Illness Behaviour & Somatic Symptom Checklist scales were categorized in the same way as our previous prospective community study of CWP [6]. For the other scales, subjects' scores were divided by thirds. The association between new CWP and the scale scores of subjects who scored in the middle and highest thirds were compared with those in the lowest third by logistic regression analysis, adjusted for age and gender. The results are presented as odds ratios with 95% CI that under the rare disease assumption provide a valid estimate of the relative risk [27]. Risk factors found to be associated with CWP were entered into a multiple logistic regression model to examine their relative contribution to the presence of new CWP. All analyses were conducted using the STATA statistical software package (STATACorp, 1993).

Results

At baseline, 10 987 persons were mailed a questionnaire (13 subjects were deemed not able to participate by their general practitioner and were removed from the study). A total of 6792 responses were received (68.2% response rate after adjusting

for persons not resident at their listed address and subjects who had died, $n = 1032$). Of the subjects who responded, 5190 were free of CWP, of whom 4201 were eligible to be followed-up and comprised the cohort for the present study (Fig. 1).

Figure 1 shows that at the 15-month follow-up, 3185 subjects returned the questionnaire (82% after adjustment for subjects who had moved to another address or died), of whom 3001 returned a full questionnaire, 139 a short questionnaire, and 45 subjects completed a telephone questionnaire. Of these, pain status could be determined in 3171 subjects. We compared the baseline characteristics of participants at follow-up with those subjects who were lost to follow-up due to various reasons (Table 1). Participants were older, more likely to be female, and had higher scores on Illness Behaviour and Sleep Problem scales than those who were lost to follow-up.

As shown in Table 2, of the 3171 subjects who did not have CWP at baseline, 324 (10.2%) reported new CWP at follow-up. Older female subjects were slightly more likely to report symptoms. Table 3 shows the age- and sex-adjusted univariate associations of all the baseline psychosocial scores with the presence of new CWP. Subjects in the highest third based on their scores for the psychosocial factors were at the greatest odds for reporting new CWP. For all scales there was evidence of a 'dose-response' effect across the thirds of the distributions. The strongest effects were in the illness behaviour score and the Sleep Problem Scale. In a multivariate analysis (Table 3), three factors made independent contributions to the odds of new CWP, namely illness behaviour, somatic symptoms and sleep problems.

Of the subjects who were free of CWP at baseline, a substantial proportion reported some pain at baseline: while 1326 (41.8%) reported no pain, 1771 (55.6%) reported pain that was not widespread, while 74 (2.3%) had widespread pain that was not chronic. We expected that individuals with pain would be at greater odds of new CWP at follow-up. This was indeed the case: 38 (2.9%) of subjects with no pain developed CWP, 270 (15.3%) of those with some pain and 16 (21.2%) of those with widespread but not chronic pain at baseline developed new CWP. Indeed, compared with those reporting no pain, those with some pain [OR = 6.1, 95% CI (4.3, 8.6)] and those with widespread pain [OR = 9.4, 95% CI (5.0, 17.9)] had an increased odds of developing CWP after adjusting for the effects of age and gender. These relationships were not simply a reflection of increased rates of somatization among those with some pain at baseline: no differences were observed in scores on the somatic symptom, illness behaviour and sleep scales. After adjusting for the presence of pain at baseline the somatic symptom checklist [OR = 2.2, 95% CI (1.4, 3.6)], illness behaviour scale [highest third OR = 5.2, 95% CI (3.8, 7.2)] and sleep scale [OR = 2.5, 95% CI (1.8, 3.4)] remained independent, albeit attenuated, predictors of symptom onset.

Finally, we examined the point prevalence of new CWP by the combination of those three factors significantly associated in the multivariate analysis with prevalent cases at follow-up, i.e. somatic symptoms, illness behaviour and sleep problems. As shown in Table 4, there was evidence of an additive effect. Subjects who scored in the highest range of all three factors had

