# THE ASSOCIATION BETWEEN COMPENSATION AND OUTCOME AFTER INJURY.

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# DECLARATION

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of the university or other institute of higher learning.

This research was developed by the author, with guidance from the two supervisors. Assistance with data collection and statistical analysis, and specific advice, where provided, is outlined in the acknowledgements. All other work, including all writing, was performed by the author alone.

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**ABSTRACT**

Work-related injuries and road traffic injuries are common causes of morbidity and are major contributors to the burden of disease worldwide. In developed countries, these injuries are often covered under compensation schemes, and the costs of administering these schemes is high. The compensation systems have been put in place to improve the health outcomes, both physical and mental, of those injured under such systems; yet there is a widespread belief, and some evidence, that patients treated under these schemes may have worse outcomes than if they were treated outside the compensation system.

Chapter One of this thesis explores the literature pertaining to any effect that compensation may have on patient outcomes. It is noted that the concept of "compensation neurosis" dates from the nineteenth century, with such injuries as "railway spine", in which passengers involved in even minor train accidents at the time, would often have chronic and widespread symptoms, usually with little physical pathology. Other illnesses have been similarly labelled over time, and similarities are also seen in currently diagnosed conditions such as repetition strain injury, back pain and whiplash. There are also similarities in a condition that has been labelled "shell shock", "battle fatigue", and "post-traumatic stress disorder"; the latter diagnosis originating in veterans of the Vietnam War.

While there is evidence of compensation status contributing to the diagnosis of some of these conditions, and to poor outcomes in patients diagnosed with

these conditions, there is little understanding of the mechanism of this association. In contrast to popular stereotypes, the literature review shows that malingering does not contribute significantly to the effect of compensation on health outcomes. Secondary gain is likely to play an important role, but secondary gain is not simply confined to financial gain, it also includes gains made from avoidance of workplace stress and home and family duties.

Other psychosocial factors, such as who is blamed for an injury (which may lead to retribution as a secondary gain) or the injured person's educational and occupational status, may also influence this compensation effect.

The literature review concludes that while the association between compensation and health after injury has been widely reported, the effect is inconsistent. These inconsistencies are due, at least in part, to differences in definitions of compensation (for example, claiming compensation versus using a lawyer), the use of different and poorly defined diagnoses (for example, back pain), a lack of control groups (many studies did not include uncompensated patients), and the lack of accounting for the many possible confounding factors (such as measures of injury severity or disease severity, and socio-economic and psychological factors). The literature review also highlighted the variety of different outcomes that had been used in previous studies, and the paucity of literature regarding the effect of compensation on general health outcomes.

This thesis aims to explore the association between compensation status and health outcome after injury. It addresses many of the methodological issues of the previously published literature by,

- i selecting study populations of patients with measurable injuries,
- ii clearly defining and separating aspects of compensation status,
- iii including control groups of non-compensated patients with similar injuries
- iv allowing for a wide variety of possible confounders, and
- v using clearly defined outcome measures, concentrating on general health outcomes.

Before commencing the clinical studies reported in Chapters Three and Four, a systematic review and meta-analysis was performed to quantify and analyse the effect of compensation on outcome after surgery. This allowed a clearly defined population of studies to be included, and was relevant to the thesis as the surgeries were performed as treatment of patients who had sustained injuries. The study, which is reported in Chapter Two, hypothesised that outcomes after surgery would be significantly worse for patients treated under compensation schemes.

The study used the following data sources: Medline (1966 to 2003), Embase (1980 to 2003), CINAHL, Cochrane Controlled Trials Register, reference lists of retrieved articles and textbooks, and contact with experts in the field. The review included any trial of surgical intervention where compensation status was reported and results were compared according to that status, and no restrictions were placed on study design, language or publication date. Data extracted were study type, study quality, surgical procedure, outcome, country of origin, length and completeness of follow-up, and compensation type. Studies were selected by two unblinded independent reviewers, and data were extracted by two reviewers independently.

Data were analysed using Cochrane Review Manager (version 4.2). Two hundred and eleven papers satisfied the inclusion criteria. Of these, 175 stated that the presence of compensation (worker's compensation with or without litigation) was associated with a worse outcome, 35 found no difference or did not describe a difference, and one paper described a benefit associated with compensation.

A meta-analysis of 129 papers with available data (20,498 patients) revealed the summary odds ratio for an unsatisfactory outcome in compensated patients to be 3.79 (95% confidence interval 3.28 to 4.37, random effects model). Grouping studies by country, procedure, length of follow-up, completeness of follow-up, study type, and type of compensation showed the association to be consistent for all sub-groups.

This study concludes that compensation status is associated with poor outcome after surgery, and that this effect is significant, clinically important and consistent. Therefore, the study hypothesis is accepted. However, as data were obtained from observational studies and were not homogeneous, the summary effect should be interpreted with caution.

Determination of the mechanism for the association between compensation status and poor outcome, shown in the literature review (Chapter One) and the systematic review (Chapter Two) required further study. Two studies were designed to further explore this association and these are reported in Chapters Three and Four.

The retrospective study reported in Chapter Three, the Major Trauma Outcome Study (MTOS), aimed to explore the association between physical, psychosocial, and compensation-related factors and general health after major physical trauma. The primary hypothesis predicted significantly poorer health outcomes in patients involved in pursuing compensation, allowing for possible confounders and interactions. The study also examined other health outcomes that are commonly associated with compensation, and examined patient satisfaction.

Consecutive patients presenting to a regional trauma centre with major trauma (defined as an Injury Severity Score greater than 15) were surveyed between one and six years after their injury. The possible predictive factors measured were: general patient factors (age, gender, the presence of chronic illnesses, and the time since the injury), injury severity factors (injury severity score, admission to intensive care, and presence of a significant head injury), socio-economic factors (education level, household income, and employment status at the time of injury and at follow-up), and claim-related factors (whether a claim was pursued, the type of claim, whether the claim had settled, the time to settlement, the time since settlement, whether a lawyer was used, and who the patient blamed for the injury). Multiple linear regression was used to develop a model with general health (as measured by the physical and mental component summaries of the SF-36 General Health Survey) as the primary outcome. The secondary outcomes analysed were: neck pain, back pain, post-traumatic stress disorder, and patient satisfaction.

On multivariate analysis, better physical health was significantly associated with increasing time since the injury, and with lower Injury Severity Scores. Regarding psychosocial factors, the education level and household income at the time of injury were not significantly associated with physical health, but pursuit of compensation, having an unsettled claim, and the use of a lawyer were strongly associated with poor physical health.

Measures of injury severity or socio-economic status were not associated with mental health. However, the presence of chronic illnesses and having an unsettled compensation claim were strongly associated with poor mental health.

Regarding the secondary outcomes, increasing neck pain and back pain were both significantly associated with lower education levels and the use of a lawyer, but not significantly associated with claiming compensation. The severity of symptoms related to post-traumatic stress disorder was not associated with measures of injury severity, but was significantly and independently associated with the use of a lawyer, having an unsettled compensation claim, and blaming others (not themselves) for the injury. The strongest predictor of patients' dissatisfaction with their progress since the injury was having an unsettled compensation claim, and as with the other secondary outcomes, patient satisfaction was not significantly associated with injury severity factors.

Factors relating to the compensation process were among the strongest predictors of poor health after major trauma, and were stronger predictors than measures of injury severity. The hypothesis that general physical and mental health would be poorer in patients involved in seeking compensation for their injury was accepted. This study concludes that the processes involved with claiming compensation after major trauma may contribute to poor health outcomes.

The prospective study reported in Chapter Four, the Motor Vehicle Accident Outcome Study (MVAOS), aimed to explore the effect of compensation related factors on general health in patients suffering major fractures after motor vehicle accidents (MVAs). The study hypothesized that general health would be poorer in patients claiming compensation for their injuries.

Patients presenting to 15 hospitals with one or more major fractures (any long bone fracture, or fracture of the pelvis, patella, calcaneus or talus) after a motor vehicle accident were invited to participate in this prospective study. Initial data was obtained from the patient and the treating doctors. Both the patients and treating surgeons were followed up with a final questionnaire at six months post injury. General factors (age, gender, treating hospital, country of birth, presence of chronic illnesses and job satisfaction), injury factors (mechanism of injury, number of fractures, and the presence of any non-orthopaedic injuries), socioeconomic factors (education level, income, and employment status), and compensation-related factors (whether a claim was made, the type of claim, whether a lawyer was used, and who was blamed for the injury) were used as explanatory variables. The primary outcome was general health as measured by the physical and mental component summaries of the SF-36 General Health Survey. The secondary outcomes were neck pain, back pain, and patients' ratings of satisfaction with progress and of recovery. Multiple linear regression was used to develop predictive models for each outcome.

Completed questionnaires were received from 232 (77.1%) of the 301 patients included in the study. Poor physical health at six months was strongly associated with increasing age, having more than one fracture, and using a lawyer, but not with pursuit of a compensation claim. Poor mental health was associated with using a lawyer and decreasing household income.

Increasing neck pain and back pain were both associated with the use of a lawyer and with lower education levels. Higher patient satisfaction and patient-rated recovery were both strongly associated with blaming oneself for the injury, and neither were associated with pursuit of compensation.

Although the use of a lawyer was a strong predictor of the primary outcomes, the pursuit of a compensation claim was not remotely associated with these outcomes, and therefore the study hypothesis was rejected.

The studies reported in this thesis are compared in the final chapter, which concludes that poor health outcomes after injury are consistently and strongly associated with aspects of the compensation process, particularly the pursuit of a compensation claim, involvement of a lawyer, and having an unsettled claim. Compensation systems may be harmful to the patients that these systems were designed to benefit. Identification of the harmful features present in compensation systems my allow modification of these systems to improve patient outcomes.

## ABBREVIATIONS

| | |
|---|---|
| AIS | Abbreviated Injury Scale |
| ABS | Australian Bureau of Statistics |
| ANOVA | Analysis of variance |
| CI | Confidence interval |
| CTP | Compulsory Third Party insurance |
| DF | Degrees of freedom |
| DSM | Diagnostic and Statistical Manual of Mental Disorders |
| HAVS | Hand Arm Vibration Syndrome |
| ISS | Injury Severity Score |
| LEAP | Lower Extremity Assessment Project |
| MMPI | Minnesota Multiphasic Personality Inventory |
| MRI | Magnetic resonance imaging |
| MBA | Motor bike accident |
| MBC | Motor bike crash |
| MCS | Mental component summary (of the SF-36) |
| M-H | Mantel-Haenszel |
| MTOS | Major Trauma Outcome Study |
| MVA | Motor vehicle accident |
| MVC | Motor vehicle crash |
| MVAOS | Motor Vehicle Accident Outcome Study |
| PCS | Physical component summary (of the SF-36) |
| PTSD | Post-traumatic stress disorder |
| RSI | Repetition strain injury |

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|---|---|
| SD | Standard deviation |
| SF-36 | Short-Form 36 General Health Survey |
| TOS | Thoracic outlet syndrome |
| WRAP | Work-related arm pain |

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# CHAPTER ONE. INTRODUCTION AND LITERATURE REVIEW.

## 1.1 Introduction

This thesis aims to examine the association between compensation status and outcome after injury.

In some areas of health care practice, it is commonly believed that patients treated under compensation schemes, such as worker's compensation or third party compensation, have poor outcomes compared to uncompensated patients, and that this association holds regardless of the outcome measure used (such as pain, physical function, or mental well being). The negative association between compensation and outcome after injury has led some to suggest that the compensation system is iatrogenic.[1 2] This stands in contrast with the general aim of compensation systems: to benefit those who have been injured. As the literature review will show, many aspects of this association have been studied, usually with varying results.

Despite the proposal of numerous theories to explain the mechanism of the association, and, alternatively, attempts to explain any association by attributing it to methodological flaws or bias within the studies, the association remains poorly understood. Despite many studies relating to this subject, there is no consensus regarding the presence of, or mechanism for, any effect that compensation may have on health outcomes. This poor understanding is in some part due to the difficulty in obtaining high-level clinical evidence due to the nature of compensation systems.

The quality of research into the association between compensation and outcome has been restricted by several factors. Much of the research is based on medical conditions diagnosed by subjective complaints (for example, whiplash, low back pain, and post-traumatic stress disorder) making the diagnosis less reliable, as the reporting of symptoms (on which the diagnosis is based) may be easily influenced by psychosocial factors. The reported prevalence of these diagnoses often varies significantly between patient groups due to these factors, and also due to poorly defined (or changing) diagnostic criteria.

Study quality is also influenced by the types of studies performed. While many controlled trials have been performed comparing outcomes in compensated to uncompensated patients, studies of this type cannot be randomised. Therefore, confounding may occur due to the observational nature of these studies. Studies of this type also have potential selection bias between the groups of patients, for example, due to greater disease severity in compensated patients. Furthermore, much of the research comparing compensated to uncompensated patients is retrospective, increasing the potential for measurement bias.

The measurement of compensation as an exposure variable makes interpretation of this research difficult, as compensation itself has many forms, such as workers' compensation, litigation, and third party compensation, and may also include non-financial forms of compensation.

This research explores the association between compensation status and health outcome in several ways, each reported in a separate chapter. By clarifying the association between compensation and outcome, it is hoped that the medical management of compensated patients will be improved. It is also possible that this research can guide policy decisions by governments and insurers regarding compensation for injury.

### 1.2 Definitions

Before further discussion regarding the subject of the thesis and its relevance, some definitions are necessary.

In Australia, compensation is defined as the receipt of payment (financial or otherwise) as a consequence of loss[3] (for example, loss of income, a body part, or quality of life). Here, recompense (compensation made, as for loss, injury or wrong)[3] or reparation (the making of amends for wrong or injury done)[3] would be suitable synonyms, although the latter may also be interpreted as "restoration to good condition"[3] which is not always the case in compensation cases.

Compensation then, can be seen simply as 'something good' given to counter 'something bad'. In this regard, when considering illness, compensation is a form of secondary gain: the receipt of benefits as a result of loss (of quality of life, income, physical function etc.). As compensation is usually calculated and provided in financial terms, it is a more objective form of secondary gain compared to other forms such as the social benefits from assuming the sick role (for example, increased attention, or avoidance of obligations or unpleasant activities), which are difficult to measure.

For the purposes of this thesis, compensation will be defined as the receipt of payment ***as a result of*** harm, loss or suffering (to the recipient). For conciseness and brevity, I have included persons claiming compensation (i.e., the ***potential*** receipt of compensation) under the umbrella of 'compensated patients' throughout this work, although the analysis will, in part, attempt to discern any difference in outcome between those who claim compensation, those who are entitled to claim compensation, and those who receive compensation.

Compensation is usually given through workers' compensation (in the case of work injuries), third party compensation (in some motor vehicle collisions), or public liability and personal injury litigation (for example, injuries at shopping centres). Combinations also exist, for example worker's compensation cases are often pursued through the courts, similar to personal injury litigation. In New South Wales (NSW), where the studies in chapters 3 and 4 take place, all motor vehicles are covered with compulsory third party (CTP) insurance through private insurance companies. This insurance covers any person injured in a motor vehicle collision, who is not the driver at fault. The insurance is covered by the CTP insurer of the vehicle at fault. Therefore, drivers at fault (for example, single vehicle collisions) are not covered by CTP insurance. This system differs from systems elsewhere (for example, Victoria and New Zealand) where all persons involved in motor vehicle accidents are covered by insurance, regardless of fault (so-called ‘no fault’ insurance). It is this difference that allows this research to be done in NSW, i.e. it allows a comparison of compensated and uncompensated patients who have been subjected to similar injuries (motor vehicle collisions).

There will be some overlap between the various compensated groups listed in the previous paragraph and, unless otherwise specified, they will be treated as one group referred to as ‘compensated patients’.

Injury is defined as any physical damage to the body caused by violence or accident.[3] In the literature regarding compensation, however, the definition of injury is not clear. In many cases the energy transferred to the patient may be mild or may not cause identifiable physical damage but because an event occurred and a complaint (often pain) was reported after the event, an injury is assumed. Similarly, many work related ‘injuries’ are often not associated with any particular violent episode. Therefore, in the literature review, the definition of injury will vary between articles.

The broader definition of injury to include “harm of any kind” or a “wrong or injustice done or suffered’[3] will not be used, as any non-physical definition will be contentious. In fact, the vague definition of both the injury and the diagnosis in many studies regarding compensation make interpretation difficult. This research aims to avoid this criticism by using an inclusion threshold: a bone fracture in the prospective study (Chapter Four), and an Injury Severity Score (ISS) threshold in the retrospective study (Chapter Three).

Unfortunately, many labels have been given to the association between compensation and poor outcome, although not always implicating compensation per se. These labels have been used over centuries to explain conditions of unexplained illness after injury and, often, the label of one condition changes over time due the changes in theories and beliefs regarding the underlying mechanism. These terms include post traumatic neurosis, accident neurosis, post accident anxiety syndrome, litigation neurosis, occupational neurosis, compensation neurosis, and compensation hysteria.[4]

The current term “post traumatic stress disorder” (PTSD), which has been listed in the Diagnostic and Statistical Manual of Mental Disorders[5] (DSM) since 1980, is related to these earlier conditions and is an example of a condition with changing definitions. The definition does not refer to compensation but may be diagnosed in patients claiming compensation, and may in fact be the basis of a claim for compensation. The definition of PTSD relates the condition to “confrontation with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others”.[5] The condition may be over-diagnosed, particularly since the definition of the traumatic event was expanded in the current edition of the DSM (DSM-IV) to include any event which is distressing to the patient, including events involving other persons, or news of an event, rather than events which would be distressing to almost anyone.[5-7] Furthermore, the diagnosis is based on subjective reports from the patients and therefore cannot be expected to be perfectly consistent.

The terms covered in the previous two paragraphs will be used as they are provided in the literature under discussion but will not be further defined, as they relate to theories of aetiology, which are not the subject of this thesis. The definition of conditions as physical, psychogenic, neurophysiologic, behavioural or hysterical is variable and is confused by changing and imprecise terminology. It is not the purpose of this thesis to clarify the debate over the possible mechanism of compensation related illness. Rather, the intention is to explore the history, and the strength and validity of any association. However, a detailed examination of the subject is not possible without some discussion of the theories regarding aetiology and this can be found later in this chapter.

The outcomes used in this research will be clearly defined in the methods section of each chapter.

### 1.3 Professional Background

I am an orthopaedic trauma surgeon in both public and private practice and I was trained, and continue to work, in the Sydney metropolitan area.

As a medical student between 1980 and 1984, with clinical attachments at teaching hospitals in Sydney, I was not made aware of any association between compensation and outcome beyond that of any layperson. My awareness of the problem was derived mainly from the media, which indicated that some patients feigned illness in order to receive financial compensation, that back pain and neck pain were the usual conditions feigned, and that these conditions were a major health problem and a diagnostic dilemma.

I had very little professional involvement with compensated patients during my resident years, consequently, I did not become interested in exploring the association between compensation and outcome until after I had completed advanced surgical training and entered private practice as an orthopaedic surgeon in 1997. Although I was aware of colleagues' opinions regarding exaggerated claims and poor surgical outcomes in compensated patients, I did not realise until I treated my own patients, that these patients not only appear to have genuinely poor outcomes, they often have symptoms of depression and anxiety. That the knowledge of this phenomenon was widespread is demonstrated by the fact that many successful orthopaedic surgeons and psychiatrists refuse to see any compensated patients, despite the fact that treating these patients can be financially rewarding (in NSW, doctors are paid on a different scale for workers compensation cases, outside the publicly-funded Medicare system and private health insurance system). Research into the beliefs and attitudes of orthopaedic surgeons has revealed that many surgeons feel that workers' compensation is a negative predictor for outcome, even for routine operations like anterior cruciate ligament reconstruction.[8]

My later knowledge of epidemiology, gained through my enrolment in a Master of Medicine in Clinical Epidemiology at Sydney University, allowed me to better analyse the current literature on compensation and surgery. This resulted in my desire to formally examine the relationship between compensation and outcome using epidemiological methods.

### 1.4 Problem Statement

Compensation payments make up a significant financial burden on society. In Australia, workers compensation is compulsory, and the mean premium varies between 2% and 4.5% of gross wages. In NSW alone, the total premiums collected for workers compensation and compulsory third party insurance are $2.6 billion and $1.4 billion, respectively.[9 10]

The high cost of compensation insurance has led to restructuring of compensation administration in many parts of the world such as Canada, New Zealand and Victoria. In New South Wales, the legislation regarding workers compensation and third party motor vehicle insurance has been modified due to the high costs of maintaining these systems, as well as concerns regarding the relative distribution of compensation payments for the injured, and for administrative, legal, and medical costs.[11 12]

Despite concerns regarding the costs involved in compensation systems, there is little evidence that these systems are associated with improved health for those involved, compared to patients who may be similarly injured but not eligible to access compensation. In fact, as the literature review will show, patients treated under compensation systems often have worse health outcomes than comparable non-compensated patients, despite the increased input into treatment and rehabilitation in compensated patients.

The possible negative effects have been raised in the literature, and addressed in detail in the three editions of Occupational Musculoskeletal Disorders,[13] by Nortin Hadler, a rheumatologist and Professor of Medicine in North Carolina. Locally, the problem was addressed in a review jointly authored by the Australasian Faculty of Occupational Medicine and the Royal Australasian College of Physicians entitled Compensable Injuries and Health Outcomes.[14] These publications, along with the many articles referred to by them will be discussed in the literature review but the conclusions of these authors is that there is a real and considerable negative effect of compensation on health, that research regarding the mechanisms of any association is “fragmentary and inconclusive”,[14] but that psychosocial factors are important. The Australian review concludes that further research is required regarding the impacts on health outcomes.

As the following literature review will show, compensation has been associated with poor health outcomes after treatment, with the development of particular conditions, and with poor general health regardless of specific diagnoses. All of these aspects of the association between compensation and health, however, have been challenged and the literature on this subject is inconsistent.

The problem to be addressed in this thesis is the negative association between the receipt or potential receipt of compensation and general health after injury. If present, this may represent a considerable financial problem for government and public compensation schemes, and a considerable health problem for patients covered and treated under such systems.

The literature relevant to this topic is covered in the following section, and the specific aims of this thesis follow that review.

### 1.5 Literature Review

#### 1.5.1 Historical Perspective

The negative effect of compensation (or, more loosely, any secondary gain) on outcome after injury is not a recent phenomenon. Numerous historical accounts exist, and specific conditions which demonstrate this association are discussed in more detail later in this chapter.

There is some common ground between the effect of compensation on outcome and the effect of occupation on outcome, particularly since the introduction of workers' compensation. One of the earliest recordings of how outcome may be influenced by occupation (rather than compensation) is attributed to Dupuytren, who was quoted in a thesis published in 1836.[15] He compares the outcome of soldiers injured in battle to labourers, farmers or artisans: the latter having "a profound sorrow, a dark hopelessness".[16]

Paradoxically, although others, like Dupuytren, have found that soldiers injured in battle have been shown to have less pain and suffering compared to civilians with similar injuries,[16 17] it is in soldiers that the earliest reports of illness related to compensation are found. The term "shell-shock" from World War I refers to illness related to conditions of battle. Similar reports from earlier conflicts can be found. In the American Civil War, this condition was referred to as "soldier's heart" or "neurasthenia",[18] the latter being a term used in the 19$^{th}$ Century to indicate exhaustion of the nervous system.[19] The condition was initially attributed to physical injury, usually on some part of the nervous or cardiovascular system, caused by the physical effects of gunfire or gas.[20] Lack of physical evidence, and possibly the concomitant rise of psychiatry and psychology, led to these war-related conditions being labelled as psychogenic in nature after World War I.

In World War II, a similar condition was seen, although this time labelled as "battle fatigue"[20] or "battle neurosis".[19] It was in World War II that it became clear that the development of this condition was not related to the degree of exposure to the conditions of battle, and that many soldiers diagnosed with battle fatigue were uninjured or had not been exposed to gunfire. Of further interest was the fact that the incidence varied with such factors as unit morale, effectiveness of leadership, and secondary gain.[21] The role of secondary gain was widely accepted[19] but, as is the case today, the form of secondary gain often varied, and included not only financial gain but also non-financial gains from assumption of the sick role. In another similarity with current debates, it was not known whether secondary gain actually caused the condition, or only prolonged or exacerbated an underlying condition.

The condition of battle fatigue was considered a form of post-traumatic neurosis but it was not until after the Vietnam War that the condition was labelled post-traumatic stress disorder. Since 1980, post-traumatic stress disorder has been given the legitimacy afforded by the Diagnostic and Statistical Manual of Mental Disorders (DSM), although, like the diagnoses it replaced, its legitimacy has been questioned.[22]

Historically, many other forms of post-traumatic neuroses have been reported, often associated with secondary gain. Probably the earliest widely reported form of post-traumatic neurosis related to compensation was “railway spine”. This was the name given to the widespread finding of chronic back pain and disability associated with railway injuries in the mid and late nineteenth century, mainly in England.

Railway spine was characterised by a variety of physical disorders attributed to a railway accident, in patients with no significant organic injury. The symptoms varied and included back pain, limb pain, headache, fatigue, dizziness, memory loss, and sensory changes and weakness in the limbs. At the time, the mechanism for the condition was still thought to be organic, but without identification of an organic process, theories developed which matched the thinking of the time. The condition was thought to be a form of neurasthenia: irritation of the nervous system secondary to the physical shock of the accident. Various terms were used for this such as spinal concussion, traumatic neurosis and nervous shock.[19]

In the nineteenth century, neurosis, neurasthenia and nervous shock were seen as physical disorders of the nervous system, separate to mental or emotional shock. Although the mechanism was thought to be through the physical impact of the collision, some element of exaggeration was suspected in claims for railway spine.[23] Later, near the end of the nineteenth century, psychological theories were developed for conditions such as railway spine and shell shock.

