

Predicting New Onset of Widespread Pain Following a Motor Vehicle Collision

GWENLLIAN WYNNE-JONES, GARETH T. JONES, NICOLA J. WILES, ALAN J. SILMAN,
and GARY J. MACFARLANE

ABSTRACT. *Objective.* To determine, in a group of persons involved in a motor vehicle collision, the contributions of pre-collision health and psychological factors, the social environment, collision-specific factors, and post-collision symptoms, to the new onset of widespread pain (WP).

Methods. A prospective cohort study of persons, registered with an insurance company, who had recently experienced a motor vehicle collision. Participants were sent a questionnaire to assess pre-collision health, collision-specific factors, post-collision health, and WP. Those reporting WP prior to the collision were excluded from followup. At 12 months, participants were sent a followup questionnaire to ascertain one-month period prevalence of (new onset) WP.

Results. In total 957 individuals took part in the baseline survey and were eligible for followup. Subsequently, 695 (73%) completed a questionnaire at 12 months, of whom 54 (7.8%) reported new WP. Few collision-specific factors predicted the onset of WP. In contrast, post-collision physical symptoms (rate ratio 2.5, 95% confidence interval 1.2–5.1), pre-collision health-seeking behavior (RR 3.6, 95% CI 1.6–7.9), pre-collision somatization (RR 1.7, 95% CI 0.99–2.8), and perceived initial injury severity (RR 1.7, 95% CI 0.9–3.3), in addition to older age (RR 3.3, 95% CI 1.5–7.1), were all independently predictive of new onset WP. In combination, these factors accounted for about a 20-fold difference in the risk of new onset WP.

Conclusion. We identified 5 factors that independently predict the onset of WP following a motor vehicle collision. Early identification of this “at-risk” group may allow the targeting of preventive management in those at highest risk of developing future symptoms. (First Release Mar 15 2006; J Rheumatol 2006;33:968–74)

Key Indexing Terms:

EPIDEMIOLOGY

WIDESPREAD PAIN

ONSET

TRAUMA

Widespread pain (WP) is a common condition reported by roughly 16% of the population at any one time¹. Of those with WP, 80% will develop chronic symptoms² representing a con-

siderable burden, both to individuals and their families, as well as to healthcare resources.

Individuals with WP often report symptom-onset subsequent to a physically traumatic event³, and there is some research evidence to suggest that this may be the case. Al-Allaf, *et al* examined 136 outpatients with fibromyalgia (FM; of which WP is the cardinal symptom), and reported that 39% had experienced physical trauma in the previous 6 months, compared to only 24% of an age and sex matched control group⁴. Similarly, Greenfield, *et al* showed that 23% of a series of 127 outpatients with FM reported that a physical trauma preceded their symptoms, including motor vehicle collisions, physical injury, and surgery⁵. The association between trauma and risk of new WP has also been examined prospectively. Buskila, *et al* demonstrated that the onset of FM was 13 times more frequent following neck injury than following lower extremity injury⁶, and more recently, Wynne-Jones, *et al* demonstrated that the risk of new onset WP at 6 months was twice as high in persons exposed to a motor vehicle collision, compared to non-exposed individuals⁷. These authors also showed that even after adjusting for psychological distress and somatic symptom-reporting, persons involved in a collision experienced a 40% increase in the risk of WP.

Despite this emerging body of evidence, there is a paucity of data examining, among persons who have experienced a

From the Primary Care Sciences Research Centre, Keele University, Keele; Aberdeen Pain Research Collaboration, Epidemiology Group, University of Aberdeen, Aberdeen; Academic Unit of Psychiatry, Department of Community Based Medicine, University of Bristol, Bristol; and the ARC Epidemiology Unit, The University of Manchester, Manchester, United Kingdom.

Supported by the Association of British Insurers and the Arthritis Research Campaign. However, the opinions in this report, content, and choice of words are those of the authors alone. G. Wynne-Jones was funded by a Medical Research Council PhD studentship.

