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Original article

Impact of musculoskeletal pain on insomnia onset: a prospective cohort study

Nicole K. Y. Tang[1,2], John McBeth[2], Kelvin P. Jordan[2], Milisa Blagojevic-Bucknall[2], Peter Croft[2] and Ross Wilkie[2]

Abstract

Objective. Pain, the most common manifestation of rheumatological conditions, is highly prevalent among older adults, with worse health outcomes found in those with co-morbid insomnia. Proactive prevention of insomnia may reduce the overall disease burden of pain and rheumatological conditions. To inform such development, this study examined the role of pain, physical limitation and reduced social participation in predicting and mediating insomnia onset.

Methods. A prospective cohort study was conducted involving 6676 individuals $\geqslant$50 years of age who completed questionnaires at baseline and a 3-year follow-up. Participants were classified into none, some and widespread pain according to the ACR criteria. Logistic regression was used to examine the relationship between baseline pain and insomnia onset at 3 years. Path analysis was used to test for the mediating role of physical limitation and social participation restriction.

Results. Some [adjusted odds ratio (AOR) 1.57 (95% CI 1.15, 2.13)] and widespread [2.13 (1.66, 3.20)] pain increased the risk of insomnia onset at 3 years, after adjusting for age, gender, socio-economic class, education, anxiety, depression, sleep and co-morbidity at baseline. The combination of physical limitation and reduced social participation explained up to 68% of the effect of some pain on insomnia onset and 66% of the effect of widespread pain on insomnia onset.

Conclusion. There was a dose–response association between the extent of pain at baseline and insomnia onset at 3 years that was substantially mediated by physical limitation and reduced social participation. Targeting physical limitation and social participation in older people with pain may buffer co-morbid insomnia, reducing the overall disease burden.

Key words: musculoskeletal, widespread pain, insomnia, sleep, cohort study, physical function, social participation.

CLINICAL SCIENCE

Introduction

The World Health Organization (WHO) recognizes the significant contribution of musculoskeletal conditions to the global burden of disease, and pain is the key driver [1]. Pain is a common reason for consultation and a potentially manageable problem, but evidence suggests that it is not well controlled and its consequences are underevaluated [1]. The majority of patients seeking treatment for pain report insomnia of a severity that warrants clinical attention [2–4]. Disruption of sleep can aggravate pain and inflammatory processes, reduce endogenous pain-inhibitory responses and dampen mood and the perception of well-being [5–7]. In the longer term, untreated insomnia is associated with an increased incidence of depression, anxiety and substance misuse [8], as well as a range of adverse but preventable health outcomes [9–11].

Existing treatment options for pain-related insomnia are limited. Hypnotics (e.g. zolpidem) and pharmacological agents with hypnotic qualities (e.g. amitriptyline) are commonly used as the first-line treatment [12], although evidence to support their efficacy and safety beyond 6–12 months is limited [13, 14]. The prolonged use of hypnotics is problematic in older adults, with increased risks of falls,

[1]Department of Psychology, University of Warwick, Coventry and [2]Arthritis Research UK Primary Care Centre, Primary Care Sciences, Keele University, Staffordshire, UK.

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Correspondence to: Nicole K. Y. Tang, Department of Psychology, University of Warwick, Coventry CV4 7AL, UK.
E-mail: n.tang@warwick.ac.uk

dementia and mortality [15–17]. Psychological treatments offer an alternative approach for treatment and prevention. Targeting the mechanisms perpetuating insomnia, cognitive-behavioural treatment for insomnia is achieving clinically significant improvements in sleep despite ongoing pain [18–19]. It is also possible to prevent pain-related insomnia by addressing the cognitive-behavioural factors mediating its onset early.

