

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

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

Study Design.

Best evidence synthesis.
Objective. To undertake a best evidence synthesis on the burden and determinants of whiplash-associated disorders (WAD) after traffic collisions.

Summary of Background Data. Previous best evidence synthesis on WAD has noted a lack of evidence regarding incidence of and risk factors for WAD. Therefore there was a warrant of a reanalyze of this body of research.

Methods. A systematic search of Medline was conducted. The reviewers looked for studies on neck pain and its associated disorders published 1980–2006. Each relevant study was independently and critically reviewed by rotating pairs of reviewers. Data from studies judged to have acceptable internal validity (scientifically admissible) were abstracted into evidence tables, and provide the body of the best evidence synthesis.

Results. The authors found 32 scientifically admissible studies related to the burden and determinants of WAD. In the Western world, visits to emergency rooms due to WAD have increased over the past 30 years. The annual cumulative incidence of WAD differed substantially between countries. They found that occupant seat position and collision impact direction were associated with WAD in one study. Eliminating insurance payments for pain and suffering were associated with a lower incidence of WAD injury claims in one study. Younger ages and being a female were both associated with filing claims or seeking care for WAD, although the evidence is not consistent. Preliminary evidence suggested that headrests/car seats, aimed to limiting head extension during rear-end collisions had a preventive effect on reporting WAD, especially in females.

Conclusion. WAD after traffic collisions affects many people. Despite many years of research, the evidence regarding risk factors for WAD is sparse but seems to include personal, societal, and environmental factors. More research including, well-defined studies with accurate denominators for calculating risk, and better consideration of confounding factors, are needed.

Key words: neck injury, whiplash-associated disorders, traffic collision, systematic review, epidemiology.

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

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 Lena W Holm, DrMedSc, Institute of Environmental Medicine, Division of Epidemiology, Karolinska Institutet, PO Box 210, SE-171 77 Stockholm, Sweden; E-mail: Lena.Holm@ki.se

Whiplash injuries occur primarily after motor vehicle collisions, although they can also occur in other settings, such as work and sports. The Québec Task Force on Whiplash-Associated Disorders defined whiplash as “an acceleration-deceleration mechanism of energy transferred to the neck that results in soft tissue injury that may lead to a variety of clinical manifestations including neck pain and its associated symptoms.”¹ That task force also coined the term “whiplash-associated disorders” (WAD) to describe the clinical entities related to the injury, and to distinguish them from the injury mechanism.

It is likely that WAD results from cervical sprain or strain. The exact pathophysiology is not known, and there may or may not be damage to soft tissue, including

the joints, ligaments and/or the muscles in the neck, posterior shoulder and upper thoracic regions. Cervical fractures are generally excluded from WAD, even though they sometimes occur as a result of a whiplash acceleration/deceleration mechanism. Although traffic collision is the most common cause of WAD, the disorder can also occur as a result of falls or other mishaps.²

A range of biomechanical research has been published exploring possible mechanisms of injury to the neck. One key area of interest is to determine the minimum threshold of force during an impact that is required to produce WAD. That literature includes experimental studies of crash tests using dummies, animals or human cadavers, and volunteers.³⁻⁵ The experimental literature using dummies, animals or human cadavers was outside of the mandate of the Neck Pain Task Force, and we did not include those studies in our best evidence synthesis.

The occurrence of WAD is based on a combination of factors, including exposure to a whiplash mechanism, followed by the appearance of symptoms or clinical signs. However, there is no gold standard diagnostic test, such as radiograph, computerized tomography (CT) or magnetic resonance imaging (MRI), to detect WAD. The clinical diagnosis is also confused by the high prevalence of neck pain and other WAD-like symptoms in the general population and in the working population.^{2,6}

In the introductory paragraphs of this report of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders, details of the background of the Task Force and its purpose are presented.^{6a} The overall objective was to conduct a systematic search and a critical review of the literature to produce a body of the best evidence on neck pain and its associated disorders. This includes findings on incidence, prevalence, risk, diagnosis, prognosis, treatment, and prevention. This article reports a best evidence synthesis on incidence, prevalence and risk factors of WAD after traffic collisions, *i.e.*, for neck pain after traffic collisions. Other results of the best evidence on neck pain and its associated disorders are reported elsewhere.^{7-10c,17-19}

■ Materials and Methods

Design and Data Collection

The literature search and critical review strategy are outlined in detail elsewhere.⁷ Briefly, we systematically searched the electronic library database Medline for literature published from 1980 through 2005 on neck pain and its associated disorders (including neck pain after traffic collisions); we updated our search with key articles published in 2006 and early 2007, and we also systematically checked reference lists for other potentially relevant articles. Our electronic search strategy is described in more detail elsewhere,⁷ and is available in detail online.

