Eur Spine J (2008) 17 (Suppl 1): S83-S92
DOI 10.1007/s00586-008-0628-7

Course and Prognostic Factors for Neck Pain in Whiplash-Associated Disorders (WAD)

Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders

Linda J. Carroll, PhD,*† Lena W. Holm, DrMedSc,‡ Sheilah Hogg-Johnson, PhD,¶‖ Pierre Côté, DC, PhD,**††‡‡§ J. David Cassidy, DC, PhD, DrMedSc,**††‡‡ Scott Haldeman, DC, MD, PhD,§§¶¶ Margareta Nordin, PT, DrMedSc,‖‖*** Eric L. Hurwitz, DC, PhD,††† Eugene J. Carragee, MD, FACS,‡‡‡§§§ Gabrielle van der Velde, DC,¶†‡‡¶¶¶ Paul M. Peloso, MD, MSc, FRCP(C),‖‖‖ and Jaime Guzman, MD, MSc, FRCP(C)****††††

Study Design. Best evidence synthesis.

Objective. To perform a best evidence synthesis on the course and prognostic factors for neck pain and its associated disorders in Grades I–III whiplash-associated disorders (WAD).

Summary of Background Data. Knowledge of the course of recovery of WAD guides expectations for recovery. Identifying prognostic factors assists in planning management and intervention strategies and effective compensation policies to decrease the burden of WAD.

Methods. The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and its Associated Disorders (Neck Pain Task Force) conducted a critical review of the literature published between 1980 and 2006 to assemble the best evidence on neck pain and its associated disorders. Studies meeting criteria for scientific validity were included in a best evidence synthesis.

Results. We found 226 articles related to course and prognostic factors in neck pain and its associated disorders. After a critical review, 70 (31%) were accepted on scientific merit; 47 of these studies related to course and prognostic factors in WAD. The evidence suggests that approximately 50% of those with WAD will report neck pain symptoms 1 year after their injuries. Greater initial pain, more symptoms, and greater initial disability predicted slower recovery. Few factors related to the collision itself (for example, direction of the collision, headrest type) were prognostic; however, postinjury psychological factors such as passive coping style, depressed mood, and fear of movement were prognostic for slower or less complete recovery. There is also preliminary evidence that the prevailing compensation system is prognostic for recovery in WAD.

Conclusion. The Neck Pain Task Force undertook a best evidence synthesis to establish a baseline of the current best evidence on the course and prognosis for WAD. Recovery of WAD seems to be multifactorial.

Key words: neck pain, systematic review, epidemiology, prognosis, whiplash.

From the *Department of Public Health Sciences, and †the Alberta Centre for Injury Control and Research, School of Public Health, University of Alberta, Canada; ‡Institute of Environmental Medicine, Karolinska Institutet, Sweden; §Institute for Work & Health, Toronto, Canada; ¶Institute for Work and Health, Toronto, Canada; ‖Department of Public Health Sciences, University of Toronto, Canada; **Departments of Public Health Sciences, and Health Policy, Management and Evaluation, University of Toronto, Canada; ††Centre of Research Expertise in Improved Disability Outcomes (CREIDO), University Health Network Rehabilitation Solutions, Toronto Western Hospital, Toronto, Canada; ‡‡Division of Health Care and Outcomes Research, Toronto Western Research Institute, Toronto, Canada; §§Department of Neurology, University of California, Irvine, CA; ¶¶Department of Epidemiology, School of Public Health, University of California, Los Angeles, CA; ‖‖Departments of Orthopaedics and Environmental Medicine and Program of Ergonomics and Biomechanics, School of Medicine and Graduate School of Arts and Science, New York University, NY; ***Occupational and Industrial Orthopaedic Center (OIOC), New York University Medical Center, NY; †††Department of Public Health Sciences, John A. Burns School of Medicine, University of Hawaii at Mānoa, Honolulu, HI; ‡‡‡Department of Orthopaedic Surgery, Stanford University School of Medicine, Standford, CA; §§§Orthopaedic Spine Center and Spinal Surgery Service, Stanford University Hospital and Clinics, Standford, CA; ¶¶¶Department of Health Policy, Management and Evaluation, University of Toronto, Canada; ‖‖‖Endocrinology, Analgesia and Inflammation, Merck & Co. Rahway, NJ; ****Department of Medicine, University of British Columbia, Canada; and ††††Occupational Health and Safety Agency for Healthcare in British Columbia, Canada.

The manuscript submitted does not contain information about medical device(s)/drug(s).

Corporate/Industry, Foundation, and Professional Organizational funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

Address correspondence and reprint requests to Linda J. Carroll, 4075 RTF, University of Alberta, Edmonton, Alberta, Canada T6G 2E1; E-mail: lcarroll@ualberta.ca

Neck pain after traffic collisions is common; the most recent figures suggest that more than 300 persons (per 100,000 in the population) are seen in emergency departments every year.[1] Whiplash is a mechanism of injury, consisting of acceleration-deceleration forces to the neck. In 1995, the Québec Task Force on Whiplash-associated Disorders (WAD) coined the term 'WAD' to describe the symptom sequelae of this injury. This cluster of symptoms includes neck pain, along with other symptoms of the injury such as dizziness and pain in other parts of the body.[2] WAD is thought to result from cervical sprain or strain, probably from soft tissue damage to ligaments and muscles in the neck. Although joints may also be involved, the term WAD generally does not include cervical fractures, and we excluded this degree of injury from our discussions on the course of recovery and prognostic factors for recovery in WAD. Thus our mandate involves Grades I-III WAD (described in more detail later).

The 'course' of recovery from WAD refers to 2 key questions: Are neck pain and associated symptoms likely to resolve, and, if so, within what time frame? These questions are of vital interest to all stakeholders, including individuals with WAD and their families, their health care providers, those who develop and implement policy and regulations, and researchers who study WAD.

Likewise, the determinants of that course of recovery (prognostic factors) are important in planning effective health policies, health care interventions, and lifestyle changes. However, knowledge about the course of recovery in WAD-related neck pain is also informative in determining the effectiveness of interventions [*i.e.*, it may help demonstrate whether a particular intervention improves (or worsens) the usual course of recovery of WAD]. Identifying factors associated with poor prognosis after WAD can also provide a useful target for intervention studies; this may also provide information about those most likely to benefit from such an intervention.

A plethora of widely varying evidence and opinion exists on these issues, resulting in a certain amount of confusion among stakeholders. This makes it imperative to conduct a considered and thorough examination of the scientific evidence and also look at the strength of that evidence.