Two factors hypothesized to be mediating the pain–insomnia onset relationship are physical limitation and reduced social participation, which are common consequences of pain, particularly in older adults [20, 21]. Participation in social and physical activities is theoretically sleep-promoting because engagement in activities generates sleep pressure and brings exposure to light and stimulation that are essential for entraining the circadian rhythm [22, 23]. However, it is unclear whether restrictions in physical and social activity explain the increased risk of insomnia onset in older adults with pain. The aim of this study was to investigate the longitudinal impact of pain on insomnia onset and to examine the role of physical limitation and reduced social participation in mediating the pain–insomnia link in community-dwelling older adults.

Methods

Study design

A population-based prospective cohort study was conducted. A detailed protocol is available [24]. In brief, all individuals $\geqslant$50 years of age (n = 19 818) registered with six general practices in North Staffordshire, UK, were mailed a baseline questionnaire. The North Staffordshire Local Research Ethics Committee granted approval.

Assessment

Pain

For this analysis, participants were categorized according to their experience of pain at baseline. Participants who experienced pain lasting for $\geqslant$1 day in the past month were asked to shade their pain on a blank body manikin (front and back views) and were categorized into widespread pain, some pain or no pain. Widespread pain was classified according to the ACR criteria used in their definition for FM [25], which require pain to be present in the left and right hand sides of the body, above and below the waist and in the axial skeleton. The some pain group was those participants reporting pain that did not satisfy the criteria for widespread pain. These methods to determine the location and extent of pain are standard in population-based studies of pain and have been shown to be valid and reliable [26].

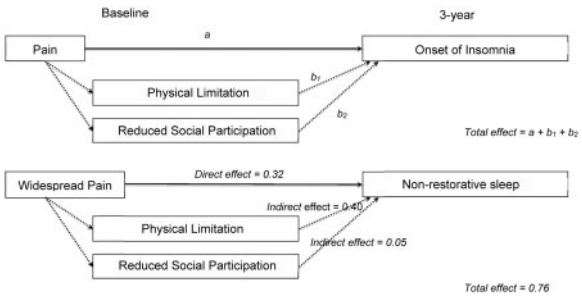
Insomnia

Insomnia was measured at baseline and at 3-year follow-up using the Jenkins Sleep Questionnaire (JSQ) [27]. The questionnaire asks about recent problems with sleep and contains items on the most commonly occurring symptoms of poor sleep quality: sleep onset (During the past 4 weeks did you have trouble falling asleep?), sleep maintenance (During the past 4 weeks did you wake up several times per night?), early awakening (During the past 4 weeks did you have trouble staying asleep, including waking up far too early?) and non-restorative sleep (During the past 4 weeks did you wake up after your usual amount of sleep feeling tired and worn out?). Participants indicate the number of days in the past month that they have experienced difficulties in each of the four sleep components on a 3-point scale: not at all, on some nights or on most nights. For this analysis, on most nights was used to define insomnia for each item. Insomnia onset at 3 years was defined for each individual insomnia item separately; persons with the symptom on most nights at baseline were first excluded and onset was defined as movement from no problems or problems experienced on some nights at baseline to problems on most nights 3 years later [28]. Items were individually analysed because insomnia is defined by 'a repeated difficulty with sleep initiation, duration, consolidation or quality' [29]. We also examined the risk of any insomnia onset, as indicated by the above defined movement, in any of the four insomnia items.

Potential mediators

The role of baseline physical limitation and reduced social participation in mediating the association between baseline pain and onset of insomnia at 3 years was examined for the identification of prevention targets (Fig. 1). Physical limitation was measured using the physical functioning (PF) scale of the Medical Outcomes Study 36-item Short Form Health Survey (SF-36). Scores range from 0 to 100, with higher scores indicating better function [30]. Items measured the limitation in the individual's capacity to complete basic tasks such as lifting and walking. Reduced social participation was measured using the Keele Assessment of Participation (KAP) [31], which measures taking part in 11 activities in accordance with the WHO framework (e.g. work and attending social and community events) [32]. Participants were considered to

Fig. 1 Path diagram



Top panel: a = direct effect; b_1 or b_2 = indirect effect; $a + b_1 + b_2$ = total effect. Bottom panel: Example findings using widespread pain as the predictor and physical limitation and reduced social participation as the mediators of non-restorative sleep.

have reduced social participation if they reported that they were not taking part during the previous 4 weeks 'as and when [they] wanted' for 'some of the time' or less. The resulting 11 binary items were then summed to give a total score ranging from 0 to 11. The KAP has demonstrated adequate levels of reliability and validity for use in population studies [39].