Whatever the mechanism behind railway spine, the association with compensation was indisputable. By validating the physical nature of the disease (albeit with theories only), the medical community smoothed the way for sufferers to successfully sue the railway companies and by the 1860's the railway companies were paying out large sums in compensation for this condition and were losing almost every personal injury case that went to court.[24 25] The difficulty with assessment of the cases (due to lack of physical evidence of injury) was noted at the time and, interestingly, a proposal for an independent review panel, consisting of physicians, surgeons and a legal advisor was suggested, similar to solutions proposed and implemented for compensation cases today.[23] When the theories regarding aetiology of the condition turned towards psychological factors rather than physical, claims became harder to support and the reporting of railway spine subsided.

Similar conditions, which pre-date railway spine, are less well documented. Steel nib syndrome was the earliest recorded epidemic of writer's cramp and was reported as occurring among male clerks of the English Civil Service in the 1830s. It was attributed to the introduction of new technology (the steel nib). Like other traumatic neuroses, regional variations in incidence occurred, and a relationship between use (or overuse) of the limb, and symptoms, could not be established. Reynolds, in 1869 noted that symptoms of steel nib syndrome could be developed by close contact with other cases and that the development was related to aspects of the will and described the "secondary influence the brain has over the relations established in the body". Similarly, Gower noted in his monograph ***Diseases of the nervous system***, that writer's cramp “is a disease easily imagined by those who have witnessed the disorder”.[26]

Steel nib syndrome was, however, only one form of a disorder termed writer’s cramp (or scrivener’s palsy). Other forms occurred, often named after the occupation in which the condition was seen, for example hammerman’s palsy, milker’s cramp and tailor’s cramp. Like steel nib syndrome, telegraphist’s cramp was blamed, by some, on new technology: the introduction of the telegraph (and accompanying Morse code key) in the late nineteenth century.[27 28]

Any connection between these early occupational upper limb disorders and compensation, however, is not well documented, as many of these conditions pre-dated the introduction of the workers’ compensation system. The conditions were, however, used as bargaining tools in negotiations with employers and governments. In Britain, workers successfully lobbied the Industrial Diseases Committee to have telegraphist’s cramp listed on the schedule of compensable diseases in 1908, so that it would be covered by recently introduced workers’ compensation. There is evidence that the provision of compensation increased the incidence (or at least the reporting) of this condition. By 1911, telegraphist’s cramp was epidemic in Britain, and attempts to control it using regular rest breaks or different machines were largely ineffective.[27] The study of this condition was interrupted by the development of wireless communication and the telephone but it showed similarities with previous work-related upper limb conditions, and those that followed.

One of the earliest work-related conditions with detailed reporting and a strong link to compensation was condition known as miner's nystagmus. Miner's nystagmus was another example of a condition with few physical signs (even nystagmus was not necessary for the diagnosis), and which relied on patients' symptoms for diagnosis. Like shell shock and railway spine, it involved many symptoms, including headache and fatigue. It was initially thought to be due to poor lighting conditions in coal mines. Present from the 1890's to the early twentieth century, it was clearly used as a bargaining tool between workers and their employers, and compensation and secondary gain were implicit in the negotiations.[29]

Although it can be argued that these conditions were associated with secondary gain through their use as negotiating tools, the clearest evidence of (financial) secondary gain in these conditions was usually from one of two sources: litigation through the court system (such as in railway spine), or benefits through the worker's compensation system (such as in miner's nystagmus).

Worker's compensation dates back to the second half of the nineteenth century when the introduction of workmen's accident insurance spread across Europe from Germany.[30] It was not adopted in the United States (state by state) until the turn of the century, and only under the condition that the employers would be exempt from litigation under common law, and that costs could be passed on to the consumer. Worker's compensation was introduced in New South Wales in 1926 through the Workers Compensation Act.

More recently, Repetitive Strain Injury (RSI), a condition that bears similarities to the upper extremity occupational disorders already discussed, has been extensively studied and shown to have a strong connection with the workers compensation system.[31-44] It is of particular interest due to its high prevalence in Australia.

The incidence of RSI rose rapidly in the mid 1980's, particularly in Canberra and Sydney.[31 38] The condition was diagnosed through subjective complaints from the patients, as there were no tests available and no discernable underlying pathology, despite some theories regarding inflammation and nerve injury. At the time, it was attributed to the introduction of the computer keyboard, which replaced the typewriter in the 1980s. As computers allowed faster keystrokes than a typewriter, it was felt that the increased typing speed caused an injury to the structures in the hand and wrist.[31]

It is interesting to note the similarity between RSI and writer's cramp in that they appeared to be transmitted by line-of-sight. That is, the incidence of both diseases was largely confined to groups of workers, usually in one building or company, which led to theories relating to hysteria,[31] employee-employer relations[28] and malingering.[41]

Like many other conditions associated with compensation such as those already discussed, or whiplash, which will be discussed in detail later, physical theories have been put forward, but none were proven or widely accepted. Like these other conditions again, though, RSI was thought to be associated with many psychosocial factors. Tertiary gain by health professionals, including doctors, physiotherapists and occupational therapists, all of whom stood to gain from having RSI established as a medical, and work-related condition, was thought to be a contributing factor. The union movement concern (which resulted in workplace lectures and publications such as “The sufferer’s handbook”) and misinformation and exposure in the media were also thought to contribute.[28] As an example, one alarmist media headline at the time read: “Hi-tech epidemic. Victims of a bright new technology that maims”,[45] another read: “A crippling new epidemic in industry”,[46] and still others used words such as “torture”, “plague”, and “kills”.[39]

Although RSI reached epidemic proportions in the 1980s in Sydney and Canberra, compensation for the condition was eventually denied due to the lack of physical evidence of a disease process, combined with epidemiological evidence of the condition occurring in distinct clusters, and being unrelated to workplace conditions such as the typing speed or the number of keystrokes used.[32 39] The strength of the association with compensation is best demonstrated by the rapid decline in the incidence of the condition after claims for compensation were rejected.[28]

The relationship between compensation and illness is not new, and this historical review provides information regarding common features of illnesses that have been associated with compensation apart from the presence of secondary gain: the lack of a physical basis to support the diagnosis, and the geographic clustering of cases.

The next section covers the literature relating to specific conditions, and this is followed by a review of the literature pertaining to the effect of compensation on health in general and the possible mechanisms by which compensation may affect aspects of health.

### 1.5.2 Specific Conditions

#### 1.5.2.1 Chronic Pain

As in the past, compensation continues to be associated with many conditions. The association between compensation and chronic pain has been studied at length. Like the other conditions previously discussed, pain is purely subjective: it is not possible to confirm the diagnosis by physical examination or tests. Although much has been written on the effect of compensation on chronic pain, the nature of the association is not well understood as much of the evidence is conflicting. While there is some evidence that pain reporting is higher, and that symptoms are prolonged in compensated patients, evidence to the contrary also exists. The study methodology in these studies is poor, with significant variation in the populations and outcome tools used.

Although it is interesting to note that the majority of referrals to chronic pain clinics have pending disability claims,[47] it is difficult to determine the significance of this. It may be, particularly in the Australian system where pain clinics are often privately run, that patients who are covered with compensation are more likely to be able to afford treatment in a pain clinic. Also, pursuit of treatment in a pain clinic does not necessarily correlate with pain severity; there are many other factors that may influence referral to a pain clinic, such as pressure from an employer or an insurance company.

Mendelson, in several reviews of chronic pain and compensation[4 48 49] concluded that there was little evidence to show a difference in pain reporting (of severity or distress), pain behaviour or psychological disturbance in compensated patients. He did find, however, that compensation had an adverse effect on treatment response and that the prevalence of chronic pain complaints was positively related to compensation systems that provided pain contingent benefits. Many of these studies have considerable methodological deficiencies and represent a heterogeneous group, with different causes for pain, different treatments provided, and different outcomes measured.

Conflicting results regarding the role of compensation in chronic pain patients were also noted by Dworkin.[50 51] In Dworkin's study, a poor treatment effect was noted in compensated patients with chronic pain, but this difference was explained, in a multivariate analysis, by the high rate of unemployment (at the time of presentation) in compensated patients. Dworkin's argument is that patients who have returned to work are more likely to improve with treatment, and that the association between compensation and poor treatment response is because many of the compensated patients had not returned to work. However it is not possible to discern whether the opposite is true: that compensated patients have poor treatment outcomes, and therefore are less likely to return to work.

In a study by Talo et al,[52] workers compensation patients were significantly less likely to improve with multidisciplinary management of chronic pain than patients with other forms of compensation. Whether the claim was active or settled made no difference to the outcome, except in the workers compensation cohort where it was noted that patients with active litigation were much less likely to return to work. This latter finding is difficult to interpret, as workers compensation cases do not usually settle until after the patient has returned to work.

Two current chronic pain conditions with strong links to compensation are low back pain and neck pain (usually labelled as whiplash). Both are common conditions, often present in a medico-legal context through workers compensation or accident claims. Back pain is said to be the most common medical condition seen by medical practitioners and the most common cause for sick leave and workers compensation claims.[30 53-59]

#### 1.5.2.2 Low Back Pain

The literature regarding low back pain is difficult to summarise for the same reason as many other compensation related conditions in which the diagnosis is subjective: there is no clear diagnostic test, and this results in considerable variations in important factors such as incidence and time to recovery. Yet despite the lack of a clear diagnosis (apart from the reporting of pain in the lower back) the condition is the most common cause of work-related illness claims, and account for the greatest proportion of costs related to workers compensation.[55 60-65]

Low back pain has been associated with many factors unrelated to the physical condition or age of the patient. Considerable differences in the prevalence of back pain exist, for example, between countries, and between regions within countries.[66] More specifically, incidence of, and recovery after back pain has been associated with non-physical factors such as job satisfaction, social class, employment grade, education, job security, litigation and workers compensation.[53 57-59 67-79]

Recovery from back pain has also been shown to be adversely affected by compensation.[68 71 80-82] Response to treatment in chronic low back pain patients has also been shown to be adversely affected by pending disability claims.[83 84]

Clearly, the large number of variables that may influence back pain in the work place makes the assessment of any one factor difficult due to the large number of potential confounders that must be considered. For this reason, the methodology of much of the research into psychosocial factors and back pain has been criticised.[78]

In a large prospective cohort study from Canada, which followed 6,571 patients over 3 years, multivariate analysis of physical and psychological factors found that the risk of developing back pain was only associated with psychological stress, and that other factors such as working hours, job satisfaction, physical exertion, race, marital status, income, education or occupational class were not significant.[85] This was despite some factors such as physical exertion being positively associated with the development of back pain in the crude analysis. This study highlights the problem of the "back injury" model which is accepted by most practitioners, workers and insurers. For only with this injury model can back pain in the workplace be compensated. Studies of back pain in workers often do not find a correlation with physical activity, and most patients do not have physical evidence of acute injury. This makes any study of work-related back pain spurious, as there is often no clearly defined diagnosis, only pain. As this is a subjective diagnosis, it is open to influence from non-physical (psychosocial) factors. This is relevant to this thesis, as compensation related back pain is often work-related.

The majority of the literature regarding the association between compensation and back pain, however, relates to post-operative outcome, and is covered extensively in the systematic review in Chapter Two.

**1.5.2.3 Whiplash**

The term whiplash refers to neck pain, usually associated with a traumatic event. Most cases of whiplash are attributed to a motor vehicle injury.[56] Often, physical signs of underlying pathology are not present, and there is little evidence for an underlying physical abnormality.[56 86 87]

Neck pain is the most frequent injury sustained by motor vehicle occupants in the USA,[88] is the most common complaint after motor vehicle injury in NSW,[89 90] and accounts for over half of all injury claims in Queensland.[91] Claims for whiplash injury cost billions of dollars each year in developed countries like Australia, the United Kingdom, and the USA.[92-94]

A possible association between whiplash and compensation has been shown in recent studies from Lithuania.[95 96] The authors, neurologists from Lithuania and Norway, interviewed 202 subjects who had been involved in motor vehicle accidents 1 – 3 years previously based on records from the traffic police department. The incidence of neck pain, headache and other symptoms were compared with 202 uninjured, age and sex-matched controls chosen at random from population registers. The study found no significant difference in chronic symptoms between the two groups. Chronic neck pain and chronic headache occurred in 8.4% and 9.4% (respectively) of accident victims and 6.9% and 5.9% of controls.[95] The possible connection with compensation was that neck pain is not compensated in Lithuania, indicating that chronic neck pain after injury may not occur where compensation does not exist. However, other explanations exist: the authors suggest that the low incidence of chronic neck pain in Lithuania may be due to local cultural factors such as the lack of symptom expectation,[97] i.e. chronic neck pain after injury is not known in Lithuania and therefore, is not expected. This is supported by studies from Greece and Germany, which show no significant difference in long term symptoms after whiplash injuries (acute neck pain after motor vehicle accident) compared to uninjured controls, despite the availability of compensation through the courts and through third party insurance.[98 99] The community expectations in these areas are that whiplash does not cause long term neck symptoms, because long term neck symptoms are not common.

The Lithuania study stood in contrast to numerous studies from Scandinavia, the United Kingdom, Canada, the United States, and Australia which showed high rates of chronic neck pain after whiplash injury, ranging from 18% to 40%.[100-108] Consequently, it generated considerable debate and was also criticised for its lack of statistical power to detect a small difference.[109-112] However, as some studies have shown that up to 47% of car occupants report whiplash symptoms after an accident,[113] it would be reasonable to expect a large difference in symptoms between those who were involved in an accident and those who were not so involved.

To address criticisms, the same authors repeated the Lithuania study using a prospective controlled cohort design, with 210 accident victims and 210 controls.[96] Initial neck pain was reported in 47% of accident victims (similar to other studies), but after one year, there was no significant difference in neck pain between the accident victims (4%) and the control group (6.2%). Similar findings were noted for other symptoms such as headache and reduced neck mobility (with slightly higher rates in the control group). The study used detailed questionnaires and the follow-up rates were 95% and 92% for the two groups after one year. Again, the main concern regarding methodology was the statistical power: the study had 80% power to detect a difference in neck pain of 9%. The low incidence of chronic neck pain lowered the power of the study, but the fact that it is lower than expected, and lower than that found in previous studies, is evidence itself that chronic neck pain after injury is uncommon in this environment. The significance of this study is increased when the supporting data from the previous study is considered, as well as the lack of studies to the contrary, as previous studies have not included a population control group.

Although it is reasonable to conclude that the lack of chronic symptoms after neck injury is due to the lack of available secondary gain from compensation, there are clearly other differences between Lithuania and European and North American countries, which report a higher incidence of chronic neck pain after road traffic accidents. Other regional differences have been noted in studies of whiplash comparing incidence between countries.[101 114] Although the difference may be due to cultural differences or differences in the legal and compensation systems, conclusions are difficult to make in these cross-sectional studies.

The role of compensation in whiplash has been examined in two systematic reviews, with conflicting conclusions. The first, by Cote et al[115] updated the previous review conducted by the Quebec Task Force on Whiplash-Associated Disorders[86] and concluded that insurance and compensation systems have a large impact on recovery from acute whiplash injuries. In contrast, Scholten-Peeters et al,[116] in their systematic review, concluded that compensation did not seem to be of predictive value. These conflicting conclusions necessitate an examination of these two systematic reviews.

The review by Cote provided strict a priori criteria for inclusion and exclusion of studies in the review, based on internal and external validity, whereas the review by Scholten-Peeters did not. The latter review analysed seven articles that contained an analysis of the effect of litigation on outcome, and found that only one article had a negative association, defined as a relative risk of over 2 or less than 0.5, or significance (p value) of $< 0.05$. The study that showed a negative association,[117] of which Cote is a co-author, is the only study that was specifically designed to examine the effect of legal and compensation factors on outcome, and is the only study used to support the conclusion in the review by Cote.

This study by Cassidy et al[117], reported in the New England Journal of Medicine in 2000, compares recovery from whiplash before and after the introduction of “no-fault” legislation in Saskatchewan, Canada, which significantly reduced the ability to sue for pain and suffering. It shows a significant decrease in the number of claims, and the time to case closure, with the introduction of the new legislation. This study has been criticised for using time to case closure as a surrogate outcome for recovery,[118-121] but the use of this outcome has been justified by the authors as they were able to show a correlation between early case closure and decreased pain, decreased depressive symptoms, and improved physical functioning. Of note, the same authors showed similar findings when they applied the same methodology to study chronic low back pain after motor vehicle accidents before and after the legislative change.[68]

In a similar before-and-after study to that of Cassidy, Reilly showed a 68% reduction in the number of whiplash claims over a three year period, after a change in legislation in Victoria in 1987 which included a provision for the patients to pay the first $317 in medical expenses for any whiplash claim.[122] At the same time, the number of claims made under workers compensation, which was unaffected by the legislation, continued to rise. These findings were similar to figures from the New South Wales Motor Accidents Scheme, in which a significant reduction in the number and cost of claims for whiplash was seen after introduction of legislation to reduce the level of non-economic loss (pain and suffering) payments for minor injury claims.[90]

Care should be taken, however, in using claim statistics as epidemiological data: adjusting the threshold for a claim would be expected to decrease the claim rate, but may not affect the underlying incidence.[100 123] To overcome this problem, a clinical outcome study was performed in NSW, using the before-and-after design, on patients either side of the legislative change described above. Validated region-specific and general health scores were measured, and significant improvements in physical functioning were noted after the legislative change.[124]

The six studies in the systematic review by Scholten-Peeters that do not support the association between compensation and outcome provide very little evidence to support their conclusions. They are limited by low numbers and low power,[87 103 125] low follow-up (27% in one study[126]), lack of statistical analysis,[105 127 128] and outcomes apart from comparative rates of neck pain (such as work capacity,[125] short-term improvement after physiotherapy,[87] or improvement after settlement[129]).

The psychiatric outcome after whiplash injury has been reported in many articles by Mayou et al.[105 106 127 128 130-136] They measured outcomes one, three and five years after motor vehicle collision in a consecutive cohort of 1441 patients presenting to one emergency department. No statistical analysis of the effect on compensation is provided in these studies. However, the authors conclude that there is a trend towards a poor outcome in claimants, attributed to the stress of the compensation proceedings, as a cause of stress. They also state that these patients may have more serious physical problems. In their analysis of injury severity and outcome,[130] however, they conclude that the claim rate was not related to the injury severity score.

More recently, Joslin et al[93] have examined the association between compensation and outcome after whiplash injury and concluded that the long-term (mean follow-up of 3.5 years) functional recovery after neck injury was not related to the physical injury, but was associated with litigation.

The diagnosis of neck pain as a result of injury (whiplash) is less clear when studies do not take into account the background prevalence of neck pain in the community. It should be noted that the incidence of chronic neck pain in studies of whiplash varies from 0 – 66%,[123 125 137 138] and the background prevalence of neck pain in the community ranges from 10 to 48%.[102 123 125 137 139-145] Interestingly, Kasch et al have shown that frequent questioning regarding neck pain symptoms increases the likelihood of reporting neck pain, which may account for the increased rate in whiplash studies with multiple follow-up points.[125]

The high rate of chronic neck pain in the community, which may match the rate after motor vehicle collisions, raises the possibility of symptom misattribution whereby patients who feel neck pain may attribute their symptoms to a motor vehicle collision either because of secondary gain or because it appears more logical to them. This would be similar to the patient with a naturally occurring orthopaedic condition such as a bone tumour or osteoarthritis presenting with symptoms that they attribute to a recent fall or minor injury. This also relates to symptom expectation, which has been put forward as a possible cause for the findings in the Lithuania studies: as whiplash is not common in this country, most patients would not expect to attribute neck pain to the motor vehicle collision.[97]

The term 'whiplash' itself may contribute to the problem, as the use of the term in patients who have suffered minor neck pains after injury may lead to symptom expectation. Some authors have suggested that the indiscriminate use of the term may become a self-fulfilling prophecy.[146-148]

Many physical theories have been put forward to explain pain after whiplash, with anatomic abnormalities ranging from the cervical discs, joints[149] and spinal cord[150] to "atypical carpal tunnel syndrome".[151] To date, no theory based on physical damage has been widely accepted as a cause of pain in whiplash.[152] It appears unlikely that a single physical cause for whiplash will be found, given the lack of evidence of physical damage and the strength of evidence correlating outcome with psychosocial factors such as psychological or psychiatric illness,[127 153-157] financial compensation,[50 86 123 129] or legislative factors, as discussed above. Also, the association between whiplash and other, more general, complaints, such as low back pain, fatigue, headache, sleep disturbances and general ill health,[158 159] would appear to make local neck pathology an unlikely cause.

The difficulties found when attempting to attribute neck pain (or back pain) to a motor vehicle collision are amplified when attempting to associate neck or back pain with work-related "injuries". Work-related injuries are usually less well defined, and rarely isolated to a single violent event,[160] as is the case with motor vehicle accidents. Associations between work activities and neck and back pain are difficult to establish and inconsistent. They are also confounded, and out-weighed, by other associations such as job satisfaction, job security, decision latitude and income.[73 75 76 161-163] Many of these studies show that these psycho-social factors are stronger predictors of neck pain (and upper extremity pain) than the physical parameters of the occupation, and similar findings have been reported for low back pain.[73 77 78]

#### 1.5.2.4 Work-related arm pain

In a recent large review of 3888 patients presenting to a US hand clinic over a 12 month period, 496 (12.8%) were diagnosed with idiopathic arm pain (pain with few objective findings and no clear diagnosis).[164] Although the authors attribute the diagnosis to the strong influence of psychological and sociological factors, specific factors were not measured or correlated.
There are no studies that directly address the role of compensation in idiopathic arm pain, but the issue off compensation has been raised in several work-related upper limb conditions, which will be discussed in this section.

Work-related arm pain (WRAP) covers a number of possible diagnoses, only some of which have a well-defined pathological basis. It would include repetitive strain injury (RSI), which has been covered in the section on historical perspective, as the term is no longer used.
Winspur, in a paper that offers an opinion on the high incidence of work-related arm pain in Britain, attributes it to the acceptance of work-related arm

pain by the courts in Britain as a compensable injury, and contrasts this to the situation in Australia (post-RSI) and the US.

Carpal tunnel syndrome is a common condition that is included as a cause of WRAP. However, despite numerous reports and reviews that have drawn attention to the lack of evidence for carpal tunnel being work-related,[13 165-168] and evidence that outcomes are significantly worse in patients who are treated under workers compensation[169-175] or who have contested claims,[176-178] it continues to be subject to workers compensation claims and ranks second behind back pain as a cause for lost work time in the US.[179]

Another work-related upper limb condition, Hand-Arm Vibration Syndrome (HAVS) is an industrial injury affecting one of the largest groups of workers claiming compensation in the world.[180] Like many of the other conditions mentioned, it has clear diagnostic criteria but relies almost completely on subjective patient reporting for diagnosis, and attempts to develop reliable tests to diagnose this condition, such as the cold provocation test, have failed.[180 181]

In a review of the literature regarding work risks for upper limb pain, Bongers et al concluded that perceived high job stress was consistently associated with all upper extremity problems, and that the association between upper limb pain and high job demands was not significant.[182] The lack of evidence for this and other proposed causes for WRAP is well summarised in Hadler's "Occupational Musculoskeletal Disorders".[13]

#### 1.5.2.5 Head Injury

Recovery after head injury has also been associated with compensation status. In a meta-analysis of 17 studies and 2,353 patients examining the effects of financial incentives on recovery after closed head injury, Binder and Rohling[183] found significantly increased symptoms and disability after head injury in compensated patients. Interestingly, they predicted (and found) that compensated patients were more likely to have less severe initial injuries, were more likely to have late-onset symptoms, and were less likely to have an organic aetiology.

The analysis included three studies that found no difference but did not have sufficient data for inclusion in the meta-analysis, and assigned these studies an effect size of zero, whereas it excluded studies that reported an association but did not have sufficient data for analysis. This would have artificially decreased the overall effect size. The effect size in this analysis would also have been biased towards the null by misclassification in the seven studies examining patients involved in litigation, compared to those who are not. Misclassification may have occurred in that non-litigating patients were classified as patients without financial incentives, but non-litigants may have other, unrecorded, financial incentives such as workers compensation.

#### 1.5.2.6 Post-traumatic stress disorder

Post-traumatic stress disorder (PTSD) was only recognised as a diagnosis after the Vietnam War, where it was commonly diagnosed in military personnel. It continues to be diagnosed in a military setting, a setting where the diagnosis is associated with entitlement for compensation. In the US, the incidence of PTSD in military veterans is rising. The number of veterans awarded compensation for PTSD increased from 120,000 in 1999 to 216,000 in 2004. Costs for PTSD rose from US$1.7 billion to US$4.3 billion over the same period.[184]

Within the Veteran's Affairs organisation in the US, research has been done to determine any effect of compensation on the diagnosis or reporting of symptoms (which, in turn, influences the diagnosis). As with other conditions, reports of symptom exaggeration in those seeking compensation[185] are mixed with reports concluding that there is no difference in symptom reporting.[186] Like the conditions discussed above, difficulties with classification exist within studies of PTSD because the diagnosis is dependent on symptom reporting, and the compensation system is complex, and all veterans are potentially eligible for compensation so any distinction between those currently seeking compensation and those not seeking compensation may not be meaningful.