G. Wynne-Jones, RN, BSc (Hons), Post-doctoral Research Fellow, Primary Care Sciences Research Centre, Keele University; G.T. Jones, BSc (Hons), MSc Econ, PhD, Senior Lecturer in Epidemiology, Epidemiology Group, Department of Public Health, University of Aberdeen; N.J. Wiles, BSc (Hons), PhD, Lecturer in Epidemiology, Academic Unit of Psychiatry, Department of Community Based Medicine, University of Bristol; A.J. Silman, MD, FRCP, Professor of Rheumatic Disease Epidemiology, ARC Epidemiology Unit, The University of Manchester; G.J. Macfarlane, BSc (Hons), MBChB, PhD, MD (Hons), Professor of Epidemiology, Epidemiology Group, Department of Public Health, University of Aberdeen.

Address reprint requests to Dr G.T. Jones, Epidemiology Group, Department of Public Health, University of Aberdeen, Polwarth Building, Foresterhill, Aberdeen, AB25 2ZD, UK. E-mail: gareth.jones@abdn.ac.uk

Accepted for publication November 15, 2005.

Personal non-commercial use only. The Journal of Rheumatology Copyright © 2006. All rights reserved.

traumatic event, which factors specifically predict the onset of WP. Thus, we investigated, in a group of persons recently involved in a motor vehicle collision, the contribution of pre-collision health and psychological factors, the psychosocial work environment, collision-specific factors, and post-collision symptoms to the new onset of WP.

MATERIALS AND METHODS

Design. A prospective cohort study. Participants were recruited from a large national UK based insurance company as they made a claim following a motor vehicle collision. Participants were required to be aged 17–70 years, currently resident in the UK, and fluent in written English. Those who agreed to participate were sent a postal questionnaire.

The baseline questionnaire assessed WP in the month prior to the collision. Participants were asked whether they had experienced any aches or pains lasting for 1 day or longer in the month prior to the collision. Those who answered positively were asked to identify the location of their pain(s) on a 4-view full-body manikin. WP was identified using the criteria proposed by the American College of Rheumatology in their classification of FM⁸ — thus, WP was deemed to be present if pain was reported both above and below the waist, in both the left and right sides of the body, and in the axial skeleton. Individuals were excluded from the study if they reported WP during this 1-month pre-collision period.

The questionnaire also collected data on a number of potential risk factors for the future onset of WP: pre-collision health and psychological factors, mechanical (collision-specific) factors, and post-collision health. Because participants were asked to recall 3 distinct time periods, clear time references were given for each question: “Thinking back over the month before your collision,” “thinking about your collision,” and “since your collision.”

Baseline questionnaire

Pre-collision health and psychological factors. General psychological distress in the month prior to the collision was measured using the 12-item General Health Questionnaire (GHQ)⁹. This GHQ was scored using the Likert method and dichotomized for analysis at the median value: low (< 22) and high (≥ 22). Health anxiety and health behavior in the month prior to the collision were measured using the Illness Attitudes Scale (IAS)¹⁰. High scores on the illness behavior subscale refer to a history of frequent consultations, referrals, and receiving treatment. Participants were also asked to rate their general health (excellent to poor) and to report the number of times they had visited their primary care physician in the year preceding the collision.

In addition, participants were asked to complete the Somatic Symptoms Checklist¹¹. This 7-item instrument measured the occurrence of a number of symptoms (e.g., trouble breathing, forgetfulness, vomiting) pre-collision. Finally, for persons in current employment, work related psychosocial factors (demands, control, and support) were assessed using questions previously validated in a large occupational cohort study¹².

Collision-specific factors. Information on a number of aspects of the collision was requested, including: speed, direction, the use of safety features (seatbelt, airbag, headrest), anticipation of impending collision, and perceived collision severity.

Post-collision health. Participants were asked whether they had experienced any of the following physical symptoms since their collision: headaches, dizziness, abnormal or tingling sensations, tinnitus, problems with vision, neck, shoulder, back or arm pain, and problems with memory. Physical symptoms were scaled and analyzed as “none” versus “one or more” symptoms. Also, the perceived severity of any injuries sustained in the collision was measured using a 100 mm visual analog scale.

