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Chronic Widespread Pain after Motor Vehicle Collision Typically Occurs via Immediate Development and Non-Recovery: Results of an Emergency Department-Based Cohort Study

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Abstract

Motor vehicle collision (MVC) can trigger chronic widespread pain (CWP) development in vulnerable individuals. Whether such CWP typically develops via the evolution of pain from regional to widespread or via the early development of widespread pain with non-recovery is currently unknown. We evaluated the trajectory of CWP development (American College of Rheumatology criteria) among 948 European-American individuals who presented to the emergency department (ED) for care in the early aftermath of MVC. Pain extent was assessed in the ED and 6 weeks, 6 months, and 1 year after MVC on 100%, 91%, 89%, and 91% of participants, respectively. Individuals who reported prior CWP at the time of ED evaluation (n = 53) were excluded. Trajectory modeling identified a two-group solution as optimal, with the Bayes Factor value (138) indicating strong model selection. Linear solution plots supported a non-recovery model. While the number of body regions with pain in the non-CWP group steadily declined, the number of body regions with pain in the CWP trajectory group (192/895, 22%) remained relatively constant over time. These data support the hypothesis that individuals who

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develop CWP after MVC develop widespread pain in the early aftermath of MVC which does not remit.

1. Introduction

Population-based studies indicate that 11–13% of adults suffer from chronic widespread pain (CWP) [46], defined as pain on both the left and right side of the body, above and below the waist, and including the axial region [50]. These individuals experience substantial disability and reduced quality of life [11; 27]. The pathogenesis of CWP remains poorly understood [25; 28; 29; 35], and gaining a greater understanding of this disease is an important public health priority [19].

One known trigger of CWP is motor vehicle collision (MVC) [18; 22; 51; 52]; the natural history of CWP development after MVC is currently unknown. One common hypothesis is that chronic regional pain first develops and then progresses to CWP in vulnerable individuals [18; 23; 25; 46]. Another possibility is that in vulnerable individuals CWP develops in the early aftermath of MVC and persists [7; 18]. Accurately identifying the typical trajectory of CWP development after MVC is important both in order to gain insights into potential etiologic mechanisms and also to inform the design of future studies.

In this study, we examined the developmental trajectory of CWP among a cohort of individuals who presented to the emergency department (ED) for care after MVC and were followed prospectively for one year. Trajectory modeling was used to determine if either a progressive onset model of CWP development or an early onset/non-recovery model of CWP development provided a good fit to the data. Because evidence suggests that mechanisms related to stress-induced hyperalgesia play an important role in the pathogenesis of chronic pain after MVC (reviewed in [41]), and because such mechanisms are mediated by central neurobiological systems mechanisms with widespread effects and relatively early onset (reviewed in [41]), we hypothesized that CWP development would be characterized by a non-recovery rather than a progressive accrual model. In addition, we evaluated demographic characteristics of participants with and without CWP after MVC and co-morbid psychological and somatic symptoms in these groups.

2. Methods

2.1 Design and setting

This prospective longitudinal study enrolled patients presenting to the ED within 24 hours of MVC. Data were collected at eight EDs in four no-fault MVC litigation/insurance states, where litigation related to MVC is low, (Michigan, Massachusetts, New York, and Florida) between February 2009 and October 2011. Study participants were enrolled at research network ED sites and received an initial interview evaluation at the time of the ED visit. Follow-up assessments were performed at 6 weeks, 6 months, and one year. The study was approved by the institutional review boards of all participating hospitals, and each participant provided written informed consent. Complete information regarding study design, procedures, and methods has previously been described. [38]

2.2 Participant eligibility criteria and study sites

Patients ages 18 to 65 who presented to the ED within 24 hours after a MVC and were unlikely to require hospitalization were screened for eligibility. Patients who were admitted to the hospital, had fractures other than phalangeal fractures, had more than 4 lacerations requiring sutures or a single laceration more than 20 cm in length, or had intracranial or spinal injuries were excluded. Spinal injury was defined by the presence of a fracture, dislocation, or new neurologic deficit. Enrollment was also limited to non-Hispanic whites (the most common ethnicity at study sites) because the study included the collection of genetic data and genetic analyses are potentially biased by population stratification.[16] Patients who were not alert and oriented were also excluded, as were pregnant patients, prisoners (due to risk of coercion [5]), patients unable to read and understand English, patients taking a β-adrenoreceptor antagonist (due to concern for confounding because β-adrenergic mechanisms have been implicated in chronic pain development [32]), or patients taking opioids above a total daily dose of 20 mg of oral morphine or equivalent.