The increase in incidence over the years may not be due to changes in symptom reporting, but rather due to changes in the diagnostic criteria for PTSD. Concern has been raised regarding the change in diagnostic criteria for PTSD in the latest (fourth) edition of the DSM as it has become more subjective.[6] Whereas previously the definition of PTSD relied on a traumatic event which is “out of the ordinary” and would be “markedly distressing to almost everyone” (i.e. referenced to the population norm), the current definition requires exposure to a stressor which elicits a response of “intense fear, helplessness or horror” from the subject (i.e. referenced to the patient, or subjective).[5]

The diagnosis of PTSD has been questioned by Summerfield[7] who argues that it is an entity that has been constructed as much from sociopolitical ideas than psychiatric ones, and that PTSD may be another example of the medical community labelling (or medicalising) normal human distress and suffering.

The subjectivity of PTSD is also demonstrated in the way physician expectations influence the outcome in PTSD patients seeking compensation. In a study by Sayer and Thuras,[187] clinicians treating veterans for PTSD were found to have a more negative view of the likely treatment outcome for patients who were seeking compensation. This difference, however, may be based on valid experience, or may be part of a self-fulfilling prophecy regarding the poor outcomes for compensation-seeking patients. They also found that the negative perceptions towards the compensation-seeking group increased with increased exposure to the patients. Interestingly, the treating physicians in this study also had more negative perceptions towards compensation-seeking patients, compared to patients who were already receiving permanent compensation, even though the latter group were more severely affected, indicating that the pursuit of compensation is seen as the negative factor, not receipt of compensation.

In the general community, PTSD is one of the most prevalent categories of mental illness[188] and motor vehicle accidents are the single largest civilian cause of PTSD.[189 190]

The role of compensation in PTSD following motor vehicle accidents has been studied, with varying results. Mayou et al, in a prospective study of over one thousand consecutive patients presenting to an Oxford Emergency Department after a motor vehicle accident, found that chronic PTSD at one year[191] and 3 years[192] was associated with pursuit of litigation when allowing for other variables. This supported previous smaller studies by Blanchard et al, which showed that litigation was an independent predictor of PTSD at 4 months[193] and 1 year[190] after presenting for treatment after a motor vehicle accident.

In a study from New South Wales that argues against the influence of litigation on PTSD, Bryant and Harvey base their conclusion on their finding that settlement of compensation did not influence PTSD symptoms.[194] This reasoning is common in the literature: that if litigation or compensation influences illness, then settlement (of the litigation or compensation) should have the opposite effect on the illness. This implies that the illness is a conscious process and is a false logic: removal of a stimulus does not necessarily reverse a disease process, just as cessation of smoking does not reverse lung cancer. This fallacy is shown in Bryant and Harvey's results, which showed that patients involved in compensation (whether settled or not) had a significantly higher incidence of PTSD than those who were not so involved.

A more recent study from New South Wales, however, showed that PTSD at 18 months was not influenced by compensation status in a similar cohort of patients presenting to hospital after a motor vehicle accident.[188]

The methodological inadequacies of studies of psychiatric morbidity following motor vehicle trauma have been highlighted in a review by Blaszczynski et al,[195] citing factors such as selection bias, absence of a clear definition of PTSD, reliance on clinical judgement and failure to incorporate ratings of injury severity. Interestingly, they cite issues of compensation as being the main concern.

The literature regarding the relationship between road trauma and PTSD has also been reviewed by Matthews[196] who found evidence of the legal process contributing to the diagnosis but that the literature regarding compensation was mixed. Matthews concludes that "compensation neurosis" only occurs in a minority of patients after road trauma but bases this conclusion on the findings that malingering is not more frequent in compensated patients, and that symptoms do not appear to improve after settlement of a claim. Matthews, like Bryant and Harvey, seems to imply that the effect of compensation is a conscious one. Contrary to their conclusion, the knowledge that PTSD in compensated patients is not due to malingering and does not improve after case settlement does not discount the possibility that compensation is associated with the development of (genuine) PTSD, a finding that is supported by the literature.

### 1.5.2.7 Other Conditions

The conditions discussed above are assumed to have occurred as a result of physical trauma. Although there is disagreement regarding the role of trauma in some of these conditions, it is the assumption of a traumatic event as a precipitant that enables the conditions to be covered by compensation schemes. The role of compensation in other forms of trauma has also received some attention. A large multi-centre study of outcomes after major trauma to the leg (below the knee) called the LEAP (Lower Extremity Assessment Project) study was performed in the late 1990's and the results were reported in many formats over the following years, with two papers commenting on the role of compensation after two and seven years follow-up.[197 198]

The LEAP study was an observational study looking at predictors of outcome in consecutive patients presenting with major lower extremity trauma, which took into account many psychosocial variables. While the original intention was to assess the outcome of amputated limbs versus salvaged limbs (no difference was found), the analysis provides useful information regarding psychosocial predictors in this group.

The LEAP study found that after 2 and 7 years, the following factors were independent predictors of poor outcome, assessed by the Sickness Impact Profile (SIP[199]): increasing age, female gender, non-white race, low income, low education level, lack of health insurance, smoking, poor health prior to the injury, low self-efficacy (confidence in one's ability to resume life activities) and involvement in the legal system in an effort to obtain disability payments or compensation. Legal involvement was a significant predictor of general (physical and psychosocial) outcome after 2 years, but the effect was decreased after 7 years, and only remained significant for psychosocial functioning (rather than physical). The study does not give more information regarding the type of compensation, or the effect of compensation per se, as it only provides data regarding whether or not the legal system was used in the process.

More recently, other conditions have been put forward as work-related and, therefore, compensable. In 2004, the South Australian Workers Compensation Tribunal found that work-related stress contributed to a man's colorectal cancer, resulting in financial compensation being granted.[200 201] This decision was criticised for a lack of scientific evidence, and raises the problem of the lower standard of scientific proof required for legal proof, compared to medical proof.

In summary, although illness related to secondary gain has been reported for over 200 years, it was not until the increased availability of compensation through common law and worker's compensation in the late nineteenth and early twentieth centuries that we have been able to document an association between various illnesses and secondary gain (compensation). Each of the conditions discussed has some evidence of association with compensation but the level of evidence is low as there are no randomised controlled trials on this subject as they are not feasible, and many of the studies suffer from methodological limitations such as being retrospective, having misclassification bias (often due to poor diagnostic criteria), poorly matched controls, or untested outcome measures.

### 1.5.3 Proposed mechanisms

The previous section provided some evidence of increased reporting of illness, increased severity of illness, prolonged recovery, and poor treatment response in compensated patients. This section explores the literature pertaining to the ways in which compensation may influence illness.

The first part addresses the issue of malingering: the possibility that the effects seen are due to conscious manipulation of illness behaviour. The second part addresses physical factors that may contribute to the association. The last two parts address psychosocial factors associated with compensation which may explain the differences in illness behaviour outlined in the previous section, covering such factors as financial incentives, the role of the adversarial legal system, and the psychology of illness labelling and the promotion of illness in the medical and legal systems (tertiary gain).

#### 1.5.3.1 Malingering

An important question that is often asked is whether or not the symptoms described by patients (and therefore the syndromes diagnosed and quality of life measured) are genuine. That is, whether patients are ill or whether they are malingering.

Malingering is often suspected in injured patients claiming compensation. The fact that compensation claims often involve conditions with subjective diagnoses, such as back pain, whiplash, headaches, shoulder pain, and post-traumatic stress disorder (PTSD) raises suspicion that patients may be consciously fabricating or exaggerating symptoms for secondary gain. Certainly, it appears that the effect of compensation on outcome is mainly seen in conditions with subjective diagnostic criteria, such as those listed above.

Although the suggestion of malingering for secondary gain has been associated with many of the conditions already mentioned, it is difficult to prove. The symptoms associated with railway spine were thought by some to be manufactured or, at least, greatly exaggerated.[19 25] Sir John Collie, in his 1913 book entitled “Malingering and Feigned Sickness” detailed examination techniques to “catch out” malingerers.[202] This was approached in a more scientific manner by Waddell in a landmark paper from 1980 published in the journal ***Spine***.[203] This paper gave a statistical analysis of non-organic signs (later described as “Waddell signs”) associated with complaints of back pain.

The paper describes several signs, listed under five groups, which are not compatible with physical lesions. These signs include non-dermatomal (stocking distribution) sensory changes, altered straight-leg raising angles with the patient distracted (i.e. sitting versus lying), tenderness to light touch over the back, overreaction (including disproportionate verbalisation, facial expression, collapsing), and low back pain with axial loading of the cervical spine by hand pressure over the head. Despite the possibility of observer bias, Waddell showed these test to have good inter-observer and intra-observer reliability. These tests were intended as a prognostic indicator for patients that may have a poor outcome with treatment and this has been subsequently verified by other researchers.[59 204-206]

The Waddell signs have been used as indicators of malingering,[207] even though this was not their original purpose. Waddell originally described the signs as useful indicators for patients whose pain is not purely physical and therefore unlikely to be corrected by interventions aimed at physical correction, such as surgery. He felt that these patients had psychosocial aspects to their pain, which may require different treatment. In other words, he did not claim that the signs were indicators of conscious deception.

Other behavioural factors have been proposed as indicators of malingering, including sub maximal effort on muscle testing, lack of motivation, aspects of a patients verbal reporting of symptoms,[47] and facial expression.[208] As with the non-organic signs outlined above, however, it is difficult to prove deliberate deception with any of these signs.

An important point regarding non-organic signs, then, is that the absence of a physical basis for the complaints does not necessarily indicate malingering. In other words, demonstrating biological implausibility with regard to a patient's symptoms does not mean that the patient does not feel them, or suffer from them. This relates to the concept of disease versus illness. Patients suffer illnesses; doctors diagnose and treat diseases.[21] Illnesses are experienced by the patient and may be related to physical, psychological and social factors, whereas diseases are abnormalities in the structure and function of physical systems or parts of the body.

Although there are reports in the literature claiming that compensated patients report symptoms with increased frequency[32 183 209-211] or severity,[212] this is not always the case.[48 186 203 213 214] This debate is not important to the discussion of malingering. Whether or not the symptoms described are actually felt (rather than consciously produced or altered) is the crucial distinction to be made. Similarly, the observation that compensated patients rate their pre-injury functioning superior to non-compensated patients[215 216] does not mean that they do not believe this to be the case.

Malingering refers to the intentional, or conscious, reporting or exaggeration of symptoms for reward (secondary gain, compensation). To show that a patient is malingering requires proof, not of the irrational nature of the symptoms, but of the conscious reporting of those symptoms, or the conscious exaggeration of "real" symptoms.

Several studies have attempted to demonstrate conscious symptom exaggeration in compensated patients. Measurement of conscious symptom exaggeration usually incorporates one or more sub-scales of the Minnesota Multiphasic Personality Inventory (MMPI).[217] Lees-Haley has published several papers on this subject.[154 218-223] He developed a Fake Bad Scale[154] which is the summary score of 43 selected items from the MMPI. The items chosen were based on test responses and observation of “obvious” malingerers. The score was tested in malingerers, non-malingerers, and groups of outpatients (without injuries or compensation) who were asked to simulate emotional distress from an injury. A score of 20 or more was set as the cut-off for malingering. The scores were higher for malingerers than for non-malingerers, and similar in malingerers and patients simulating emotional distress.

Many other scales for measuring malingering based on the MMPI exist, such as the F Scale, F-K Index, Ego Strength Scale, Subtle-Obvious Scale, DIS Scale and DEB Scale.[220] They are designed to appraise the extent to which patients over-state their psychological problems and exaggerate emotional symptoms, usually by scoring much higher than the general population. Like the Fake Bad Scale, they are based on answers given by controls who are asked to “fake” the answers by consciously trying to appear psychologically abnormal, and on answers given by malingerers.

An obvious flaw with these scales is that the diagnosis of malingering in the test subjects used for validating the scales is not based on any recognised standard. Malingerers chosen to test the scales are referred to as “obvious” cases, or are cases who have been shown to be malingerers by surveillance. Choosing a patient based on the obviousness of their malingering is clearly subjective. It is likely that patients were chosen because of their likelihood to have high scores on the various scales.

Another scale, the CE scale developed by Clayer et al,[224] is based on the Illness Behaviour Questionnaire[225] and has been proposed as a measure of conscious exaggeration of symptoms. Rather than testing on normal patients mimicking malingering, it was tested on a small group (ten) of patients thought to have pain at least partly of psychosocial origin. The gold standard used was the opinion of two independent psychiatrists and correlation between the average of the psychiatrists’ opinions and the CE scale was good (correlation coefficient 0.64, $p < 0.01$). Unfortunately, as with other studies, the weakness lies in the reference standard. Correlation between the two psychiatrists was not provided, and one previous study has shown poor correlation between psychiatrists regarding their opinion on malingering.[226]

In the insurance industry, surveillance has been used as proof of malingering. However, the problem with this is that it usually only shows functional capacity; video footage of patients performing strenuous tasks does not reveal their level of pain during that task.

It is well established that normal people can fake emotional distress in general, and post-traumatic stress disorder (PTSD) specifically.[154 220 227 228]

There is also some evidence that certain scales can distinguish between patients who simulate (fake) the test and "genuine" patients.[154 220 222 227 229] This does not mean, however, that patients with similar scores to the control simulators are necessarily faking. And although some of the scales are designed to pick up patients who fake badly (Fake Bad Scale, Subtle-Obvious Scale) it remains difficult to prove that this is the case. In fact, in one study that looked at student volunteers who were instructed to feign PTSD (some with coaching and some without) no difference was found in the Fake-Bad Scale between the volunteers who were coached, those who were not coached, and "bona-fide" PTSD cases.[228] This paper has been criticised[223] for using workers compensation claimants as the bona-fide group, which leads to two possible conclusions: that the workers compensation patients were also feigning illness (in other words, they were not bona-fide cases of PTSD); or that the Fake-Bad Scale is not sensitive in detecting malingering. For our purposes, this means that information from such studies is of limited use: either compensated patients are feigning illness, or we cannot determine whether they are.

The scales used to detect malingering may be positive for other reasons. Grillo[221] found that patients with personality disorders had scores consistent with simulation (faking bad). Barsky[212] provides an overview of possible reasons for symptom amplification, including depression, anxiety, neuroses (hysterical conversion), somatizing personalities, and social and legal interventions. Consequently, the fake-bad scale has been criticised as produceing a very high rate of false positives.[228]

It is possible that the scores mentioned may be related to malingering, but the evidence is not strong. What is of note is that, although these scores may be high in “obvious” malingerers, they are not always high in patients involved with compensation or litigation. Mendelson[230] used a measure of Conscious Exaggeration from the McGill Pain Questionnaire and found no difference in the scores between chronic pain patients involved in personal injury litigation and those not seeking compensation. In a review by the same author, it was concluded that there was no evidence that patients seeking compensation exaggerate their pain.[48] This finding was confirmed by Melzak, Katz and Jeans[213] who found no difference in pain reporting using the McGill Pain Questionnaire or psychological testing (MMPI) between compensated and non-compensated patients. DeViva and Bloem[186] showed that in combat veterans seeking treatment for PTSD, scales of symptom exaggeration were not related to attempts to seek compensation.

Other measures of malingering, used in head injured patients, have been associated with compensation status. Binder[231], in 1993, showed that scores of poor effort and conscious exaggeration were increased in compensated patients with head injury. In this study, a series of 5-digit numbers were presented to the patient at the rate of one digit per second, and after an interruption they were asked to recognise the numbers by being presented with a forced choice between one correct response and one distractor such that patients with no short term memory would still score 50% by chance alone. Binder found that the group seeking compensation scored worse than the group not seeking compensation, and often scored worse than would be expected by chance. He concludes that this is evidence of conscious exaggeration of memory deficit, although the proportion of scores below chance were only 17% and 3% in the two compensated groups.

Similar findings to that of Binder were found by Millis,[232] in a study examining exaggerated memory deficits using a similar, word-based, recognition test. He found that mildly head injured patients seeking financial compensation scored significantly worse than uncompensated patients with moderate to severe head injuries. Interpretation, however, is difficult, as confounding was possible due to age differences between the two groups. Also, differences in the mean scores between the two groups does not give us information regarding the extent of symptom exaggeration, as we do not know the proportion of patients who may exaggerate, or at what cut-off one should declare malingering to be present.

Perhaps of greater interest in the trials by Binder and Millis was the finding that, of the compensated patients, those with mild head injury scored significantly worse than those with more severe brain dysfunction. This association of increased disability with less severe physical injury has been noted previously for whiplash[93] and 'railway spine'.[19] Combined with the evidence from Binder of increased symptom exaggeration with less severely injured patients, this provides some evidence for malingering.

In a review of articles on malingering, Fishbain et al[233] concluded that the methodology of studies was poor and that data on malingering is inconsistent.

However, they found that malingering probably does occur in chronic pain patients and is probably in the order of 1.25 – 10.4%.

In a review of orthopaedic and neurosurgeons opinions in the US, Leavitt and Sweet[234] found that most surgeons agreed that malingering occurred in 5% or less of patients with low back pain. A higher estimate of malingering (20 - 30%) was reported by Less-Haley[219] using the Fake-Bad Scale, but this has been reported as having a high false positive rate.[228]

Another aspect of symptoms that has been seen as proof of malingering is the improvement in symptoms after settlement of compensation. The expression "cured by a verdict" comes from a quote by Foster Kennedy,[20] a neurologist who, in 1946, wrote: "A compensation neurosis is a state of mind, born out of fear, kept alive by avarice, stimulated by lawyers, and cured by a verdict." This was reinforced in an often cited article by another neurologist, Henry Miller,[235, 236] who claimed that only 2 patients from a cohort of 50 with "accident neurosis" had ongoing neurosis two years after settlement. Apart from questions regarding the validity of the diagnosis, the methodology is not clear regarding selection of the 50 patients for review, and the review process. Another paper from the same period noted that 88 out of 100 patients with whiplash improved after settlement of the legal case and attributed the lack of improvement in the remaining twelve patients to ongoing secondary gain.[237] Like Miller's paper though, the study population (one hundred) was taken from a larger cohort of 219 patients and the selection criteria were not stated. Since then, studies involving follow-up of a pre-defined cohort of claimants, have not shown significant evidence of improvement in the patient’s condition after settlement of the case.[238-241]

The literature regarding improvement after settlement has been reviewed by Mendelson in three articles[238 242 243] and he concludes that Miller’s view, that patents improve after settlement, is not supported by the literature since that time. Mendelson, though, concentrated on return to work as a measure of recovery. Return to work is influenced by many factors other then illness. Several studies have shown that social factors are stronger predictors of sick absence and return to work than illness. This is discussed above, in relation to back pain. In any case, there is no good evidence that patients are “cured by a verdict”. This does not, however, prove that compensation was not involved in the development of the condition.

Changes in patient behaviour and perceptions of health after settlement of claims, which may provide stronger evidence of malingering, have received little attention.

Although there is evidence that malingering exists, it probably occurs in a minority of injured patients, and is insufficient to explain any association between outcome and compensation status. It can be concluded then, that compensated patients are genuinely ill, or at least perceive themselves as such. In other words, we have to take their word for it. Lack of a physical explanation for patients’ illnesses does not, in itself, imply malingering.

It appears then, that in most cases the symptoms (and therefore the illness) is real. And although there is little evidence for conscious symptom exaggeration or feigning, this does not mean that compensation status does not influence the development or course of the illness. The association between compensation (and other psychosocial variables) and many, usually subjectively diagnosed, illnesses remains; the lack of evidence of malingering merely indicates that any effect that compensation has is not being consciously manipulated.

#### 1.5.3.2 Physical factors

The association between compensation and poor outcome may be explained by physical factors, that is, different physical injuries in compensated patients may be the cause of the poor outcome. In other words, the association between compensation and poor outcome may be confounded by physical factors. This may seem intuitive in some situations: for example, for any given injury, workers in factories may be expected to experience more severe injuries than non-compensated people who are injured in their home or at sport. With objectively diagnosed conditions, this may be the case, and this can easily be accounted for in any analysis, by allowing for injury severity. In subjectively diagnosed conditions, like those usually associated with compensation, this becomes difficult as we do not know if the condition is more severe because of physical factors associated with the injury, or if it being reported as more severe because of the presence of compensation.

In any case, while there is some evidence that outcome after injury can be predicted by the physical injury (for example, scores of injury severity have been shown to predict mortality,[244-251] and illnesses such as PTSD,[191 252]), in more subjective conditions (such as PTSD), which are often associated with compensation, this is not always the case.[253] In fact, some studies have shown worse outcomes in patients with lower scores of physical injury.[183 254]

It may be argued that psychological factors, such as those involved in compensation, may cause physical disease. For example, the risk of coronary heart disease has been associated with psychological factors such as occupational stress or low job control.[62 255 256] However, the association between adverse psychological exposure and disease may not necessarily be causal, and the problems and biases inherent in studies of this type have been documented.[257 258]

The literature does not provide any evidence that the association between compensation and poor outcome is confounded (or mediated) by physical factors. In fact, in studies that do take injury severity into account, there is often little correlation between the physical severity of the injury and the perceived outcome, as psychosocial factors (including the presence of compensation) will often outweigh the influence of injury severity.

#### 1.5.3.3 Secondary gain

Secondary gain refers to personal advantages that result from the symptoms of physical disease. It does not necessarily imply that this is a conscious process, and the “gains” may include the avoidance of certain activities. It relates to the concept of the sick role, which may contain or lead to secondary gains.

The literature pertaining to secondary gain has been reviewed by Fishbain et al,[259] who concluded that secondary gain is an important determinant of illness behaviour. They also conclude, however, that the literature pertaining to the role of secondary gain via compensation is inconsistent. This may be due to an inconsistent association between secondary gain and compensation. For example, a compensated patient may be following a compulsory process and not necessarily be seeking gain, and conversely, patients may be seeking secondary gain, but, for other reasons, do not choose to seek compensation through the recognised channels.

Secondary gain as a mechanism for the association between compensation and health, may be purely financial or due to other psychosocial gains that arise from assumption of the sick role.

#### 1.5.3.3.1 Financial factors

The evidence for financial gain as a motive for pursuing compensation is found in studies that have shown variation in the uptake of compensation (usually workers compensation in the form of financial payments or sick leave) with variation in the level of benefits provided.[113 260-264] Attempts have been made to quantify the association between benefits on the one hand, and illness on the other.[265-267] In a study using randomly sampled claims from 12 US states, Worral and Appel, showed that a 10% increase in workers compensation benefits is associated with a 10% increase in claims costs, a 4% increase in claims frequency, and a 9% increase in the duration of disability.[260] Differences in health outcomes (health care utilisation and time off work) have also been shown between lump sum and continuous payments.[20 80 268]

The effect of financial incentives on surgical outcome has been studied in the United States military system, where compensation rates are standardised and not usually subjected to appeal. In a study of 348 soldiers after lumbar microdiscectomy, increasing compensation incentive (anticipated payout relative to usual income) was a significant determinant of poor surgical outcome.[269]

Similar changes in claim rates (as a surrogate for injuries) have also been demonstrated after legislative changes in compensation systems in Australia.[90 270 271] However, to attribute the changes to any one factor, such as level of compensation, is difficult due to the widespread changes which accompany such administrative changes. For example, the introduction of the Accident Compensation Commission in Victoria brought with it an increased crackdown on false claims that, on its own, may have dramatically reduced the claim rate.[270]

Leigh,[272] however, states that the influence of the size of the benefits on claim rates, while present, may be overestimated. He suggests that part of the association between benefit levels and claim rates may be due to legislators increasing benefits ***in response to*** increased claim rates. Within the workers compensation system, he also found that claim rates were higher in blue-collar workers and lower in experienced workers, thereby exposing a variable that may be responsible for confounding in studies of this type.

Outside the role of financial incentives, other psychosocial factors such as occupational factors, blame, and personality have been studied as potential mechanisms or confounders in the association between compensation and health.

#### 1.5.3.3.2 Psychosocial factors

Prior to the rise of psychology in the second half of the 19th century, several authors had attributed unexplained illnesses to neurological abnormalities, and, in the absence of identifiable pathology, several authors suggested that the complaints may be generated by an “idea” or “emotion”.[273 274] The earliest psychological theories regarding compensation related illness used the term hysteria, and early reports of the association between compensation and illness were labelled as compensation hysteria.[242] Although hysteria in the workplace has been described as far back as the eighteenth century.[275] Hysteria, or hysterical conversion, defined as illness behaviour without evidence of physical cause, has been given as the cause of compensation related illnesses such as RSI[31] and railway spine.[25]

It is interesting to compare early accounts of hysteria with compensation related illnesses from the present day. Gower,[26] in his textbook of neurology from 1888 felt that hysteria was not caused by malingering, continued even after the stimulus was removed, was often post-traumatic, and was influenced by the suggestion of symptoms and by imitation. The presentation of hysterical symptoms, however, changes with time, which is thought to be a reflection of what is thought to be legitimate disease at the time.[94 276] In Gower's textbook, paralysis was a common presentation, but he also described superficial tenderness, a common finding in hysterical (or somatoform) disorders today, such as fibromyalgia.[277]

Part of the psychology of compensation-related conditions such as whiplash and RSI may be symptom expectation or suggestion. The presentation of feelings such as dissatisfaction, anxiety and stress as complaints that fit into known patterns, such as whiplash or RSI, has been attributed to pre-existing knowledge of these conditions.[31 44 94 96 97 111 278] The important connection with compensation lies in the possibility that the stress and anxiety felt by the patients (which they then describe in terms that fit into recognised patterns of illness) may be caused by the compensation process itself. It is also important to note that labelling of symptoms as a recognisable disease is an important step in the process of obtaining compensation, and in deriving the benefits of the sick role.