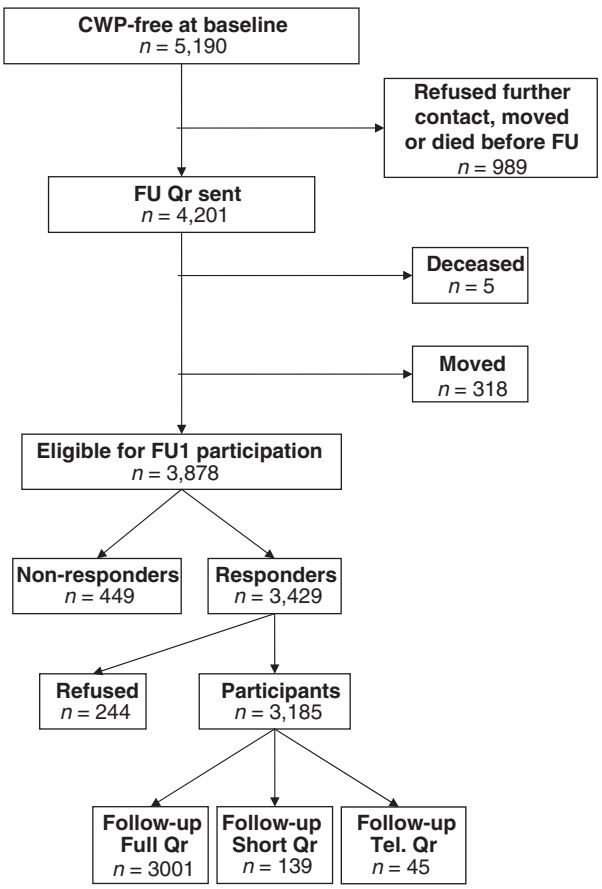

FIG. 1. Flow chart showing participation of subjects from baseline to follow-up phase.

TABLE 1. Distribution of age, gender and psychological scale scores of participants and non-participants in the follow-up questionnaire

	Participants ($n = 3185$)[a]	Non-participants ($n = 2005$)[a]	P[b]
Age			
25–30	202 (6.4)	227 (11.3)	
31–35	345 (10.8)	325 (16.2)	
36–40	407 (12.8)	319 (15.9)	
41–45	433 (13.6)	284 (14.1)	
46–50	447 (14.0)	250 (12.5)	
51–55	447 (14.0)	234 (11.7)	
56–60	479 (15.0)	186 (9.3)	
61–65	425 (13.4)	180 (9.0)	0.000
Male sex	1386 (43.5)	948 (47.3)	
Female sex	1799 (56.5)	1057 (52.7)	0.008
Psychosocial scales (possible range)			
Somatic symptoms (0–5)	0 (0–1)	0 (0–1)	0.01
Illness behaviour (0–24)	4 (2–7)	4 (2–7)	0.000
Health anxiety (0–44)	9 (5–15)	9 (4–15)	0.192
HAD anxiety (0–21)	5 (3–8)	5 (3–8)	0.714
HAD depression (0–21)	3 (1–5)	2 (1–6)	0.721
Sleep problems (0–20)	5 (2–9)	4 (2–8)	0.001
Life events (0–12)	1 (0–2)	1 (0–2)	0.158
GHQ (0–12)	0 (0–3)	0 (0–3)	0.845

[a]Values for age and sex are the number (%); those for the psychosocial scales are median (interquartile range).
[b]All P-values were determined by Mann–Whitney U-test except those for age and sex, which were determined by chi square test.

TABLE 2. Prevalence of new CWP by age and sex

	Total subjects	Number with new CWP	Prevalence (%) (95% CI)
Overall	3171	324	10.2 (9.2, 11.3)
Male	1378	136	9.9 (8.3, 11.6)
Female	1793	188	10.5 (9.1, 11.9)
Age			
25–40	1057	99	9.4 (7.7, 11.3)
41–54	1057	109	10.3 (8.5, 12.1)
55–65	1057	116	11.0 (9.1, 12.9)

TABLE 3. Psychological predictors of new CWP on univariate logistic regression, adjusted for age and gender