Research on the course of neck pain in WAD and identification of prognostic factors necessarily involves longitudinal research. This means studying a group of persons with WAD (preferably those with a recent injury), and tracking their recovery over time. Because cross-sectional studies provide a 'snapshot' in time, factors found to be associated with neck pain in such studies could be risk factors, prognostic factors, or consequences of neck pain. Findings from cross-sectional studies on WAD are reported elsewhere.[1]

In examining findings from longitudinal studies, the strength of the evidence produced by these studies should also be considered. One paradigm classifies cohort studies into a 3-level hierarchy of knowledge. This model has been used to interpret evidence obtained in prognostic studies of breast cancer, WAD, and mild traumatic brain injuries.[3–6]

- Phase I studies are descriptive and hypothesis generating, exploring crude associations between potential prognostic factors and health outcomes.
- Phase II studies are also exploratory, but use stratified or multivariable analyses to identify sets of prognostic factors.
- A Phase III study is hypothesis driven and confirmatory. The goal is to confirm or refute the independence of any apparent relationship between a particular prognostic factor and the outcome of interest, after adjusting for confounding.[7]

In the current article, we have used this hierarchy to help us interpret findings from prognostic studies of WAD. The course of neck pain and prognostic factors for recovery in the general population and in workers are presented elsewhere.[7,8] Although there may be many similarities across these populations, we believe this way of organizing our findings will be most useful to audiences. Within the studies on WAD, we further separated the tables reporting the course of recovery (for example, time to recovery) from those reporting prognostic factors for recovery.

Methods

Design and Data Collection

The literature search and critical review strategy are outlined in detail elsewhere.[9] In brief, we systematically searched the electronic library database Medline for literature published from 1980 through 2005 on neck pain and its associated disorders; we also systematically checked the reference lists of relevant articles and updated the search to include key articles for 2006 and early 2007.[9] We screened each citation for relevance to the Neck Pain Task Force mandate, using *a priori* inclusion and exclusion criteria. We made no attempt to assess the scientific quality of each study when establishing its relevance to the Neck Pain Task Force mandate. Studies were considered relevant if:

- they pertained to the assessment, incidence, prevalence, determinants or risk factors, prevention, course, prognosis, treatment and rehabilitation, and/or economic costs of neck pain
- they contained data and findings specific to neck pain and/or disorders associated with neck pain, or described a systematic review of the literature on neck pain
- they included at least 20 persons with neck pain or at risk for neck pain We excluded studies on neck pain which was associated with serious local pathology or systemic disease, such as neck pain from infections; fractures or dislocations (except where such studies informed differential diagnosis of neck pain); myelopathy; rheumatoid arthritis and other inflammatory joint diseases; or tumors.

Quality Assessment

Rotating pairs of Scientific Secretariat members performed independent, in-depth critical reviews of each article, identifying methodologic strengths and weaknesses. Where Scientific Secretariat members were authors or coauthors of an article under consideration, they were excluded from both the discussions and the decisions about scientific merit. The forms used in the methodologic appraisal of the studies can be seen at the following: (address of our web-version: available online). Our methodologic appraisal focused on sources of potential selection bias, information bias, and confounding. We also considered whether or not these biases would likely result in erroneous or misleading conclusions. After discussions of each article, decisions were made about the article's scientific merit. Studies judged to have adequate internal validity and to be methodologically rigorous, such that the results could be accepted with reasonable confidence, were considered to be scientifically admissible and were summarized in evidence tables. These evidence tables were used to formulate the best evidence synthesis.

Analysis

We classified the studies identifying prognostic factors into Phase I, II, or III studies (described earlier). We used this framework in our synthesis of the studies and in our development of summary statements of the evidence. Where the evidence from

different studies varied, more emphasis was given to evidence from well-conducted Phase III studies, and secondarily, to well-conducted Phase II studies. The best evidence synthesis consists of a qualitative integration of the studies judged to be scientifically admissible, and links all summary statements and conclusions to the evidence tables, so that the evidence which formed the basis of any statements is made clear.[10–12]

In accordance with our conceptual framework on the course and care of neck pain,[13] and similar to the organization of risk factors for new onset (incidence) of neck pain,[1,14,15] we further classified prognostic factors into the following categories:

- Demographic and socioeconomic factors: These are usually either nonmodifiable (for example, age and gender) or not easily modifiable (for example, socioeconomic status).
- Prior health or prior pain or comorbidities: These can be classified as 'impairments' according to the WHO's ICF framework.[16]
- Collision factors: These could include information about the collision, such as direction of impact, use of headrests and other safety devices, and speed of impact.
- Initial symptoms: This includes information such as grade of WAD,[2] intensity and distribution of initial postinjury pain, presence and severity of other symptoms.
- Psychological and social factors: These would include depression, anxiety and coping strategies, and interpersonal factors (*e.g.*, relationships with friends). Again, many of these factors are potentially modifiable.
- Societal factors: This would include the prevailing compensation systems and laws. Such factors are potentially modifiable, although not on an individual basis.
- Genetic factors: These are potentially important prognostic factors, although not considered modifiable.
- Health behaviors, initial interventions: This would include health lifestyle factors such as physical exercise, and type and frequency of initial clinical interventions. Such factors are also potentially modifiable.

Results

We found 226 articles in our literature search that pertained to course and prognostic factors for neck pain. After critically reviewing these studies, we judged 70 articles of sufficient scientific merit to be accepted for our best evidence synthesis. Twenty of these studies (19 distinct cohorts) pertained to the course of recovery of WAD; 29 studies pertained to prognostic factors for WAD recovery; 8 studies reported prognostic factors for outcomes other than recovery from WAD, and 3 were systematic reviews. The studies are summarized in Tables 1 to 3 (available online through Article Plus). Some studies related to more than 1 topic, and are therefore reported in more than one section.