Each citation was screened for relevance to the Neck Pain Task Force mandate, using *a priori* inclusion and exclusion criteria. However, we made no attempt to assess the scientific quality of studies when establishing this level of relevance. Studies were considered relevant if they pertained to neck pain assessment, diagnosis, incidence, prevalence, determinants,

risk factors, prevention, course, prognosis, treatment and rehabilitation; if they pertained to the economic costs of neck pain; if they contained data and findings specific to neck pain and/or disorders associated with neck pain; if they included at least 20 persons with neck pain or at risk for neck pain; or if they described a systematic review of the literature on neck pain.

We included neck pain resulting from traffic-related whiplash injuries, and work-related injuries and strains, and neck pain from sports injuries and of unknown etiology in the general population. Clinical case series were included if they were judged to be of special relevance to the Neck Pain Task Force report, for example, if they were frequently cited in the literature, or if they focused on a topic for which there was little or no information available. We excluded studies on neck pain that was associated with serious local pathology or systemic disease, such as neck pain from fractures or dislocations (except for studies on assessment, where such studies can inform differential diagnosis), infections, myelopathy; rheumatoid arthritis and other inflammatory joint diseases; or tumors.

Quality Assessment

Rotating pairs of clinician/scientist reviewers (members of the Scientific Secretariat) performed independent, in-depth, critical reviews of each article, identifying methodologic strengths, and weaknesses. After a full discussion of each article, the reviewers then made decisions about the article's scientific and clinical merit. Criteria used in the methodologic appraisal of the studies are available on the following internet site (address of our web-version). Our appraisal focused on sources of potential selection bias, information bias, and confounding; we also considered whether these biases would likely result in erroneous or misleading conclusions.

Studies judged to have adequate internal validity were included in our best evidence synthesis. We divided our syntheses of evidence regarding incidence, prevalence, risk factors, and prevention based on the following source populations: workers; people involved in traffic collisions; and finally, the general population (which included people with sports-related neck pain).

Analysis

To better delineate risk factors and evidence strength from cohort studies where risk factors were assessed, we adapted a ranking method used in previous studies and systematic reviews.⁸⁻¹² The methodology acknowledges three types of analytic approach when assessing associations between determinants and an outcome:

- Phase I studies are hypothesis-generating, descriptive investigations that explore crude associations between single factors and outcome.
- Phase II studies are also exploratory, but use stratified or multivariable analyses to identify sets of predictors.
- Phase III studies are hypothesis-driven and confirmatory. The goal is to confirm or refute hypotheses about the apparent relationship between a particular risk factor and the outcome of interest (in this case, onset of neck pain), after adjusting for confounding.

Organization of the Findings

We have organized our results for incidence of WAD based on study settings (*i.e.*, emergency room visits and insurance injury claims). 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 links summary statements and conclusions to the evidence tables, so that the evidence which formed the basis of any statements is made clear.¹³⁻¹⁵

In accordance with our conceptual framework on the course and care of neck pain,¹⁶ and similar to the organization of other papers in this supplement,^{2,6,17-19} we further classified risk 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/prior pain/comorbidities:** These can be classified as “impairments” according to the WHO’s ICF framework.²⁰
- **Collision factors:** These could include information about the collision, such as direction of impact, occupant’s seat position, and use of headrests and other safety devices. Some of the factors may be modifiable, through car design.
- **Psychological and social factors:** These would include depression, anxiety, and coping strategies, and interpersonal factors (e.g., relationships with friends). Many of these factors are potentially modifiable.
- **Societal factor:** 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 factors, although not considered modifiable.
- **Cultural factors:** This would include health lifestyle factors, and perception of health, including health expectations and beliefs. Some may be potential modifiable, others not easily modifiable.

Incidence rates, estimates of prevalence, and relative risks with confidence intervals from these studies are presented in the tables, or when possible, calculated from data contained in the papers.

Studies concerning neck pain related to mechanisms other than motor vehicle collisions are described elsewhere in the Neck Pain Task Force Report.^{2,6} However, studies containing information related to both risk of neck pain due to traffic collisions and information about risk of neck pain due to un-specific mechanisms in the general population are cited in both this article and in Hogg-Johnson *et al.*²

■ Results

Our results are organized according to study design, the subpopulation of interest and the type of evidence found in the literature. Four evidence tables summarize the admissible papers as follows: cohort studies providing evidence on incidence and risk factors (Tables 1 and 2, available online through Article Plus); experimental studies providing evidence of possible injury mechanism in low-impact, rear-end collisions (Table 3, available online through Article Plus), and cohort and cross-sectional studies providing evidence on prevalence and/or associated factors (Table 4, available online through Article Plus).