Followup. Participants who were free of WP in the month prior to their collision were eligible for followup and were recontacted by postal questionnaire 12 months subsequently. WP (by definition, new onset WP) was assessed in the same manner as at baseline.

Analysis. Initial analysis examined which baseline factors were associated with the onset of WP at followup. This was assessed using Poisson regression

models, and results are expressed as rate ratios (RR) with 95% confidence intervals (95% CI), the latter being calculated using robust estimates of standard error¹³. All analysis was conducted using Stata v8.2 (Stata Corp., College Station, TX, USA) and, unless otherwise stated, was adjusted for age and sex. Variables that, after age and sex adjustment, predicted new onset WP at $p \leq 0.2$ (as assessed using a Wald test) were offered to a forward stepwise Poisson regression modeling procedure to identify independent predictors of outcome. Variables were included in the final model at $p \leq 0.10$ and excluded at $p \geq 0.15$.

External validity. It was anticipated that many individuals reporting a collision to their vehicle insurers might be reluctant to participate in the study, resulting in a low participation rate. Therefore, the insurance company was asked to provide anonymized data (age and sex) on all claimants who were invited to participate. Additionally, some data were gathered from those individuals who initially agreed to participate but subsequently failed to return a questionnaire. A short questionnaire, either self-completed or through telephone interviews, was utilized to collect this additional information. These additional data were compared with that provided by full participants. Finally, a weighted analysis was conducted, weighting the full dataset back to the age and sex distribution of the target population.

Ethical approval for the study was obtained from the University of Manchester Committee on the Ethics of Research on Human Beings.

RESULTS

Subjects. Altogether 2665 individuals were invited to participate in the study, of whom 1907 (72%) agreed to receive a questionnaire. A group of 1499 individuals (79%) completed a baseline questionnaire, 1003 of whom (67%) completed a full-length questionnaire. The median age of the cohort was 41 years [interquartile range (IQR) 33–50 yrs] and 51% were female. Questionnaires were completed at a median of 23 days post-collision (IQR 13–39 days). Forty-six individuals (4.6%) reported WP in the month prior to the collision and were therefore excluded. The remaining 957 individuals were eligible for followup and thus composed the baseline cohort.

Followup. At 12 months, 695 participants returned a followup questionnaire with completed pain data (followup response rate 73%). Of these individuals, 54 (7.8%) reported new onset WP. Prevalence increased with age: 3.5% in persons aged 19–36 years versus 12.6% in those aged 48–70 years (chi-square for trend, in tertiles: 13.02; $p < 0.001$), although there was no difference in WP onset by sex: 7.9% in men and 7.7% in women (chi-square 0.01; $p = 0.94$).

Pre-collision health and psychological factors. Compared to persons who rated their health prior to the collision as excellent/very good, those who reported good, fair, or poor health experienced nearly a doubling in the risk of new onset WP (Table 1). Similarly, participants who had visited their primary care physician frequently in the year prior to the collision, or who scored highly on the IAS health behavior scale, were more likely to report new onset WP than other individuals (RR 3.3, 95% CI 1.4–7.7, and RR 5.0, 95% CI 2.2–11.1, respectively). High levels of health anxiety and the reporting of somatic symptoms (pre-collision) were also associated with an increase in risk. In contrast, a high level of psychological distress was associated with neither an increase nor decrease in the risk of developing WP. Similarly, there was no consis-

Table 1. Relationship between pre-collision health and psychological factors and new onset widespread pain (WP).