2.3 Study procedures

Eligible and consenting participants completed ED interview evaluations regarding pre-MVC health status, the details of the MVC, and current symptoms. Interviews were conducted by research staff at the time of the ED visit using a web-based survey with explicit definitions of variables. Before enrolling patients in the ED, each research staff member completed a study training module followed by an interview with a standardized mock ED patient. Comparison of mock ED patient data across research staff demonstrated an error rate of 1.3%. Injury characteristics and medications administered in the ED were obtained by data extraction from the ED medical record. Study personnel extracted participant injury data from the participant's medical record using a standardized web-based data extraction form with explicit definitions of all variables. All aspects of the emergency medical record, including physician notes, nursing notes, and physical orders were used for completion. Information obtained included the presence and location of any fractures, lacerations, contusions, avulsions, and abrasions. Injury information was used to generate Abbreviated Injury Severity Scores [1]. Six weeks, six months and one year after the MVC, participants completed a follow up interview online, by telephone, or via mail. Regardless of follow up type, survey content was identical. Participants were compensated $80 for completing the ED interview, $60 for completing the 6 week interview, $65 for completing the 6 month interview and $70 for completing the 1 year interview.

2.4 Measures

A number of measures were used to assess health status prior to MVC and symptoms in the ED. Complete study measures are described in full elsewhere.[38]

2.4.1 Participant demographics—Participant demographic characteristics (including age, gender, income, height, weight, and educational attainment) were obtained from the ED medical record and from participant self-report.

2.4.2 Pain assessments and pain outcome definitions—Pain extent during the month prior to the MVC and in the ED were both assessed at the time of ED evaluation; pain

extent six weeks, six months and one year after MVC was assessed at the respective timepoint. Pain extent was assessed in 19 discrete body regions evaluated in the regional pain scale[49] and in the head region. Widespread pain (WP) was defined according to the American College of Rheumatology (ACR) 1990 criteria [50]. In each body region in which the participant reported pain, average pain severity was assessed using a verbal 0 to 10 numeric rating scale (NRS). Verbal scores have advantages in acute care settings, and verbally administered NRSs have been validated as highly correlated with visual analogue scale scores. [34] Regional pain was defined by the presence of one or more body regions with a NRS score greater than zero. Number of body regions with pain at each timepoint was defined as the number of body regions with a NRS score greater than zero.

2.4.3 Psychological symptoms—Distress was assessed using the Peritraumatic Distress Inventory (PDI). This measure has high internal consistency (0.75–0.76) and test-retest reliability (0.74).[10] A PDI cut-off score of ≥23 was used to define marked distress symptoms ("distress").[36] Post-traumatic stress disorder symptoms were measured at 6-week, 6-month, and 1-year timepoints using the Impact of Events Scale – Revised (IES-R) [47]. Depressive symptoms during the week prior to the MVC were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D).[40] Based on previous evidence, CES-D scores between 16 and 25 were defined as mild depressive symptoms and scores greater than or equal to 26 were defined as severe depressive symptoms.[48; 53]

2.4.4 Somatic symptoms—Somatic symptoms during the past month were assessed at the time of the ED assessment and via telephone or web-based questionnaire six weeks, six months, and one year after MVC. The somatic symptom assessment consisted of a 21-item questionnaire that evaluated the presence and severity of the following: headache, dizziness, nausea, noise sensitivity, light sensitivity, poor concentration, taking longer to think, blurred vision, double vision, restlessness, upset stomach, fatigue, sensitive or tender skin, ringing in ears, itchy eyes or skin, racing heart, insomnia, hands trembling, feeling faint, abdominal pain, diarrhea and constipation. Participants were asked to report the severity of each symptom on a 0–10 scale, where 0 represented no problem and 10 represented a major problem. Number of somatic symptoms at each timepoint was defined as the number of somatic symptoms greater than zero.

2.5 Statistical analyses

Descriptive and trajectory analyses of extent of pain across time were performed using SAS 9.3 (SAS Institute, Cary, NC). Descriptive analysis of the whole cohort, after excluding previous WP participants, was evaluated. Trajectory analyses were performed using PROC TRAJ. This SAS procedure, developed by Jones, Nagin and Roeder, was designed to estimate clusters of individuals following similar trends over time[21]. Model fit in TRAJ is based on the Bayesian Information Criterion (BIC), a statistic that is similar to adjusted R^2 as it incorporates model complexity and overall fit. BIC value is a fit index used to compare competing models that include different numbers or shapes of trajectories. BIC values are negative, and in comparing the different models, the best fit model is the one with the smallest negative number, closest to 0. To test the inclusion of different numbers of trajectories, the estimate of the log Bayes Factor is defined with the following formula: $2\log_e$

(B_{10}) ≈ 2(ΔBIC)[21]. The difference is calculated by subtracting the BIC value of the simpler model, with a smaller number of trajectories, from the more complex model. According to the guidelines, values ranging from 0–2 are interpreted as weak evidence for the more complex model, 2 to 6 as moderate evidence, 6–10 as strong and values greater than 10 as very strong evidence. The comparisons are completed in a step-wise manner so that the two-group model is compared to the one-group, and the three-group model is compared to the two-group model. Trajectory membership for each individual was used as an identifier, and further descriptive analyses were conducted.