Psychological variable	*n*	Number with new CWP (%)	Univariate model OR (95% CI)	Multivariate model OR (95% CI)
Somatic symptoms				
0–2	2987	291 (9.7)	Referent	Referent
3–5	111	26 (23.4)	2.8 (1.8, 4.5)	1.8 (1.1, 3.1)
Illness behaviour				
0–4	1271	59 (4.6)	Referent	Referent
5–7	1111	110 (9.9)	2.3 (1.7, 3.2)	1.9 (1.3, 2.7)
8–22	740	147 (19.9)	5.2 (3.8, 7.2)	3.3 (2.3, 4.8)
Health anxiety				
0–6	1089	95 (8.7)	Referent	Referent
7–13	1069	112 (10.5)	1.2 (0.9, 1.6)	0.9 (0.7, 1.3)
14–44	894	105 (11.7)	1.4 (1.01, 1.8)	0.7 (0.5, 1.05)
HAD anxiety				
0–4	1353	94 (7.0)	Referent	Referent
5–7	867	83 (9.6)	1.4 (1.1, 2.0)	1.1 (0.8, 1.6)
8–21	912	142 (15.6)	2.6 (1.9, 3.4)	1.3 (0.9, 1.9)
HAD depression				
0–2	1554	103 (6.6)	Referent	Referent
3–5	872	98 (11.2)	1.8 (1.3, 2.4)	1.2 (0.8, 1.7)
6–20	708	119 (16.8)	2.9 (2.2, 3.8)	1.3 (0.9, 1.9)
Sleep problem				
0–3	1203	67 (5.6)	Referent	Referent
4–8	1039	109 (10.5)	2.0 (1.5, 2.8)	1.7 (1.2, 2.4)
9–20	832	139 (16.7)	3.4 (2.5, 4.6)	2.2 (1.6, 3.2)
Life events				
0	1425	113 (7.9)	Referent	Referent
1	878	84 (9.6)	1.2 (0.9, 1.6)	0.98 (0.7. 1.4)
2–9	807	120 (14.9)	2.0 (1.6, 2.7)	1.2 (0.9, 1.7)

TABLE 4. Prevalence and odds of new CWP according to exposure to psychological factors

Number of factors[a]	Total subjects	Number with CWP (%)	OR (95% CI)
0	632	20 (3.2)	Referent
1	1085	74 (6.8)	2.3 (1.4, 3.8)
2	1184	189 (16.0)	6.0 (3.7, 9.6)
3	62	17 (27.4)	12.1 (5.9, 24.7)

[a]Factors included in model: somatic symptom >2, illness behaviour >4, sleep >4.

TABLE 5. Prevalence of new CWP at follow-up, by baseline Somatic Symptom and Illness Behaviour scores

	Illness Behaviour score								
	0–4			5–7			8–24		
Somatic symptoms score	*n*	Number with new CWP	%	*n*	Number with new CWP	%	*n*	Number with new CWP	%
0–2	1228	56	4.6	1053	100	9.5	667	128	19.2
3–5	19	3	15.8	36	8	22.2	50	14	28.0

27.4% odds of new CWP, while those who scored in the lowest categories of all four factors had only 3.2% odds. Subjects scoring highly on all three scales were at 12 times higher odds of developing new prevalent CWP than those who had the lowest scores.

Discussion

We have demonstrated that psychosocial factors including multiple physical symptoms, help-seeking for health problems, sleep problems and adverse life events increase the likelihood of the onset of CWP in the next 15 months 20-fold in a community sample. The presence of one or more of these four factors, present in 78.6% of the study sample, predicted 93% of new cases of CWP. In a previous cohort, we demonstrated that the best predictors of CWP at 12 months were the Somatic Symptom Checklist and Illness Behaviour Scale [6]. Table 5 shows that in our current cohort, of those subjects scoring in the highest categories of both scales, 28.0% developed new CWP, while among those subjects who scored in the lower ranges of both these scales, 4.6% developed new CWP. These proportions compare with 20.8% and 1.4%, respectively, in the original study [6].

The strengths of the study are the prospective follow-up design, the community sample drawn from three disparate socio-demographic areas, with widely varying levels of employment and income, the use of a broad range of standardized measures and the use of blinding to establish outcome independent of baseline pain status, demographic data or psychosocial data. The study is an improvement on our first cohort study which was conducted in a smaller sample drawn from a relatively prosperous area and considered a smaller range of psychosocial factors (illness attitudes and somatization) [6].