The reviewers looked at 469 relevant studies relating to incidence, risk factors, and prevention of neck pain and its associated disorders. Of the 249 studies (53%) judged to be scientifically admissible, 32 were related to WAD after traffic collisions and are included in the best evidence synthesis.^{1,21-51} Eighteen of the studies are from Europe, eight are from North America, four are from Australia, one is from Asia, and one is a systematic review.

Cumulative Incidence

There were nine scientifically admissible studies on the annual cumulative incidence of neck injuries based on population denominators (Table 1, available online through Article Plus).^{21,25,27-29,40,41,43,46} These studies are from different time periods, different settings, and span five different countries (Sweden, the United Kingdom, the Netherlands, the United States and Canada).

Emergency Room Visits for WAD. Four of the accepted studies were from the northern part of Sweden. The two oldest studies included all mechanisms of injury, but the traffic specific incidences were given or could be calculated; thus these traffic-specific incidences are included in this article.^{25,27} The two most recent studies included motor vehicle injuries only.^{28,43} Over time, there was an increase of the number of subjects seeking emergency room health care for traffic-related WAD, with an annual cumulative incidence increasing from 83 per 100,000 inhabitants (1985–1986), to 142 per 100,000 (1988–1990), to 147 per 100,000 (1990–1991), and to 302 per 100,000 (1997–1998). The latter study also included visits to general practitioners (about 10% of the cases). A 1983–1984 hospital-based study from the United Kingdom (which included persons presenting to hospital for evaluation of WAD symptoms) reported an annual incidence of WAD of 27.8 (95% CI 23.6–32.6) per 100,000 inhabitants.⁴⁰ A Dutch study identified patients who had been exposed to a traffic collision and had attended an emergency department, complaining of neck pain.⁴⁶ Over a 20-year period, they found a 10-fold increase in such visits, from an average annual incidence of 3.4 visits per 100,000 inhabitants (1970–1974) to 40.2 visits per 100,000 (1990–1994). Quinlan *et al* reported the weighted annual incidence of emergency room visits to be 328 visits (95% CI 254–402) per 100,000 inhabitants, based on the population of the United States in the year 2000.⁴¹

Insurance Claims for WAD. Two studies from Canada used insurance data to assess the cumulative incidence of WAD. These studies included those who made a claim and/or who presented for treatment for WAD (since initiating treatment also resulted in making an insurance claim). The incidence of reported/treated injuries varied substantially, from 70 per 100,000 in the province of Québec in 1993,²¹ to approximately 600 per 100,000 in the province of Saskatchewan in 1995.²⁹ During the last 6 months period of a “tort” system (as opposed to a “no fault” system) in Saskatchewan in 1994, the incidence was 417 per 100,000. This is despite the fact that, in

those years, it was possible to for people in both provinces to make claims for disability and treatment costs. However, policy conditions may have differed in other ways between these provinces, for example, inception into the Québec cohort study was limited to those who actually received compensation, although this difference is unlikely to completely explain the large difference in incidence.

Factors Associated With WAD in Traffic Collisions

Twelve cohort studies presented factors associated with WAD after traffic collisions (Table 1; available online through Article Plus).^{11,21,24,27,29,31,32,35,38,41,46,48} Ten of these studies were Phase I studies, one was a natural experiment, and one was a Phase II study. All potential risk factors for WAD that have been investigated in scientifically admissible cohort studies are listed in Table 2 (available online through Article Plus). All studies identified WAD cases through either hospital attendance or insurance claims.

Demographics/Socioeconomic Variables in WAD

Gender. The evidence that gender is associated with risk of WAD is inconsistent, although it seems as females are at slightly greater risk. Three studies based on hospital attendance did not find any gender differences in risk for sustaining WAD^{25,27,46}; however, one other study found that female gender was associated with seeking hospital care due to WAD.⁴¹ Furthermore, one Phase I study,²¹ one natural experiment²⁹ and the Phase II study,²⁴ all based on insurance claims, found that females had a slightly increased risk of WAD compared to males. In the study by Cassidy *et al*, we recalculated the gender stratified incidence and the results reveal that the gender difference was greater during the period of no-fault system (IRR 1.6) compared to claiming under tort system (IRR = 1.1).²⁹