Course of Neck Pain in WAD

The preponderance of evidence indicates that, in adults, recovery of WAD is prolonged, with approximately half of those affected reporting neck pain symptoms 1 year after the injury. However, this should be interpreted in light of the background prevalence of neck pain. The best evidence suggests that between 20% and 40% of the general population reports having experienced neck pain during the previous month.[15]

We accepted 20 studies on course of neck pain in WAD (Table 1, available online through Article Plus).[17–36] The sampling frames used in these studies were primarily attendees to emergency departments[18–20,23,26–29,31,37] and insurance claimants.[17,21,22,33,34,36] In addition, one study used police reports,[30] one involved volunteers with WAD,[32] and one studied persons who had been judged to be permanently medically impaired due to WAD.[25]

Most studies examining recovery from WAD in adult populations suggest a prolonged recovery.[18,20–23,28,29,32–34,37] Three studies (2 studying attendees to emergency departments and 1 studying volunteers with WAD) reported that over 60% of those with WAD reported symptoms at 3 months,[28,32,37] with 37% of these symptomatic individuals experiencing moderate to very severe pain at that point in time.[28] Five studies using a longer follow-up also reported symptoms in a high proportion of persons with WAD.[18,21–23,29,34] For example, a Canadian study reported that only 50% of personal injury claimants had closed their claim by 6 months (under the no fault insurance system) and by 1 year (under the tort insurance system).[21] A subsequent study by the same authors in the same province, which used a different cohort of participants, found that 50% of personal injury claimants reported they were either 'all better' or had 'quite a bit or improvement' by approximately 4 months (under the prevailing no-fault system at that time). In that cohort, 46% had recovered (using this self-reported global index of recovery) by 6 weeks; 59% had recovered by 3 months; 66% had recovered by 6 months; and 71% had recovered by 1 year. However, this determination of recovery did not necessarily reflect a complete resolution of symptoms.[22]

Two other studies suggest that the proportion of individuals experiencing symptoms at 1 year after traffic-related WAD was even higher, with estimates varying from 44% (of those attending an emergency department) to almost 66% (of those making a traffic injury claim).[23,34] However, only 12% of subjects reported experiencing daily neck pain[23] and only 9% reported significant health impairment due to the collision.[34] Symptom reporting was greater among those who presented with neck pain accompanied by neurologic signs (equivalent to WAD III): 90% of these patients reported being symptomatic a year after their injury.[29]

Studies with longer term follow-up suggest that WAD symptoms can persist (or recur) over the long-term. One study of WAD patients seen in emergency departments found that 30 months or longer after the collision, 58% of patients had symptoms which they attributed to the injury event.[20] At 7 years postinjury, almost 40% of those making a claim for traffic-related WAD reported often or always having neck pain, compared with less than 15% of a matched cohort who had been in a car crash with no WAD.[17] This latter figure is consistent with the prevalence of 'frequent' neck pain or neck pain which 'interferes with activities' in the general popula-

tion.[15] Persons with a history of WAD were also more likely to have pain in other parts of their body and to report general ill health, sleep disturbance, and fatigue at 7 years postinjury.[38] Furthermore, 30 months after the injury, 7% of WAD patients seen in emergency departments in Umea, Sweden, were still on sick leave[20]; and 5% of WAD patients seen in emergency departments in Norway were on rehabilitation or permanent disability pension.[18] Interestingly, more than 60% of the Swedish persons judged to have a WAD-related medical impairment (10% or more) had returned to full working capacity within approximately 4 years after their injury.[25]

The exceptions (studies indicating more rapid recovery) include 1 study of Lithuanian traffic injuries in which the maximum duration of neck pain was 17 days (median 3 days)[30]; a study of Greek traffic injuries in which almost 90% of subjects had recovered by 1 month and 99% had recovered by 6 months[31]; and a Swedish study reporting that 52% of WAD injuries had resolved by 6 weeks.[26] Similarly, a study of WAD insurance claims in 1987 in the Canadian province of Québec reported that 50% of WAD claims had been closed within 1 month and that 87% had been closed within 6 months.[36] However, the same authors reported much slower claim closure in a subsequent study (also in Québec), which found that 40% to 50% of claims were still open 1 year postinjury.[33] Although these 2 studies did not validate claim closure against health recovery, findings from the latter study are consistent with those from a majority of studies which found longer duration of symptoms.

Little research exists on how children recover from WAD, and we accepted only 1 such study for our best evidence synthesis. This study suggests that recovery in children was relatively rapid, with a mean symptom duration of only 9 days, and maximum duration of symptoms of 2 months.[20]

Prognostic Factors for Recovery from WAD

We accepted 29 studies reporting factors associated with prognosis in WAD (Table 2, available online through Article Plus). These studies examined a large number of potential prognostic factors and a variety of different outcomes. For purposes of this report, we have categorized potential prognostic factors into demographic and socioeconomic factors; prior health, prior pain, comorbidities; initial pain and symptom severity; collision factors; psychological and social factors; compensation and legal factors; genetic factors; health behaviors and interventions; and cultural factors.

Demographic and Socioeconomic Factors. The evidence varies on the roles of both age and gender as a prognostic factor for recovery in WAD.

Gender. Of the *11* distinct cohorts which looked at gender as a potential prognostic factor, *7* Phase I and II cohorts (in both clinical and insurance claim samples) found longer sick leave or slower or less complete recovery in women compared to men.[18,20,21,34,36,39–41] Most of these associations were modest, reflecting at most a twofold increase in likelihood of poorer outcome. However, *1* of these studies reported that women with WAD *were* more than twice as likely as men to take sick leave, and almost *3* times more likely to take long sick leave.[20]

None of the remaining 4 studies (all Phase II, and all in clinical samples) found an association between gender and the following indexes of recovery: duration of symptoms, presence or severity of neck pain at 6 months or 1 year, or work capacity.[42–45] Three of the 4 studies which found no association between gender and recovery also adjusted for psychological factors in their statistical models[43–45]; this raises the possibility that observed gender effects in other studies may be partly explained by differences in psychological functioning. Among Swedish subjects judged to have a WAD-related, permanent medical impairment (10% or more), female gender was not a prognostic factor for partial or full work disability at follow-up.[25] However, no information was given about the possibility that gender might have played some role in judging whether subjects had a medical impairment related to WAD.

Age. Eight distinct cohorts (all Phase II studies) examined age as a prognostic factor for recovery from WAD. Five studies reported no association between age and outcome *after* WAD. Outcomes examined in these studies were: neck pain or limitations in daily activities at 2 years[39]; work capacity or time to return to work *or* activities[43]; neck pain at 1 year[44]; length of sick leave and health impairment at 1 year[34]; and interference with work or leisure.[41] In contrast, one Phase II study reported that older persons (age 50 or more) were more likely than younger persons to report symptoms at 6 months after the injury [*odds ratio* (*OR*) = 3.1].[42] Two other Phase II studies reported that older age predicted progressively slower recovery *or* slower insurance claim closure, although the effect sizes in these latter *2* studies were more modest (HRR = 0.58 and 0.86, respectively).[21,40]

Education. The evidence varied in the *3* studies examining education as a prognostic factor for recovery. One large Phase II cohort study reported that those with the lowest educational level recovered more quickly.[21] However, another Phase II study found that those with lower education reported greater pain intensity at *2*-years postinjury (compared to those with higher education),[39] and a Phase I study found greater self-reported decreases in health (which the respondent attributed to the collision) in those with lower education at *1*-year postinjury.[34] It is possible that occupation mediates the relationship between educational level and recovery of WAD. There were no scientifically admissible studies on the role of occupation type on WAD recovery; however, there is some evidence that occupation has an influence on prognosis in non-WAD neck pain.[8]

Prior Health or Prior Pain or Comorbidities. The evidence varies regarding the prognostic role of preinjury pain and preinjury health in recovery from WAD. According to *2* Phase II studies, prior neck pain was a strong predictor of neck pain at 1 year postcollision (adjusted OR = 4.5)[44] and of interference with work or leisure at 16 months postcollision (OR = 3.2).[41] In addition, prior sick leave for neck pain predicted longer sick leave after a traffic-related WAD (Phase I study, estimate of effect not given).[18] Another study (Phase I) reported that people with prior headaches and neck pain were almost *3* times more likely to experience cervicogenic headaches 1 year after a whiplash injury versus those without prior symptoms.[23] However, *2* other Phase II studies showed that prior health and prior pain did not predict poorer outcome.[21,42] Differences in findings did not *seem* to be related to the source of the study participants.