Exposure	Total	New Onset WP, n (%)	Age and Sex Adjusted RR (95% CI)
Perceived general health			
Excellent/very good	516	31 (6.0)	1.0
Good	139	17 (12.2)	1.8 (1.03–3.2)
Fair/poor	38	6 (15.8)	1.9 (0.8–4.3)
No. of visits to primary care in past year			
None	164	6 (3.7)	1.0
1	173	9 (5.2)	1.4 (0.5–3.8)
2	149	12 (8.1)	2.1 (0.8–5.5)
≥ 3	209	27 (12.9)	3.3 (1.4–7.7)
IAS—Health behavior (tertiles)			
Low (0–2)	250	7 (2.8)	1.0
Medium (3–5)	246	18 (7.3)	2.5 (1.1–5.8)
High (≥ 6)	194	29 (14.9)	5.0 (2.2–11.1)
IAS—Health anxiety (tertiles)			
Low (0–3)	267	15 (5.6)	1.0
Medium (4–8)	209	17 (8.1)	1.3 (0.6–2.5)
High (≥ 9)	209	21 (10.0)	1.5 (0.8–2.9)
Somatic Symptoms Checklist			
0–1 symptoms	590	39 (6.6)	1.0
≥ 2 symptoms	105	15 (14.3)	2.4 (1.4–4.2)
Psychological distress (GHQ; dichotomized at median)			
Low (< 22)	450	35 (7.8)	1.0
High (≥ 22)	243	19 (7.8)	1.0 (0.6–1.7)

RR: rate ratio, IAS: Illness Attitudes Scale¹⁰, GHQ: General Health Questionnaire⁹.

tent evidence to suggest that workplace psychosocial factors were associated with an increase or decrease in the risk of new onset WP (Table 2).

Collision-specific factors. None of the collision-specific factors was significantly associated with the onset of WP. In particular, self-reported speed and awareness of impending collision were unrelated to the risk of WP onset (Table 3). However, participants who perceived their collision to be severe (2nd or 3rd tertile) experienced approximately a doubling in the risk of symptoms, while those hit from the side were less likely to develop WP than those hit from the rear (RR 0.5, 95% CI 0.2–1.008).

Although measured, too few participants reported airbag deployment, having a nonadjustable headrest, or lack of seat-belt use to allow meaningful analysis of these variables.

Post-collision health. Participants who reported any adverse physical symptoms after their collision experienced a significant increase in the risk of developing WP (RR 3.7, 95% CI 2.1–6.5). Also, those who perceived their initial injuries to be more severe experienced an increase in risk (Table 4). The occurrence of neck pain both pre- and post-collision was associated with an increase in the risk of WP onset. The greatest risk was observed in participants who reported neck pain at both timepoints; these persons experienced a 3-fold increase in risk (RR 3.3, 95% CI 1.5–6.8).

Multivariable analysis. Four factors, plus age, remained sta-

tistically independent predictors of new onset WP: the number of physical symptoms post-collision, pre-collision health behavior, pre-collision somatic symptom reporting, and perceived injury severity (Table 5, Model 1).

As a crude estimate of model performance, a count was made of how many of these factors each individual was exposed to. Non-dichotomous variables were split such that persons in the highest tertile were considered as “exposed,” while those in the lower 2 tertiles were considered “unexposed.” The risk of new onset WP was then examined with respect to this new summary variable. In combination, the variables in the multivariable model were able to identify individuals with about a 20-fold difference in the risk of developing WP (Figure 1): fewer than 2% of persons with a score of zero (those with none of the factors in the final model) developed WP; whereas among those 4 or more factors, 36% experienced future WP.

External validity. As described, of the 1907 individuals who initially agreed to participate in the study, only 1003 subsequently returned a full-length baseline questionnaire. A subgroup (n = 496) of the remaining 904 individuals went on to return a shortened version of the questionnaire, but were unable to be included in the main analysis due to missing data on pre-collision WP. There were few systematic differences between short- and full-length questionnaire participants. No differences were observed regarding pre-collision general

Table 2. Relationship between work and work-related psychosocial factors and new onset widespread pain (WP).