Putative confounders

Putative confounders were demographic (age, gender) and socio-economic (occupational class: professional/managerial, semi-routine, routine), educational attainment (further education, or not), baseline anxiety and depression and co-morbidity. Anxiety and depression were measured using the Hospital Anxiety and Depression Scale; raw scores were used to categorize individuals as non-cases (0–7), possible cases (8–10) and probable cases (11–21) [33]. General practitioners in the study populations used the Read system to code all morbidity encounters in actual consultations [34, 35]. Morbidity data (i.e. symptoms and diseases) in this system are grouped under 19 main Read chapters. Data collected at the second hierarchical level or above were used to identify morbidity and relate to at least one consultation for a given morbidity category in the 18 months prior to baseline (repeat consultations for the same morbidity were not included). The number of morbidities consulted for were then summed to give a total score ranging from 0 to 19. For the prediction of the onset of each insomnia symptom, disturbance in the other three areas of sleep at baseline was included as potential confounders of the pain–insomnia onset link.

Statistical analysis

First, we examined whether there was a dose–response relationship between the extent of baseline pain and insomnia onset at 3 years. Associations between baseline pain and the onset of each insomnia symptom at 3 years, unadjusted and then adjusted for putative confounders, were examined using logistic regression. All putative confounders were significantly associated with baseline pain and insomnia onset at 3 years and were entered in each multivariable logistic regression model.

Path analysis (i.e. an extended form of multiple regression that tests whether dependent variables are on a pathway to the occurrence of an outcome [36]) was then used to test mediation of the association between baseline pain and insomnia onset at 3 years by physical limitation and reduced social participation at baseline. A series of models were built to estimate (i) the total effect of baseline pain on insomnia onset at 3 years without including the mediators, (ii) the direct effect (i.e. the effect of baseline pain on insomnia onset at 3 years adjusting for the mediators) and (iii) the indirect effect (i.e. the difference between the total effect of baseline pain on insomnia onset and the direct effect). The indirect effect indicates the amount of mediation and the extent to which each putative mediator explains the link between baseline pain and insomnia onset at 3 years [37]. The Karlson–Holm–Breen method of decomposition was adopted to separate the total effect in a logistic model into direct (some and widespread pain) and indirect (physical limitation and reduced social participation) effects [38]. The proportion of mediation is calculated by dividing the indirect effect by the total effect [38], and this can be interpreted as the proportion of pain effects that might be explained by physical limitation and/or reduced social participation. For each of the four JSQ items and for any insomnia, the first model examined the total effect of baseline pain on insomnia onset at 3 years. Putative confounders were then added to the models for each insomnia symptom. Physical limitation and reduced social participation were added separately and then simultaneously and indirect effects were estimated for each. Results were reported as standardized β coefficients.

Sensitivity to subject attrition and missing data was examined via probability-weighted analysis for survey data [39]. As there were no differences in inference between the original and weighted analyses, the former are reported.

Results

At baseline, responses were received from 13 986 persons and 9457 (68%) gave consent to take part in the follow-up study. At the 3-year follow-up there were 579 exclusions due to deaths, departures, terminal illness or severe psychiatric illness. The remaining 8878 were sent a 3-year follow-up questionnaire and 7230 (81%) completed responses were received. Of this group, 6676 (representing 71% of consented participants) had complete pain data at baseline and 3 years and were used for the analysis (Fig. 2).