3. Results

3.1 Cohort characteristics

A total of 10,629 patients were screened, 1,416 were eligible, 969 consented to study participation and 948 completed baseline evaluation. Slightly more than 60% of participants were females, more than three quarters had some education past high school, and more than half worked full time (Table 1). The median age of study participants was 36 (range 18–65). Consistent with eligibility criteria, all participants were discharged to home after ED evaluation. Ninety three percent (710/948) of the study participants had musculoskeletal strain only, and nearly all participants (939/948, 99%) had an Abbreviated Injury Scale score of 1. Fractures were present in 1/948 (<1%, phalanx fracture) participants; a small laceration was present in 53/948 (6%) participants. Six week, six month, and one year follow-up assessments were completed on 859/948 (91%), 840/948(87%), and 861/948 (91%) of enrolled patients, respectively.

3.2 WP outcomes in the cohort over time

Widespread pain prior to the MVC was reported by 53/948 (6%) of participants; these patients were excluded from subsequent analyses. MVC-related WP was present in 238/895 (27%) at the ED, 160/808 (20%) participants six weeks after MVC, 97/790 (12%) participants six months after MVC, and 78/809 (10%) participants one year after MVC.

3.3 Identifying trajectories of widespread pain development

Fit indices (Table 2) were used to identify the trajectory model for CWP development that best corresponded to the data. Bayesian information criterion values identified a two-group solution as optimal, with the Bayes Factor value (138) for the two-group linear solution indicating very strong model selection [55]. As shown in Table 221.5% of participants were classified as having a high probability of a CWP trajectory (“CWP” group), and 78.5% participants were classified as having a low probability of a CWP trajectory (“non-CWP” group). Demographic characteristics of participants with and without a CWP trajectory are shown in Table 1. Those developing CWP after MVC were more likely to be older, female, to have a higher BMI, and to have lower educational attainment (Table 1).

3.4 CWP development after MVC is most consistent with a non-recovery trajectory

Two-group linear solution plots supported a non-recovery model of CWP development (Figure 1). More than half of individuals (99/192, 52%) in the CWP group met widespread pain criteria in the ED; mean time between MVC and ED presentation among study

participants was 1.2 hours. Little change in the probability of widespread pain was observed across follow-up time points within the CWP group, indicating little aggregate improvement or progression over the course of the year (Fig. 2).

3.5 Relatively few individuals experienced a substantial increase in number of body regions with pain over time

As shown in Figure 3, among individuals with widespread pain one year after MVC, the distribution of change in number of body regions between six weeks and one year is relatively symmetric around zero (47/78 [60%] had experienced an increase in the number of body regions with pain and 31/78 [40%] had experienced no change or a decrease in the number of body regions with pain). As shown in Table 3, individuals with a CWP trajectory already had substantially more regions of pain than those without a CWP trajectory at the time of ED evaluation, and while the number of body regions with pain in the non-CWP group steadily declined over time, the number of body regions with pain in the CWP trajectory group remained relatively constant. Only 23/78 (30%) of participants meeting criteria for CWP at one year had an increase of 4 or more body regions with pain (out of 20 regions assessed) between six weeks and one year (“spread of pain group”, Fig. 3). The pain characteristics in the ED and at week 6 of individuals in the spread of pain group much more closely resembled those who consistently had widespread pain across follow-up time points than those without widespread pain (Table 4).

3.6 Somatic symptoms, depressive symptoms, and posttraumatic stress symptoms among those in the low vs. high probability of widespread pain groups

Somatic symptoms, depressive symptoms, and posttraumatic stress symptoms among those who did and did not develop CWP after MVC are shown in Table 3. Reported depressive symptoms during the month prior to MVC were similar in the two groups, but depressive symptoms in the widespread pain group at follow up time points were markedly higher. Similarly, the number of reported somatic symptoms in the month prior to MVC was similar among those who did and did not develop CWP after MVC, but the number of somatic symptoms increased markedly among those developing CWP at 6 weeks, 6 months and 1 year. Individuals developing CWP had greater peritraumatic distress symptoms in the ED and greater posttraumatic stress disorder (PTSD) symptoms across follow-up timepoints. At six months, 49/170 (29%) of individuals in the CWP group vs. 58/614 (9%) in the non-CWP group had substantial PTSD symptoms ($\chi = 42.42$, $p < 0.001$). At one year, 51/174 (30%) of individuals in the CWP group vs. 55/633 (9%) in the non-CWP group had substantial PTSD symptoms ($\chi = 50.87$, $p < 0.001$).