We found no scientifically admissible study or studies which addressed the role of disc degeneration on recovery in WAD.

Collision Factors. Most of the evidence from scientifically admissible studies indicates that collision-related factors (outlined below) are not prognostic of recovery in WAD. Evidence for this statement is derived from *11* Phase I and Phase II studies examining whether characteristics of the collision itself or vehicle characteristics were prognostic for recovery from WAD. Such factors included: the person*'s* position in the vehicle; his *or* her awareness of the impending collision; use *or* type of headrest; use *or* type of seat belt; direction of the collision; whether the person's head was turned or facing forward; and self-reported speed differential between vehicle or vehicles involved in the collision.

Seven Phase II studies found no association between any of these factors (as assessed by self-report), and recovery.[21,34,39,41–43,45] One Phase II study reported a better prognosis (as determined by faster claim closure) for persons injured in rear-end collision injuries, and also for those whose injuries were sustained in vehicles other than trucks or buses.[36] However, the latter study used an administrative database; thus, no data were available describing patients' initial pain and symptom severity, nor was it possible to directly assess patients' recovery. Conversely, another Phase I study (of attendees to an emergency department) studying direction of the collision (*i.e.*, rear-end *vs.* another direction) reported the opposite outcome: those injured in rear-end collisions had a worse prognosis, as determined by longer sick leave.[20]

Two Swedish studies directly assessed vehicle characteristics instead of relying on self-report. One study (Phase I) concluded that those who were injured while driving a vehicle with a tow bar (an apparatus which equips vehicles for towing trailers or other objects) had a poorer prognosis over the long-term, although the effect size was quite modest (RR for tow bars was 1.2).[46] The other study (a small Phase I study) reported that sustaining an injury during a crash involving greater mean acceleration (assessed using a crash recorder, effect size not reported) was prognostic of longer symptom duration.[46,47] However, these were both Phase I studies and the observed associations may have been confounded by other prognostic factors.

Initial Symptoms

WAD Grade. In the few studies examining this issue, there was consistent evidence that those with Grade III WAD recovered more slowly than those with Grade I WAD. WAD grading was proposed by Spitzer et al in the 1995 Québec Task Force on WAD,[2] partly as a result of earlier research by Norris and Watt[29] The Québec Classification of WAD is a clinical classification system which grades symptoms as follows:

- Grade 0 WAD refers to no neck complaints and no physical signs (that is, no WAD injury, and thus outside the mandate of the Neck Pain Task Force).
- Grade I WAD refers to injuries involving complaints of neck pain, stiffness or tenderness, but no physical signs.
- Grade II WAD refers to neck complaints accompanied by decreased range of motion and point tenderness (musculoskeletal signs).
- Grade III WAD refers to neck complaints accompanied by neurologic signs such as decreased or absent deep tendon reflexes, weakness and/or sensory deficits.
- Grade IV WAD refers to injuries in which neck complaints are accompanied by fracture or dislocation (and thus outside the mandate of the Neck Pain Task Force)
- Other symptoms such as deafness, dizziness, tinnitus, headache, memory loss, dysphagia, and temporomandibular joint pain can be present in all grades.

Four studies performed subsequent to the publication of the Québec Task Force Report on WAD specifically examined the association between WAD grade and recovery. Other studies in which the primary goal was to assess the adequacy of the classification system are included elsewhere (in the best evidence synthesis of assessment or classification of neck pain).[48]

One Phase I study of children with WAD reported that those with Grade II WAD had slightly longer symptom duration than those with Grade I WAD (19.7 *vs.* 6.4 days).[19] Another Phase I study, this time in adults, reported that 3 years after an injury, WAD III patients were more likely than WAD I patients to report significant changes in health (OR = 3.3), which the patients attributed to the injury. Differences between WAD II and WAD I patients were less marked in that study.[34] (This study did not attempt to validate patients' reports of prior health status or whether symptoms at follow-up could actually be attributed to the injury.) A Phase II cohort study reported that adults with Grades II or III WAD had more work or leisure limitations than the

comparison group at 16 months, although it should be noted that the comparison group included those with no neck complaints after the injury (Grade 0 WAD).[41] That study did not provide a comparison of Grades II and III WAD patients. However, a Phase II study using WAD grading based on self-reported signs and symptoms (rather than clinical assessment) reported that increasing grades of WAD predicted increasingly higher pain intensity and disability (higher scores on the Disability Rating Index) 2 years later (OR for Grade II = 1.5, and for Grade III = 2.4).[39]

Other Indices of Symptom Severity. There is consistent evidence that persons who report more frequent or more severe postinjury symptoms and greater pain intensity had a poorer prognosis for recovery of WAD. Seven distinct cohorts of persons with WAD assessed these indexes of injury severity which included: initial neck pain and stiffness; headache intensity; type, frequency and intensity of other postinjury symptoms; and early limitations in *activities* of daily living.