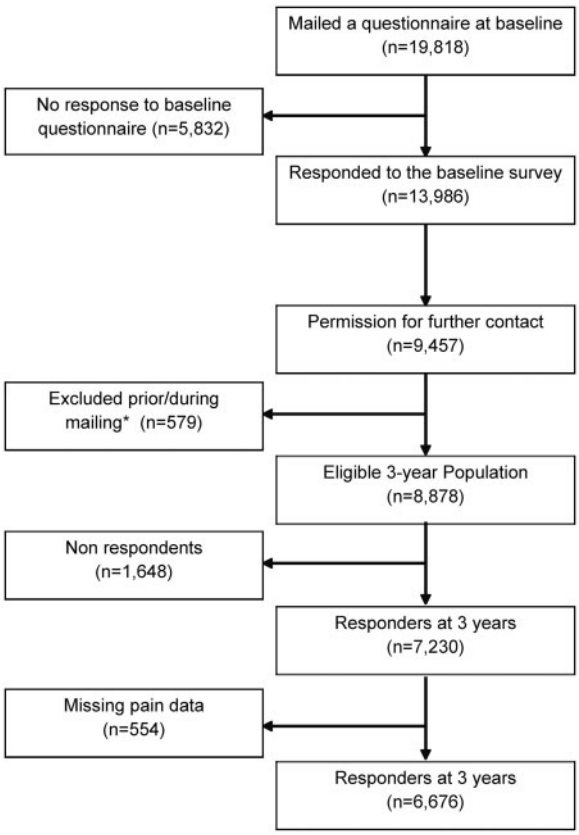
Participant characteristics

At baseline median age overall was 64.2 years (S.D. 9.2), 55.0% were women and 86.4% had a high school education only (Table 1). A total of 1767 (26.5%) participants had no pain, 3074 (46.0%) had some pain and 1835 (27.5%) had widespread pain. There was a cross-sectional dose–response relationship between prevalent pain extent and each prevalent insomnia symptom (all $P < 0.001$).

Association between baseline pain extent and insomnia onset at 3 years

Widespread and some pain at baseline were significantly associated with the subsequent onset of any and each of the four insomnia symptoms at 3 years (Table 2); for example some and widespread pain were associated with the onset of non-restorative sleep [unadjusted OR 2.09 (95%CI 1.59, 2.75) and OR 3.98 (95%CI 3.00, 5.27) respectively]. These associations remained largely unchanged when adjusted for age, gender, educational attainment and occupational class. Further adjustment for anxiety, depression, comorbidity and other baseline sleep disturbance slightly attenuated the associations [adjusted OR 1.57 (95%CI 1.15, 2.13) and OR 2.31 (95%CI 1.66, 3.20) respectively].

FIG. 2 Flow diagram of participants



*Exclusion due to deaths, departures, terminal illness or severe psychiatric illness.

Association between baseline pain extent and insomnia onset at 3 years, via physical limitation and reduced social participation

Baseline physical limitation and reduced social participation mediated the association between baseline pain and the onset of any and each of the four insomnia symptoms (Table 3). For example, the standardized β coefficient for the total effect of widespread pain on the onset of non-restorative sleep was 0.78 (S.E. 0.17). After inclusion of physical limitation as a mediator, the direct effect was 0.30 (0.18) and the indirect effect was 0.47 (0.06). With inclusion of reduced social participation as the mediator instead, the direct effect was 0.70 (0.17) and the indirect effect was 0.10 (0.02). With the inclusion of both physical limitation and reduced social participation, the indirect effect of these mediators were 0.40 (0.07) and 0.05 (0.02) respectively. Inclusion of both physical limitation and reduced social participation explained 58% of the total effect of widespread pain on the onset of non-restorative sleep.

Findings for any and the other insomnia items were comparable and a similar pattern of findings was observed for the effect of some pain on insomnia onset.

Discussion

In this longitudinal study of older adults with pain there was a dose–response association between pain and insomnia onset 3 years later, and importantly, physical limitation and reduced social participation appeared to play a role in mediating the pain–insomnia association. Both widespread and some pain increased the risk of insomnia onset. The total effect of pain on the onset of each insomnia symptom was up to twice as strong in participants with widespread pain compared with those with some pain. The combination of physical limitation and reduced social participation mediated a substantial proportion of the total effect of pain on insomnia onset, with physical limitation being a stronger mediator than reduced social participation.