It has been suggested that there is an underlying psychological difference (such as depression, hysteria, and hypochondriasis) between compensated versus non-compensated patients.[206] Some studies testing psychological predictors of outcome after treatment have shown a difference in outcome between patients grouped by different psychological profiles,[155 178 205 206 279 280] although others have not.[281] However, most of these studies measure the psychological status long after the initial injury, which raises the question of whether the patient was psychologically predisposed to the condition, or whether the injury (or the compensation process and its accompanying social environment) caused the psychological condition.

Unfortunately, in studies that show an association between pre-injury psychological status and post-injury psychological status, the pre-injury status is usually provided by the patient, which leads to potential bias. For example, studies of stress disorders after motor vehicle accidents show that patients with a history of psychological problems such as PTSD and depression are more likely to develop stress disorders post injury.[193 282-284] If compensated are also more likely to develop PTSD (by reporting more symptoms relating to PTSD), it is possible that they are also more likely to report a past history of

PTSD and depression. This recall bias is likely to affect the association for prospective cohort studies as well as case-control studies, as the pre-injury status depends on patient recall and not on prospectively collected data.

While the role of psychopathology, for example depression and neuroses, in predisposing patients to poor outcome remains unclear, there is evidence that some aspects of the psychosocial environment influence outcome. Factors such as socioeconomic status and work factors such as employment grade and job satisfaction have been shown to influence both the reporting and the outcome of some conditions.[67 69 70 72 73 76 77 79 85 130 158 182 285-292] The compensation process itself, particularly if the process is long or adversarial has been blamed for poor outcomes after injury.[81 96 132 192 241 293 294]

Blame may also play a role in outcome after injury. It appears that blaming others (particularly an employer) for one's condition, rather than accepting responsibility, or blaming no-one, leads to poor outcome and resistance to treatment,[72 110 295-297] not only in subjective conditions such as whiplash and back pain, but in patients paralysed after severe injuries.[298] It may not be possible, however, to dissect out any effect of blame from the effect of compensation, as those who are entitled to compensation are usually not at fault. This is thought to be a significant factor in the improved outcomes seen in "no-fault" compensation systems, and may be an explanation for the association between compensation and whiplash.[110]

Blame and fault relate to the psychological concept of attribution theory, which concerns the affective and behavioural consequences that result from the perceived causes of events.[299 300] Williams and others offer an explanation of how patients' reaction to trauma may be influenced by causal attribution.[299] In simple terms, blaming a railroad company for one's injury because they are more interested in deadlines than patient safety can create feelings (such as anger) which may influence behaviour. They also offer other cognitive processes, such as hindsight bias, as explanations for the variance in response to traumatic events. While these may be valid explanations for the difference in response found in compensated patients, they do not tell us if these processes are also driven by secondary gain or some other aspect of the compensation process. In fact, some of the aspects of blame, such as a desire for revenge on the person or organisation seen as causing the injury, may be regarded as forms of secondary gain.

#### 1.5.3.4 Tertiary gain

Although much has been written about the role of secondary gain and how the psychosocial environment may influence the patient, another, external, cause for the association should be considered: tertiary gain. Tertiary gain of illness was first proposed by Dansak[301] who defined it as "gains sought or attained from a patient's illness by someone other than the patient". In the context of compensation for injury, tertiary gain may benefit lawyers and medical and allied health practitioners.

The involvement of a lawyer is associated with poor outcome after chronic pain,[302] surgery,[178 303 304] work place injury,[61 260 305] and road traffic injuries.[68 117 198 306 307] Diminished use of lawyers has also been suggested as one of the reasons for the benefits associated with no-fault insurance schemes.[68 117] Lees-Haley attributes the effect of legal representation to "coaching" by the lawyers, where plaintiffs are advised how to answer questions from doctors and psychologists to increase the reported disability.[308]

Although retention of a lawyer is associated with poor outcome, cause and effect is difficult to determine without clear information regarding the temporal relationship between the two factors. While it can be argued that those who retain the services of a lawyer are less satisfied with their condition and have a poor outcome as a consequence of retaining the lawyer, the opposite may also be argued: that those who are dissatisfied or who have a poor outcome are more likely to retain the services of a lawyer. Causal association is also difficult to establish due to the obvious association with other factors, for example, cases involving legal representation will usually involve more engagement with the adversarial legal system, the cases will take longer, and exposure to other stress-related events, such as attendance for medical reports and in court, may be increased. For example, in a study of outcome after carpal tunnel surgery within the workers compensation system,[176] patients with contested claims had a significantly worse outcome than those with uncontested claims, the latter group having similar outcomes to uncompensated patients. This would indicate that the compensation process (involvement with the adversarial legal system) may be more important than the financial gain, although it could also be argued that those seeking compensation through the courts may be seeking greater financial gain. Other authors attribute the effect of compensation to the process, rather than the financial gain.[131 132 293 308-310] Lees-Haley describes the model personal injury plaintiff as an "unhappy somatizer involved in a social context which encourages rationalization, projection of blame, and complaining".[308] Put more succinctly, Hadler notes that "It is hard, if not impossible, to get well if you have to prove you are sick",[309] and, in a direct comment on the compensation process, states that "the algorithm for recourse … promulgated by the Workers' Compensation system is dangerous, if not iatrogenic".[310] This may represent tertiary gain, in that the processes of claiming compensation may be thought to benefit other parties, such as lawyers and insurance companies.

Tertiary gain from doctors and other health practitioners may also contribute to illness in compensated patients. Kwan and others offer a broad definition of tertiary gain to include any gain received by caregivers, such as status and recognition, and describe tertiary gain as a natural phenomenon, with the gains being normal consequences of the caregiver role.[311] However, specific examples exist of how compensated patients may be diagnosed or treated differently by doctors, so that financial or academic gains may be achieved. In a study examining diagnosis and treatment of thoracic outlet syndrome, Cherington and Cherington[312] found significant small area variations in the rates of surgery for thoracic outlet syndrome (TOS). They found that in Colorado, workers compensation patients were more likely to be diagnosed with TOS than uninsured patients, and that workers compensation patients were far more likely to be treated surgically once diagnosed. 27% of patients treated surgically were treated under workers compensation, and 3% were uninsured. Of the patients treated non-operatively, 7% were under workers compensation and 29% uninsured. Increased use of health services by patients treated under workers compensation compared to private insurance has been attributed to the higher medical fees provided under the workers compensation scheme.[313]

The labelling of many of the conditions closely tied to compensation, such as railway spine, RSI and whiplash, by the medical profession is thought to represent tertiary gain. Even though the provision of a diagnosis, in the form of a medical label such as whiplash or RSI, provides some secondary gain to the patients by validating their illness and therefore leading to secondary benefits such as monetary compensation or withdrawal from obligations at work or in the home, it also leads to tertiary gains for the health professional, such as financial gain from ongoing treatment and support of other practitioners.[278 311]

The labelling of conditions may carry with it the expectation of symptoms by the patient and expose them to harm by treating them as diseased patients.[277 314] The labelling of RSI was thought to be an issue in the epidemic of RSI,[31] in whiplash where it was suggested that the diagnosis is more disabling than the injury,[146] and in idiopathic arm pain where making a diagnosis is thought to be harmful.[164]

Tertiary gain may contribute to the incidence of illness and poor outcomes seen in compensated patients not only by diagnosing (labelling) illnesses,[314] but by promoting illness through further investigations, referral to other health practitioners, and through treatments provided.

The role of investigations in the propagation of illness has been studied by Jarvik et al[315] with respect to magnetic resonance imaging (MRI). In this study, 380 patients referred to a primary carer for regional back pain (no neurological symptoms) were randomised to receive a plain radiograph or an MRI as their initial investigation. No other intervention was provided as part of the study, apart from usual care by the general practitioner. Back-related disability and general health at 12 months were similar for both groups, however the group who were randomised to receive an MRI had higher treatment costs, were more likely to undergo spinal surgery (10 operations compared to 4), and were therefore more likely to have treatment complications. The higher likelihood of treatment associated with medical investigations has also been shown elsewhere.[316 317]

While this does not provide direct evidence of tertiary gain, it does provide some evidence of the tendency for doctors to fall back on a medical model of disease, and to use what they know and what they have at hand. In other words, to medicalise patients' complaints. Medicalisation refers to the interpretation of various processes of human life as medically defined conditions. The term, and its causes and implications, were first brought into public discussion by Ivan Illich in 1975.[318-320] Illich noted the widening scope of medical care, and an association between increased therapy and increased harm to patients, largely through iatrogenesis. Medicalisation has been considered a problem with various aspects of health, such as childbirth,[321] old age,[322] and the process of dying,[323] but compensation related conditions such as back pain, PTSD and whiplash have all been held up as examples of medicalisation:[7 13 55 114 148 324 325] of the medical community taking general complaints, many of which may simply be expressions of dissatisfaction, and of attempting to fit them into known constructs by the use of investigations and, once placed into the most appropriate diagnostic category, of instigating treatments. Indeed, the study described above, which compared plain radiographs to MRI scans for low back pain, may be seen as an example of medicalisation, as both of these investigations have been shown to be not predictive of back pain.[326-328]

Although the increased illness reporting in compensated patients may, in part, be mediated by increased treatment provided by health practitioners, it may also be true that more investigations and treatment are provided to compensated patients because they exhibit more illness behaviour. Also, there is less financial restriction on providing health related activities in compensated patients.

The interplay between compensation and other psychosocial factors may be difficult to untangle. Many psychosocial factors that are known to be related to general health, such as occupational factors (job satisfaction,[67 69 70 73 76-78 158 269 285 289 292 329] job control,[78 256] income,[76 330] occupational prestige or social class,[76 79 158 292 330] education,[330 331] and psychological stress,[67 69 76 78 182 255 258 286] are likely to be associated with compensation. For example, self-employed persons in Australia often do not have workers compensation, those on high incomes may be less likely to pursue compensation, less educated people may be more likely to be employed in jobs of lower prestige and jobs that are more likely to result in injury (and therefore subsequent claims for compensation),[70] and, interestingly, those suffering psychological stress may be more likely to suffer a work-related injury.[332 333]

### 1.5.4 Summary of literature

An association between compensation status and poor outcome after injury has been widely, although inconsistently, reported. The association between compensation and outcome after injury is not new, and is not confined to any particular type of compensation and is not geographically confined.

The literature relating to the association with compensation is often flawed by the absence of a control group of non-compensated patients, by retrospective design, and by difficulties in establishing the diagnosis. Interestingly, most of the literature on this topic concerns diagnoses or conditions, such as whiplash, chronic pain, low back pain, head injury and arm pain, which rely on subjective complaints, particularly pain. Not only do these diagnoses rely on patient-reported symptoms, they are often diagnoses that have no accepted pathological findings.

Regarding the mechanism of any association, there is evidence that compensation may influence outcome by secondary gain (financial or psychosocial). There is very little evidence of pre-existing psychological factors acting as predictors of outcome, but this may be due to the practical difficulties associated with establishing such a connection.

Tertiary gain, through the control of the process afforded to doctors and lawyers, also appears to be an important mechanism by which compensation may be associated with increased incidence of illness, and poor outcome after treatment. It is likely, however, that compensation produces poor outcome by a combination of the processes described, for example, it is reasonable to conclude that, in seeking secondary gain, patients are forced to use legal representation and deal with the adversarial legal system. These factors, along with social and cultural pressures from workmates, employers, friends and family, and anxiety from increased involvement with medical practitioners may lead them to escalate pain behaviours.

Difficulties with methodology in studies of compensation, for reasons such as selection bias, recall bias, misclassification bias (from poorly defined definitions of exposure and outcome variables) has resulted in significant variation in reporting the presence and magnitude of any association, and makes arguments for a causal relationship more difficult to sustain. It has been argued,[50] for example, that compensation may be a consequence of chronic pain and illness, rather than a cause, and that the observational studies so far performed are unable to distinguish between cause and effect.

The lack of prospective studies, and difficulties controlling for confounding from injury factors, demographic factors, and other psychosocial variables also makes the determination of cause and effect difficult. Furthermore, differing definitions of compensation make it difficult to determine whether the association is due to legal involvement, blaming others, pursuit of compensation, or related to case settlement.

Due to the nature of the compensation systems, trauma is assumed to be a common precipitant for these conditions; many of the conditions discussed relate to the trauma of industrial injury or to motor vehicle trauma. The role of physical trauma, however, has been argued, particularly in the case of back pain, whiplash, and work-related arm pain.

There is a need for more research into compensation status and outcome after injury. A systematic literature review of the association between compensation and outcome after injury or after surgery has not been performed. In order to accurately measure the association, studies with multivariate analysis, to allow for other psychosocial and physical factors likely to influence outcome, are required. Clear definitions of compensation and validated outcomes are also required.

Regarding research aimed at determining cause and effect, an effort must be made to examine the decision to pursue compensation. If the decision to pursue compensation is influenced by outcome factors, or by factors influencing the outcome, cause and effect cannot be concluded. In this case, using entitlement to compensation as the main exposure variable (rather than pursuit of compensation) may allow more robust conclusions regarding cause and effect, as entitlement to compensation is less likely to be voluntarily influenced by patients.

## 1.6 Statement of objectives

### 1.6.1 Aim

This thesis aims to explore aspects of the association between compensation and outcome after injury. Secondarily, it will explore any association between compensation status and the development or diagnosis of some conditions (back pain, neck pain, PTSD, and fracture non-union), and any association between compensation status and the relationship between surgeon and patient assessment of satisfaction and recovery.

Any effect of compensation will be adjusted for important confounders, such as demographic factors, socio-economic status, and injury severity factors, where possible. If an association between compensation and outcome is found, it will be further explored by sub-group analysis of compensation type (third party or workers' compensation), by comparing outcome in those who pursued compensation to those who were entitled to compensation, and by examining the effect of case settlement.

Other aspects of the compensation process will be studied, namely perception of fault and use of a lawyer. Some correlation between these factors and compensation status is expected, but the presence of some overlap in these factors will allow exploration of confounding and interaction, to determine which factor is the strongest predictor of outcome.

The study does not aim to determine the mechanism of any association, although this will be included in the discussion, and the evidence for a cause-and-effect relationship, regardless of the mechanism, will be examined.

### 1.6.2 Study hypotheses

The primary study hypothesis is that the receipt (or potential receipt) of compensation influences health outcomes after injury in a negative manner. Specific hypotheses are listed in each of the three main sections (Chapters Two, Three and Four), and the general aim of each of these sections is listed below.

1. To investigate the association between compensation status and outcome after surgery reported in the published literature by performing a systematic review, and to quantify and further analyse this association by using meta-analysis. This will be reported in Chapter Two.
2. To identify predictors of poor outcome after major trauma, by studying a consecutive cohort of major trauma patients and developing

statistical models of health outcomes using general, injury-related, psychosocial, and compensation-related factors as possible predictor variables. This study will be reported in Chapter Three.

3. To determine the association between compensation status and outcome, adjusting for baseline factors, by prospectively following a cohort of patients suffering fractures in motor vehicle accidents. This study will be presented in Chapter Four.

The main health outcomes used will be general health, as measured by the physical component summary (PCS) and the mental component summary (MCS) of the SF-36 General health Survey. Other health outcomes will include neck pain, back pain, PTSD, and patient satisfaction.

# CHAPTER TWO. A SYSTEMATIC REVIEW AND META-ANALYSIS OF THE EFFECT OF COMPENSATION ON OUTCOME AFTER SURGERY.

## 2.1 Introduction

As discussed in Chapter One, there is conflicting evidence for a relationship between compensation status and outcome after injury. A review of the effect of compensation-related factors on outcome after any injury has not been performed and would be difficult due to inconsistencies in the definition of injury, and in the types of treatment provided.

As the surgery performed for compensated patients is usually provided to treat an injury, a review of outcomes after surgery is a reasonable method of testing outcomes after injury. It has the advantage of controlling for diagnosis (as most patients undergoing a certain procedure do so for particular diagnoses) and controlling for treatment (as all patients in each study are undergoing the same treatment (operation). It can be argued, however, that many conditions treated surgically in compensated patients, such as back pain and shoulder pain, may not be entirely caused by injury.

Surgery for patients treated under compensation schemes is commonplace, particularly in the fields of spine, shoulder and knee surgery, where the treatment is usually based on a perceived injury in the course of work, or, less commonly, as a result of a motor vehicle accident. Any difference in outcome between compensated and non-compensated patients is important as it may influence clinical decision making regarding surgery. Also, a negative effect of compensation on outcome after surgery would raise questions regarding the validity of the diagnosis if the surgical correction of the perceived problem (for example, a herniated disc) failed to relieve the symptoms.

The association between compensation status and outcome after surgery has not previously been subjected to systematic review or meta-analysis, however systematic reviews regarding the effect of compensation have been performed in other areas. The two previous systematic reviews, mentioned in Chapter One, relate to chronic pain[210] and outcome after head injury.[183]
The first of these studies, by Rohling and Binder, was published in 1995 and examined the effect of compensation on the reporting and outcome after treatment of chronic pain. It analysed 32 papers, 72% of which were related to low back pain, and showed that compensation was associated with increased reporting of pain and decreased treatment efficacy. The association remained significant when both liberal and conservative estimates were used.
The second systematic review, by the same authors (Binder and Rohling) was published in 1996 and examined the association between compensation and outcome after head injury. The authors found that compensated patients demonstrated significantly more abnormalities and disabilities pertaining to their head injuries, despite having less severe injuries.

### 2.1.1 Aims and specific hypotheses

This systematic review aims to examine the association between compensation and outcome after surgery in a systematic manner and, through meta-analysis, to provide an estimate of the size and precision of any effect. Further, through examination of any heterogeneity by subgroup analysis and meta-regression, this study aims to explore possible mechanisms for the effect and important associations that may provide indicators for future research.

The study hypothesis states that compensated patients are more likely to have an unsatisfactory outcome after surgery, compared to non-compensated patients, and that when subjected to meta-analysis, this association will be statistically significant.

## 2.2 Materials and methods

As it is not possible to perform a randomised controlled trial for compensation, the studies under review can only provide observational data. Therefore, the so-called ‘MOOSE’ criteria were used to guide the conduct of the study, as recommended by leading medical journals and the CONSORT initiative.[334] The MOOSE criteria are based on a report entitled Meta-Analysis Of Observational Studies in Epidemiology (MOOSE)[335] which provides a checklist to standardize the methodology of reviews based on observational data.

Studies to be included in the review included any trial of surgical intervention where compensation status was reported and results were compared according to that status. A compensated patient was defined as any patient receiving worker’s compensation payments for their condition, or undergoing litigation as a result of their pre-operative condition. Papers must have included at least one compensated patient and one non-compensated patient and must have the patients’ compensation status ascertained prior to surgery. No language restriction was applied, and no limit was set for the time since publication. Although the electronic searches were date-limited by design, earlier studies, usually gathered from reference lists, were included.

Surgical intervention was defined as any surgery performed on patients regardless of the specialty of the surgeons. Studies examining the effect of injections of local anaesthetic or steroids, application of splints, physical therapies, or rehabilitation were excluded. Studies of intra-discal injections of chymopapain (usually for lumbar disc pathology) were included, as this procedure is usually performed by a surgeon, and usually performed in an operating room.

The abstracted outcome used, where available, was a region-specific outcome score (e.g., Low Back Outcome Score[336], Harris Hip Score[337]). If this was not provided, the following outcomes were abstracted (in order of preference): a general functional score, a general health outcome score (e.g., SF-36[338]), a patient satisfaction score, or a pain score. Outcome scores measuring time to return to work were excluded as this is influenced by confounding factors such as job characteristics and social factors,[72 339] and has been shown to be a poor measure of outcome for compensated patients[340]. In patients treated under workers compensation for example, there is no financial urgency to return to work and, furthermore, the employers and insurers are often reluctant to have a patient return to work unless they have significantly recovered. These factors would bias the results if return to work was used as an outcome variable. Also, return to work is not necessarily related to the health or quality of life of the patient.

Outcome was classified as satisfactory or unsatisfactory as given by the authors. If this was not provided, outcome scores of “excellent” or “good” were classified as satisfactory, and outcomes labelled as “fair”, “poor” or “failure” were classified as unsatisfactory according to usual reporting of surgical studies.

The electronic database search strategy used “compensation” as a text word, or the medical subject heading “compensation and redress”, combined with “surgery” or “surgical procedures, operative”. Searches were not limited to any particular form of clinical study: randomised controlled trials, cohort studies, case-control studies, and case series were all included. Animal studies were excluded. Unpublished studies were not included. The initial electronic search strategies are given in Tables 2.1, 2.2, and 2.3.

Table 2.1. Medline search strategy and results (15 July 2003).

| Search number | Search History | Results |
|---|---|---|
| 1 | compensation.tw. | 14732 |
| 2 | exp “Compensation and Redress”/ | 865 |
| 3 | exp SURGERY/ or surgery.mp. | 367218 |
| 4 | exp SURGICAL PROCEDURES, OPERATIVE/ | 1282369 |
| 5 | 1 or 2 | 15498 |
| 6 | 3 or 4 | 1455756 |
| 7 | 5 and 6 | 1753 |
| 8 | Limit 7 to (human and yr=1966-2002 and (classical article or clinical trial or clinical trial, phase i or clinical trial, phase ii or clinical trial, phase iii or clinical trial, phase iv or controlled clinical trial or journal article or meta analysis or multicenter study or randomized controlled trial)) | 1189 |

Table 2.2. Embase search strategy and results (15 July 2003).

| Search number | Search History | Results |
|---|---|---|
| 1 | compensation.tw. | 10680 |
| 2 | exp WORKMAN COMPENSATION/ | 1870 |
| 3 | exp SURGERY/ or surgery.mp. | 1023755 |
| 4 | exp SURGICAL PROCEDURES, OPERATIVE/ | 942391 |
| 5 | 1 or 2 | 11526 |
| 6 | 3 or 4 | 1023755 |
| 7 | 5 and 6 | 1389 |
| 8 | Limit 7 to (human and yr=1966-2002 and (article or conference paper or journal or proceeding or report or review)) | 923 |

Table 2.3. CINAHL search strategy and results (5 Sept 2003)

| Search number | Search History | Results |
|---|---|---|
| 1 | compensation.tw. | 1264 |
| 2 | Liability, Legal/ or WORKER'S COMPENSATION/ or Occupational-Related Injuries/ or Insurance, Liability/ | 9337 |
| 3 | surgery.mp. or Surgery, Operative/ | 15286 |
| 4 | 1 or 2 | 10122 |
| 5 | 3 and 4 | 165 |
| 6 | limit 5 to yr=1966-2002 | 159 |

Articles were sourced from Medline (1966 to 2003), Embase (1980 to 2003), CINAHL, Cochrane Controlled Trials Register, reference lists of retrieved articles and textbooks, and through contact with experts in the field. Duplicate articles were dealt with by inclusion of only the most recent publication. Authors were not contacted.

Two authors reviewed all abstracts and selected articles for retrieval. Retrieved articles were reviewed independently by the same authors (for inclusion criteria and for data extraction) and differences were resolved by discussion. The reviewers were not blinded to any aspect of the studies (e.g., journal type, author names, or institution). The reviewers abstracted the data onto a proforma (Figure 2.1) in the order given on the form, so that data regarding the comparative results of compensated and non-compensated patients was abstracted last.

Each article that met the inclusion criteria was reviewed for compensation type (worker's compensation, litigation or both), publication year, country of origin, surgical procedure, whether or not the article was designed specifically to look for a compensation effect, length of follow-up and outcome. Country of origin was taken as the country in which the study was performed. Length of follow-up was grouped as follows: 0-6 months, 7-12 months, 12-24 months, and over 24 months. Outcome was recorded as the numbers of patients with satisfactory and unsatisfactory outcomes in each group (compensated and uncompensated) according to the criteria given above. If continuous outcomes were reported, the total number of patients, the mean score and the standard deviation for each group was recorded where possible.

Figure 2.1.

**Meta-analysis: Compensation vs Outcome after Surgical Intervention**

Data Collection Form

| No | Auth | Year | Jrnl | Cntry | Body Region | Proc | Specific for WC? | Study type | Pro vs Retro | Compo type | Blinded outcome | Outcome tool | % F/U | Results | C n | C N | NC n | NC N |
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| | Last name, initial | | | | spine shoulder nerve other | | Y/N | **R**CT **C**ohort **S**eries | Y/N | WC, TP, both | Y/N | | | **B**etter/**W**orse & **S**/**NS** | n=unsat., N=group total | | | |

Methodology was examined by recording study type, completeness of follow-up, randomisation concealment, and blinding of outcome assessment. Study type was classified two ways: firstly as either randomised controlled trial, cohort study or case series, and secondly as prospective or retrospective. Completeness of follow-up was classified dichotomously as less than 80%, or 80% or more.

The extracted items of data were selected as they were possible confounders or causes of heterogeneity in the association under investigation. Sub-group analyses according to these variables, and according to sample size, were planned a priori, to explore any heterogeneity. The author's stated conclusions regarding the effect of compensation (regardless of the statistical findings) were also recorded.