4. Discussion

The results of this study suggest that the pathogenic trajectory of CWP after MVC is characterized by the immediate development of widespread pain which persists, rather than the gradual progression of pain from regional to widespread. Approximately 1 in 5 individuals presenting to the ED after MVC had a CWP trajectory, and there was little change in the probability of widespread pain across follow-up time points within this group. Similarly, individuals with a CWP trajectory had a high number of body regions with pain in

the ED which subsequently changed little over time. In addition, among individuals with a CWP trajectory the change in number of body regions with pain between six weeks and one year after MVC was relatively evenly distributed around zero. A subset of individuals within the CWP trajectory group experienced a substantial progression in the number of body regions with pain, reporting four or more new body regions with pain at one year compared to the six-week timepoint (47/78, [60%]). This data could seem to support a progressive onset model of CWP development, however such individuals had pain characteristics in the early aftermath of MVC (in the ED in the hours after MVC and at six weeks) that much more closely resembled those with CWP than those without CWP.

To our knowledge, the only other study to examine the natural history of CWP after MVC was conducted by Holm *et al*, who examined widespread pain outcomes over time among 266 individuals who filed an MVC-related insurance claim in the Canadian province of Saskatchewan [18]. The design of this study differed markedly from the present study, making comparison of study findings difficult. Holm *et al* designed their study based on the accumulation model of CWP development[18], and only individuals completing initial insurance claim evaluation with pain limited to a portion of the head, neck, and/or back regions were eligible for study participation. Those with more extensive neck or back pain (> 5 of 11 areas of pain in these regions) or any pain in a location outside these regions were not enrolled[18]. Even among this subset of individuals with limited neck and/or back pain in the early aftermath of injury, the investigators still found that CWP most often developed before the first follow-up assessment [18]. Of note, only 845/7462 (11%) of those who reported locations of body pain on their initial insurance claim had the limited distribution of pain required for potential study eligibility. “The others either had different pain patterns or already had more widespread pain when making their insurance claim.”[18] We believe that these other patterns of pain or more widespread pain among the great majority of individuals experiencing MVC are also consistent with a non-recovery model.

Other studies examining the natural history of pain over time in the general population have found that the extent of pain exhibits natural variation [4; 37]. Thus, an individual who meets criteria for CWP at a follow-up timepoint might not have exceeded the CWP threshold if assessed on a different day. However, results of both previous studies and the present study indicate that this natural variation occurs across a relatively narrow range. For example, in the study of Papgeorgiou et al [37], only 2% of participants without initial pain developed CWP over a seven-year follow-up period, and in the present study within-group mean scores varied over time but remained distinctly different between groups (Table 3).

Findings of the present study supporting a non-recovery model of CWP development are consistent with increasing evidence that pain outcomes after stress exposures such as MVC are mediated by central neurobiological mechanisms [2; 6; 8; 30; 32; 33; 43; 44]. This evidence includes data that pain in the immediate aftermath of other stressful events such as sexual assault is located in many body regions, most of which experienced no tissue trauma, and that the distribution of body regions with pain continues to expand in the initial week after stress exposure[33]. These data are consistent with evidence that stress exposure can result in widespread changes in sensory processing due to mechanisms such as the

sensitization of peripheral afferents by persistently elevated levels of catecholamines and glucocorticoids[24] and by endogenous opioid-induced hyperalgesia[2; 26].

Results of the present study indicates that older age, female sex, increased BMI, and lower educational attainment are associated with vulnerability to CWP are consistent with previous studies [3; 15; 20; 35; 45]. Our finding that crash-related factors such as amount of vehicle damage showed little association with CWP outcomes is also consistent with previous studies[12; 14; 17], as are results of the present study that individuals developing CWP after MVC are much more likely to develop substantial co-morbid somatic symptoms, PTSD symptoms, and depressive symptoms [7; 13; 31].

CWP is a syndrome defined by the extent/distribution rather than severity of pain, and as noted above, has been shown to result in substantial disability and reduced quality of life [11; 27]. Extent and severity of pain, both in the present study and in other studies [42], have been shown to be only moderately correlated (e.g., correlation between overall pain severity and number of body regions with pain in the present study was 0.38 in the ED, 0.53 at week 6, 0.53 at month 6, and 0.52 at year 1). Biopsychosocial factors contributing to extent vs. severity of pain remain poorly understood, and are an important target for future study.