The aetiological role of pain in insomnia onset was often inferred from patients' own attribution [40] and cross-sectional findings that showed increased insomnia symptoms with the presence of pain [41–42]. A handful of recent studies adopted a longitudinal design to clarify the temporal order of the association [43–45], with one reporting an odds ratio (OR) of 1.64 for the incidence of insomnia. The focus of these studies was on young and middle-aged adults and the length of the observation was restricted to within 15 months, which might be too short to capture the medium- to long-term impact of pain as its chronicity increases. Findings of the current study provided evidence of a link between pain and insomnia onset over 3 years specific to older adults. The generally higher ORs observed in the current study suggest that older adults with some (OR 1.81–2.09, adjusted OR 1.41–1.78) and widespread (OR 2.47–3.98, adjusted OR 1.52–2.55) pain may be particularly vulnerable to the development of co-morbid insomnia.

The observed mediating role of physical limitation echoes previous findings that reduced physical capacity is linked to poor outcomes in older adults with pain. This links to existing conceptualizations of primary and pain-related insomnia, in which dysregulation of physical activity is considered a behavioural pathway to non-restorative sleep [46–51]. The current findings indicate that physical activity and social participation are potential targets for the management for older adults with pain, although we note that the PF measures perceived limitations in physical capacity rather than the amount of physical activity. Medical approaches to managing pain are important and targeting pain is going to make some contribution to preventing or reducing sleep disturbance. However, if pain persists, our results suggest that an important option for intervention might be to maintain or improve older people's physical capacity and social participation for their potential to avert insomnia onset. Physiotherapy, exercise classes and psychological interventions that address the barriers to physical activities are example treatments that may improve physical functioning despite the presence of underlying medical conditions. Notably, improving social participation offers the opportunity of reducing insomnia with or without improving

Table 1 Subject characteristics at baseline overall and by pain extent

	Overall ($n = 6676$)	No pain ($n = 1767$)	Some pain ($n = 3074$)	Widespread pain ($n = 1835$)	*P*-value
Age, mean (S.D.), years	64.2 (9.2)	64.36 (9.3)	64.37 (9.3)	63.71 (8.9)	0.04
Gender, female	3670 (55.0)	917 (51.9)	1644 (53.5)	1109 (60.4)	<0.001
No further education	5667 (86.4)	1455 (83.7)	2625 (86.8)	1587 (88.2)	<0.001
					<0.001
Occupational class					
Managerial/professional	2619 (39.2)	754 (42.7)	1218 (39.6)	647 (35.3)	
Lower supervisory/semi-routine	1963 (29.4)	514 (29.1)	882 (28.7)	567 (30.9)	
Routine	1720 (25.8)	415 (23.5)	800 (26.0)	505 (27.5)	
Anxiety					
Possible case	1360 (20.4)	253 (14.3)	624 (20.3)	483 (26.3)	<0.001
Probable case	1241 (18.6)	169 (9.6)	541 (17.6)	531 (28.9)	
Depression					<0.001
Possible case	742 (11.1)	83 (4.7)	326 (10.6)	333 (18.2)	
Probable case	541 (8.1)	71 (4.0)	216 (7.0)	254 (13.8)	
Co-morbidity					<0.001
0–3	3862	1217	1769	876	
⩾4	2276 (37)	383 (23.9)	1078 (37.8)	815 (48.2)	
Physical limitation (0–100), mean (S.D.)	65.3 (30.3)	83.9 (20.8)	65.9 (28.3)	46.6 (30.0)	<0.001
Reduced social participation (0–11), mean (S.D.)	1.15 (1.80)	0.58 (1.13)	1.05 (1.66)	1.87 (2.25)	<0.001
Insomnia at baseline					
Trouble falling asleep[a]	879 (13.4)	102 (5.8)	356 (11.8)	421 (23.3)	<0.001
Wake up several times per night[a]	2009 (30.7)	298 (17.2)	898 (29.9)	813 (45.0)	<0.001
Trouble staying asleep[a]	1302 (20.2)	175 (10.2)	558 (18.8)	569 (32.0)	<0.001
Wake up feeling tired and worn out[a]	1069 (16.4)	96 (5.6)	429 (14.3)	544 (30.2)	<0.001

All values are *n* (%) unless otherwise indicated. [a]Each insomnia symptom at baseline is defined as disturbance on most nights. Analysis of variance test for age and chi-square for gender, education and each insomnia symptom.