Age. In a Phase II study, younger age was associated with a slightly higher risk of WAD (IRR point estimates were less than 1.2 for all age groups), compared with age 55 years and older.²⁴ A recalculation of the age stratified incidence in a large natural experiment showed a much stronger (unadjusted) association between younger age and being treated for or claiming for WAD, especially when claiming under the tort insurance system (IRR for ages 18–23 = 4.6 under tort and IRR = 3.5 under the no fault system).²⁹

Prior Health/Prior Pain/Co-Morbidities in WAD. In one Phase I study, subjects who recalled having more than 1 day a month of neck pain before a rear-end collision had a higher incidence of acute neck pain after the collision.³⁸ We found no scientifically admissible studies examining whether degenerative changes in the cervical spine increase the risk of WAD after a traffic collision. However, the fact that *younger* age groups are *more* likely to seek health care or file a claim for WAD (see above) argues

against that association, since degenerative changes are highly associated with increasing age.

Collision Factors in WAD. One Phase II study of persons filing an traffic-injury insurance claim reported that WAD was associated with being the driver or the front seat passenger (*vs.* being a rear seat passenger) and being exposed to a rear-end or frontal collision (*vs.* a side collision).²⁴ A Phase I study (of insurance claims) found that persons exposed to rear-end collisions and seated in cars equipped with tow-bars were not at higher risk for reporting WAD.³⁵ Results from one Phase I study (of insurance claims) suggest that the type of restraint used to secure children in vehicles was not associated with neck injury in children (recalculated by the Neck Pain Task Force).⁴⁸ However, differences in the subjects' age might have impacted on the findings, since the different child restraints are age specific.

Two studies (of insurance claims) assessed the effect of equipment aimed at limiting head extension during rear-end collisions (Table 1, available online through Article Plus).^{31,32} One study found female drivers, but not males, had a lower rate of WAD if they were in cars equipped with "good-rated" (*vs.* "poor-rated") head restraints.³¹ The other study found that active (*i.e.*, activated automatically in case of a rear end collision and not movable by the driver/passenger) devices such as active head rests and seat backs were associated with an overall 43% significant reduction in WAD claims in favor of the devices.³² The effect was greater in females.

Psychological and Social Factors in WAD. We found no scientifically admissible study or studies examining the effect of psychological or social factors in the onset of WAD after traffic collision.

Compensation, Legal, Societal Factors in WAD. One study found that, at the population level, changing the insurance system from tort (involving compensation for pain and suffering) to no-fault (higher health care, income replacement and other benefits, but no compensation for pain and suffering) was associated with a 40% lower incidence rate of filing an insurance claim or seeking health care treatment for WAD after traffic collision.²⁹

Genetic Factors in WAD. We found no scientifically admissible study or studies examining the effect of genetic factors on the onset of WAD after traffic collision.

Cultural Factors in WAD. We found no scientifically admissible studies examining the effect of cultural influence on the onset of WAD after traffic collision.

Experimental Studies

We found two admissible experimental studies involving exposing volunteers to low-speed collision and recording the onset of neck pain and associated disorders (Table 3, available online).^{26,30} Approximately one-third of subjects exposed to such a low-speed collision (where the speed change at impact was between 4 and 8 km/h) re-

ported transient WAD symptoms.²⁶ However, so did one-quarter of subjects exposed to a sham collision.³⁰

Other Health Factors and WAD

We included 12 studies which looked at various health-related factors and their associations with previous motor vehicle collision (Table 4, available online through Article Plus).^{22,23,33,34,36,37,39,42,47,50,51}

Three of these studies were cohort studies describing the incidence and/or prevalence of factors associated with WAD.^{33,37,51} One study describes the prevalence of various precollision health conditions and incidence of postinjury symptoms affecting neck injury insurance claimants in Saskatchewan.³³ Both sexes also had a high incidence of WAD symptoms other than neck pain, although in general, females reported more postinjury symptoms than males. For example, in addition to neck pain, 86.1% of women reported postinjury headaches as a result of the collision (as did 78.4% of men); 64.6% of women reported low back pain (as did 61.9% of men); and 20% of women reported reduced or painful jaw movement (as did 13.2% of men). Females also reported a higher prevalence than males of preinjury health complaints, such as prior neck/shoulder pain, headaches, and low back pain.³³ One study examined the incidence of temporomandibular disorders, treated by dentists in Australia and reported to a motor insurer. These were uncommon: The incidence of treated temporomandibular disorders in WAD claims was 0.05% over a five-year period.⁵¹ One study by Matsumoto *et al*, found that cervical lordosis and angular kyphosis were equally prevalent in normal controls and in patients with acute WAD (presenting to hospital after the injury).³⁷