4.1 Limitations

A number of limitations should be considered when interpreting our results. First, our study was limited to individuals who were discharged to home from the ED after evaluation. CWP development may differ substantially in those with severe injuries who are admitted to the hospital. However, because more than 90% of individuals evaluated in the ED after MVC are discharged to home after evaluation[39], and because many individuals who experience an MVC do not present to the ED for care[38], we believe the spectrum of injury examined in the present study accounts for the great majority of individuals developing CWP. Another limitation of the present study is that only European Americans aged 18 to 65 were enrolled. Further studies are needed which evaluate the trajectory of CWP development in other ethnic groups and in older individuals. Finally, self-report information obtained from study participants might have been inaccurate or incomplete. However, baseline data were obtained at the time of initial evaluation, 6 weeks prior to the first assessment of persistent pain outcomes.

4.2 Conclusions

Our study results indicate that a non-recovery model most accurately describes the pathogenesis of CWP after MVC. Individuals developing CWP after MVC generally have an extensive burden of body pain beginning in the immediate aftermath of MVC, which does not remit, rather than experiencing a gradual extension of pain from regional to widespread over time. As Professor George E. P. Box famously said, “Essentially, all models are wrong, but some are useful [9].” We do not believe that a non-recovery model applies to each and every individual who ever has or ever will suffer from CWP after MVC. However, our data suggest that a non-recovery model is an accurate characterization of CWP in most individuals. Future studies evaluating CWP development after stress exposure should begin in the early aftermath of the event, so that both non-recovery and accumulation models of

pathogenesis can be evaluated. In addition, the results of the present study suggest that future preventive intervention studies for this common and morbid outcome might best be initiated in the early aftermath of stress exposure, with the goal helping a greater proportion of individuals with widespread pain or a high burden of overall body pain transition to recovery.

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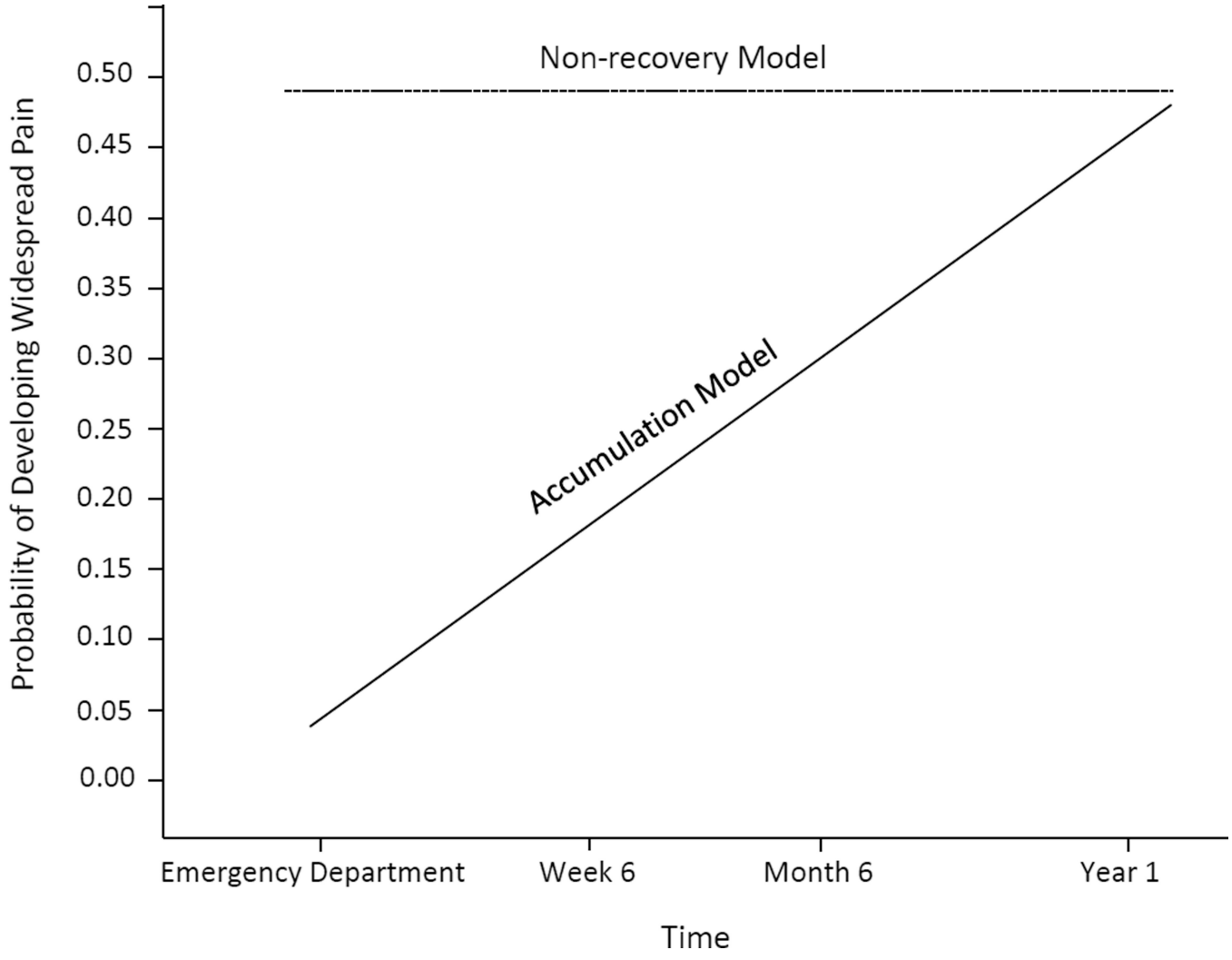