Table 2 Associations between baseline pain extent and the onset of insomnia at 3 years in participants without the relevant insomnia symptom at baseline

	Pain status	*N* (%)	Crude OR (95% CI)	Adjusted OR[a] (95% CI)	Adjusted OR[b] (95% CI)
Trouble falling asleep	No pain ($n = 1562$)	59 (3.8)	1	1	1
	Some ($n = 2502$)	166 (6.6)	1.81 (1.34, 2.45)	1.73 (1.26, 2.39)	1.30 (0.93, 1.82)
	Widespread ($n = 1300$)	129 (9.9)	2.81 (2.04, 3.85)	2.67 (1.91, 3.73)	1.61 (1.12, 2.32)
Wake up several times per night	No pain ($n = 1351$)	162 (12.0)	1	1	1
	Some ($n = 1984$)	399 (20.1)	1.85 (1.52, 2.25)	1.85 (1.51, 2.28)	1.54 (1.24, 1.91)
	Widespread ($n = 927$)	234 (25.2)	2.48 (1.99, 3.09)	2.55 (2.02, 3.21)	1.78 (1.39, 2.29)
Trouble staying asleep	No pain ($n = 1396$)	101 (7.2)	1	1	1
	Some ($n = 2225$)	283 (12.7)	1.87 (1.47, 2.37)	1.79 (1.39, 2.29)	1.41 (1.08, 1.85)
	Widespread ($n = 1109$)	182 (16.4)	2.52 (1.95, 3.26)	2.55 (1.95, 3.34)	1.59 (1.18, 2.14)
Wake up feeling tired and worn out	No pain ($n = 1535$)	72 (4.7)	1	1	1
	Some ($n = 2434$)	227 (9.3)	2.09 (1.59, 2.75)	2.07 (1.56, 2.76)	1.57 (1.15, 2.13)
	Widespread ($n = 1667$)	196 (16.4)	3.98 (3.00, 5.27)	3.84 (2.85, 5.16)	2.31 (1.66, 3.20)
Any insomnia	No pain ($n = 1278$)	259 (20.3)	1	1	1
	Some ($n = 1952$)	661 (33.9)	2.01 (1.71, 2.38)	2.04 (1.71, 2.42)	1.46 (1.21, 1.75)
	Widespread ($n = 977$)	476 (48.7)	3.74 (3.11, 4.50)	3.78 (3.11, 4.59)	1.80 (1.47, 2.22)

OR: odds ratio. [a]Adjusted for age, gender, education and occupational class. [b]Adjusted for age, gender, education, occupational class, anxiety, depression, other insomnia symptoms at baseline and co-morbidity.

physical capacity and may extend beyond clinical management, e.g. the provision of good access to transport and resources for maintaining social networks in the community.

The prospective design and the use of path analysis enabled the testing of physical limitation and social participation as mechanisms linking pain and insomnia onset at 3 years. However, caution must be exercised when

Table 3 The pathway from baseline pain to the onset of insomnia at 3 years via physical disability and social participation