Cochrane Review Manager (version 4.2) software[341] was used to analyse the data. Data were entered by one author (IH) and checked by another (JM). Odds ratios (OR) and 95% confidence intervals (CI) were calculated for dichotomous outcomes (satisfactory / unsatisfactory). Standard mean difference was given for continuous outcomes and these were analysed separately from the dichotomous outcomes due to the statistical difficulties of combining dichotomous and continuous variables. Continuous and dichotomous outcomes were combined in the meta-regression.

The results were examined for heterogeneity by examining the forest plot, comparing the summary odds ratio using random and fixed effect models, and by using statistical tests for heterogeneity. Funnel plots were used to look for publication bias.

Meta-regression, to investigate any association between the recorded variables and the effect size, was performed using Bayesian hierarchical methods[342] and the software Winbugs[343], and was performed separately from the RevMan analysis. Two variables, year of publication and sample size, were analysed in meta-regression only, as they were continuous variables and therefore not suitable for subgroup analysis in RevMan. At the suggestion of a journal reviewer, meta-regression was also used to perform a post hoc analysis of the effect of primary versus revision surgery, on the effect of compensation.

The literature search began on 13 July, 2003 with the electronic search, restricted to articles published before 1 January, 2003. The search was repeated, and the results updated, in May 2004 to include articles published before 1 January, 2004.

## 2.3 Results

### 2.3.1 Study retrieval

The Medline searches found 1,192 studies, Embase 928, CINAHL 159 and the Cochrane trial register found 315 studies. All studies found by CINAHL and Cochrane, and the majority of studies found by Embase were also found by the Medline search. No new articles were found from textbook reference lists or contact with experts in the field. Sixty-six extra articles were retrieved via references in the articles retrieved from the electronic search.

Most studies retrieved electronically could be excluded from information provided in the abstract, obviating the need for retrieval of the full article. For example, some studies initially retrieved electronically referred to aspects of respiratory compensation or compensation of renal function and were unrelated to the topic of the review. Studies found from reference lists were inspected electronically where possible and excluded, where possible, based on information provided in the abstract. Other articles that initially appeared relevant were excluded on reviewing the abstract if they only included compensated patients in the study population, or alternatively, if they excluded compensated patients.

The final number of complete articles retrieved for review (from all sources) was 305, of which 211 satisfied the inclusion criteria. Most were excluded because, although they may have included a mixture of compensated and non-compensated patients in the population, the comparative results for these two groups were not given. Two studies were excluded because compensation was provided only if patients had a poor outcome, i.e., the compensation status was not known prior to surgery, and was dependent on outcome.

There were four randomised controlled trials, 45 cohort studies (this included any study containing a control group, including case-control studies) and 162 case series. None of the randomised controlled trials randomised patients to receive or not receive compensation, therefore, the data from these studies pertaining to the difference in outcome between compensated and non-compensated patients were observational, as for the other studies. Twenty-four of the cohort studies were specifically designed to look at outcomes in compensated versus non-compensated patients. There were no studies in which treatment was allocated according to compensation status.
None of the randomised controlled trials stated that randomisation was concealed. No study stated that the outcome assessor was blinded to compensation status.

Of the 211 studies, 175 described a worse outcome in the compensation group, 30 articles described no difference between the groups, 5 of the articles provided results but did not comment on the difference in the text, and one study described better outcome in the compensation group. This was based on statements made in the text of the studies, regardless of what figures were provided in the text or tables.

Of the studies included in the meta-analysis, 31 different previously reported outcome tools were used, the most frequently used were the SF-36 and the Oswestry low back disability questionnaire (seven studies each), and the majority of the others were region-specific outcome scores. Most papers used their own outcome tool, 15 used measures of pain, and 12 used measures of patient satisfaction. Due to the wide variety of different outcome tools used, analysis of the results using the outcome tool as a variable was not possible.

### 2.3.2 Overall association

One hundred and twenty nine papers had dichotomous outcome scores available for comparison and were included in the meta-analysis. References for these studies are provided in Appendix 1. These studies included information on 7,244 compensated and 13,254 non-compensated patients. All except five of the included studies showed a positive association between compensation and poor outcome. None of the 129 studies with dichotomous outcomes showed an equivalent outcome. None of the five studies showing a negative association reached statistical significance. The summary odds ratio (OR) for an unsatisfactory outcome in compensated patients was 3.79 (95% CI: 3.28 to 4.37, random effects model).

The results for each study and the overall estimate are shown as a forest plot in Figure 2.2. Due to the size of this forest plot, it is provided as a single figure (Figure 2.2), in order to provide a pictorial overview of the results, and in an identical but enlarged format (Figures 2.3a and 2.3b) over two pages to allow it to be read.

The OR using a fixed effect model was 3.12 (95% CI: 2.90 to 3.36). The difference in the OR between the random effects and fixed effect models indicates some degree of heterogeneity. The chi squared test for heterogeneity was significant ($chi^2$ = 308.88, 127 DF, $p<0.00001$) however this test has excessive power in large meta-analyses and the P value does not reasonably describe the extent of heterogeneity in the results[344]. The $I^2$ value was 58.9%, indicating the proportion of the total variation due to heterogeneity[344].

Figure 2.2. Results and forest plot for all studies in the meta-analysis.

| Study | Compensated n/N | Not compensated n/N | Weight % | OR (random) 95% CI |
|---|---|---|---|---|
| Campbell 1947 | 19/52 | 2/70 | 0.59 | 19.58 [4.30, 89.08] |
| Greenwood 1952 | 13/23 | 8/43 | 0.84 | 5.69 [1.84, 17.55] |
| Raaf 1959 | 43/224 | 20/316 | 1.40 | 3.52 [2.00, 6.17] |
| Gurdjian 1961 | 71/177 | 178/738 | 1.63 | 2.11 [1.49, 2.97] |
| Slepian 1966 | 158/474 | 93/704 | 1.67 | 3.28 [2.46, 4.39] |
| Aronson 1968 | 8/24 | 4/44 | 0.70 | 5.00 [1.32, 18.96] |
| DePalma 1972 | 31/86 | 53/143 | 1.41 | 0.96 [0.55, 1.67] |
| Stauffer 1972 | 15/25 | 50/138 | 1.07 | 2.64 [1.10, 6.32] |
| White 1973 | 6/16 | 15/49 | 0.80 | 1.36 [0.42, 4.43] |
| Hudgins 1974 | 52/73 | 14/73 | 1.17 | 10.44 [4.82, 22.59] |
| Burke 1976 | 15/21 | 17/67 | 0.87 | 7.35 [2.46, 21.98] |
| Graham 1976 | 12/16 | 15/24 | 0.65 | 1.80 [0.44, 7.31] |
| LaMont 1976 | 12/29 | 17/96 | 1.04 | 3.28 [1.33, 8.12] |
| Law 1978 | 26/29 | 11/23 | 0.63 | 9.45 [2.22, 40.24] |
| Finnegan 1979 | 10/16 | 3/51 | 0.58 | 26.67 [5.69, 124.93] |
| Javid 1980 | 11/20 | 23/104 | 0.96 | 4.30 [1.59, 11.64] |
| Weir 1980 | 39/162 | 62/398 | 1.52 | 1.72 [1.09, 2.70] |
| Rish 1984 | 55/115 | 14/90 | 1.28 | 4.98 [2.53, 9.80] |
| Strefling 1984 | 43/105 | 9/123 | 1.16 | 8.78 [4.02, 19.21] |
| Javid 1985 | 7/18 | 13/87 | 0.85 | 3.62 [1.19, 11.06] |
| Dellon 1986 | 6/40 | 0/20 | 0.21 | 7.72 [0.41, 144.36] |
| Maciunas 1986 | 17/61 | 33/180 | 1.28 | 1.72 [0.88, 3.38] |
| Sutton 1986 | 10/26 | 32/163 | 1.07 | 2.56 [1.06, 6.17] |
| Branch 1987 | 32/82 | 10/90 | 1.15 | 5.12 [2.32, 11.32] |
| Bray 1987 | 13/26 | 10/24 | 0.85 | 1.40 [0.46, 4.28] |
| Friedman 1987 | 4/13 | 8/29 | 0.64 | 1.17 [0.28, 4.88] |
| Hill 1987 | 45/129 | 36/206 | 1.46 | 2.53 [1.52, 4.21] |
| Ritts 1987 | 6/8 | 11/27 | 0.47 | 4.36 [0.74, 25.74] |
| Christie 1988 | 12/23 | 21/87 | 0.99 | 3.43 [1.32, 8.90] |
| Hawkins 1988 | 8/35 | 6/73 | 0.83 | 3.31 [1.05, 10.44] |
| Herron 1988 | 18/34 | 7/57 | 0.92 | 8.04 [2.84, 22.71] |
| Javid 1988 | 17/42 | 16/172 | 1.14 | 6.63 [2.97, 14.80] |
| Silvers 1988 | 9/199 | 8/341 | 0.98 | 1.97 [0.75, 5.20] |
| Zucherman 1988 | 23/38 | 7/39 | 0.91 | 7.01 [2.47, 19.93] |
| Hanley 1989 | 16/26 | 4/24 | 0.70 | 8.00 [2.11, 30.34] |
| Hanley 1989 1 | 8/16 | 4/87 | 0.65 | 20.75 [5.10, 84.36] |
| Hawkins 1989 | 32/38 | 5/13 | 0.65 | 8.53 [2.07, 35.21] |
| Snyder 1989 | 6/8 | 14/47 | 0.50 | 7.07 [1.27, 39.41] |
| Biondi 1990 | 20/30 | 4/15 | 0.67 | 5.50 [1.39, 21.71] |
| Gartsman 1990 | 17/56 | 12/96 | 1.11 | 3.05 [1.33, 7.00] |
| Ogilvie-Harris 1990 | 15/28 | 6/24 | 0.80 | 3.46 [1.06, 11.33] |
| Paulos 1990 | 3/18 | 7/48 | 0.61 | 1.17 [0.27, 5.13] |
| Silvers 1990 | 27/82 | 43/141 | 1.38 | 1.12 [0.62, 2.01] |
| Davis 1991 | 23/91 | 56/427 | 1.42 | 2.24 [1.29, 3.88] |
| Ellman 1991 | 3/17 | 4/48 | 0.54 | 2.36 [0.47, 11.83] |
| Green 1991 | 16/25 | 12/28 | 0.86 | 2.37 [0.78, 7.18] |
| Schaffer 1991 | 3/16 | 8/80 | 0.63 | 2.08 [0.49, 8.88] |
| Tregonning 1991 | 56/66 | 72/170 | 1.21 | 7.62 [3.64, 15.95] |
| Javid 1992 | 13/38 | 14/140 | 1.08 | 4.68 [1.96, 11.15] |
| Lauerman 1992 | 13/22 | 12/21 | 0.78 | 1.08 [0.32, 3.64] |
| Albert 1993 | 6/11 | 9/66 | 0.67 | 7.60 [1.91, 30.19] |
| Hanel 1993 | 0/6 | 0/4 | | Not estimable |
| Jerosch 1993 | 19/21 | 13/23 | 0.51 | 7.31 [1.37, 38.98] |
| Knox 1993 | 4/5 | 7/12 | 0.28 | 2.86 [0.24, 33.90] |
| Rotman 1993 | 7/18 | 1/3 | 0.26 | 1.27 [0.10, 16.81] |
| Silvers 1993 | 5/44 | 10/200 | 0.84 | 2.44 [0.79, 7.52] |
| Greenough 1994 | 58/85 | 17/40 | 1.17 | 2.91 [1.34, 6.31] |
| Hutchinson 1994 | 10/26 | 2/21 | 0.52 | 5.94 [1.13, 31.15] |
| Mont 1994 | 10/10 | 16/63 | 0.21 | 60.45 [3.35, 1089.73] |
| Myerson 1994 | 8/11 | 9/25 | 0.57 | 4.74 [1.00, 22.52] |
| Olsewski 1994 | 12/24 | 0/37 | 0.21 | 75.00 [4.13, 1360.71] |
| Paulos 1994 | 2/4 | 0/14 | 0.17 | 29.00 [1.05, 801.98] |
| Polk 1994 | 19/34 | 10/39 | 0.96 | 3.67 [1.37, 9.86] |
| Silvers 1994 | 19/29 | 7/53 | 0.86 | 12.49 [4.14, 37.66] |
| al-Qattan 1994 | 13/34 | 5/99 | 0.84 | 11.64 [3.74, 36.20] |
| Frieman 1995 | 12/57 | 1/17 | 0.36 | 4.27 [0.51, 35.48] |
| Hallock 1995 | 41/52 | 19/48 | 1.06 | 5.69 [2.36, 13.74] |
| Higgs 1995 | 92/113 | 27/53 | 1.23 | 4.22 [2.06, 8.64] |
| MacKay 1995 | 17/46 | 9/108 | 1.04 | 6.45 [2.60, 15.98] |
| Misamore 1995 | 11/24 | 7/83 | 0.85 | 9.19 [3.01, 28.03] |
| Novak 1995 | 22/51 | 3/19 | 0.68 | 4.05 [1.05, 15.64] |
| Ricciardi 1995 | 0/6 | 2/11 | 0.18 | 0.29 [0.01, 7.14] |
| Herron 1996 | 37/89 | 12/186 | 1.23 | 10.32 [5.02, 21.22] |
| Lee 1996 | 11/60 | 19/193 | 1.14 | 2.06 [0.92, 4.61] |
| Parker 1996 | 9/10 | 5/13 | 0.30 | 14.40 [1.37, 150.81] |
| Strickland 1996 | 1/16 | 2/46 | 0.28 | 1.47 [0.12, 17.36] |
| Taylor 1996 | 273/1502 | 263/2674 | 1.75 | 2.04 [1.70, 2.44] |
| Ghazavi 1997 | 7/21 | 11/105 | 0.86 | 4.27 [1.42, 12.86] |
| Glowacki 1997 | 4/24 | 2/21 | 0.46 | 1.90 [0.31, 11.61] |
| Jebson 1997 | 6/15 | 2/9 | 0.43 | 2.33 [0.36, 15.30] |
| Johannsen 1997 | 12/24 | 2/36 | 0.53 | 17.00 [3.31, 87.22] |
| Penta 1997 | 43/61 | 25/42 | 1.12 | 1.62 [0.71, 3.71] |
| Schnee 1997 | 17/20 | 6/32 | 0.59 | 24.56 [5.40, 111.68] |
| Tomaras 1997 | 4/18 | 13/167 | 0.75 | 3.38 [0.97, 11.78] |
| Vaccaro 1997 | 13/13 | 2/11 | 0.18 | 102.60 [4.41, 2388.93] |
| Davies 1998 | 8/76 | 0/163 | 0.22 | 40.58 [2.31, 712.85] |
| Greenough 1998 | 52/57 | 46/63 | 0.89 | 3.84 [1.31, 11.24] |
| Hulsizer 1998 | 9/11 | 3/19 | 0.41 | 24.00 [3.36, 171.54] |
| Iskandar 1998 | 0/2 | 6/26 | 0.18 | 0.63 [0.03, 14.90] |
| Katz 1998 | 51/101 | 36/99 | 1.40 | 1.79 [1.01, 3.14] |
| Klekamp 1998 | 19/27 | 5/27 | 0.74 | 10.45 [2.92, 37.39] |
| Mont 1998 | 30/42 | 5/42 | 0.83 | 18.50 [5.86, 58.37] |
| Siambanes 1998 | 5/5 | 3/4 | 0.15 | 4.71 [0.15, 151.48] |
| Slotman 1998 | 7/17 | 8/31 | 0.75 | 2.01 [0.57, 7.07] |
| Westkaemper 1998 | 6/25 | 5/22 | 0.68 | 1.07 [0.28, 4.16] |
| Agazzi 1999 | 18/34 | 6/37 | 0.86 | 5.81 [1.93, 17.52] |
| Deshmukh 1999 | 11/17 | 9/27 | 0.73 | 3.67 [1.02, 13.14] |
| Hawkins 1999 | 5/8 | 1/11 | 0.27 | 16.67 [1.36, 204.03] |
| Kucharzyk 1999 | 12/27 | 8/103 | 0.91 | 9.50 [3.33, 27.07] |
| Sotereanos 1999 | 15/22 | 2/6 | 0.42 | 4.29 [0.63, 29.23] |
| Straub 1999 | 6/65 | 2/35 | 0.52 | 1.68 [0.32, 8.79] |
| Tribus 1999 | 2/6 | 0/7 | 0.17 | 8.33 [0.32, 215.68] |
| Albert 2000 | 10/28 | 2/11 | 0.50 | 2.50 [0.45, 13.91] |
| Antoniou 2000 | 10/19 | 7/22 | 0.74 | 2.38 [0.67, 8.49] |
| Arcand 2000 | 17/22 | 0/13 | 0.20 | 85.91 [4.36, 1692.79] |
| Atlas 2000 | 85/132 | 70/191 | 1.51 | 3.13 [1.97, 4.96] |
| Concannon 2000 | 6/84 | 0/107 | 0.21 | 17.80 [0.99, 320.69] |
| Connor 2000 | 9/20 | 2/16 | 0.49 | 5.73 [1.02, 32.10] |
| Flemister 2000 | 10/48 | 3/35 | 0.67 | 2.81 [0.71, 11.08] |
| Hostin 2000 | 4/24 | 3/24 | 0.54 | 1.40 [0.28, 7.06] |
| Lin 2000 | 3/8 | 0/8 | 0.18 | 10.82 [0.46, 252.79] |
| Lonner 2000 | 4/5 | 2/27 | 0.25 | 50.00 [3.63, 688.34] |
| Marks 2000 | 23/51 | 15/52 | 1.13 | 2.03 [0.90, 4.58] |
| Slosar 2000 | 30/73 | 21/60 | 1.25 | 1.30 [0.64, 2.63] |
| Taylor 2000 | 23/47 | 60/188 | 1.31 | 2.04 [1.07, 3.91] |
| Barrett 2001 | 7/24 | 10/115 | 0.87 | 4.32 [1.45, 12.90] |
| Geel 2001 | 11/15 | 2/17 | 0.44 | 20.63 [3.19, 133.45] |
| Hawkins 2001 | 26/47 | 27/71 | 1.20 | 2.02 [0.95, 4.26] |
| Lew 2001 | 3/10 | 4/37 | 0.50 | 3.54 [0.64, 19.45] |
| Mobbs 2001 | 14/84 | 14/140 | 1.15 | 1.80 [0.81, 3.99] |
| Moro 2001 | 2/3 | 6/22 | 0.26 | 5.33 [0.41, 70.20] |
| Asch 2002 | 23/63 | 15/111 | 1.20 | 3.68 [1.74, 7.77] |
| Buckley 2002 | 88/129 | 64/206 | 1.50 | 4.76 [2.97, 7.65] |
| Noerdlinger 2002 | 4/10 | 4/5 | 0.27 | 0.17 [0.01, 2.09] |
| Novak 2002 | 15/67 | 15/54 | 1.12 | 0.75 [0.33, 1.72] |
| Shinners 2002 | 2/11 | 1/30 | 0.27 | 6.44 [0.52, 79.64] |
| Spangehl 2002 | 13/23 | 4/28 | 0.69 | 7.80 [2.04, 29.84] |
| Jebson 2003 | 1/6 | 2/14 | 0.25 | 1.20 [0.09, 16.44] |
| Madan 2003 | 11/21 | 4/53 | 0.70 | 13.48 [3.56, 51.01] |
| Total (95% CI) | 7238 | 13250 | 100.00 | 3.79 [3.28, 4.37] |

Total events: 2671 (Compensated), 2143 (Not compensated)
Test for heterogeneity: Chi² = 308.80, df = 127 (P < 0.00001), I² = 58.9%
Test for overall effect: Z = 18.35 (P < 0.00001)

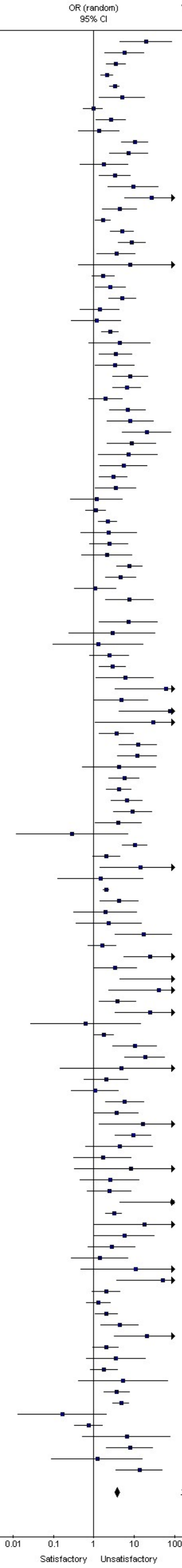
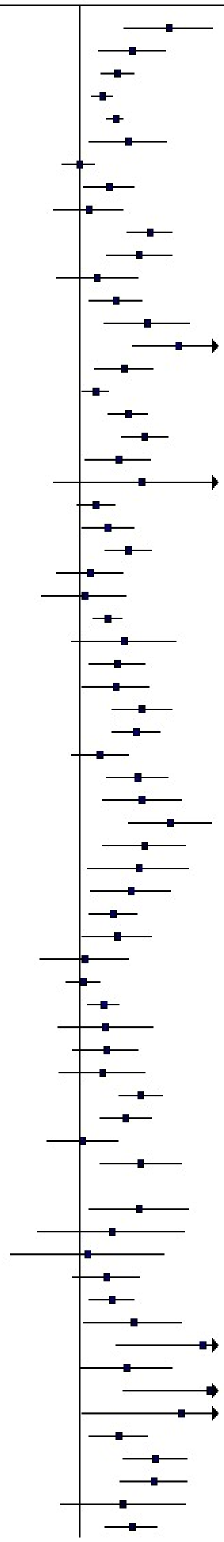
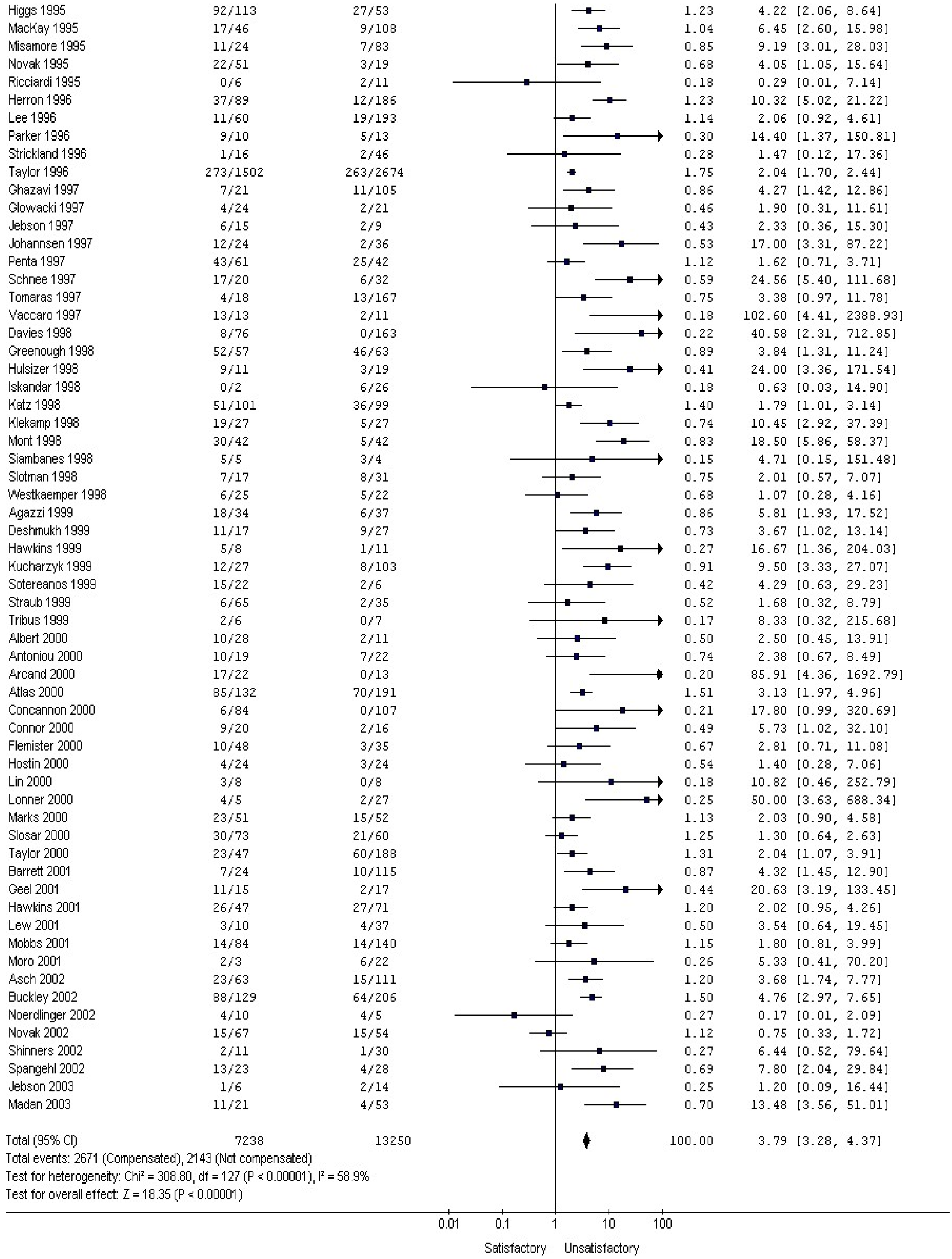