Fig. 1.
Candidate trajectory models of widespread pain development after motor vehicle collision assessed in the study.

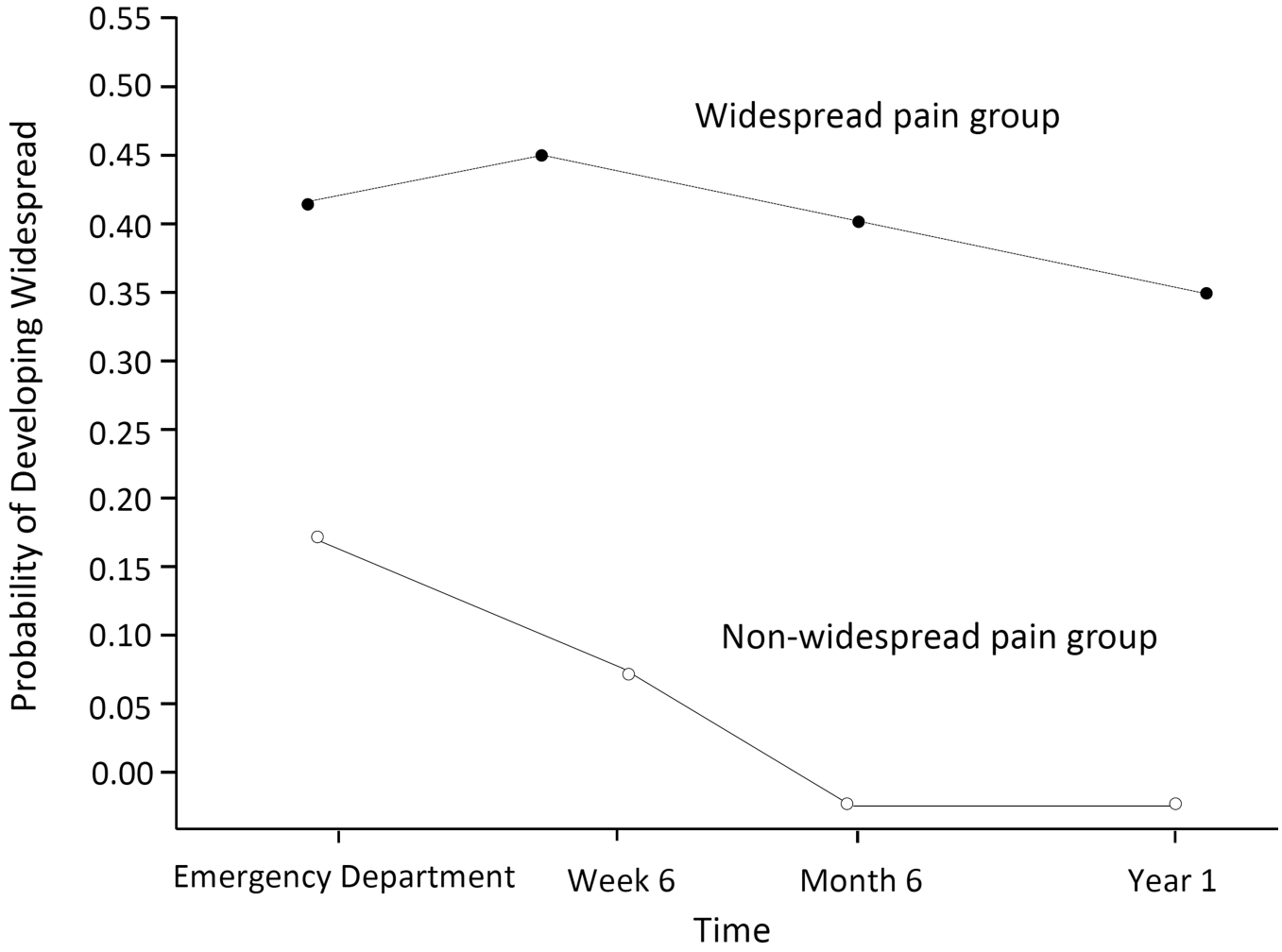

Fig. 2.
Trajectory analysis performed using study data indicated that widespread pain after MVC develops via non-recovery.