Nine cross-sectional studies/case series described associations between various health complaints and having persistent WAD.^{22,23,34,36,39,42,45,47,50} Three of these studies looked at cognitive functioning/malingering in patients with chronic WAD.^{34,39,42} We conclude from these studies that no evidence exists to show that poor cognitive functioning in patients seeking treatment for chronic WAD is the result of demonstrable brain damage; instead, these deficits may be linked to a chronic health condition (including chronic pain). A study by Guez *et al* found no evidence of malingering on MMPI in 21 volunteers with chronic WAD.³⁴ However, in another study which differentiated litigants and nonlitigants, litigants were found to obtain higher scores on a malingering test.⁴² However, this should not be taken to mean that litigation leads to malingering or that those who litigate are malingerers.

Four studies looked at associations between reporting a WAD and different psychological states.^{22,23,36,47} One cross-sectional study assessed coping in WAD patients who had been referred to a tertiary rehabilitation clinic 3–22 months after their injury, and found that pain interference was associated with type of coping strategies used, but pain severity was not associated with any particular type of coping.³⁶ Another cross-sectional study

reported that in patients with chronic pain and work disability after WAD, depression and use of the coping style of “catastrophizing” were associated with reduced health quality of life.²² Wenzel *et al* reported greater anxiety and depression in those who reported that they had experienced a whiplash trauma at least 2 years previously: No such association was found for more recent whiplash trauma.²³ Finally, in a study of patients referred to a specialty research unit for chronic (more than 3 months) neck pain attributed to a motor vehicle collision, those with chronic headaches and neck pain have similar psychological profiles (on testing using the SCL-90) to those with neck pain but no headaches. In the subgroup of patients with headaches, those with non-traumatic headaches had higher (signifying more psychological distress) subscale scores than the normative sample; whereas those with WAD-related headaches had higher scores than the normative sample on only four subscales and on the global severity index.⁴⁷

Another study found that within 1 month of subjects seeking emergency, hospital or private practice care for WAD, there was a positive associations between the early severity of pain and various outcomes, including generalized hypersensitivity, cervical range of motion, reported dizziness and psychological distress.⁵⁰ One study found positive associations between dizziness and balance in patients with chronic WAD, who had been referred to a specialty whiplash clinic.⁴⁵ It is important to note that we cannot generalize any of the prevalence estimates from these cross-sectional studies to other populations, since the study samples are selective. It is also important to note that neither temporal relationships nor causation can be determined by cross-sectional studies.

Discussion

Our review of the literature found consistent evidence that the annual cumulative incidence of hospital visits due to WAD in traffic collisions has increased in some western countries during the past 30 years. It is unclear if the observed increase is a true population increase in the frequency and/or severity of WAD, since it may reflect a change in care-seeking behavior for suspected neck injuries after traffic collisions. For example, Versteegen *et al* found a greater than fourfold increase in hospital visits due to reported neck sprains from causes other than car collisions during the last two decades of the 20th century.⁵² Alternatively, the increase might possibly reflect changes in how such injuries are recorded in health records. Estimates of the occurrence of WAD using insurance data may be less prone to those biases. However, ascertaining frequency of WAD through insurance claims may be prone to other biases, for instance, insurance systems where there are no benefits for the person responsible for the collision may underestimate the frequency of WAD since they would have fewer claims, as may insurance systems where insurance claim access is lim-

ited, or where payments for compensation result in a significant increase of the insurance premium.

The evidence of gender as a risk factor for seeking health care or making a claim for WAD is not consistent. However, the four strongest of the seven studies suggest that females have a slightly increased risk.^{21,24,29,41} Interestingly, neck pain is also more common in females in both the general population and in working populations.^{2,6} There is also preliminary evidence that neck pain before a collision might be a risk factor for acute neck pain after a rear-end collision.³⁸ That might explain the apparent gender difference in risk (*i.e.*, it might be that females are more vulnerable to WAD because they are more likely to have experienced previous episodes of neck pain, or perhaps already have neck pain at the time of the collision).

Younger persons seem to be at greater risk of making insurance claims and/or being treated for WAD. The strength of this association was variable, and ranged from an increased risk of only 20% (or less)²⁴ to an over fourfold increase in risk for the youngest age group (aged 18–23).²⁹ There is also a suggestion that the strength of the relationship between age and WAD claims/clinical care for WAD might depend on the characteristics of the insurance compensation system. These findings also suggest that there is not a strong association between degenerative cervical changes and risk of WAD, although this question should be studied directly.