Table 2.3a. Enlarged overall forest plot, top half.



| Study | Compensated n/N | Not compensated n/N | OR (random) 95% CI | Weight % | OR (random) 95% CI |
|---|---|---|---|---|---|
| Campbell 1947 | 19/52 | 2/70 | | 0.59 | 19.58 [4.30, 89.08] |
| Greenwood 1952 | 13/23 | 8/43 | | 0.84 | 5.69 [1.84, 17.55] |
| Raaf 1959 | 43/224 | 20/316 | | 1.40 | 3.52 [2.00, 6.17] |
| Gurdjian 1961 | 71/177 | 178/738 | | 1.63 | 2.11 [1.49, 2.97] |
| Slepian 1966 | 158/474 | 93/704 | | 1.67 | 3.28 [2.46, 4.39] |
| Aronson 1968 | 8/24 | 4/44 | | 0.70 | 5.00 [1.32, 18.96] |
| DePalma 1972 | 31/86 | 53/143 | | 1.41 | 0.96 [0.55, 1.67] |
| Stauffer 1972 | 15/25 | 50/138 | | 1.07 | 2.64 [1.10, 6.32] |
| White 1973 | 6/16 | 15/49 | | 0.80 | 1.36 [0.42, 4.43] |
| Hudgins 1974 | 52/73 | 14/73 | | 1.17 | 10.44 [4.82, 22.59] |
| Burke 1976 | 15/21 | 17/67 | | 0.87 | 7.35 [2.46, 21.98] |
| Graham 1976 | 12/16 | 15/24 | | 0.65 | 1.80 [0.44, 7.31] |
| LaMont 1976 | 12/29 | 17/96 | | 1.04 | 3.28 [1.33, 8.12] |
| Law 1978 | 26/29 | 11/23 | | 0.63 | 9.45 [2.22, 40.24] |
| Finnegan 1979 | 10/16 | 3/51 | | 0.58 | 26.67 [5.69, 124.93] |
| Javid 1980 | 11/20 | 23/104 | | 0.96 | 4.30 [1.59, 11.64] |
| Weir 1980 | 39/162 | 62/398 | | 1.52 | 1.72 [1.09, 2.70] |
| Rish 1984 | 55/115 | 14/90 | | 1.28 | 4.98 [2.53, 9.80] |
| Strefling 1984 | 43/105 | 9/123 | | 1.16 | 8.78 [4.02, 19.21] |
| Javid 1985 | 7/18 | 13/87 | | 0.85 | 3.62 [1.19, 11.06] |
| Dellon 1986 | 6/40 | 0/20 | | 0.21 | 7.72 [0.41, 144.36] |
| Maciunas 1986 | 17/61 | 33/180 | | 1.28 | 1.72 [0.88, 3.38] |
| Sutton 1986 | 10/26 | 32/163 | | 1.07 | 2.56 [1.06, 6.17] |
| Branch 1987 | 32/82 | 10/90 | | 1.15 | 5.12 [2.32, 11.32] |
| Bray 1987 | 13/26 | 10/24 | | 0.85 | 1.40 [0.46, 4.28] |
| Friedman 1987 | 4/13 | 8/29 | | 0.64 | 1.17 [0.28, 4.88] |
| Hill 1987 | 45/129 | 36/206 | | 1.46 | 2.53 [1.52, 4.21] |
| Ritts 1987 | 6/8 | 11/27 | | 0.47 | 4.36 [0.74, 25.74] |
| Christie 1988 | 12/23 | 21/87 | | 0.99 | 3.43 [1.32, 8.90] |
| Hawkins 1988 | 8/35 | 6/73 | | 0.83 | 3.31 [1.05, 10.44] |
| Herron 1988 | 18/34 | 7/57 | | 0.92 | 8.04 [2.84, 22.71] |
| Javid 1988 | 17/42 | 16/172 | | 1.14 | 6.63 [2.97, 14.80] |
| Silvers 1988 | 9/199 | 8/341 | | 0.98 | 1.97 [0.75, 5.20] |
| Zucherman 1988 | 23/38 | 7/39 | | 0.91 | 7.01 [2.47, 19.93] |
| Hanley 1989 | 16/26 | 4/24 | | 0.70 | 8.00 [2.11, 30.34] |
| Hanley 1989 1 | 8/16 | 4/87 | | 0.65 | 20.75 [5.10, 84.36] |
| Hawkins 1989 | 32/38 | 5/13 | | 0.65 | 8.53 [2.07, 35.21] |
| Snyder 1989 | 6/8 | 14/47 | | 0.50 | 7.07 [1.27, 39.41] |
| Biondi 1990 | 20/30 | 4/15 | | 0.67 | 5.50 [1.39, 21.71] |
| Gartsman 1990 | 17/56 | 12/96 | | 1.11 | 3.05 [1.33, 7.00] |
| Ogilvie-Harris 1990 | 15/28 | 6/24 | | 0.80 | 3.46 [1.06, 11.33] |
| Paulos 1990 | 3/18 | 7/48 | | 0.61 | 1.17 [0.27, 5.13] |
| Silvers 1990 | 27/82 | 43/141 | | 1.38 | 1.12 [0.62, 2.01] |
| Davis 1991 | 23/91 | 56/427 | | 1.42 | 2.24 [1.29, 3.88] |
| Ellman 1991 | 3/17 | 4/48 | | 0.54 | 2.36 [0.47, 11.83] |
| Green 1991 | 16/25 | 12/28 | | 0.86 | 2.37 [0.78, 7.18] |
| Schaffer 1991 | 3/16 | 8/80 | | 0.63 | 2.08 [0.49, 8.88] |
| Tregonning 1991 | 56/66 | 72/170 | | 1.21 | 7.62 [3.64, 15.95] |
| Javid 1992 | 13/38 | 14/140 | | 1.08 | 4.68 [1.96, 11.15] |
| Lauerman 1992 | 13/22 | 12/21 | | 0.78 | 1.08 [0.32, 3.64] |
| Albert 1993 | 6/11 | 9/66 | | 0.67 | 7.60 [1.91, 30.19] |
| Hanel 1993 | 0/6 | 0/4 | | | Not estimable |
| Jerosch 1993 | 19/21 | 13/23 | | 0.51 | 7.31 [1.37, 38.98] |
| Knox 1993 | 4/5 | 7/12 | | 0.28 | 2.86 [0.24, 33.90] |
| Rotman 1993 | 7/18 | 1/3 | | 0.26 | 1.27 [0.10, 16.81] |
| Silvers 1993 | 5/44 | 10/200 | | 0.84 | 2.44 [0.79, 7.52] |
| Greenough 1994 | 58/85 | 17/40 | | 1.17 | 2.91 [1.34, 6.31] |
| Hutchinson 1994 | 10/26 | 2/21 | | 0.52 | 5.94 [1.13, 31.15] |
| Mont 1994 | 10/10 | 16/63 | | 0.21 | 60.45 [3.35, 1089.73] |
| Myerson 1994 | 8/11 | 9/25 | | 0.57 | 4.74 [1.00, 22.52] |
| Olsewski 1994 | 12/24 | 0/37 | | 0.21 | 75.00 [4.13, 1360.71] |
| Paulos 1994 | 2/4 | 0/14 | | 0.17 | 29.00 [1.05, 801.98] |
| Polk 1994 | 19/34 | 10/39 | | 0.96 | 3.67 [1.37, 9.86] |
| Silvers 1994 | 19/29 | 7/53 | | 0.86 | 12.49 [4.14, 37.66] |
| al-Qattan 1994 | 13/34 | 5/99 | | 0.84 | 11.64 [3.74, 36.20] |
| Frieman 1995 | 12/57 | 1/17 | | 0.36 | 4.27 [0.51, 35.48] |
| Hallock 1995 | 41/52 | 19/48 | | 1.06 | 5.69 [2.36, 13.74] |

Figure 2.3b. Enlarged overall forest plot, bottom half.




Full references are provided in Appendix 1.

Odds ratios are given in the tables and are the preferred method of reporting the effect estimate in this study. However, some information may be gained by examining the alternative forms of summarizing the effect estimate. From RevMan, the overall relative risk (RR) for a poor outcome in compensated patients was 2.26 (95% CI: 2.06 – 2.48, random effects model). The risk difference (RD) was 0.25 (95% CI: 0.22 – 0.28, random effects model).

The risk difference may be used to calculate the attributable risk percent. This is the proportion of poor outcomes in the compensated group that are attributable to the exposure variable (compensation). Using the pooled data, the incidence of poor outcomes in the compensated and uncompensated groups is 36.9% (2,671 / 7,238) and 16.2% (2,143 / 13,250), respectively. Therefore, the attributable risk percent is 56.2%. This indicates that over half of the poor outcomes seen in the compensated group can be attributed to their compensation status.

As RevMan is unable to perform meta-regression, this was performed separately using different statistical software.[343] The meta-regression was performed using Bayesian methods, and the overall effect size was larger, and with a narrower confidence interval, as expected. The overall effect size using these methods was an odds ratio of 4.06 (95% CI: 3.49 to 4.78) for a poor outcome in compensated patients, a relative risk of 2.71 (95%CI: 2.49 to 2.97), and a risk difference of 0.28.

### 2.3.3 Sub-group analyses and heterogeneity

Sub-group analyses were performed to examine possible causes of heterogeneity. The positive association between compensation and unsatisfactory outcome was seen in all sub-groups, with little variation in the magnitude of the association. The results for each sub-group are summarised in Table 2.4, and forest plots are provided where indicated. The significance of the difference between subgroups was analysed by meta-regression and these results are provided below.

Analysis according to study type (Figures 2.4 – 2.6) showed a stronger association in randomised controlled trials, compared to either cohort studies or case series. However, there were only two randomised controlled trials in the analysis, which meant that the confidence interval for the effect estimate was wide and the difference seen between study types was likely to be due to chance. This variable was also analysed on meta-regression and the difference was found to be not significant. Although the use of randomisation may indicate better methodology, it must be remembered that the exposure variable being analysed (compensation status) was not the variable randomised.

Table 2.4. Sub-group analyses of the association between compensation status and unsatisfactory outcome.

| Subgroup | Studies | Odds ratio | 95% C.I. |
|---|---|---|---|
| ***Study type*** | | | |
| Randomised controlled trial | 2 | 5.03 | 3.22 – 7.86 |
| Cohort study | 30 | 3.58 | 2.74 – 4.67 |
| Case series | 97* | 3.87 | 3.25 – 4.61 |
| ***Minimum time to follow-up*** | | | |
| 0 – 6 months | 21 | 3.81 | 2.72 – 5.34 |
| 7 – 12 months | 30 | 4.02 | 3.08 – 5.25 |
| 13 – 24 months | 34* | 4.36 | 3.17 – 6.01 |
| Over 24 months | 30 | 3.44 | 2.60 – 4.55 |
| ***Completeness of follow-up*** | | | |
| 80% or more | 111* | 3.84 | 3.30 – 4.47 |
| Less than 80% | 18 | 3.61 | 2.39 – 5.47 |
| ***Prospective versus retrospective*** | | | |
| Prospective | 15 | 3.60 | 2.70 – 4.80 |
| Retrospective | 114* | 3.84 | 3.27 – 4.50 |
| ***Procedure*** | | | |
| Lumbar spine discectomy | 24 | 4.77 | 3.51 – 6.50 |
| Lumbar spine fusion | 19 | 4.33 | 2.81 – 6.62 |
| Shoulder acromioplasty | 13 | 4.48 | 2.71 – 7.40 |
| Carpal tunnel decompression | 10 | 4.24 | 2.43 – 7.40 |
| Lumbar intradiscal chymopapain injection | 9 | 3.67 | 2.45 – 5.51 |
| ***Country of origin*** | | | |
| U.S.A. | 106* | 3.77 | 3.20 – 4.43 |
| Canada | 12 | 4.02 | 2.65 – 6.09 |
| All Europe | 6 | 7.42 | 4.37 – 12.60 |
| Australia | 5 | 2.23 | 1.49 – 3.35 |
| ***Study designed to assess compensation effect*** | | | |
| Yes | 16 | 3.60 | 2.50 – 5.20 |
| No | 113* | 3.85 | 3.29 – 4.51 |
| ***Compensation type*** | | | |
| Worker’s compensation only | 86* | 3.89 | 3.26 – 4.64 |
| Worker’s compensation and litigation | 43 | 3.69 | 2.88 – 4.73 |
| ***Revision versus primary surgery*** | | | |
| Primary surgery only | 81 | 3.66 | 3.07 – 4.36 |
| Revision surgery only | 19 | 5.54 | 3.47 – 8.83 |

*includes one study with an unestimatable odds ratio (no unsatisfactory outcomes)

Figure 2.4. Forest plot of results according to the study type for randomised controlled trials.

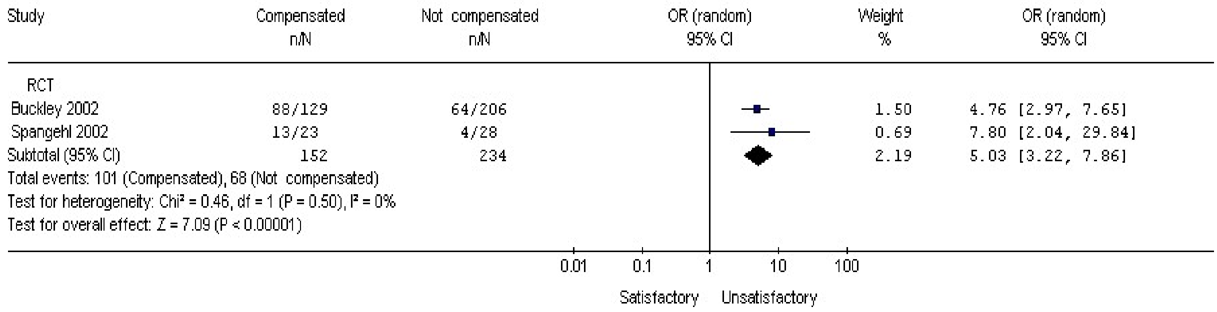



Figure 2.5. Forest plot of results according to the study type for cohort studies.

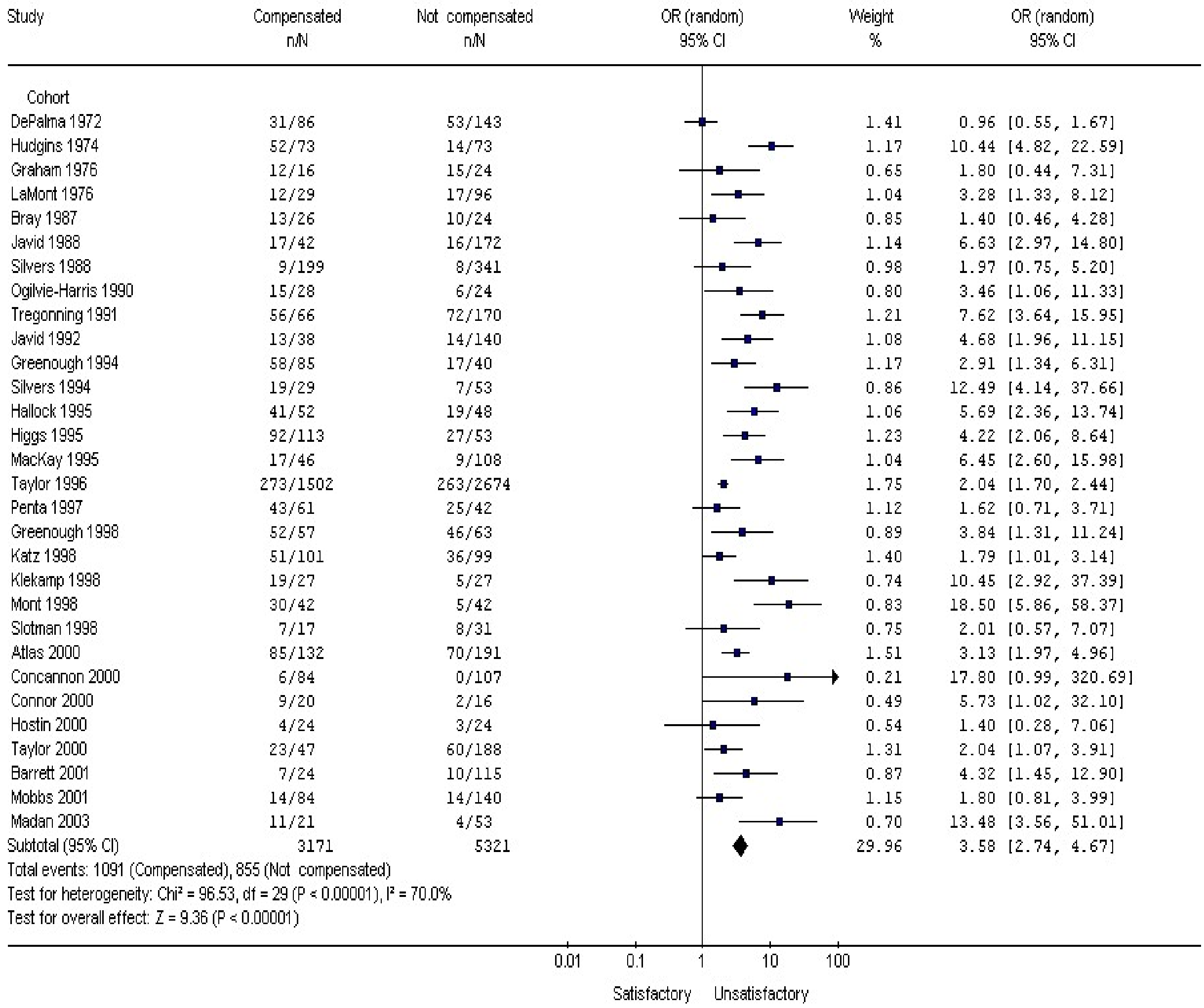
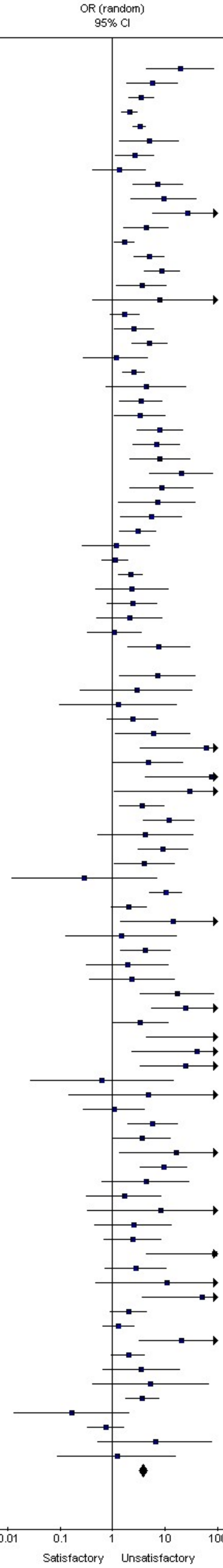


Figure 2.6. Forest plot of results according to the study type for case series.

| Study | Compensated n/N | Not compensated n/N | Weight % | OR (random) 95% CI |
|---|---|---|---|---|
| **Case series** | | | | |
| Campbell 1947 | 19/52 | 2/70 | 0.59 | 19.58 [4.30, 89.08] |
| Greenwood 1952 | 13/23 | 8/43 | 0.84 | 5.69 [1.84, 17.55] |
| Raaf 1959 | 43/224 | 20/316 | 1.40 | 3.52 [2.00, 6.17] |
| Gurdjian 1961 | 71/177 | 178/738 | 1.63 | 2.11 [1.49, 2.97] |
| Slepian 1966 | 158/474 | 93/704 | 1.67 | 3.28 [2.46, 4.39] |
| Aronson 1968 | 8/24 | 4/44 | 0.70 | 5.00 [1.32, 18.96] |
| Stauffer 1972 | 15/25 | 50/138 | 1.07 | 2.64 [1.10, 6.32] |
| White 1973 | 6/16 | 15/49 | 0.80 | 1.36 [0.42, 4.43] |
| Burke 1976 | 15/21 | 17/67 | 0.87 | 7.35 [2.46, 21.98] |
| Law 1978 | 26/29 | 11/23 | 0.63 | 9.45 [2.22, 40.24] |
| Finnegan 1979 | 10/16 | 3/51 | 0.58 | 26.67 [5.69, 124.93] |
| Javid 1980 | 11/20 | 23/104 | 0.96 | 4.30 [1.59, 11.64] |
| Weir 1980 | 39/162 | 62/398 | 1.52 | 1.72 [1.09, 2.70] |
| Rish 1984 | 55/115 | 14/90 | 1.28 | 4.98 [2.53, 9.80] |
| Strefling 1984 | 43/105 | 9/123 | 1.16 | 8.78 [4.02, 19.21] |
| Javid 1985 | 7/18 | 13/87 | 0.85 | 3.62 [1.19, 11.06] |
| Dellon 1986 | 6/40 | 0/20 | 0.21 | 7.72 [0.41, 144.36] |
| Maciunas 1986 | 17/61 | 33/180 | 1.28 | 1.72 [0.88, 3.38] |
| Sutton 1986 | 10/26 | 32/163 | 1.07 | 2.56 [1.06, 6.17] |
| Branch 1987 | 32/82 | 10/90 | 1.15 | 5.12 [2.32, 11.32] |
| Friedman 1987 | 4/13 | 8/29 | 0.64 | 1.17 [0.28, 4.88] |
| Hill 1987 | 45/129 | 36/206 | 1.46 | 2.53 [1.52, 4.21] |
| Ritts 1987 | 6/8 | 11/27 | 0.47 | 4.36 [0.74, 25.74] |
| Christie 1988 | 12/23 | 21/87 | 0.99 | 3.43 [1.32, 8.90] |
| Hawkins 1988 | 8/35 | 6/73 | 0.83 | 3.31 [1.05, 10.44] |
| Herron 1988 | 18/34 | 7/57 | 0.92 | 8.04 [2.84, 22.71] |
| Zucherman 1988 | 23/38 | 7/39 | 0.91 | 7.01 [2.47, 19.93] |
| Hanley 1989 | 16/26 | 4/24 | 0.70 | 8.00 [2.11, 30.34] |
| Hanley 1989 1 | 8/16 | 4/87 | 0.65 | 20.75 [5.10, 84.36] |
| Hawkins 1989 | 32/38 | 5/13 | 0.65 | 8.53 [2.07, 35.21] |
| Snyder 1989 | 6/8 | 14/47 | 0.50 | 7.07 [1.27, 39.41] |
| Biondi 1990 | 20/30 | 4/15 | 0.67 | 5.50 [1.39, 21.71] |
| Gartsman 1990 | 17/56 | 12/96 | 1.11 | 3.05 [1.33, 7.00] |
| Paulos 1990 | 3/18 | 7/48 | 0.61 | 1.17 [0.27, 5.13] |
| Silvers 1990 | 27/82 | 43/141 | 1.38 | 1.12 [0.62, 2.01] |
| Davis 1991 | 23/91 | 56/427 | 1.42 | 2.24 [1.29, 3.88] |
| Ellman 1991 | 3/17 | 4/48 | 0.54 | 2.36 [0.47, 11.83] |
| Green 1991 | 16/25 | 12/28 | 0.86 | 2.37 [0.78, 7.18] |
| Schaffer 1991 | 3/16 | 8/80 | 0.63 | 2.08 [0.49, 8.88] |
| Lauerman 1992 | 13/22 | 12/21 | 0.78 | 1.08 [0.32, 3.64] |
| Albert 1993 | 6/11 | 9/66 | 0.67 | 7.60 [1.91, 30.19] |
| Hanel 1993 | 0/6 | 0/4 | | Not estimable |
| Jerosch 1993 | 19/21 | 13/23 | 0.51 | 7.31 [1.37, 38.98] |
| Knox 1993 | 4/5 | 7/12 | 0.28 | 2.86 [0.24, 33.90] |
| Rotman 1993 | 7/18 | 1/3 | 0.26 | 1.27 [0.10, 16.81] |
| Silvers 1993 | 5/44 | 10/200 | 0.84 | 2.44 [0.79, 7.52] |
| Hutchinson 1994 | 10/26 | 2/21 | 0.52 | 5.94 [1.13, 31.15] |
| Mont 1994 | 10/10 | 16/63 | 0.21 | 60.45 [3.35, 1089.73] |
| Myerson 1994 | 8/11 | 9/25 | 0.57 | 4.74 [1.00, 22.52] |
| Olsewski 1994 | 12/24 | 0/37 | 0.21 | 75.00 [4.13, 1360.71] |
| Paulos 1994 | 2/4 | 0/14 | 0.17 | 29.00 [1.05, 801.98] |
| Polk 1994 | 19/34 | 10/39 | 0.96 | 3.67 [1.37, 9.86] |
| al-Qattan 1994 | 13/34 | 5/99 | 0.84 | 11.64 [3.74, 36.20] |
| Frieman 1995 | 12/57 | 1/17 | 0.36 | 4.27 [0.51, 35.48] |
| Misamore 1995 | 11/24 | 7/83 | 0.85 | 9.19 [3.01, 28.03] |
| Novak 1995 | 22/51 | 3/19 | 0.68 | 4.05 [1.05, 15.64] |
| Ricciardi 1995 | 0/6 | 2/11 | 0.18 | 0.29 [0.01, 7.14] |
| Herron 1996 | 37/89 | 12/186 | 1.23 | 10.32 [5.02, 21.22] |
| Lee 1996 | 11/60 | 19/193 | 1.14 | 2.06 [0.92, 4.61] |
| Parker 1996 | 9/10 | 5/13 | 0.30 | 14.40 [1.37, 150.81] |
| Strickland 1996 | 1/16 | 2/46 | 0.28 | 1.47 [0.12, 17.36] |
| Ghazavi 1997 | 7/21 | 11/105 | 0.86 | 4.27 [1.42, 12.86] |
| Glowacki 1997 | 4/24 | 2/21 | 0.46 | 1.90 [0.31, 11.61] |
| Jebson 1997 | 6/15 | 2/9 | 0.43 | 2.33 [0.36, 15.30] |
| Johannsen 1997 | 12/24 | 2/36 | 0.53 | 17.00 [3.31, 87.22] |
| Schnee 1997 | 17/20 | 6/32 | 0.59 | 24.56 [5.40, 111.68] |
| Tomaras 1997 | 4/18 | 13/167 | 0.75 | 3.38 [0.97, 11.78] |
| Vaccaro 1997 | 13/13 | 2/11 | 0.18 | 102.60 [4.41, 2388.93] |
| Davies 1998 | 8/76 | 0/163 | 0.22 | 40.58 [2.31, 712.85] |
| Hulsizer 1998 | 9/11 | 3/19 | 0.41 | 24.00 [3.36, 171.54] |
| Iskandar 1998 | 0/2 | 6/26 | 0.18 | 0.63 [0.03, 14.90] |
| Siambanes 1998 | 5/5 | 3/4 | 0.15 | 4.71 [0.15, 151.48] |
| Westkaemper 1998 | 6/25 | 5/22 | 0.68 | 1.07 [0.28, 4.16] |
| Agazzi 1999 | 18/34 | 6/37 | 0.86 | 5.81 [1.93, 17.52] |
| Deshmukh 1999 | 11/17 | 9/27 | 0.73 | 3.67 [1.02, 13.14] |
| Hawkins 1999 | 5/8 | 1/11 | 0.27 | 16.67 [1.36, 204.03] |
| Kucharzyk 1999 | 12/27 | 8/103 | 0.91 | 9.50 [3.33, 27.07] |
| Sotereanos 1999 | 15/22 | 2/6 | 0.42 | 4.29 [0.63, 29.23] |
| Straub 1999 | 6/65 | 2/35 | 0.52 | 1.68 [0.32, 8.79] |
| Tribus 1999 | 2/6 | 0/7 | 0.17 | 8.33 [0.32, 215.68] |
| Albert 2000 | 10/28 | 2/11 | 0.50 | 2.50 [0.45, 13.91] |
| Antoniou 2000 | 10/19 | 7/22 | 0.74 | 2.38 [0.67, 8.49] |
| Arcand 2000 | 17/22 | 0/13 | 0.20 | 85.91 [4.36, 1692.79] |
| Flemister 2000 | 10/48 | 3/35 | 0.67 | 2.81 [0.71, 11.08] |
| Lin 2000 | 3/8 | 0/8 | 0.18 | 10.82 [0.46, 252.79] |
| Lonner 2000 | 4/5 | 2/27 | 0.25 | 50.00 [3.63, 688.34] |
| Marks 2000 | 23/51 | 15/52 | 1.13 | 2.03 [0.90, 4.58] |
| Slosar 2000 | 30/73 | 21/60 | 1.25 | 1.30 [0.64, 2.63] |
| Geel 2001 | 11/15 | 2/17 | 0.44 | 20.63 [3.19, 133.45] |
| Hawkins 2001 | 26/47 | 27/71 | 1.20 | 2.02 [0.95, 4.26] |
| Lew 2001 | 3/10 | 4/37 | 0.50 | 3.54 [0.64, 19.45] |
| Moro 2001 | 2/3 | 6/22 | 0.26 | 5.33 [0.41, 70.20] |
| Asch 2002 | 23/63 | 15/111 | 1.20 | 3.68 [1.74, 7.77] |
| Noerdlinger 2002 | 4/10 | 4/5 | 0.27 | 0.17 [0.01, 2.09] |
| Novak 2002 | 15/67 | 15/54 | 1.12 | 0.75 [0.33, 1.72] |
| Shinners 2002 | 2/11 | 1/30 | 0.27 | 6.44 [0.52, 79.64] |
| Jebson 2003 | 1/6 | 2/14 | 0.25 | 1.20 [0.09, 16.44] |
| Subtotal (95% CI) | 3915 | 7695 | 67.85 | 3.85 [3.24, 4.58] |