Exposure	Total	New Onset WP, n (%)	Age and Sex Adjusted RR (95% CI)
Employment (full- or part-time)			
Yes	595	42 (7.1)	1.0
No	100	12 (12.0)	1.3 (0.7–2.4)
Boring work*			
Never/occasionally	527	34 (6.5)	1.0
Half or all the time	67	8 (11.9)	1.9 (0.9–3.8)
Hectic or fast work*			
Never/occasionally	401	31 (7.7)	1.0
Half or all the time	193	11 (5.7)	0.7 (0.4–1.4)
Stressful work*			
Never/occasionally	485	38 (7.8)	1.0
Half or all the time	109	4 (3.7)	0.5 (0.2–1.3)
Able to make decisions at work*			
Very often/often	520	39 (7.5)	1.0
Sometimes/seldom	73	3 (4.1)	0.6 (0.2–1.8)
Learn new things at work*			
Very often/often	368	22 (6.0)	1.0
Sometimes/seldom	226	20 (8.8)	1.4 (0.8–2.5)
Support from supervisor*			
Very satisfied/satisfied	338	26 (7.7)	1.0
Very dissatisfied/dissatisfied	190	11 (5.8)	0.8 (0.4–1.5)
Support from colleagues*			
Very satisfied/satisfied	454	31 (6.8)	1.0
Very dissatisfied/dissatisfied	101	8 (7.9)	1.0 (0.3–3.0)

* Data available only from persons in full- or part-time employment. RR: rate ratio.

Table 3. Relationship between collision-specific factors and new onset widespread pain (WP).

Exposure	Total	New Onset WP, n (%)	Age and Sex Adjusted RR (95% CI)
Speed of collision			
Stationary	269	25 (9.3)	1.0
Low (\leq 11 mph)*	200	10 (5.0)	0.5 (0.3–1.004)
High (\geq 12 mph)*	226	19 (8.4)	0.9 (0.5–1.6)
Anticipation of impending collision			
No	412	34 (8.3)	1.0
Yes	260	19 (7.3)	1.0 (0.6–1.8)
Perceived collision severity (tertiles of 100 mm VAS)			
Low (1–11 mm)	237	12 (5.1)	1.0
Medium (12–28 mm)	228	21 (9.2)	1.9 (0.95–3.7)
High (29–100 mm)	230	21 (9.1)	1.9 (0.97–3.7)
Direction of collision			
From rear	256	23 (9.0)	1.0
From front	186	16 (8.6)	1.0 (0.5–1.8)
Shunt (rear and front)	18	2 (11.1)	1.3 (0.3–5.5)
From side	200	9 (4.5)	0.5 (0.2–1.008)
Other	12	2 (16.7)	2.0 (0.6–6.7)

* Dichotomized at median speed of all moving vehicles. VAS: visual analog scale. RR: rate ratio.

health (chi-square 0.16, $p = 0.89$), collision direction (chi-square 8.09, $p = 0.15$), or neck pain pre- or post-collision (chi-square 0.74, $p = 0.39$, and chi-square 0.53, $p = 0.47$, respectively). However, nonparticipants were younger (median age 33 and 40 yrs, respectively; Mann-Whitney $Z = 7.56$, $p < 0.001$)

and a greater proportion were male (66% and 49%, respectively; chi-square 19.68, $p < 0.001$). However, weighting the final analysis back to the original age and sex distribution of the target population revealed similar results to the main findings and did not alter the study conclusions (Table 5, Model 2).

Table 4. Relationship between post-collision health and new onset widespread pain (WP).

Exposure	Total	New Onset WP, n (%)	Age and Sex Adjusted RR (95% CI)
Physical symptoms			
None	397	15 (3.8)	1.0
≥ 1 symptom	295	39 (13.2)	3.7 (2.1–6.5)
Perceived injury severity (100 mm VAS)			
No injuries (0 mm)	463	25 (5.4)	1.0
Low (1–15 mm)*	114	10 (8.8)	1.8 (0.9–3.5)
High (16–100 mm)*	118	19 (16.1)	3.3 (1.9–5.6)
Neck pain			
None	464	23 (5.0)	1.0
Pre-collision only**	39	4 (10.3)	2.0 (0.7–5.5)
Post-collision only	153	20 (13.1)	2.8 (1.6–5.0)
Pre- and post-collision	37	7 (18.9)	3.3 (1.5–6.8)

* Dichotomized at median value of all scores ≥ 1 mm. ** Reported neck pain at some point in the month prior to the collision. VAS: visual analog scale, RR: rate ratio.