The greatest potential threat to the validity of the current findings was in the attrition of the cohort at the various stages of investigation. Of those subjects free of CWP who sent in completed questionnaires at baseline, a substantial number was lost to follow-up because of moving house, death, non-response and refusals. We were concerned about any effect of such non-participation, and therefore undertook an analysis comparing those who participated with those who did not. As is usual in epidemiological surveys, those who participated were more likely to be older and female, two factors associated with the reporting of CWP. It is possible that we have therefore over-estimated the rate of CWP in our population. Assuming that all subjects who did not respond were likely to be free of CWP, we have calculated

the minimum prevalence rate of CWP at follow-up to be 6.2% [95% CI (5.6%, 6.9%)]. Non-responders also had higher scores on Illness Behaviour and Sleep Problem Scales. These differences would only affect the internal comparisons in the present study if the relationship between the illness behaviour and sleep problem scores and the prevalence of new CWP were different in those subjects who participated in the study compared with those who did not. It is possible that people who seek help from doctors for their health problems are also more likely to participate in research on those health complaints. The external validity of the study is likely to be influenced by the fact that the follow-up sample was restricted to 58% of the subjects who had sent in completed baseline questionnaires, although this would not have affected the internal validity of the study. Given that help seeking for health problems was related to the development of CWP and if help seeking for health problems is related to participation in research on those health problems, then the prevalence rate of new CWP in the community may not be as high as 10%.

Another potential confound in the study is that some of these patients presenting with CWP may have an underlying physical pathology causing their symptoms. The sample was restricted to an <65 yr age group, and the prevalence of new CWP was little influenced by age or gender. A substantial effect of underlying physical pathology would probably be reflected in a strong age effect on the prevalence of new CWP so it is unlikely that underlying physical pathology would bias the association between psychosocial factors and the development of new CWP.

Our follow-up period was 15 months. This is a relatively short follow-up period in the context of a chronic disorder such as CWP, which has a 'low turnover'. We realize that by making the follow-up period short, we may have missed some subjects who may have gone on to develop CWP in the future. Conversely, given the chronicity of the outcome, we are unlikely to have missed many subjects who developed CWP during the follow-up period, but were not 'in-state' at the time of the follow-up survey.

There are a number of psychosocial factors we have not considered that have been previously reported in clinical samples with fibromyalgia. Psychosocial factors include previous sexual, physical and emotional abuse and neglect experiences as a child or an adult [12, 28], positive affect and information processing disposition [29], previous lifetime pain experience [30], and lifetime illicit drug abuse and eating disorders [28]. All of these are difficult to establish in a valid way in a large-scale community study. It is unclear whether these factors would explain more new cases of CWP, or whether they would contribute to the psychosocial predictors of CWP that we have established. For instance, sexual assault is related to somatization, illness attitudes and help seeking for health problems in primary care samples [31].

In summary, we have shown that in a group of persons free of CWP, psychosocial factors are important predictors of the future development of CWP. These data lend further support to the hypothesis that psychological factors precede the onset of CWP and may relate to its origin. The low rate of symptom onset among the minority of subjects not exposed to these factors is notable.

Acknowledgements

The authors are grateful for the participation and help of the doctors, staff and patients of the three general practices in Greater Manchester, and Joanne Bradley and Karen Schafheutle for survey administration. This study was supported by the Arthritis Research Campaign, Chesterfield, United Kingdom.

The authors have declared no conflicts of interest.