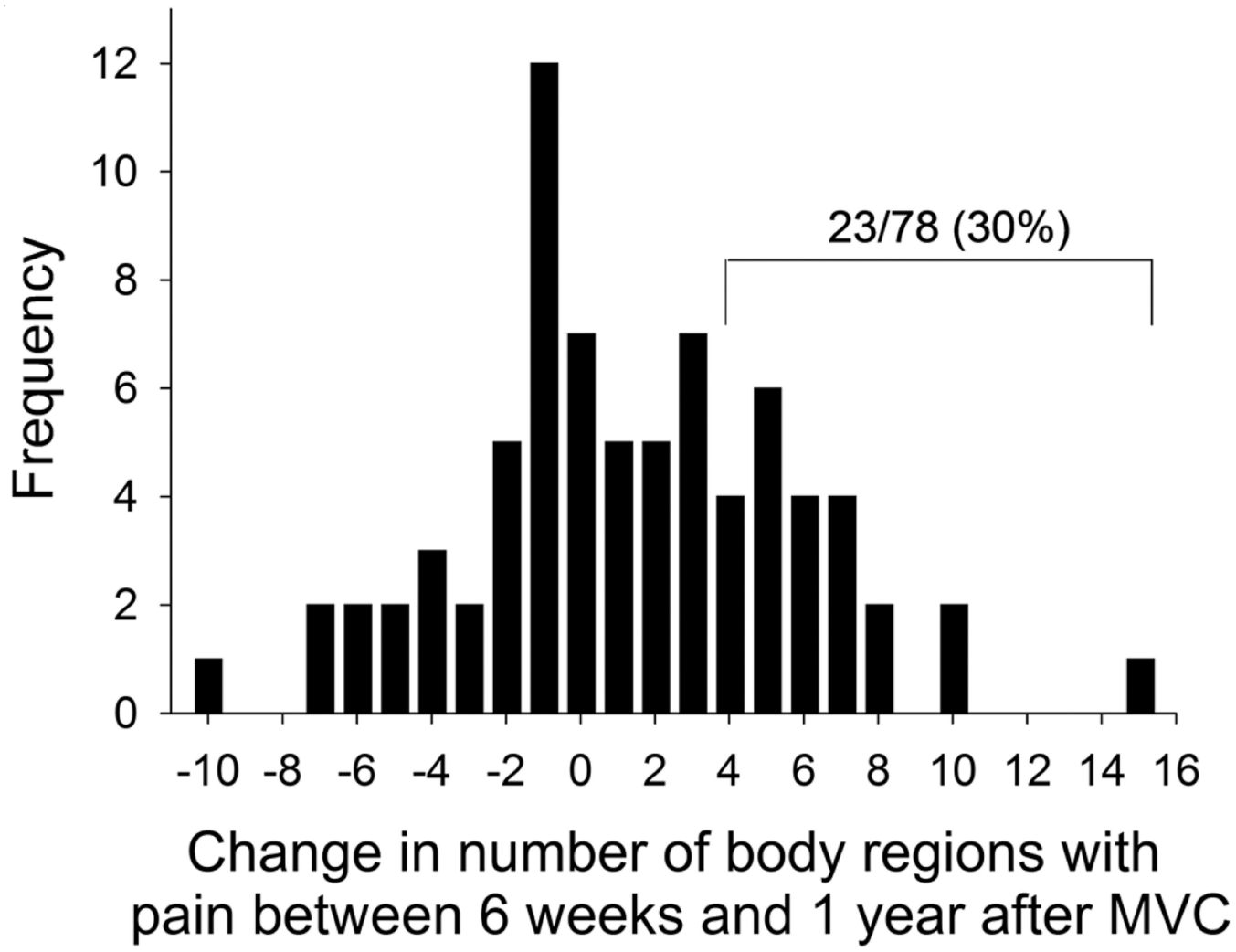

Fig. 3.

Change in number of body regions with pain between six weeks and one year after MVC among individuals with widespread pain at one year follow-up after MVC. Only 30% of individuals experienced an increase of ≥ 4 body regions with pain between these timepoints (“spread of pain” group).