Table 5. Multivariable model — independent predictors of widespread pain onset.

Exposures	Rate Ratio (95% CI)	
	Model 1 (unweighted)	Model 2 (weighted*)
Age (tertiles), yrs		
19–36	1.0	1.0
37–47	2.1 (0.95–4.6)	1.8 (0.8–4.1)
48–70	3.4 (1.6–7.1)	3.0 (1.4–6.4)
Sex		
Male	1.0	1.0
Female	0.8 (0.5–1.3)	0.8 (0.5–1.3)
No. of physical symptoms (post-collision)		
None	1.0	1.0
≥ 1 symptom	2.5 (1.2–5.1)	2.3 (1.1–5.1)
IAS—Health behavior (pre-collision)		
Low (0–2)	1.0	1.0
Medium (3–5)	2.1 (0.9–4.8)	2.1 (0.9–4.9)
High (≥ 6)	3.6 (1.6–7.9)	3.4 (1.5–7.7)
Somatic Symptoms Checklist (pre-collision)		
0–1 symptom	1.0	1.0
≥ 2 symptoms	1.7 (0.99–2.8)	1.7 (0.98–2.8)
Perceived injury severity (100 mm VAS)		
No injuries (0 mm)	1.0	1.0
Low (1–15 mm)	0.9 (0.4–2.1)	0.97 (0.4–2.4)
High (16–100 mm)	1.7 (0.9–3.3)	1.8 (0.9–3.5)

* Weighted to the age/sex distribution of the target population. IAS: Illness Attitudes Scale¹⁰.

DISCUSSION

We have demonstrated that among persons free of WP who are involved in a motor vehicle collision, approximately 8% develop WP in the subsequent 12 months. Further, we have shown that the strongest predictors of new onset WP in this group are factors relating to pre-collision health (health behavior, number of somatic symptoms), the number of physical symptoms in the month following the collision, and self-rating of initial injury. In contrast, mechanical (collision-related) factors are much less important.

In interpreting these results there are a number of method-

ological issues that need to be considered. Although 72% of eligible drivers agreed to participate in the study, only 53% of these individuals subsequently returned a full-length baseline questionnaire. Nonparticipants were more likely to be male and young. The crucial issue, however, is not whether or not there was differential participation across (for example) age/sex strata, but whether this may have introduced bias to the study results. There was no difference in the occurrence of new onset WP by sex, although prevalence increased with age. It is possible, therefore, that because of the underrepresentation of young persons in the sample we have overestimated

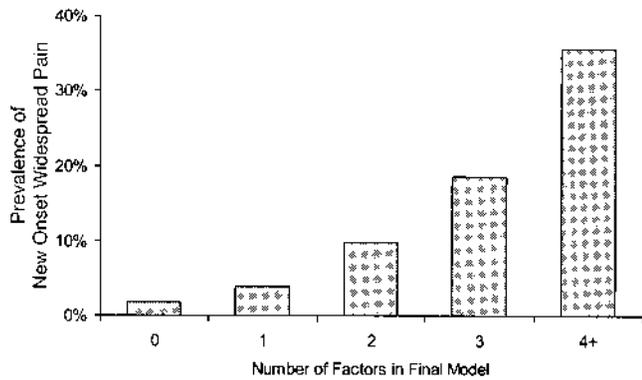


Figure 1. Performance of the multivariable model.

the occurrence of WP in the group as a whole. However, further analysis, weighting the prevalence estimate for each age/sex stratum by the inverse of the sampling proportion for that stratum, revealed a slight overestimation in men (7.9% in the study sample, 7.7% in the weighted sample) but an underestimation in women (7.7% and 8.0%, respectively) and the prevalence of new onset WP in the group as a whole would be unchanged. Further, a subsequent regression analysis, weighting the data back to the age/sex distribution of the target population, did not greatly affect the risk estimates obtained from the main analysis. Other than age and sex, there were no significant differences between participants and nonparticipants with respect to other known variables. Therefore, insofar as could be ascertained, we believe that nonparticipation has not introduced a bias into our study. In addition, followup of the 957 baseline participants was good (73%), and nonrespondents at followup did not differ from respondents in terms of age or sex (data not shown). There is little evidence, therefore, to suggest that our study suffered from bias introduced by selective loss to followup.