All studies assessing these indexes consistently showed that greater initial self-reported symptom severity was associated with slower or less complete recovery.[21,23,39,42,43,49–52] However, 1 small study found that radiologic and neurologic findings in patients presenting at a hospital emergency department did not predict severity and duration of symptoms when initial psychological factors were considered.[45]

Psychological and Social Factors. The evidence suggests that psychological factors (listed below) are prognostic of recovery in WAD. This evidence comes from ***6*** studies (***5*** Phase II studies and ***1*** Phase III study) which examined the role of various early, postinjury psychological factors in the patient's subsequent recovery. However, among these studies, there was little uniformity in the psychological construct being examined. One Phase II study reported that very early coping behavior (assessed within days of the injury) did not predict presence of neck pain 1 year after the injury.[44] However, this is not necessarily inconsistent with findings from a Phase III study of insurance claimants, which indicated that the use of passive coping later in the recovery period (coping measured at 6 weeks postinjury) predicted 55% slower recovery. The latter study also reported that, in the presence of depressive symptomatology, subjects who used passive coping recovered 75% slower than those who employed nonpassive coping strategies.[53] The same study found that depressed mood itself predicted 32% slower recovery from WAD.[53]

Several other prognostic psychological factors were identified in Phase II studies. They included feelings of helplessness in controlling the consequences of pain, which made it 2.5 times more likely that the person would report high pain levels 2 years after injury. Having such feelings also doubled the odds that the person would report high levels of disability (measured by the Disability Rating Index).[39] Other prognostic psychological factors identified in the Phase II studies (of insurance claimants and those seeking health care) were: fear of movement,[50] catastrophizing,[50] and initial postinjury anxiety (no effect sizes provided).[45]

One Phase II study did not find associations between health-related psychological functioning, as measured by the Millon Behavioral Health Inventory, and work capacity after traffic-related neck injuries.[43]

Societal Factors. There was evidence from **2** longitudinal studies that prevailing compensation and legal factors were prognostic factors in recovery from WAD. In **1** Phase II study, claim closure took twice as long when insurance claims were made under a tort insurance system versus under a no-fault system.[21] In a tort insurance system, people who are injured in a traffic collision are entitled to limited benefits (such as health care and income replacement); they may also sue the driver at fault for the collision for additional expenses and for pain and suffering. In a no-fault insurance system, claimants or entitled to benefits, regardless of fault, but they are not allowed to sue for pain and suffering.

Although claim closure should not necessarily be equated with health recovery, in the Phase II study mentioned above, the authors found that improvements in self-reported physical functioning, pain intensity, and mood were each independent predictors of claim closure.[21,54] (This does not imply that, for any particular individual, the *point* of claim closure reflects the *point* at which WAD symptoms have resolved.) The same study found that seeking legal advice early in the recovery process was associated with delayed claim closure (HRR = 0.6 in both systems; in other words, claims closed 67% more quickly with no lawyer involvement).[21] These findings are partially supported by the results of the second Phase II study. Its findings suggest that subjects who initiated lawsuits within the first month of their injury were more likely to have reduced work capacity at follow-up (however, this finding did not achieve statistical significance). While the OR for this factor was 1.5, the number of persons initiating lawsuits was small, and precision around these estimates is low and include unity, suggesting caution is needed before making firm conclusions.[43]

Genetic Factors. We found no scientifically admissible study or studies that examined genetic factors in prognosis for recovery from neck pain related to WAD.

Health Behaviors, Interventions

We found no scientifically admissible studies examining the effect of preinjury physical fitness or exercise on recovery of WAD.

The outcome of interventions for WAD is reported in more detail elsewhere[55]; however, evidence suggests that frequent early health care use is prognostic of poorer outcome.

Three studies report information on the prognostic role of interventions. In a study of traffic-related WAD

claimants (using Phase II and Phase III analyses), type and intensity of initial postinjury health care were associated with speed of recovery. Those with more frequent health care visits, those seeing chiropractors, and those who consulted specialists or chiropractors and general practitioners closed their insurance claims up to 40% more slowly (as above, claim closure was found to be predicted by improvements in self-reported physical functioning, pain intensity, and mood).[21,56,57] These findings were consistent with the results of another Phase III cohort study of insurance claimants, in which attendance at a province-wide system of community-based rehabilitation program did not enhance patients' recovery. In fact, those who attended these programs recovered 30% to 50% more slowly than those who did not attend (this study used a self-reported global measure of recovery and controlled for the effect of demographic, socioeconomic and collision-related factors, prior health and initial pain, and symptom severity).[22] However, patient choice of several therapy methods (soft collar, active physical therapy, passive physical therapy, manipulation, or weak analgesics) at the first postinjury health care visit was not associated with recovery.[43]

We found no scientifically admissible study or studies that examined the effect of initial physical fitness on recovery of WAD.

Cultural Factors. We found no scientifically admissible study or studies which directly assessed the impact of cultural factors on recovery of WAD.

Prognostic Factors for Other Outcomes of WAD

We accepted 9 studies examining prognostic factors for outcomes other than recovery and pain reduction after WAD (Table 3, available online through Article Plus).

Tender Points, Pain Threshold, Sensitization, Muscle Reactivity. One follow-up study found that WAD patients had lower pressure pain thresholds and greater tenderness in the trapezius area than a comparison group with ankle injuries at 1 week and 3 months postinjury; however, by 6 months, there was no difference between these groups.[58] When WAD patients were retrospectively divided into *2* groups – those who recovered within a *1*-year period and those who did not – both the recovered WAD patients and the ankle-injured patients had similar threshold to time to peak pain throughout the *1*-year follow-up period. However, WAD patients who failed to recover within 1 year had reduced threshold to time to peak pain during the first 6 months postinjury (reaching peak pain at approximately double the speed compared *to* other subject groups). At 12 months, all *3* groups had similar threshold time to peak pain.[59] In another study, no elevated muscle reactivity was observed in WAD patients at any time during a *3*-month follow-up, although baseline disability (measured using the Neck Pain Disability Index) was associated with surface electromyography (EMG) findings of the upper trapezius muscle at 3 months (i.e., those with high pain disability showed the greatest reduction in recruitment in these muscles during isometric exercise).[49]

Widespread Body Pain, Fibromyalgia. Three studies provide evidence about the onset of widespread body pain or fibromyalgia in persons with WAD.[60–62] One Phase II study examined risk factors for onset of widespread body pain (defined as pain both above and below the waist, in both sides of the body and in the axial skeleton) 1 year after a traffic injury (27% of these injuries had resulted in neck pain).[62] Having post-injury neck pain increased this risk threefold, after adjusting for age and gender. However, experiencing neck pain after the collision did not increase the risk of new onset widespread body pain after adjusting for collision-specific factors, work characteristics, pre- and postinjury health, and psychosocial factors.

Another cohort (Phase I study) of personal injury claimants – this one including only subjects with postcollision localized neck pain (with or without head pain or back pain) – found that 21% developed more extensive body pain (that is, pain in more areas of their body) at some point during the 1-year follow-up. This occurred more frequently in women, and also in subjects with poor prior health, greater initial symptomatology (including pain intensity), and more depressive symptoms. However, most of these subsequently improved (64%), and most sustained this improvement throughout the course of the study.[61]

A Phase I cohort study examining the role of 'soft tissue' neck injuries in onset of Fibromyalgia Syndrome (FMS) found that 21.6% of adults reporting occupational 'soft tissue' injuries to the neck (three-quarters of whom had sustained a traffic-related WAD) were diagnosed with FMS 6 to18 months after the injury. In contrast, only 1.7% of a comparison group with lower extremity fractures developed FMS by the same criteria.[60] Neck injury patients who developed FMS were more likely to be female and, at follow-up, to characterize the impact of the trauma more negatively than patients who did not go on to develop FMS.