			Trouble falling asleep	Wake up several times per night	Trouble staying asleep	Wake up feeling tired and worn out	Any insomnia
Physical limitation	Some pain	Total effect	0.25 (0.18)	0.42 (0.11)	0.35 (0.14)	0.35 (0.16)	0.34 (0.10)
		Direct effect	0.10 (0.18)	0.28 (0.12)	0.20 (0.14)	0.10 (0.17)	0.20 (0.10)
		Indirect effect	0.15 (0.04)	0.14 (0.03)	0.15 (0.03)	0.24 (0.04)	0.14 (0.02)
	Widespread pain	Total effect	0.47 (0.19)	0.61 (0.13)	0.50 (0.16)	0.78 (0.17)	0.59 (0.11)
		Direct effect	0.17 (0.21)	0.33 (0.14)	0.20 (0.17)	0.30 (0.18)	0.32 (0.12)
		Indirect effect	0.30 (0.07)	0.28 (0.05)	0.30 (0.06)	0.47 (0.06)	0.28 (0.04)
Reduced social participation	Some pain	Total effect	0.26 (0.17)	0.43 (0.11)	0.34 (0.14)	0.43 (0.16)	0.37 (0.09)
		Direct effect	0.25 (0.17)	0.41 (0.11)	0.32 (0.14)	0.39 (0.16)	0.35 (0.09)
		Indirect effect	0.01 (0.01)	0.02 (0.01)	0.02 (0.01)	0.04 (0.02)	0.02 (0.01)
	Widespread pain	Total effect	0.47 (0.19)	0.57 (0.13)	0.45 (0.15)	0.80 (0.17)	0.58 (0.11)
		Direct effect	0.44 (0.19)	0.52 (0.13)	0.40 (0.15)	0.70 (0.17)	0.52 (0.11)
		Indirect effect	0.03 (0.02)	0.05 (0.02)	0.05 (0.02)	0.10 (0.02)	0.06 (0.02)
Physical limitation and reduced social participation	Some pain	Total effect	0.25 (0.18)	0.42 (0.11)	0.35 (0.14)	0.34 (0.16)	0.33 (0.10)
		Direct effect	0.10 (0.18)	0.28 (0.12)	0.21 (0.14)	0.11 (0.17)	0.20 (0.10)
		Indirect effect	0.15 (0.04)	0.14 (0.03)	0.14 (0.03)	0.23 (0.04)	0.13 (0.03)
		Via physical limitation	0.16 (0.04)	0.13 (0.03)	0.13 (0.03)	0.21 (0.04)	0.12 (0.02)
		Via reduced social participation	−0.004 (0.01)	0.01 (0.01)	0.01 (0.01)	0.02 (0.01)	0.01 (0.01)
	Widespread pain	Total effect	0.47 (0.19)	0.61 (0.13)	0.49 (0.16)	0.76 (0.17)	0.59 (0.11)
		Direct effect	0.16 (0.21)	0.33 (0.14)	0.20 (0.17)	0.32 (0.18)	0.32 (0.12)
		Indirect effect	0.31 (0.07)	0.27 (0.05)	0.29 (0.06)	0.44 (0.06)	0.27 (0.04)
		Via physical limitation	0.32 (0.08)	0.26 (0.06)	0.27 (0.06)	0.40 (0.07)	0.25 (0.05)
		Via reduced social participation	−0.01 (0.01)	0.01 (0.01)	0.02 (0.02)	0.05 (0.02)	0.02 (0.01)

All values are the standardized β coefficient (S.E.). Indirect effects disentangled into contributions from each pathway variable. Adjusted for age, gender, educational attainment, occupational class, anxiety, depression, other insomnia symptoms at baseline and co-morbidity. Indirect effect indicates the amount of the total effect of baseline pain on the onset of insomnia at 3 years explained by the pathway variable(s).

drawing causal inference, even though the structure of the pathway was time specific and informed by theories of insomnia [46–51]. We focused on physical limitation and social participation as potential mediators because we were interested in identifying mechanisms that were amenable to change (i.e., potential targets for intervention). In the analysis we adjusted for co-morbidity; however, this may not account for medications that induce or hinder sleep. Data on the pathway variables were collected by self-reports. Although standard validated instruments were used, they were susceptible to reporting biases and there might be differences between subjective and objective measures of these variables that merit further investigation [52].