Although the study was prospective in design, and thus recall bias would not ordinarily be a concern, baseline data were collected shortly after involvement in a motor vehicle collision. It is possible, of course, that some participants may have overestimated their health status prior to the collision by attributing any ill health post-collision to the collision itself. However, any measurement error introduced in this manner would serve to bias our results toward the null. Thus, all risk estimates we present may actually be underestimates of the true risk associated with these factors. Further, measurements of pre-collision health that are more objective, and therefore less prone to bias (e.g., the number of visits to primary care in the year prior to the collision), were still associated with large and significant increases in the risk of developing WP. In contrast, the measurement of several of our variables may have biased results away from the null — for example: perceived collision severity may have been influenced by symptoms immediately post-collision, thus augmenting any potential association with new onset WP. However, these variables were not, on multivariable analysis, independent predictors of

new onset WP and this is not, therefore, a major cause of concern.

Previous studies have reported an association between various kinds of physical trauma (motor vehicle collision, physical injury, and surgery) and the occurrence of WP⁴⁻⁷. Specifically, previous work has shown a 40% increase in the risk of WP in persons involved in a motor vehicle collision, after adjusting for the effects of psychological distress⁷. However, our study is the first to examine the relationship between a number of pre-collision health and psychosocial factors, collision-specific, and post-collision factors and the risk of WP onset in a group of individuals known to have experienced a motor vehicle collision. Others have shown that 23%⁵ and 39%⁴ of persons with FM report prior involvement in a physically traumatic event. However, these studies recruited participants (patients) from specialist rheumatology clinics and were more likely, therefore, to identify recurrent or persistent cases. In contrast, our study recruited individuals who were free of WP and looked longitudinally at the onset of WP post-collision.

There is little work examining the onset of WP in the general population. McBeth, *et al*, in a community-based sample, demonstrated that roughly 6% of persons developed chronic WP over a 12-month period¹⁴, whereas our findings (7.8%) are slightly higher. The majority of participants in our study experienced relatively minor collisions. However, these results are consistent with the hypothesis that the risk of WP in a “trauma” cohort is greater than that of the general population. Further, our findings are similar to those of a study that recruited participants in an identical manner and showed the onset of WP over a 6-month period to be 8% in persons involved in a motor vehicle collision⁷.

Because our study is the first prospective study of the risk factors for WP onset in persons involved in a motor vehicle collision, there is little directly comparable literature. Previous work has shown that the risk of WP in persons who have been exposed to a motor vehicle collision is twice that of unexposed individuals, but the excess risk is attenuated by adjustment for prior levels of psychological distress and the occurrence of somatic symptoms⁷. In addition, McBeth, *et al* demonstrated that health behavior and the occurrence of somatic symptoms were associated with approximately a 9-fold and a 3-fold increase in the risk of new onset WP, respectively, although they failed to demonstrate a strong relationship with psychological distress¹⁴.

In addition to aspects of prior health, we have demonstrated that the occurrence of physical symptoms immediately post-collision significantly, and independently, predict the onset of WP at 12 months. Persons with one or more symptoms (headaches, dizziness, tinnitus, problems with vision, etc.) were 3 times more likely to report new onset WP than other individuals. These symptoms have also been shown to be associated with poor whiplash prognosis¹⁵, and there is increasing evidence that the outcome of whiplash injury is a

function of many factors, but includes psychological and psychosocial elements¹⁶⁻¹⁸. Our study adds to this and highlights the relative unimportance of collision-specific factors in the etiology of WP following a motor vehicle collision.