Table 1

Demographic characteristics of all participants, participants with a post-MVC chronic widespread pain trajectory, and participants with a non-chronic widespread pain trajectory.[a]

	All	Non-widespread pain	Widespread pain	p-value
Age, mean (SD)	35.4 (13.0)	34.9 (13.0)	37.4 (13.0)	0.02
Female, n (%)	540 (60.3)	411 (59.0)	129 (67.0)	0.02
BMI, mean (SD)	27.6 (6.0)	27.4 (6.0)	28.5 (7.0)	0.03
Education, n (%)				0.02
High school or less	214 (23.9)	161 (23.0)	53 (28.0)	
Some college or trade school	348 (38.9)	272 (39.0)	76 (39.6)	
College/post-graduate degree	332 (37.1)	269 (38.3)	63 (32.8)	
Annual income, n (%)				0.52
Below $20,000	111 (13.0)	83 (13.3)	28 (16.4)	
$20,000 to $40,000	167 (21.0)	132 (21.1)	35 (20.5)	
$40,000 to $80,000	263 (33.0)	205 (32.7)	58 (33.9)	
>$80,000	256 (32.0)	206 (32.9)	50 (29.2)	
Works full time, n (%)	526 (59.0)	415 (59.0)	111 (57.8)	0.41
Damage to vehicle from MVC, n (%)				
None-minor	115 (13.0)	95 (14.0)	20 (10.9)	0.28
Moderate	269 (31.0)	204 (30.0)	65 (35.5)	
Severe	479 (56.0)	381 (56.0)	98 (53.6)	

MVC, motor vehicle collision; BMI, body mass index; SD, standard deviation

[a]Trajectories assigned using SAS Procedure PROC TRAJ

Table 2

Model fit indices for various group solutions for trajectories of chronic widespread pain development after motor vehicle collision from the emergency department, week 6, month 6 and year 1 (n = 895).

	BIC	ΔBIC	BF	Probability to each Group Assignment				
n trajectories				Group 1	Group 2	Group 3	Group 4	Group 5
1	−1483			100%				
2	−1414	72	138	78.5%	21.5%			
3	−1421	−7	−14	31.8%	45.9%	22.2%		
4	−1430	−9	−18	30.1%	27.2%	39.7%	3.0%	
5	−1443	−13	−26	23.1%	10.6%	30.1%	33.2%	3.0%

BIC, Bayesian Information Criterion; BF, Bayes Factor

Table 3

Number of body regions with pain, number of somatic symptoms, and depressive and posttraumatic stress disorder (PTSD) symptoms in the ED and six weeks, six months, and one year after MVC.

		Non-widespread Pain Trajectory	Widespread Pain Trajectory	
		mean (SD[d])		p-value
Number of body regions with pain				
	ED	3.7 (3.1)	6.2 (4.0)	<0.001
	week 6	2.2 (2.6)	7.4 (4.5)	<0.001
	month 6	1.0 (1.7)	6.2 (4.8)	<0.001
	year 1	0.8 (1.5)	5.2 (4.4)	<0.001
Depressive symptoms[a]	ED	8.6 (0.4)	9.3 (0.7)	0.69
	week 6	9.8 (0.6)	21.5 (0.9)	<0.001
	month 6	9.1 (0.5)	17.8 (0.9)	<0.001
	year 1	8.7 (0.5)	16.4 (1)	<0.001
Number of somatic symptoms[b]				
	ED	2.6 (0.1)	2.9 (0.3)	0.23
	week 6	5.0 (0.2)	9.5 (0.5)	<0.001
	month 6	4.5 (0.2)	8.2 (0.5)	<0.001
	year 1	4.7 (0.2)	8.4 (0.5)	<0.001
PTSD[c] symptoms				
	ED	17.5 (0.5)	22.5 (0.8)	<0.001
	week 6	16.5 (0.9)	32.4 (1.5)	<0.001
	month 6	10.4 (0.8)	25.4 (1.4)	<0.001
	year 1	9.1 (0.8)	24.3 (1.4)	<0.001

[a] Assessed with Center for Epidemiologic Studies Depression Scale (CES-D).

[b] Assessed using a 21 item somatic symptom scale.

[c] Assessed with the Peritraumatic Distress Inventory in the ED, and with Impact of Events Scale Revised (IES-R) at week 6, month 6 and year 1.

[d] SD, standard deviation

Table 4

The pain characteristics in the ED and at week 6 of individuals in the spread of pain group (≥ 4 increase in body regions with pain between six weeks and one year) much more closely resembled those who consistently had widespread pain across follow-up time points than those without widespread pain.

	ED			Week 6		
	No WP	Spread of Pain	WP	No WP	Spread of Pain	WP
Maximum pain severity, mean	6.0	7.7	7.7	4.0	6.1	7.3
Body regions with pain	4.1	4.9	7.0	2.9	3.4	7.3
Pain inference	-	-	-	14.2	34.3	35.3