Total events: 1479 (Compensated), 1220 (Not compensated)
Test for heterogeneity: Chi² = 201.55, df = 95 (P < 0.00001), I² = 52.9%
Test for overall effect: Z = 15.18 (P < 0.00001)



Analysis according to minimum time to follow-up revealed similar odds ratios for each of the four groups, with overlapping of all confidence intervals. This was also found to be not significant on meta-regression. Analysis according to completeness of follow-up (less than 80%, compared to 80% or more) revealed similar odds ratios and was also found to be not significant on meta-regression. Comparing prospective studies to retrospective studies also revealed similar odds ratios and was not significant on meta-regression.

Due to the association between compensation and injury, most studies dealt with orthopaedic, plastic and spinal surgery. The forest plots for the most common procedures are shown in Figures 2.7, 2.8, 2.9, 2.10, 2.11 and 2.12. The odds ratios for the six most common procedures (in order: lumbar discectomy, lumbar spine fusion, shoulder acromioplasty, carpal tunnel release, lumbar intradiscal injection of chymopapain, and cervical spine fusion) were similar. The differences in the effect for each of these treatment groups was not significant on meta-regression.

Analysis according to geographic origin showed a stronger association in European studies, and a weaker association in studies from Australia. Both of these groups, however, contained a small number of studies (Table 2.4, and Figures 2.13, 2.14, 2.15 and 2.16) and the differences were not significant on meta-regression.

Figure 2.7. Forest plot for subgroup analysis of lumbar discectomy.

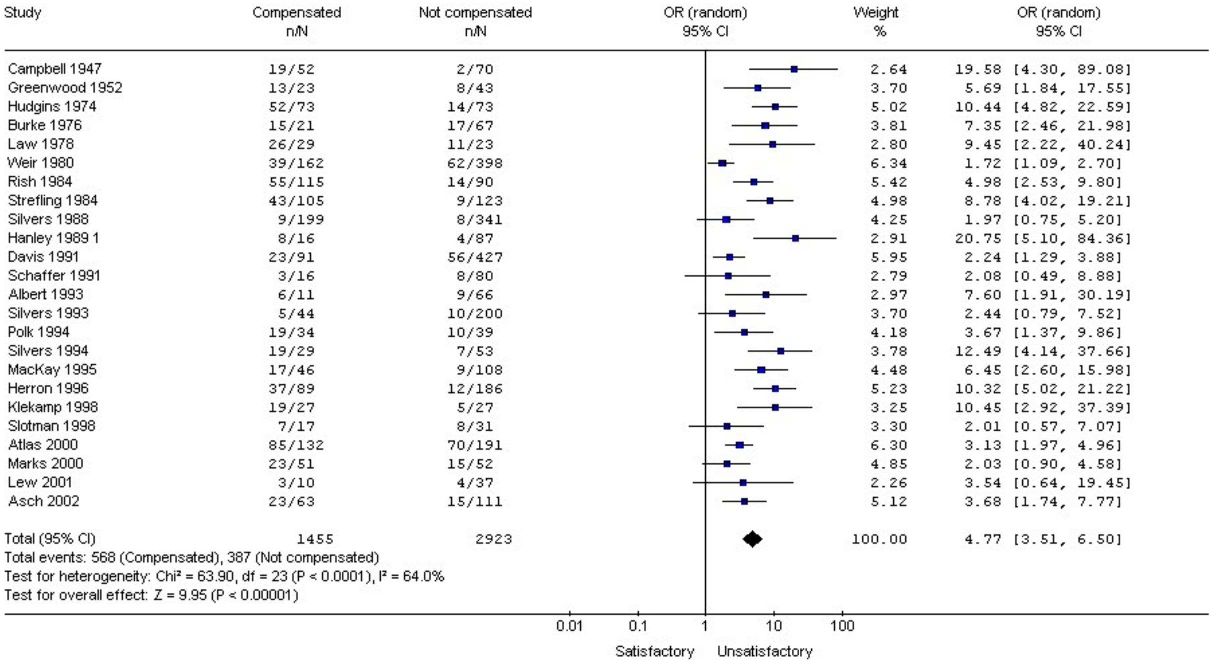



Figure 2.8. Forest plot for subgroup analysis of acromioplasty.

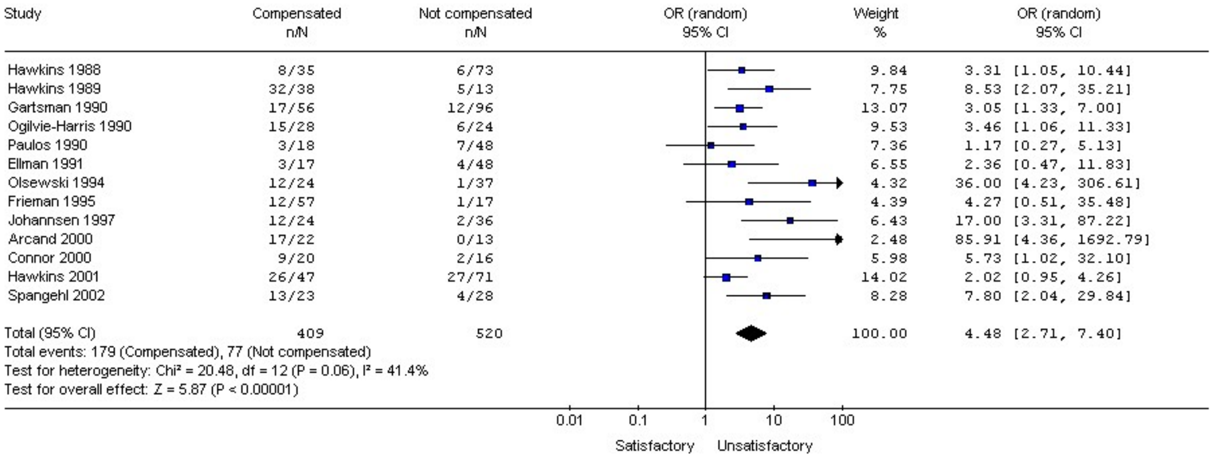



Figure 2.9. Forest plot for subgroup analysis of cervical spine fusion.

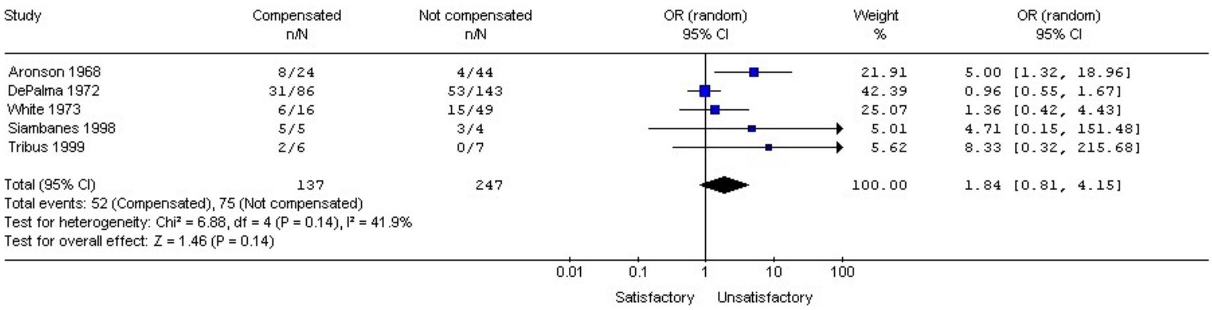


Figure 2.10. Forest plot for subgroup analysis of intradiscal chymopapain.

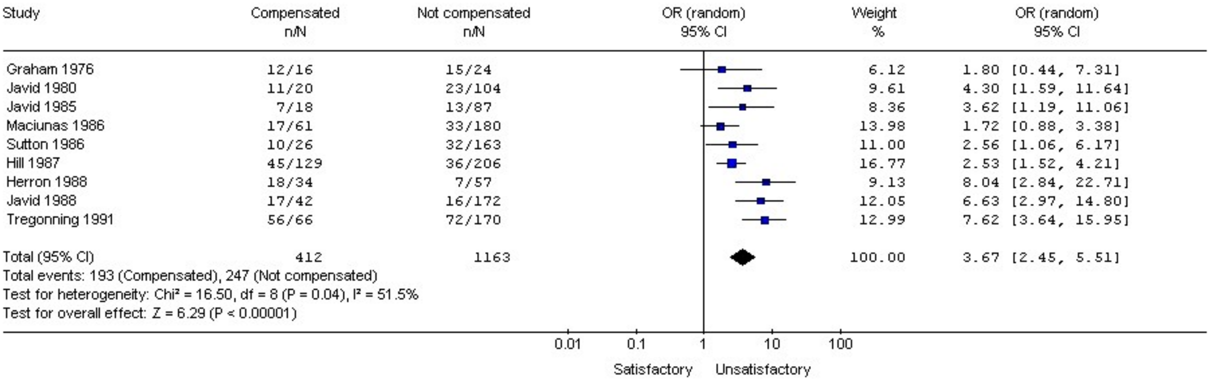



Figure 2.11. Forest plot for subgroup analysis of carpal tunnel decompression.

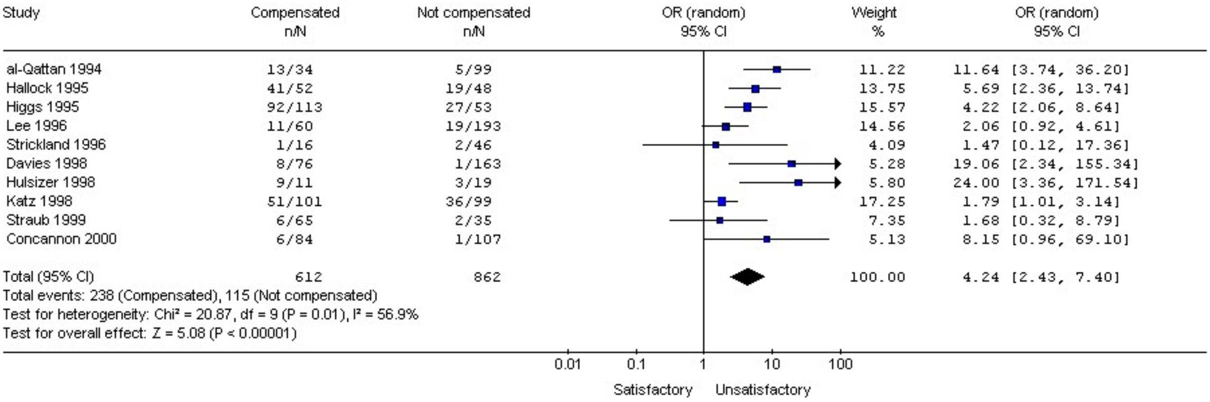



Figure 2.12. Forest plot for subgroup analysis of lumbar fusion.

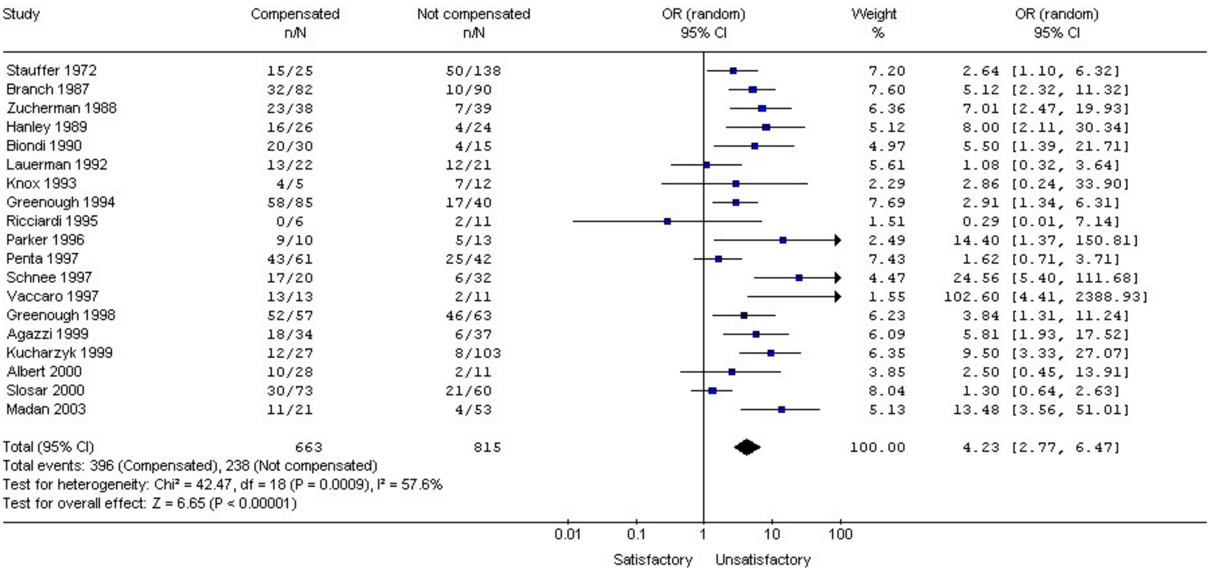
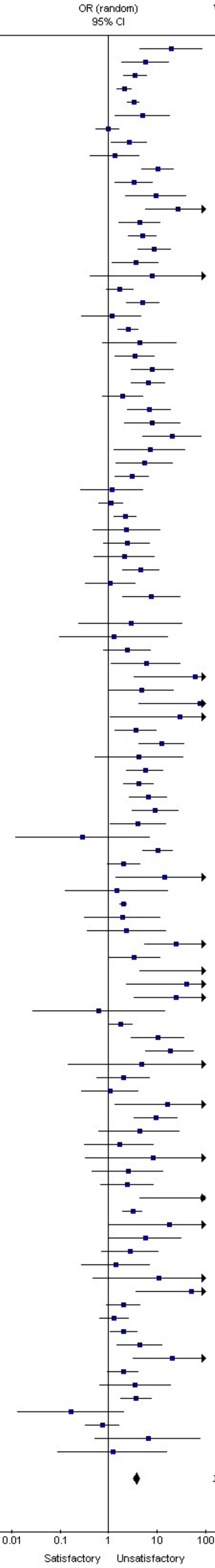


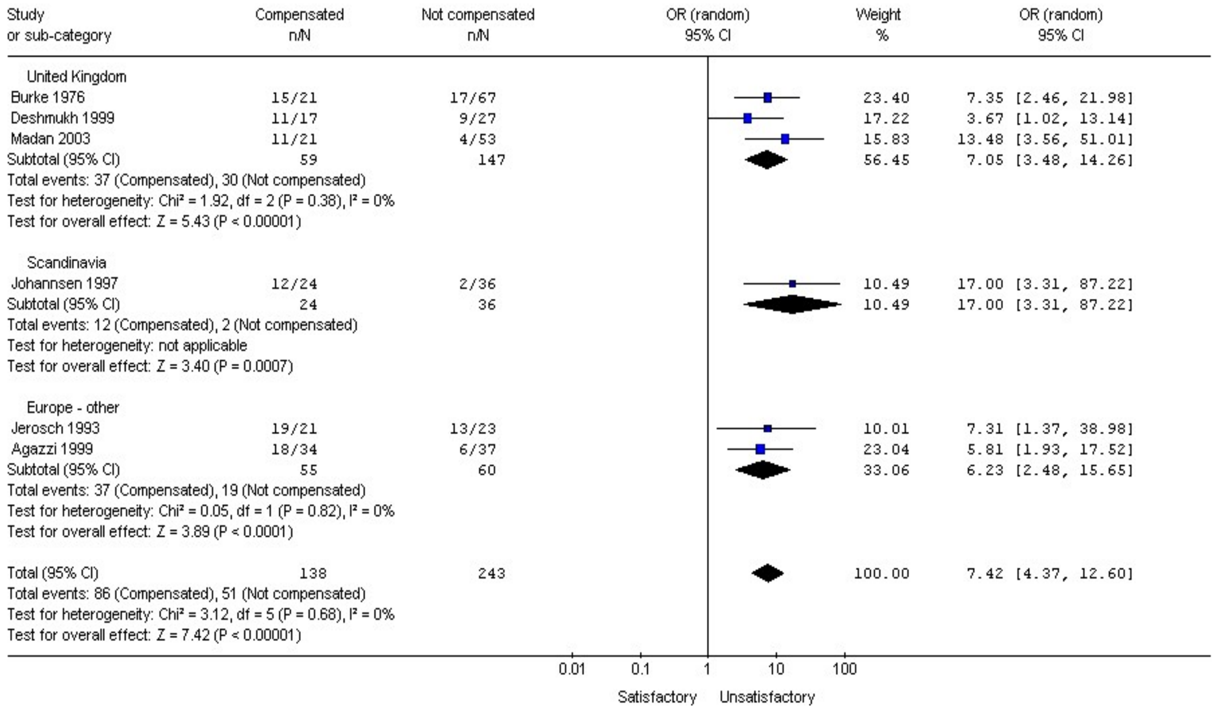
Figure 2.13. Forest plot for subgroup analysis of studies from the USA.