In summary, there are numerous anecdotal reports to suggest that physical trauma is associated with an increase in the risk of WP (or FM, of which WP is the predominant feature), although only recently has robust scientific evidence been able to support this. However, few studies have attempted to identify which factors predict the onset of symptoms in a group of individuals known to have experienced a physically traumatic event. We have demonstrated that, in a group of individuals involved in a motor vehicle collision, mechanical (collision-specific) factors, such as speed and direction of collision, are relatively poor predictors of WP onset. In contrast, WP is best predicted by aspects of prior (pre-collision) health and health behavior, and by the occurrence of physical symptoms and injury severity post-collision. In combination, these factors, plus age, can account for a 20-fold difference in the risk of developing WP. We believe that the early identification of this "at-risk" group may enable the targeting of preventive management in those with the highest risk of future symptoms.

ACKNOWLEDGMENT

We would like to thank the staff, in particular Jane Ogle and Angela Robson, at Direct Line Insurance, Manchester, who invited drivers to take part in the study. We are also very grateful to Mark Blything for assistance with study conduct.

REFERENCES

1. Croft PR, Rigby AS, Boswell R, Schollum J, Silman AJ. The prevalence of chronic widespread pain in the general population. *J Rheumatol* 1993;20:710-3.
2. Macfarlane GJ, McBeth J, Silman AJ. Widespread body pain and mortality: prospective population based study. *BMJ* 2001;323:662-5.
3. Wolfe F. Post-traumatic fibromyalgia: a case report narrated by the patient. *Arthritis Care Res* 1994;7:161-5.
4. Al-Allaf AW, Dunbar KL, Hallum NS, Nosratzadeh B, Templeton KD, Pullar T. A case-control study examining the role of physical trauma in the onset of fibromyalgia syndrome. *Rheumatology* Oxford 2002;41:450-3.
5. Greenfield S, Fitzcharles MA, Esdaile JM. Reactive fibromyalgia syndrome. *Arthritis Rheum* 1992;35:678-81.
6. Buskila D, Neumann L, Vaisberg G, Alkalay D, Wolfe F. Increased rates of fibromyalgia following cervical spine injury. A controlled study of 161 cases of traumatic injury. *Arthritis Rheum* 1997;40:446-52.
7. Wynne-Jones G, Macfarlane GJ, Silman AJ, Jones GT. Does physical trauma lead to an increase in the risk of new onset widespread pain? *Ann Rheum Dis* 2006;65:391-3.
8. Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. *Arthritis Rheum* 1990;33:160-72.
9. Goldberg DP, Williams P. A user's guide to the General Health Questionnaire. Windsor: NFER-Nelson; 1988.
10. Kellner R, Abbott P, Winslow WW, Pathak D. Fears, beliefs and attitudes in DSM-III hypochondriasis. *J Nerv Ment Dis* 1987;175:20-5.
11. Othmer E, DeSouza C. A screening test for somatization disorder (hysteria). *Am J Psychiatry* 1985;142:1146-9.
12. Nahit ES, Pritchard CM, Cherry NM, Silman AJ, Macfarlane GJ. The influence of work related psychosocial factors and psychological distress on regional musculoskeletal pain: a study of newly employed workers. *J Rheumatol* 2001;28:1378-84.
13. Greenland S. Model-based estimation of relative risks and other epidemiologic measures in studies of common outcomes and in case-control studies. *Am J Epidemiol* 2004;160:301-5.
14. 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.
15. Atherton K, Wiles NJ, Lecky FE, et al. Predictors of persistent neck pain after whiplash injury. *Emerg Med J* 2006;in press.
16. Richter M, Ferrari R, Otte D, Kuensebeck HW, Blauth M, Krettek C. Correlation of clinical findings, collision parameters, and psychological factors in the outcome of whiplash associated disorders. *J Neurol Neurosurg Psychiatry* 2004;75:758-64.
17. Hendriks EJ, Scholten-Peters GG, van der Windt DA, Neeleman-van der Steen CW, Oostendorp RA, Verhagen AP. Prognostic factors for poor recovery in acute whiplash patients. *Pain* 2005;114:408-16.
18. Sterling M, Jull G, Vicenzino B, Kenardy J, Darnell R. Physical and psychological factors predict outcome following whiplash injury. *Pain* 2005;114:141-8.