Psychological Outcomes. Three studies examined psychological outcomes in WAD. One study reported that 42% of people with traffic-related WAD developed depressive symptomatology within 6 weeks. This symptomatology was persistent throughout the **1**-year follow-up in 18% of subjects; it followed a recurrent course in another 18%. Those with self-reported prior mental health problems (i.e., in the 6 months before injury) were more likely to experience postinjury depressive symptomatology, and to have a persistent or recurrent course.[63] Similarly, patients reporting to an occupational clinic with 'soft tissue' neck injuries were reported to experience more subsequent depression and poorer quality of life than a comparison group of patients with lower extremity fractures.[60] In addition, high initial pain intensity and longer duration of WAD symptoms predicted decreased self-perceived health status, lowered quality of

life, greater depression, and higher psychological impact of event scores at 6 months (effect sizes not provided).[45]

■ Discussion

We critically reviewed 226 studies on the course and prognostic factors for neck pain. Of these, 70 studies were judged to be scientifically admissible, and 44 studies (plus 3 systematic reviews) were specifically related to recovery in WAD. Of the 28 studies reporting prognostic factors, 11 were Phase I studies; 13 were Phase II; and 4 were Phase III studies. As stated earlier, the most compelling evidence arises from Phase III studies which can determine the independent strength of any relationship between a potential prognostic factor and the outcome. Evidence from Phase II studies is less compelling, because the intent of these studies is to explore the role of a set of predictors, rather than to confirm the independence of these predictors. Phase I studies do not consider confounding and thus can only suggest relationships which might be confirmed in other studies.

Most scientifically admissible studies suggest that half of those with WAD report neck symptoms 1 year after their injury. These findings should be considered in the context of the background prevalence of neck pain in the population. The best evidence suggests that 20% and 40% of the general population has experienced neck pain during the previous month, and it is possible that some of the symptoms attributed to the whiplash injury simply reflect the background prevalence of neck pain one would expect in the local general population.[15]

The course of recovery in WAD is also similar to findings about the course of neck pain in the general population and in workers.[7,8] However, it has also been demonstrated that a history of WAD symptoms can be a risk factor for neck pain up to 7 years after the injury.[38]

Two of the studies with discrepant findings (rapid recovery) were performed in Lithuania and Greece,[30,31] and it has been hypothesized that cultural differences (*e.g.*, culturally-based differences in expectations for symptoms and recovery)[64,65] may explain longer recovery times noted in other jurisdictions. This is quite plausible, however, neither the rapid recovery reported in some studies, nor the prolonged recovery reported in other studies is well understood. Differences in culture, beliefs and attitudes are an interesting potential explanation. However, the differences in reported recovery rates may be due to other factors. Examples include methodologic issues such as differences in sampling frames (or differences in the accuracy and comprehensiveness of the sampling frames) and sampling procedures, and differences in case definitions or measurement procedures; differences in the context in which an injury occurs (for example, differences in access to health care or type of health care provided); or differences in policies relevant to compensation for traffic collisions, *etc.* We accepted (as scientifically admissible) 1 study on WAD in a pediatric group which found rapid recovery from this injury in children.[19] However, these findings need to be reproduced in other cohorts before making any firm conclusions.

Findings varied regarding the effect of age and gender on recovery in WAD. However, even in those studies which identified age and gender as prognostic, the effects were modest, suggesting that neither age nor gender plays a major role in outcome. Findings also varied about the prognostic role of preinjury neck pain. The inconsistency may be due to the fact that preinjury neck pain was only assessed retrospectively, after the injury. This increases the risk for misclassification, either through failures of recall or through reporting bias, and suggests that the role of preinjury neck pain should be properly assessed in studies with good quality measurement of prior neck pain.

There is consistent evidence that increased initial symptom severity (such as greater initial pain, greater number of symptoms, more parts of the body in pain, pain-related limitations) is prognostic of poorer outcome. WAD Grades I and II show some modest predictive validity, and WAD Grade III showed stronger predictive validity, although few studies specifically examined this as a prognostic factor.

In the studies which adjusted for initial pain and symptom severity, collision factors did not seem to be associated with recovery after WAD. However, most studies relied on self-reports of collision and vehicle factors, and there may be misclassification due to inaccurate recall. These inaccuracies in recall are likely to be non-differential, which would bias the findings toward the null. The 2 studies which did not depend on participants' recall or judgment about collision-specific factors demonstrated a small, negative long-term (but not short-term) effect related to the presence of a tow-bar on the vehicle which was struck during the collision.[46] There was a similar finding among subjects involved in crashes with higher levels of mean acceleration.[47] However, both were Phase I studies, with no adjustments made for potentially important confounders, so these results should be considered preliminary at this point.

Coping behaviors used by subjects within the first few days of injury did not seem to impact on their recovery; however, 1 Phase III study showed that later passive coping (assessed at 6 weeks) was a strong and independent predictor of slowed recovery, especially in the presence of depression.[53] Other psychological prognostic factors included helplessness, depression, fear of movement, catastrophizing, and anxiety, which also predicted slower recovery. None of the above studies examined the role of injury severity (*e.g.*, clinical pathology) on the associations between the psychological factors and recovery, and these findings may not generalize to those with more severe injuries.

The prognostic role of compensation and litigation was assessed in only 2 studies, and therefore should be verified in other jurisdictions; however, preliminary evidence suggests that these seemed to be prognostic.[21,43] There is also some evidence that greater health care uti-

lization in the first month after a whiplash injury was associated with slower recovery.[56,57] Because this was a large, population-based cohort study of traffic injury claimants, it should be noted that these findings may not apply to individual cases; and it is likely that the optimal type and frequency of acute WAD health care varies by injury severity (*e.g.*, WAD Grade) and patient characteristics (*e.g.*, age, gender, health).

Other substantial predictors of recovery were: initial postinjury pain intensity; number and severity of injury-related symptoms; and WAD Grade III (*vs.* WAD Grade I).

In conclusion, WAD is an important issue: it is both a frequent traffic injury, and can have a prolonged recovery. The course of WAD shows remarkable similarity to the course of neck pain in the general population and in workers, both of which show a persistent or recurrent course.[7,8] Like recovery of neck pain in the general population and in workers, recovery in WAD is multifactorial.