Despite the fact that the first case series on neck pain after a whiplash mechanism was published more than 50 years ago,⁵³ the knowledge about risk factors for sustaining such injury is still lacking. We found no Phase III study or studies on this topic during our search of the literature. We only found one Phase II study, in which the authors had adjusted for a limited number of potential confounders, and there was one population-based natural experiment.

One reason for this sparseness of evidence and inconsistency in the study findings might be that it is difficult to obtain accurate and similar denominators to calculate risk (incidence). Rather than using all vehicle occupants exposed to collisions as the denominator (which would be the most appropriate denominator, but is difficult information to obtain), researchers have used proxies, such as registered licensed drivers,²¹ population censuses,^{21,25,27,29,41} or persons involved in collisions where at least one person was injured.²⁴ Although recognizing the difficulties in collecting appropriate data, especially on uninjured occupants in car crashes or on injured individuals electing not to seek health care or compensation, we think that studies that include both collisions with injuries and collisions without injuries are of utmost importance for the assessment of risk. Such studies should have clearly stated hypotheses and include proper controls for potential confounding factors, such as age, gender, premorbid conditions, and for collision-specific circumstances.

One potentially important factor in risk of WAD is the severity of impact, but to date no method exists to assess this in a standardized way. Although we found

preliminary evidence of a positive, protective effect of various preventive devices in passenger cars in rear-end collisions, such findings must be confirmed in larger settings, and with consideration of the persons preinjury health, age, gender, and other potential confounders.

The two experimental studies included in our best evidence synthesis raise an interesting hypothesis: do low-speed collisions cause any significant damage to cervical soft tissues, or might fearfulness and/or cervical muscle tension fully or partially explain the development of some WAD symptoms after low-impact exposure? However, the current evidence does not allow us to draw any conclusions about a specific injury mechanism, if one exists. We believe findings from such experiments must be confirmed by conducting methodologically sound studies involving real-life crashes.

We found no scientifically admissible evidence supporting common statements that awareness of the collision and head position at the time of injury are relevant to WAD risk. Nor did we find scientifically admissible evidence that persons with cervical degeneration are higher risk for WAD than those without this condition. Furthermore, we need to determine the minimum threshold of impact forces (for sustaining WAD) in real-life crashes. It is possible, for example, that this threshold differs in different populations (*e.g.*, gender, age, prior neck pain).

In conclusion, in the western world, the annual incidence of reported WAD is likely at least 300 per 100,000 inhabitants. The incidence of reported WAD in these geographic areas has increased over the past 30 years, but we found no clear explanation for this increase. Risk factors for WAD are not well established, and there is a need for more research into this area, both regarding the influence of personal factors such as for instance premorbid conditions, and collision factors. This should include large, well-defined Phase III studies, and should expand to include subjects exposed to a whiplash mechanism, whether or not they sustain a WAD, and whether or not they elect to file claims or seek health care.

■ Key Points

- The incidence of reported WAD in western countries has increased over the past 30 years.
- The annual incidence varied, but in North America and western Europe, the incidence is likely to be at least 300 per 100,000 inhabitants.
- The evidence of determinants for WAD is sparse, but personal, societal, and environmental factors are of importance.
- There is consistent evidence from five studies that in the western world, the incidence of emergency room attendance due to WAD after a traffic collision has increased in the general population during the last 30 years.

- There is preliminary evidence that persons in younger ages and persons with prior neck pain were at higher risk of seeking care or filing a claim for WAD. The strongest studies suggest that females were at slightly increased risk, although the evidence is not consistent.
- There is evidence from one large study (a population-based natural experiment, elimination of insurance payment for pain and suffering (change from a “tort” system, where compensation for pain and suffering is available to a “no-fault” system, where insurance benefits were increased but no pain and suffering compensation was available) was associated with fewer insurance claims for WAD.
- There is preliminary evidence for the efficacy of whiplash protection devices aimed at limiting passenger head extension in rear-end collision. One study (using insurance claims to identify WAD) found female drivers, but not males, had a lower rate of WAD if they were in cars equipped with “good-rated” (*vs.* “poor-rated”) head restraint position. Another study of insurance claims found that devices such as active head rests and seat backs were associated with reduction in WAD insurance claims; this positive effect was greater in female drivers.
- We found no evidence on the effect on crash severity for the onset of WAD. We found no evidence supporting the common statement that awareness of the collision and head position at the time of injury were important in the onset of WAD. Nor did we find evidence supporting the common statement that persons with spinal degenerative changes have a higher risk for WAD.



tables

Tables available online through Article Plus.