References

1. Hudson JI, Hudson MS, Pliner LF, Goldenberg DL, Pope HG,Jr. Fibromyalgia and major affective disorder: a controlled phenomenology and family history study. Am J Psychiatry 1985;142:441–6.
2. Bennett RM, Burckhardt CS, Clark SR, O'Reilly CA, Wiens AN, Campbell SM. Group treatment of fibromyalgia: a 6 month outpatient program. J Rheumatol 1996;23:521–8.
3. Yunus M, Masi AT, Calabro JJ, Miller KA, Feigenbaum SL. Primary fibromyalgia (fibrositis): clinical study of 50 patients with matched normal controls. Semin Arthritis Rheum 1981;11:151–71.
4. Croft P, Rigby AS, Boswell R, Schollum J, Silman A. The prevalence of chronic widespread pain in the general population. J Rheumatol 1993;20:710–3.
5. Hunt IM, Silman AJ, Benjamin S, McBeth J, MacFarlane GJ. The prevalence and associated features of chronic widespread pain in the community using the 'Manchester' definition of chronic widespread pain. Rheumatology 1999;38:275–9.
6. McBeth J, MacFarlane GJ, Benjamin S, Silman AJ. Features of somatization predict the onset of chronic widespread pain: results of a large population-based study. Arthritis Rheum 2001;44:940–6.
7. McBeth J, Silman AJ. Unraveling the association between chronic widespread pain and psychological distress: an epidemiological approach. J Psychosom Res 1999;47:109–14.
8. Landis CA, Lentz MJ, Rothermel J, Buchwald D, Shaver JL. Decreased sleep spindles and spindle activity in midlife women with fibromyalgia and pain. Sleep 2004;27:741–50.
9. Rizzi M, Sarzi-Puttini P, Atzeni F *et al*. Cyclic alternating pattern: a new marker of sleep alteration in patients with fibromyalgia? J Rheumatol 2004;31:1193–9.
10. Landis CA, Lentz MJ, Tsuji J, Buchwald D, Shaver JL. Pain, psychological variables, sleep quality, and natural killer cell activity in midlife women with and without fibromyalgia. Brain Behav Immun 2004;18:304–13.
11. Landis CA, Frey CA, Lentz MJ, Rothermel J, Buchwald D, Shaver JL. Self-reported sleep quality and fatigue correlates with actigraphy in midlife women with fibromyalgia. Nurs Res 2003;52:140–7.
12. Walker EA, Keegan D, Gardner G, Sullivan M, Bernstein D, Katon WJ. Psychosocial factors in fibromyalgia compared with rheumatoid arthritis: II. Sexual, physical, and emotional abuse and neglect. Psychosom Med 1997;59:572–7.
13. Aaron LA, Bradley LA, Alarcon GS *et al*. Perceived physical and emotional trauma as precipitating events in fibromyalgia. Associations with health care seeking and disability status but not pain severity. Arthritis Rheum 1997;40:453–60.
14. Benjamin S, Morris S, McBeth J, MacFarlane GJ, Silman AJ. The association between chronic widespread pain and mental disorder: a population-based study. Arthritis Rheum 2000;43:561–7.
15. MacFarlane GJ, Croft PR, Schollum J, Silman AJ. Widespread pain: is an improved classification possible? J Rheumatol 1996;23:1628–32.
16. Wolfe F, Smythe HA, Yunus MB *et al*. The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum 1990;33:160–72.
17. Othmer E, DeSouza C. A screening test for somatization disorder (hysteria). Am J Psychiatry 1985;142:1146–9.
18. American Psychiatric Association. Americam Psychiatric Association diagnostic and statistical manual. Washington DC: 1980.
19. Kellner R, Abbott P, Winslow WW, Pathak D. Fears, beliefs, and attitudes in DSM-III hypochondriasis. J Nerv Ment Dis 1987;175:20–5.
20. Speckens AE, Spinhoven P, Sloekers PP, Bolk JH, van Hemert AM. A validation study of the Whitely Index, the Illness Attitude Scales, and the Somatosensory Amplification Scale in general medical and general practice patients. J Psychosom Res 1996;40:95–104.

21. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand 1983;67:361–70.
22. Jenkins CD, Stanton BA, Niemcryk SJ, Rose RM. A scale for the estimation of sleep problems in clinical research. J Clin Epidemiol 1988;41:313–21.
23. Croft P, Schollum J, Silman A. Population study of tender point counts and pain as evidence of fibromyalgia. Br Med J 1994;309:696–9.
24. Yunus MB. A comprehensive medical evaluation of patients with fibromyalgia syndrome. Rheum Dis Clin North Am 2002;28:201–2vi.
25. Brugha T, Bebbington P, Tennant C, Hurry J. The list of threatening experiences: a subset of 12 life event categories with considerable long-term contextual threat. Psychol Med 1985;15:189–94.
26. Tennant C, Andrews G. A scale to measure the stress of life events. Aust N Z J Psychiatry 1976;10:27–32.
27. Greenland S, Thomas DC. On the need for the rare disease assumption in case-control studies. Am J Epidemiol 1982;116:547–53.
28. Boisset-Pioro MH, Esdaile JM, Fitzcharles MA. Sexual and physical abuse in women with fibromyalgia syndrome. Arthritis Rheum 1995;38:235–41.
29. Potter PT, Zautra AJ, Reich JW. Stressful events and information processing dispositions moderate the relationship between positive and negative affect: implications for pain patients. Ann Behav Med 2000;22:191–8.
30. Poyhia R, Da Costa D, Fitzcharles MA. Previous pain experience in women with fibromyalgia and inflammatory arthritis and nonpainful controls. J Rheumatol 2001;28:1888–91.
31. Stein MB, Lang AJ, Laffaye C, Satz LE, Lenox RJ, Dresselhaus TR. Relationship of sexual assault history to somatic symptoms and health anxiety in women. Gen Hosp Psychiatry 2004;26:178–83.