■ Key Points

- Recovery of traffic-related WAD (Grades I-III) is prolonged with only half of those affected reporting no neck pain symptoms 1 year later. Children may recover more quickly.
- Most collision-specific factors (including position in the vehicle, whether the head was turned or straight ahead, awareness of the impending collision, use and type of headrest, direction of the collision) were not prognostic for recovery in WAD.
- Recovery is slower in those with greater initial symptom severity.
- Psychological factors such as postinjury psychological distress and passive types of coping were prognostic of poorer recovery. There was also preliminary evidence that compensation or legal factors are associated with recovery.
- There is consistent evidence that, on average, frequent, early health care use was associated with poorer recovery. However, the optimal type and frequency of acute WAD health care likely varies by injury severity and patient characteristics.

tables

Tables available online through Article Plus.

Acknowledgments

We are indebted to Ms. Oksana Colson and Ms. Leah Phillips (MA) for their administrative assistance and to Mr. Stephen Greenhalgh (MA, MLIS), Ms. C. Sam Cheng (MLIS) and Ms. Lori Giles-Smith (MLIS), research librarians, for their assistance in the work of the Neck Pain Task Force. The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and its Associated Disorders is supported by a grant to the University of Alberta from: the National Chiropractic Mutual Insurance Company and the Canadian Chiropractic Protective Association; Jalan Pacific Inc.; Länsförsäkringar; and the Insurance Bureau of Canada; and by a grant to Karolinska Institute in Stockholm, Sweden, from the Swedish Whiplash Commission. Dr. Côté is supported by the Canadian Institutes of Health Research through a New Investigator Award and by the Institute for Work and Health through the Workplace Safety and Insurance Board of Ontario. Dr. van der Velde is supported by the Canadian Institutes of Health Research through a Fellowship Award. Dr. Carroll is supported by a Health Scholar Award from the Alberta Heritage Foundation for Medical Research. Dr. Cassidy is supported by an endowed research chair from the University Health Network.

References

1. Holm LW, Carroll LJ, Cassidy JD, et al. The burden and determinants of neck pain in whiplash-associated disorders after traffic collisions. Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S52–S59.
2. Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its management. *Spine* 1995;20:1S–73S.
3. Altman DG, Lymann GH. Methodological challenges in the evaluation of prognostic factors in breast cancer. *Breast Cancer Res Treat* 1998;52:289–303.
4. Carroll LJ, Cassidy JD, Côté P. Depression as a risk factor for onset of an episode of troublesome neck and low back pain. *Pain* 2004;107:134–9.
5. Carroll LJ, Cassidy JD, Peloso PM, et al. Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *J Rehabil Med* 2004;43:84–105.
6. Côté P, Cassidy JD, Carroll L, et al. A systematic review of the prognosis of acute whiplash and a new conceptual framework to synthesize the literature. *Spine* 2001;26:E445–E458.
7. Carroll LJ, Hogg-Johnson S, van der Velde G, et al. Course and prognostic factors for neck pain in the general population. Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S75–S82.
8. Carroll LJ, Hogg-Johnson S, Côté P, et al. Course and prognostic factors for neck pain in workers. Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S93–S100.
9. Carroll LJ, Cassidy JD, Peloso PM, et al. Methods for the best evidence synthesis on neck pain and its associated disorders. The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S33–S38.
10. Slavin RE. Best evidence synthesis: an alternative to meta-analytic and traditional reviews. *Educ Res* 1986;15:5–11.
11. Slavin RE. Best evidence synthesis: an intelligent alternative to meta-analysis. *J Clin Epidemiol* 1995;48:9–18.
12. van der Velde G, van Tulder M, Côté P, et al. The sensitivity of review results to methods used to appraise and incorporate trial quality into data synthesis. *Spine* 2007;32:796–806.
13. Guzman J, Hurwitz EL, Carroll LJ, et al. A conceptual model for the course and care of neck pain. Results of The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S14–S23.
14. Côté P, van der Velde G, Cassidy JD, et al. The burden and determinants of neck pain in workers. Results of the Bone and Joint 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S60–S74.
15. Hogg-Johnson S, van der Velde G, Carroll LJ, et al. The burden and determinants of neck pain in the general population. Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S39–S51.
16. World Health Organization. International Classification of Function, Disability and Health: ICF. Geneva: WHO; 2001.
17. Berglund A, Alfredsson L, Cassidy JD, et al. The association between expo-