Acknowledgments

The authors are indebted to Ms. Oksana Colson and Ms. Leah Phillips (M.A.) for their administrative assistance; to Mr. Stephen Greenhalgh (M.A., 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; and to Ms. Evelyne Michaels for her assistance in editing the manuscripts. The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders was supported by grants from the following: National Chiropractic Mutual Insurance Company (USA); Canadian Chiropractic Protective Association (Canada); State Farm Insurance Company (USA); Insurance Bureau of Canada; *Länsförsäkringar* (Sweden); *The Swedish Whiplash Commission*; Jalan Pacific Inc. (Brazil); Amgen (USA). All funds received were unrestricted grants. Funders had no control in planning,

research activities, analysis or results. The report was not released to grantors prior to publication and no approval was required from funders regarding the final report. Dr. Côté is supported by the Canadian Institutes of Health Research through a New Investigator Award and by the Institute for Work & 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. 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;20:15–73S.
2. 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.
3. Cholewicki J, Panjabi MM, Nibu K, et al. Head kinematics during in vitro whiplash simulation. *Accid Anal Prev* 1998;30:469–79.
4. Tencer F, Mirza S, Bensek K. Internal loads in the cervical spine during motor vehicle rear-end impacts: the effect of acceleration and head-to-head restraint proximity. *Spine* 2002;27:34–42.
5. Siegmund GP, Myers BS, Davis MB, et al. Mechanical evidence of cervical facet capsule injury during whiplash: a cadaveric study using combined shear, compression, and extension loading. *Spine* 2001;26:2095–101.
6. 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.
- 6a. Haldeman S, Carroll LJ, Cassidy JD. The empowerment of people with neck pain. The bone and joint decade 2000–2010 task force on neck pain and its associated disorders. *Spine* 2008;33(Suppl):S8–S13.
7. 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.
8. Altman DG, Lyman GH. Methodological challenges in the evaluation of prognostic factors in breast cancer. *Breast Cancer Res Treat* 1998;52:289–303.
9. 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.
10. 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.
- 10a. Hurwitz EL, Carragee EJ, van der Velde G, et al. Treatment of neck pain: noninvasive interventions. *Spine* 2008;33(Suppl):S123–S152.
- 10b. 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.
- 10c. Carragee EJ, Hurwitz EL, Cheng I, et al. Treatment of neck pain: injections and surgical interventions: results of the bone and joint decade 2000–2010 task force on neck pain and its associated disorders. *Spine* 2008;33(Suppl):S153–S169.
11. Cassidy JD, Carroll LJ, Peloso PM, et al. Incidence, risk factors and prevention of mild traumatic brain injury: results of the WHO collaborating centre task force on mild traumatic brain injury. *J Rehabil Med* 2004;43:28–60.
12. 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.
13. Slavin RE. Best evidence synthesis: an alternative to meta-analytic and traditional reviews. *Educ Res* 1986;15:5–11.
14. Slavin RE. Best evidence synthesis: an intelligent alternative to meta-analysis. *J Clin Epidemiol* 1995;48:9–18.
15. 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.