The generalizability of the data may be limited by the characteristics of the study sample; the area covered by the study is more deprived in terms of health, education and employment, but has fewer barriers to housing and services, than England as a whole. As in all longitudinal studies, there was some attrition and missing data throughout the 3 years. The individuals in the sample analysed in this study were younger and healthier than those who were lost to follow-up, but they were less likely to have sleep disturbance at baseline. Sensitivity analysis was performed to investigate the impact of subject attrition and missing data using probability-weighted analyses for survey data [39], and there were no differences in the observed associations between the original and weighted analyses.

Rheumatology key messages

- Widespread and some pain are associated with insomnia onset.
- The frequency of older adults with pain means sleep disturbance is common.
- Physical limitation and reduced social participation are potential targets to prevent insomnia onset in older adults with pain.

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Clinical vignette

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Gouty tophi: here today gone tomorrow

An 80-year-old female presented with a 3 week history of four rapidly enlarging, painful white lumps on her fingers. They soon burst, discharging masses of thick white material (Fig. 1A). Eleven days later, large discharging tophi were diagnosed (Fig. 1B).

She recalled an episode of podagra 20 years previously and had noticed one or two small symptomless white lumps on her fingers. Her urate was 532 μmol/l and her estimated glomerular filtration rate (eGFR) was 50 ml/min. Microscopy showed typical sodium urate crystals.

Risk factors included bendroflumethiazide, stage 3 chronic kidney disease and alcohol intake of 14 U/week, which had not changed for years. A single dose of 7.5 mg of i.v. rasburicase (non-pegylated recombinant uricase) was given. Immediately after infusion her urate decreased to $<$100 μmol/l (lowest level detectable). On allopurinol, her urate has remained between 317 and 321 μmol/l.

Three months post-infusion, most of the tophaceous material had disappeared (Fig. 1C).

This case is remarkable for the very rapid appearance of multiple discharging tophi without an obvious provoking factor. We were surprised at the lack of information in the literature about such a urate storm. The rapid disappearance and healing of the tophi was equally remarkable. The single dose of rasburicase, which reduced the urate to below detectable levels, no doubt played a part here.

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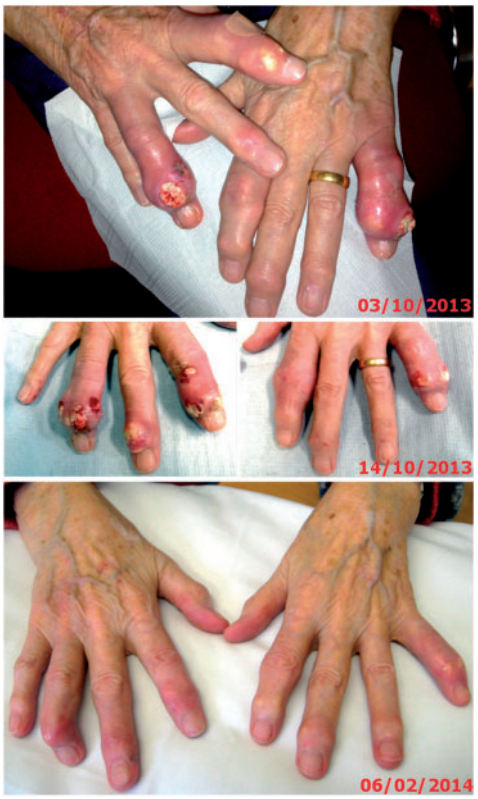
Raisa Mandokhel[1], Gwilym Pritchard[2] and Jeremy Jones[1]

[1]Peter Maddison Rheumatology Centre, Llandudno General Hospital, Llandudno and [2]Liverpool House Surgery, Waunfawr, Gwynedd, UK.

Fig. 1 Serial photographs of the patient's hands showing the extent and rapid resolution of gouty tophi



(**A**) 3 October 2013; (**B**) 14 October 2013; (**C**) 6 February 2014.

Correspondence to: Raisa Mandokhel, Peter Maddison Rheumatology Centre, Llandudno General Hospital, Llandudno LL30 1LB, UK.
E-mail: raisa.khangul@gmail.com