sure to a rear-end collision and future neck or shoulder pain: a cohort study. *J Clin Epidemiol* 2000;53:1089–94.
18. Borchgrevink GE, Lereim I, Royneland L, et al. National health insurance consumption and chronic symptoms following mild neck sprain injuries in car collisions. *Scand J Soc Med* 1996;24:264–71.
19. Boyd R, Massey R, Duane L, et al. Whiplash associated disorder in children attending the emergency department. *Emerg Med J* 2002;19:311–4.
20. Bylund PO, Bjornstig U. Sick leave and disability pension among passenger car occupants injured in urban traffic. *Spine* 1998;23:1023–8.
21. Cassidy JD, Carroll LJ, Côté P, et al. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *N Engl J Med* 2000;342:1179–86.
22. Cassidy JD, Carroll LJ, Côté P, et al. Does multidisciplinary rehabilitation benefit whiplash recovery? Results of a population-based incidence cohort study. *Spine* 2007;32:126–31.
23. Drottning M, Staff PH, Sjaastad O. Cervicogenic headache (CEH) after whiplash injury. *Cephalalgia* 2002;22:165–71.
24. Gargan M, Bannister G, Main C, et al. The behavioural response to whiplash injury. *J Bone Joint Surg Br* 1997;79:523–6.
25. Holm L, Cassidy JD, Sjogren Y, et al. Impairment and work disability due to whiplash injury following traffic collisions. An analysis of insurance material from the Swedish Road Traffic Injury Commission. *Scand J Public Health* 1999;27:116–23.
26. Jonsson H, Cesarini K, Sahlstedt B, et al. Findings and outcome in whiplash-type neck distortions. *Spine* 1994;19:2733–43.
27. Kasch H, Bach FW, Stengaard-Pedersen K, et al. Development in pain and neurologic complaints after whiplash: a 1-year prospective study. *Neurology* 2003;60:743–9.
28. Mayou R, Bryant B. Psychiatry of whiplash neck injury. *Br J Psychiatry* 2002;180:441–8.
29. Norris SH, Watt I. The prognosis of neck injuries resulting from rear-end vehicle collisions. *J Bone Joint Surg Br* 1983;65:608–11.
30. Obelieniene D, Schrader H, Bovim G, et al. Pain after whiplash: a prospective controlled inception cohort study. *J Neurol Neurosurg Psychiatry* 1999;66:279–83.
31. Partheni M, Constantoyannis C, Ferrari R, et al. A prospective cohort study of the outcome of acute whiplash injury in Greece. *Clin Exp Rheumatol* 2000;18:67–70.
32. Sterling M, Jull G, Vicenzino B, et al. Development of motor system dysfunction following whiplash injury. *Pain* 2003;103:65–73.
33. Suissa S, Giroux M, Gervais M, et al. Assessing a whiplash management model: a population-based non-randomized *intervention study. J Rheumatol* 2006;33:581–7.
34. Miettinen T, Airaksinen O, Lindgren KA, et al. Whiplash injuries in Finland–the possibility of some sociodemographic and psychosocial factors to predict the outcome after one year. *Disabil Rehabil* 2004;26:1367–72.
35. Miettinen T, Leino E, Airaksinen O, et al. Whiplash injuries in Finland: the situation 3 years later. *Eur Spine J* 2004;13:415–8.
36. Suissa S, Harder S, Veilleux M. The Quebec whiplash-associated disorders cohort study. *Spine* 1995;20:12S–20S.
37. Gargan MF, Bannister GC. The rate of recovery following whiplash injury. *Eur Spine J* 1994;3:162–4.
38. Berglund A, Alfredsson L, Jensen I, et al. The association between exposure to a rear-end collision and future health complaints. *J Clin Epidemiol* 2001;54:851–6.
39. Berglund A, Bodin L, Jensen I, et al. The influence of prognostic factors on neck pain intensity, disability, anxiety and depression over a 2-year period in subjects with acute whiplash injury. *Pain* 2006;125:244–56.
40. Harder S, Veilleux M, Suissa S. The effect of socio-demographic and crash-related factors on the prognosis of whiplash. *J Clin Epidemiol* 1998;51:377–84.
41. Sterner Y, Toolanen G, Gerdle B, et al. The incidence of whiplash trauma and the effects of different factors on recovery. *J Spinal Disord Tech* 2003;16:195–9.
42. Hartling L, Pickett W, Brison RJ. Derivation of a clinical decision rule for whiplash associated disorders among individuals involved in rear-end collisions. *Accid Anal Prev* 2002;34:531–9.
43. Kasch H, Bach FW, Jensen TS. Handicap after acute whiplash injury: a 1-year prospective study of risk factors. *Neurology* 2001;56:1637–43.
44. Kivioja J, Jensen I, Lindgren U. Early coping strategies do not influence the prognosis after whiplash injuries. *Injury* 2005;36:935–40.
45. Richter M, Ferrari R, Otte D, et al. Correlation of clinical findings, collision parameters, and psychological factors in the outcome of whiplash associated disorders. *J Neurol Neurosurg Psychiatry* 2004;75:758–64.
46. Krafft M, Kullgren A, Tingvall C, et al. How crash severity in rear impacts influences short- and long-term consequences to the neck. *Accid Anal Prev* 2000;32:187–95.
47. Krafft M, Kullgren A, Ydenius A, et al. Influence of crash pulse characteristics on whiplash associated disorders in rear impacts – crash recording in real life crashes. *Traffic Inj Prev* 2002;3:141–9.
48. Nordin M, Carragee EJ, Hogg-Johnson S, et al. Assessment of neck pain and its associated disorders. Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *Spine* 2008;33(Suppl):S101–S122.
49. Nederhand MJ, Hermens HJ, IJzerman MJ, et al. Chronic neck pain disability due to an acute whiplash injury. *Pain* 2003;102:63–71.
50. Nederhand MJ, IJzerman MJ, Hermens HJ, et al. Predictive value of fear avoidance in developing chronic neck pain disability: consequences for clinical decision making. *Arch Phys Med Rehabil* 2004;85:496–501.
51. Suissa S, Harder S, Veilleux M. The relation between initial symptoms and signs and the prognosis of whiplash. *Eur Spine J* 2001;10:44–9.
52. Suissa S. Risk factors of poor prognosis after whiplash injury. *Pain Res Manag* 2003;8:69–75.
53. Carroll LJ, Cassidy JD, Côté P. The role of pain coping strategies in prognosis after whiplash injury: passive coping predicts slowed recovery. *Pain* 2006;124:18–26.
54. Côté P, Hogg-Johnson S, Cassidy JD, et al. The association between neck pain intensity, physical functioning, depressive symptomatology and time-to-claim-closure after whiplash. *J Clin Epidemiol* 2001;54:275–86.
55. Hurwitz EL, Carragee EJ, van der Velde G, et al. Treatment of neck pain: non-invasive interventions. Results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and its Associated Disorders. *Spine* 2008;33(Suppl):S123–S152.
56. Côté P, Hogg-Johnson S, Cassidy JD, et al. Early aggressive care and delayed recovery from whiplash: isolated finding or reproducible results? *Arthritis Care Res* 2007;57:861–8.
57. Côté P, Hogg-Johnson S, Cassidy JD, et al. Initial patterns of clinical care and recovery from whiplash injuries: a population-based cohort study. *Arch Intern Med* 2005;165:2257–63.
58. Kasch H, Stengaard-Pedersen K, Arendt-Nielsen L, et al. Pain thresholds and tenderness in neck and head following acute whiplash injury: a prospective study. *Cephalalgia* 2001;21:189–97.
59. Kasch H, Qerama E, Bach FW, et al. Reduced cold pressor pain tolerance in non-recovered whiplash patients: a 1-year prospective study. *Eur J Pain* 2005;9:561–9.
60. Buskila D, Neumann L, Vaisberg G, et al. Increased rates of fibromyalgia following cervical spine injury. A controlled study of 161 cases of traumatic injury. *Arthritis Rheum* 1997;40:446–52.
61. Holm LW, Carroll LJ, Cassidy JD, et al. Widespread pain following whiplash-associated disorders: incidence, course, and risk factors. *J Rheumatol* 2007;34:193–200.
62. Wynne-Jones G, Jones GT, Wiles NJ, et al. Predicting new onset of widespread pain following a motor vehicle collision. *J Rheumatol* 2006;33:968–74.
63. Carroll LJ, Cassidy JD, Côté P. Frequency, timing, and course of depressive symptomatology after whiplash. *Spine* 2006;31:E551–E556.
64. Ferrari R, Obelieniene D, Russel AS, et al. Laypersons' expectation of the sequelae of whiplash injury. A cross-cultural comparative study between Canada and Lithuania. *Med Sci Monit* 2002;8:CR728–CR734.
65. Ferrari R, Lang C. A cross-cultural comparison between Canada and Germany of symptom expectations for whiplash injury. *J Spinal Disord Tech* 2005;18:92–7.