16. 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.
17. 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.
18. Carroll LJ, Holm LW, Hogg-Johnson S, et al. 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. *Spine* 2008;33(Suppl):S83–S92.
19. 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.
20. World Health Organization. *International Classification of Function, Disability and Health: ICF*. Geneva: WHO; 2001.
21. Suissa S, Harder S, Veilleux M. The Quebec whiplash-associated disorders cohort study. *Spine* 1995;20:125–20S.
22. Peolsson M, Gerdle B. Coping in patients with chronic whiplash-associated disorders: a descriptive study. *J Rehabil Med* 2004;36:28–35.
23. Wenzel H, Haug T, Mykletun A, et al. A population study of anxiety and depression among persons who report whiplash traumas. *J Psychosom Res* 2002;3:831–5.
24. Berglund A, Alfredsson L, Jensen I, et al. Occupant- and crash-related factors associated with the risk of whiplash injury. *Ann Epidemiol* 2003;13:66–72.
25. Bjornstig U, Hildingsson C, Toolanen G. Soft-tissue injury of the neck in a hospital based material. *Scand J Soc Med* 1990;18:263–7.
26. Brault JR, Wheeler JB, Siegmund GP, et al. Clinical response of human subjects to rear-end automobile collisions. *Arch Phys Med Rehabil* 1998;79:72–80.
27. Bring G, Bjornstig U, Westman G. Gender patterns in minor head and neck injuries: an analysis of casualty register data. *Accid Anal Prev* 1996;28:359–69.
28. Bylund PO, Bjornstig U. Sick leave and disability pension among passenger car occupants injured in urban traffic. *Spine* 1998;23:1023–8.
29. 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.
30. Castro WHM, Meyer SJ, Becke MER, et al. No stress—no whiplash? Prevalence of whiplash symptoms following exposure to a placebo rear-end collision. *Int J Legal Med* 2001;114:316–22.
31. Farmer CM, Wells JK, Werner JV. Relationship of head restraint positioning to driver neck injury in rear-end crashes. *Accid Anal Prev* 1999;31:719–28.
32. Farmer CM, Wells JK, Lund AK. *Effects of Head Restraint and Seat Redesign on Neck Injury Risk in Rear-End Crashes*. Arlington, VA: Insurance Institute for Highway Safety; 2002.
33. Ferrari R, Russell AS, Carroll LJ, et al. A re-examination of the whiplash associated disorders (WAD) as a systemic illness. *Ann Rheum Dis* 2005;64:1337–42.
34. Guez M, Brannstrom R, Nyberg L, et al. Neuropsychological functioning and MMPI-2 profiles in chronic neck pain: a comparison of whiplash and non-traumatic groups. *J Clin Exp Neuropsychol* 2005;27:151–63.
35. 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.
36. LaChapelle DL, Hadjistavropoulos HD, McCreary DR, et al. Contributions of pain-related adjustment and perceptions of control to coping strategy use among cervical sprain patients. *Eur J Pain* 2001;5:405–13.
37. Matsumoto M, Fujimura Y, Suzuki N, et al. Cervical curvature in acute whiplash injuries: prospective comparative study with asymptomatic subjects. *Injury* 1998;29:775–8.
38. 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.
39. Olsnes BT. Neurobehavioral findings in whiplash patients with long-lasting symptoms. *Acta Neurol Scand* 1989;80:584–8.
40. Otremski I, Marsh JL, Wilde BR, et al. Soft tissue cervical spinal injuries in motor vehicle accidents. *Injury* 1989;20:349–51.
41. Quinlan KP, Annett JL, Myers B, et al. Neck strains and sprains among motor vehicle occupants—United States, 2000. *Accid Anal Prev* 2004;36:21–7.
42. Schmand B, Lindeboom J, Schagen S, et al. Cognitive complaints in patients after whiplash injury: the impact of malingering. *J Neurol Neurosurg Psychiatry* 1998;64:339–43.
43. Sterner Y, Toolanen G, Gerdle B, et al. The incidence of whiplash trauma and the effects of different factors on recovery. *J Spinal Disord Techn* 2003;195–9.
44. Tingvall C. Children in cars. Some aspects of the safety of children as car passengers in road traffic accidents. *Acta Paediatrica Scandinavica* 1987;339 (Suppl):1–35.
45. Treleaven J, Jull G, Lowchoy N. Standing balance in persistent whiplash: a comparison between subjects with and without dizziness. *J Rehabil Med* 2005;37:224–9.
46. Versteegen GJ, Kingma J, Meijler WJ, et al. Neck sprain in patients injured in car accidents: a retrospective study covering the period 1970–1994. *Eur Spine J* 1998;7:195–200.
47. Wallis BJ, Lord SM, Barnsley L, et al. The psychological profiles of patients with whiplash-associated headache. *Cephalalgia* 1998;18:101–5.
48. Aldman B, Gustafsson H, Nygren A, et al. Child restraints. A prospective study of children as car passengers in road traffic accidents with respect to restraint effectiveness. *Acta Paediatr Scand* 1987;76.
49. Gustafsson H, Nygren A, Tingvall C. Children in cars. An epidemiological study of injuries to children as car passengers in road traffic accidents. *Acta Paediatr Scand* 1987;76 (Suppl 339).
50. Sterling M, Jull G, Vicenzino B, et al. Characterization of acute whiplash-associated disorders. *Spine* 2004;29:182–8.
51. Probert TC, Wiesenfeld D, Reade PC. Temporomandibular pain dysfunction disorder resulting from road traffic accidents—an Australian study. *Int J Oral Maxillofac Surg* 1994;23:338–41.
52. Versteegen GJ, Kingma J, Meijler WJ, et al. Neck sprain not arising from car accidents: a retrospective study covering 25 years. *Eur Spine J* 1998;7:201–5.
53. Gay J, Abbot K. Common Whiplash injuries of the neck. *JAMA* 1953;152:1698–704.