| Study | Compensated n/N | Not compensated n/N | Weight % | OR (random) 95% CI |
|---|---|---|---|---|
| Campbell 1947 | 19/52 | 2/70 | 0.75 | 19.58 [4.30, 89.08] |
| Greenwood 1952 | 13/23 | 8/43 | 1.06 | 5.69 [1.84, 17.55] |
| Raaf 1959 | 43/224 | 20/316 | 1.74 | 3.52 [2.00, 6.17] |
| Gurdjian 1961 | 71/177 | 178/738 | 2.00 | 2.11 [1.49, 2.97] |
| Slepian 1966 | 158/474 | 93/704 | 2.06 | 3.28 [2.46, 4.39] |
| Aronson 1968 | 8/24 | 4/44 | 0.88 | 5.00 [1.32, 18.96] |
| DePalma 1972 | 31/86 | 53/143 | 1.75 | 0.96 [0.55, 1.67] |
| Stauffer 1972 | 15/25 | 50/138 | 1.34 | 2.64 [1.10, 6.32] |
| White 1973 | 6/16 | 15/49 | 1.01 | 1.36 [0.42, 4.43] |
| Hudgins 1974 | 52/73 | 14/73 | 1.47 | 10.44 [4.82, 22.59] |
| LaMont 1976 | 12/29 | 17/96 | 1.30 | 3.28 [1.33, 8.12] |
| Law 1978 | 26/29 | 11/23 | 0.80 | 9.45 [2.22, 40.24] |
| Finnegan 1979 | 10/16 | 3/51 | 0.73 | 26.67 [5.69, 124.93] |
| Javid 1980 | 11/20 | 23/104 | 1.20 | 4.30 [1.59, 11.64] |
| Rish 1984 | 55/115 | 14/90 | 1.59 | 4.98 [2.53, 9.80] |
| Strefling 1984 | 43/105 | 9/123 | 1.45 | 8.78 [4.02, 19.21] |
| Javid 1985 | 7/18 | 13/87 | 1.07 | 3.62 [1.19, 11.06] |
| Dellon 1986 | 6/40 | 0/20 | 0.27 | 7.72 [0.41, 144.36] |
| Maciunas 1986 | 17/61 | 33/180 | 1.59 | 1.72 [0.88, 3.38] |
| Branch 1987 | 32/82 | 10/90 | 1.44 | 5.12 [2.32, 11.32] |
| Friedman 1987 | 4/13 | 8/29 | 0.81 | 1.17 [0.28, 4.88] |
| Hill 1987 | 45/129 | 36/206 | 1.81 | 2.53 [1.52, 4.21] |
| Ritts 1987 | 6/8 | 11/27 | 0.60 | 4.36 [0.74, 25.74] |
| Christie 1988 | 12/23 | 21/87 | 1.25 | 3.43 [1.32, 8.90] |
| Herron 1988 | 18/34 | 7/57 | 1.15 | 8.04 [2.84, 22.71] |
| Javid 1988 | 17/42 | 16/172 | 1.43 | 6.63 [2.97, 14.80] |
| Silvers 1988 | 9/199 | 8/341 | 1.23 | 1.97 [0.75, 5.20] |
| Zucherman 1988 | 23/38 | 7/39 | 1.15 | 7.01 [2.47, 19.93] |
| Hanley 1989 | 16/26 | 4/24 | 0.88 | 8.00 [2.11, 30.34] |
| Hanley 1989 1 | 8/16 | 4/87 | 0.83 | 20.75 [5.10, 84.36] |
| Snyder 1989 | 6/8 | 14/47 | 0.63 | 7.07 [1.27, 39.41] |
| Biondi 1990 | 20/30 | 4/15 | 0.85 | 5.50 [1.39, 21.71] |
| Gartsman 1990 | 17/56 | 12/96 | 1.39 | 3.05 [1.33, 7.00] |
| Paulos 1990 | 3/18 | 7/48 | 0.78 | 1.17 [0.27, 5.13] |
| Silvers 1990 | 27/82 | 43/141 | 1.71 | 1.12 [0.62, 2.01] |
| Davis 1991 | 23/91 | 56/427 | 1.75 | 2.24 [1.29, 3.88] |
| Ellman 1991 | 3/17 | 4/48 | 0.69 | 2.36 [0.47, 11.83] |
| Green 1991 | 16/25 | 12/28 | 1.08 | 2.37 [0.78, 7.18] |
| Schaffer 1991 | 3/16 | 8/80 | 0.79 | 2.08 [0.49, 8.88] |
| Javid 1992 | 13/38 | 14/140 | 1.35 | 4.68 [1.96, 11.15] |
| Lauerman 1992 | 13/22 | 12/21 | 0.98 | 1.08 [0.32, 3.64] |
| Albert 1993 | 6/11 | 9/66 | 0.85 | 7.60 [1.91, 30.19] |
| Hanel 1993 | 0/6 | 0/4 | | Not estimable |
| Knox 1993 | 4/5 | 7/12 | 0.36 | 2.86 [0.24, 33.90] |
| Rotman 1993 | 7/18 | 1/3 | 0.33 | 1.27 [0.10, 16.81] |
| Silvers 1993 | 5/44 | 10/200 | 1.06 | 2.44 [0.79, 7.52] |
| Hutchinson 1994 | 10/26 | 2/21 | 0.66 | 5.94 [1.13, 31.15] |
| Mont 1994 | 10/10 | 16/63 | 0.27 | 60.45 [3.35, 1089.73] |
| Myerson 1994 | 8/11 | 9/25 | 0.72 | 4.74 [1.00, 22.52] |
| Olsewski 1994 | 12/24 | 0/37 | 0.27 | 75.00 [4.13, 1360.71] |
| Paulos 1994 | 2/4 | 0/14 | 0.21 | 29.00 [1.05, 801.98] |
| Polk 1994 | 19/34 | 10/39 | 1.21 | 3.67 [1.37, 9.86] |
| Silvers 1994 | 19/29 | 7/53 | 1.09 | 12.49 [4.14, 37.66] |
| Frieman 1995 | 12/57 | 1/17 | 0.46 | 4.27 [0.51, 35.48] |
| Hallock 1995 | 41/52 | 19/48 | 1.33 | 5.69 [2.36, 13.74] |
| Higgs 1995 | 92/113 | 27/53 | 1.54 | 4.22 [2.06, 8.64] |
| MacKay 1995 | 17/46 | 9/108 | 1.30 | 6.45 [2.60, 15.98] |
| Misamore 1995 | 11/24 | 7/83 | 1.07 | 9.19 [3.01, 28.03] |
| Novak 1995 | 22/51 | 3/19 | 0.87 | 4.05 [1.05, 15.64] |
| Ricciardi 1995 | 0/6 | 2/11 | 0.23 | 0.29 [0.01, 7.14] |
| Herron 1996 | 37/89 | 12/186 | 1.53 | 10.32 [5.02, 21.22] |
| Lee 1996 | 11/60 | 19/193 | 1.42 | 2.06 [0.92, 4.61] |
| Parker 1996 | 9/10 | 5/13 | 0.39 | 14.40 [1.37, 150.81] |
| Strickland 1996 | 1/16 | 2/46 | 0.36 | 1.47 [0.12, 17.36] |
| Taylor 1996 | 273/1502 | 263/2674 | 2.14 | 2.04 [1.70, 2.44] |
| Glowacki 1997 | 4/24 | 2/21 | 0.59 | 1.90 [0.31, 11.61] |
| Jebson 1997 | 6/15 | 2/9 | 0.55 | 2.33 [0.36, 15.30] |
| Schnee 1997 | 17/20 | 6/32 | 0.75 | 24.56 [5.40, 111.68] |
| Tomaras 1997 | 4/18 | 13/167 | 0.95 | 3.38 [0.97, 11.78] |
| Vaccaro 1997 | 13/13 | 2/11 | 0.24 | 102.60 [4.41, 2388.93] |
| Davies 1998 | 8/76 | 0/163 | 0.28 | 40.58 [2.31, 712.85] |
| Hulsizer 1998 | 9/11 | 3/19 | 0.52 | 24.00 [3.36, 171.54] |
| Iskandar 1998 | 0/2 | 6/26 | 0.23 | 0.63 [0.03, 14.90] |
| Katz 1998 | 51/101 | 36/99 | 1.73 | 1.79 [1.01, 3.14] |
| Klekamp 1998 | 19/27 | 5/27 | 0.93 | 10.45 [2.92, 37.39] |
| Mont 1998 | 30/42 | 5/42 | 1.04 | 18.50 [5.86, 58.37] |
| Siambanes 1998 | 5/5 | 3/4 | 0.20 | 4.71 [0.15, 151.48] |
| Slotman 1998 | 7/17 | 8/31 | 0.94 | 2.01 [0.57, 7.07] |
| Westkaemper 1998 | 6/25 | 5/22 | 0.86 | 1.07 [0.28, 4.16] |
| Hawkins 1999 | 5/8 | 1/11 | 0.35 | 16.67 [1.36, 204.03] |
| Kucharzyk 1999 | 12/27 | 8/103 | 1.14 | 9.50 [3.33, 27.07] |
| Sotereanos 1999 | 15/22 | 2/6 | 0.54 | 4.29 [0.63, 29.23] |
| Straub 1999 | 6/65 | 2/35 | 0.66 | 1.68 [0.32, 8.79] |
| Tribus 1999 | 2/6 | 0/7 | 0.22 | 8.33 [0.32, 215.68] |
| Albert 2000 | 10/28 | 2/11 | 0.63 | 2.50 [0.45, 13.91] |
| Antoniou 2000 | 10/19 | 7/22 | 0.93 | 2.38 [0.67, 8.49] |
| Arcand 2000 | 17/22 | 0/13 | 0.26 | 85.91 [4.36, 1692.79] |
| Atlas 2000 | 85/132 | 70/191 | 1.87 | 3.13 [1.97, 4.96] |
| Concannon 2000 | 6/84 | 0/107 | 0.27 | 17.80 [0.99, 320.69] |
| Connor 2000 | 9/20 | 2/16 | 0.63 | 5.73 [1.02, 32.10] |
| Flemister 2000 | 10/48 | 3/35 | 0.85 | 2.81 [0.71, 11.08] |
| Hostin 2000 | 4/24 | 3/24 | 0.69 | 1.40 [0.28, 7.06] |
| Lin 2000 | 3/8 | 0/8 | 0.23 | 10.82 [0.46, 252.79] |
| Lonner 2000 | 4/5 | 2/27 | 0.32 | 50.00 [3.63, 688.34] |
| Marks 2000 | 23/51 | 15/52 | 1.41 | 2.03 [0.90, 4.58] |
| Slosar 2000 | 30/73 | 21/60 | 1.55 | 1.30 [0.64, 2.63] |
| Taylor 2000 | 23/47 | 60/188 | 1.62 | 2.04 [1.07, 3.91] |
| Barrett 2001 | 7/24 | 10/115 | 1.10 | 4.32 [1.45, 12.90] |
| Geel 2001 | 11/15 | 2/17 | 0.56 | 20.63 [3.19, 133.45] |
| Hawkins 2001 | 26/47 | 27/71 | 1.50 | 2.02 [0.95, 4.26] |
| Lew 2001 | 3/10 | 4/37 | 0.64 | 3.54 [0.64, 19.45] |
| Asch 2002 | 23/63 | 15/111 | 1.50 | 3.68 [1.74, 7.77] |
| Noerdlinger 2002 | 4/10 | 4/5 | 0.34 | 0.17 [0.01, 2.09] |
| Novak 2002 | 15/67 | 15/54 | 1.40 | 0.75 [0.33, 1.72] |
| Shinners 2002 | 2/11 | 1/30 | 0.35 | 6.44 [0.52, 79.64] |
| Jebson 2003 | 1/6 | 2/14 | 0.32 | 1.20 [0.09, 16.44] |
| Total (95% CI) | 6206 | 11373 | 100.00 | 3.75 [3.19, 4.41] |

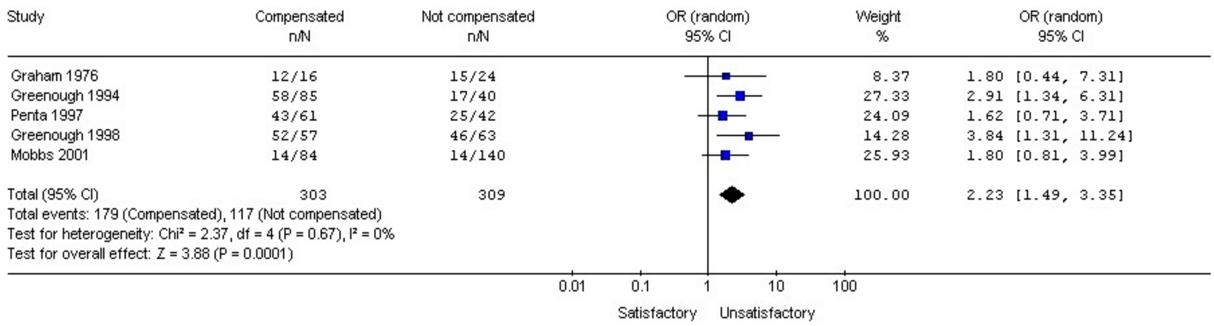
Total events: 2110 (Compensated), 1692 (Not compensated)
Test for heterogeneity: Chi² = 262.01, df = 104 (P < 0.00001), I² = 60.3%
Test for overall effect: Z = 16.03 (P < 0.00001)



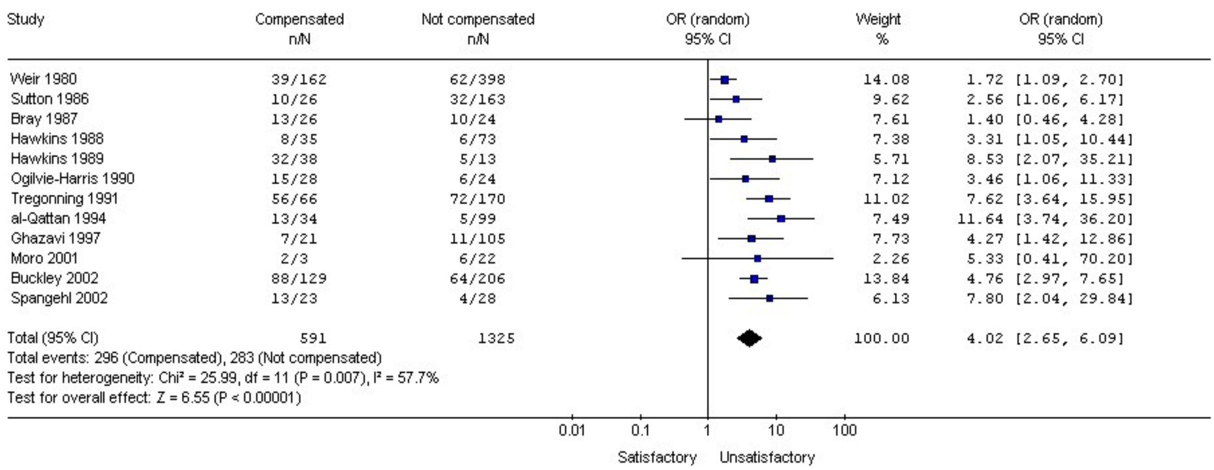
## Figure 2.14. Forest plot for subgroup analysis of studies from Europe.




## Figure 2.15. Forest plot for subgroup analysis of studies from Australia.




## Figure 2.16. Forest plot for subgroup analysis of studies from Canada.



The OR for studies looking only at worker's compensation patients (not litigation) was similar to studies looking at patients treated under worker's compensation or litigation (Table 2.4). There were no studies that examined only litigating patients (i.e., excluding workers' compensation). There was no significant difference on meta-regression.

The OR for studies looking specifically at the effect of compensation (i.e., studies designed as a compensation versus non-compensation cohort) was similar to the OR in studies not specifically designed to examine the effect of compensation (Table 2.4).

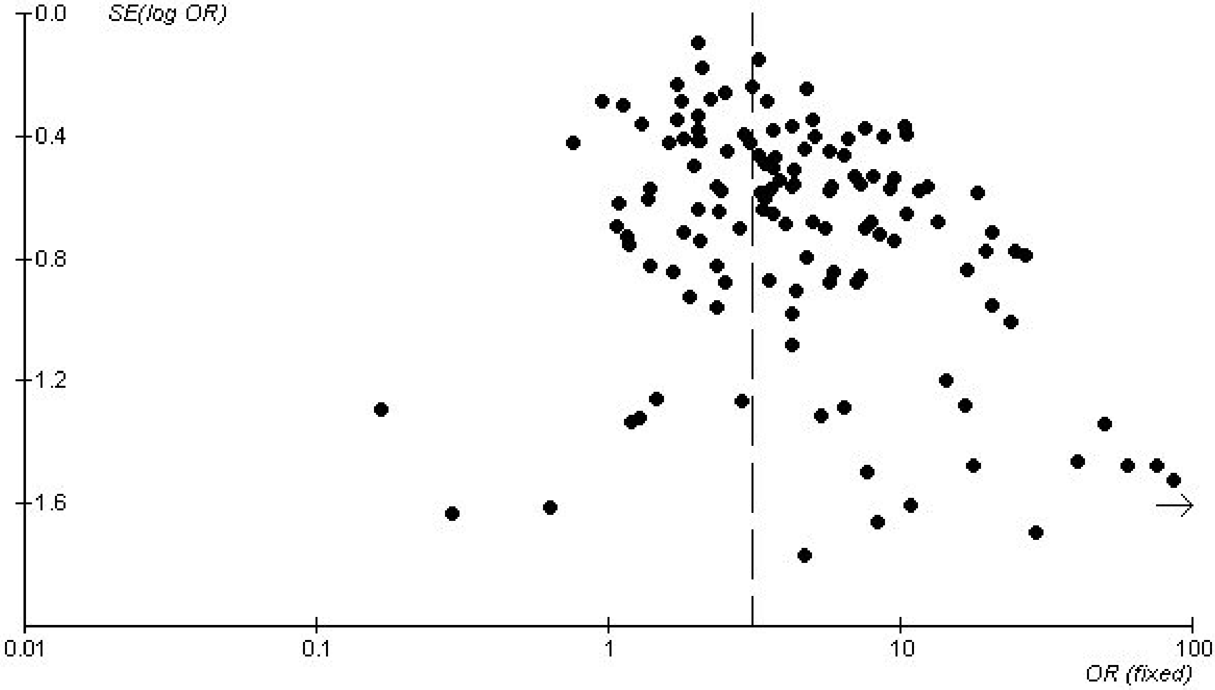
Univariate meta-regression of the sub-groups analysed above found them not to be significantly associated with the effect of compensation on the risk of an unsatisfactory outcome. On meta-regression, the year of publication was found not to be significantly associated with the effect size. The effect of sample size, however, was such that the effect size decreased with increasing sample size of the studies.

A post hoc analysis was performed to assess the effect of revision versus primary surgery (Table 2.4). This showed the association between compensation and poor outcome to be stronger in studies of revision surgery. Analysis of this variable by meta-regression, which included studies with mixed populations according to the ratio of revision to primary cases, showed this association to be strongly significant.

In general, the level of heterogeneity decreased in the sub-groups. Greater homogeneity was seen in the prospective studies ($p = 0.03$), randomised controlled trials ($p = 0.46$), and in the European and Australian studies ($p = 0.68$ and $p = 0.67$, respectively). Grouping by procedure also reduced heterogeneity, however the heterogeneity remained high for other sub-groups. A funnel plot of all included studies (Figure 2.17) revealed some asymmetry in studies with larger standard errors (smaller populations).

Of thirteen studies reporting continuous outcomes, ten noted a statistically significant association between compensation status and poor outcome, and three noted no significant difference. This is consistent with the findings of the studies with dichotomous outcomes. As only four of these studies provided the means and standard deviations necessary to calculate standard mean difference, a meta-analysis was not undertaken for this group.

Figure 2.17. Funnel plot of all studies included in the meta-analysis.*




*Arrow denotes one point outside the scale. Vertical dashed line denotes summary odds ratio.

## 2.4 Discussion

This systematic review and meta-analysis of the surgical literature shows a strong association between compensation status and poor outcome after surgery. The association is maintained when allowing for type of intervention, type of compensation, country of origin, date of publication, or aspects of methodology (length and completeness of follow-up, prospective versus retrospective, and study type).

The strength of the association was also seen in individual papers, as many of the papers found that compensation status was the most significant predictor of outcome when compared to all other diagnostic and demographic variables,[173 178 345-366] or treatment variables.[174 347 349 351 354 367-377]

Although the results are not homogeneous, they are consistent, as 123 of the 129 studies included in the meta-analysis showed a positive association between compensation status and poor outcome (Figure 2.2), and 175 of the 211 studies included in the systematic review concluded that compensation was associated with poorer outcome, with only one study concluding the opposite.

Some variability in the association was expected considering the differences in the procedures, the compensation systems, the populations and the outcome measures. As expected though, heterogeneity in the sub-groups was less than in the overall analysis, particularly when sub-grouped by procedure or geography.

The findings are consistent with previous meta-analytic reviews of the effect of compensation on outcome after treatment for chronic pain[210] and recovery from head injury[183], which found compensation to be associated with poor outcome.

The only significant findings on meta-regression, which analysed the significance of the differences according to the other variables measured, was that the effect size was smaller in larger studies, and that the effect size was larger for revision surgery.

The association with revision surgery should be interpreted with caution as this analysis was performed post hoc. It may, however, be supportive of the effect of compensation on outcome, in that patients who had already failed to improve from surgery, failed due to their compensation status, rather than any effect of the surgery, and would therefore be more likely to fail with repeat surgery.

Publication bias may be either due to the selective publication of studies showing an association, or the selective reporting of an association only when it exists. This may lead to an increase in the estimate of the association.[378 379] The decrease in the effect size in larger studies may represent publication bias, and this finding confirms the appearance of the funnel plot (Figure 2.17) which shows a bias towards a stronger association in smaller studies (larger standard error). The asymmetry of the funnel plot may reflect underreporting of studies showing a positive association.

If publication bias were present, due to the increased likelihood of the association being reported if it is positive, one would expect to find an increased effect size in studies not specifically concerned with the association (where reporting is optional), compared to studies specifically designed to look at the association (which would report the association regardless of the direction or magnitude of the effect). This was shown in this meta-analysis (Table 2.4), where the summary odds ratio for studies designed to examine the effect of compensation was 3.60, compared to 3.85 for other studies. This provides some evidence of publication bias, however the difference is small, with wide overlap of the confidence intervals, and it is not significant on meta-regression. Also, the association remained strong in both groups, and visual inspection of the funnel plot indicates that allowance for publication bias would not be enough to reduce the effect size to a relative risk of 1.

As this study relies on observational data, confounding should be considered. Confounding may occur due to differences in the two patients groups, such as differences in expectations, demands, socioeconomic status, and job description. Allowance for these factors is not possible in this analysis, however, previous studies incorporating multivariate analysis have shown that the association between compensation and outcome is maintained when allowing for demographic factors, diagnostic factors, and treatment factors.[349-351 356]

Selection bias may explain the association if compensated patients had increased disease severity. This has not been shown previously but was not determinable in the studies included in this review. Therefore this is a potential cause of bias. The opposite may also be true however: that compensated patients are more likely to be diagnosed with, and undergo treatment for, certain conditions, given similar presentations. Previous studies have shown that compensated patients are more likely to be diagnosed with conditions such as whiplash, repetitive strain injury and thoracic outlet syndrome,[349-351 356 380-383] and are more likely to undergo surgery.[383]

However, if surgeons are more likely to diagnose compensated patients with surgical conditions, or more likely to treat them surgically (possibly for financial gain, or because of over-reporting of symptoms from the patients), this may still bias the results towards the effect seen. For example, compensated patients with non-specific arm pain (incorrectly) diagnosed with carpal tunnel syndrome would be less likely to improve after surgery than a non-compensated patient with true carpal tunnel syndrome.

The outcomes used were mainly patient-based, but some outcomes were scored by observers, and in those cases it was not stated whether the observers were blinded to the patient's compensation status. This may introduce bias and increase the negative effect of compensation in that the observers may have expected poorer outcomes in compensated patients. Negative perceptions towards compensated patients have been reported in patients with post-traumatic stress disorder.[187]

It should be noted that patients' compensation status was known prior to surgery in the studies included in this analysis. Two papers were excluded because compensation status was dependent on the outcome of the surgery.

The inclusive search strategy used (all techniques, all countries, no time limit) resulted in a wide variety of populations, procedures and geographical regions being incorporated in the final analysis. This was intentional, as the aim was to look for an overall 'compensation effect' and then perform sub-group analysis and meta-regression to look for bias introduced by using such inclusive criteria. The analysis showed that these factors did not influence the effect.

The exclusion of unpublished studies may result in an increased estimate of effect. This has been shown by McAuley,[384] but Egger et al,[385] in a similar study, did not find this to significantly influence the estimate of effect. Unpublished studies are also more difficult to find on electronic searches, and any attempt to find all significant unpublished studies would be incomplete.

Limitations of the search strategy should also be discussed. The search strategy relied on the word 'compensation' being used in the title or abstract, or on the article being listed under the subheading 'compensation and redress'. It is possible that articles exist that report on the difference in outcome between compensated and non-compensated patients, yet do not fit these criteria. Several such articles were found in references provided in the articles retrieved electronically. However, it is unlikely that many such articles were missed, as the extra articles retrieved from references were usually repeatedly referred to in multiple articles. Also, no other reasonable search strategy could be found.

A general limitation of meta-analysis is that it is dependent on adequate data from the original papers to calculate a summary statistic. The meta-analysis only included data from 129 of the 211 studies included in the review, introducing the possibility of bias due to under-representation. However, if all 211 papers are considered, only one of these concluded that compensated patients had better outcomes, and 175 concluded that the outcome was worse in compensated patients. It is likely, therefore, that the studies used in the meta-analysis are representative of the group as a whole.

Lack of standardized reporting of outcomes is a limitation in this meta-analysis. The majority of the studies (114 out of 129) reported the outcome as dichotomous. In the remaining studies, the outcomes were dichotomised according to the process described in the methods section. The method, grouping “excellent” and “good” as satisfactory, was the same as that used by the majority of the authors who reported dichotomous outcomes. Furthermore, although the results were often represented as dichotomous, these divisions were based on a diverse range of outcome tools; this may have led to some inconsistency in classification of outcomes. Unless reporting of surgical outcome becomes standardized, this will always be a problem when combining studies. However, it is unlikely that manipulation of the cut-points for categorizing the outcomes occurred in these studies, as it was usually based on previous studies and accepted standards, and was usually decided a priori. Due to the strength of the association, it is likely that any alteration in the cut-points used to categorise the outcomes would not have changed the direction of the association, and that the association would have remained statistically significant, although some change in the magnitude of the effect estimate may be expected.

Several of the variables measured in this analysis were categorised, which may have led to information loss, and may then have rendered a significant association non-significant. They were categorized because this was required for subgroup analysis in RevMan. However, these variables (minimum follow-up and completeness of follow-up) were not remotely significant on meta-regression, so they were not reclassified as continuous. Aside from the differences in coding of the outcome, the outcome tools varied widely between studies.

There may be some problem with using minimum time to follow-up as a measure of the length of follow-up; it may be that mean or median follow-up would have been a more accurate measure of the overall time to follow-up. The inclusion of such data was constrained by the papers, which usually reported the minimum and maximum follow-up, and not the mean or median. The statistical technique used for the meta-analysis was the Cochrane Review Manager (RevMan). This provided the summary statistics and the figures provided in this chapter. It is not possible to perform meta-regression using RevMan, so another software program was used. Although the effect estimates were higher using this second analysis, we prefer to use the estimates from RevMan as they are more conservative, and this was the method described a priori.

Previous studies have shown a dose - response relationship, by correlating health outcomes with the level of compensation.[265 267 272 293 386-388] If compensation causes poor outcome, consideration should be given to the mechanism. The effect of compensation on outcome may be related to psychological factors related to the injury and the compensation process,[153 206 299 380 389] as well as secondary gain (from financial benefits and/or the benefits of assuming the sick role),[269 390] and tertiary gain (maintenance of health care utilization to benefit legal and health care practitioners).[278 301 311] The adversarial nature of litigation and compensation insurance may also contribute to the association.[128 176 261 391] A discussion of possible mechanisms is provided in Chapter One.

Differences in the effect between sub-groups may provide information about the mechanism for the association. Although most sub-groups showed similar estimates of effect, the largest differences were seen between geographic regions (Table 2.4), suggesting that disparities in the compensation and legal systems between countries may be important. As the confidence intervals for the region-specific estimates overlapped, however, these variations may be due to sampling error rather than true differences. Furthermore, meta-regression did not show the country of origin to be significantly associated with the magnitude of the association.

Conclusions regarding cause and effect cannot be drawn from this research; stronger evidence is required from prospective studies with matched controls. Further research may also provide information regarding the mechanism of the association, which, in turn, may guide change in these health systems to improve outcomes. If the findings of this review are borne out in later prospective studies, it may assist in identifying patients who are at risk of a poor outcome.

The findings of this review are also relevant to users and providers of compensation based health systems, as poor outcome after therapeutic intervention for compensated patients impacts on productivity and business costs, as well as quality of life for the patients involved.

The findings of the systematic review and meta-analysis support the overall hypothesis of this thesis. The hypothesis states that outcomes after injury are worse for patients treated under compensation schemes. Although the patients included in each of the papers in this analysis were surgical patients, they can be assumed to have experienced an injury, as this is the basis on which compensation is usually claimed. A superficial inspection of the types of surgical procedures supports this, as the most common procedures were for spine and shoulder conditions, and carpal tunnel disease, and these conditions are usually considered to be injuries, particularly when they occur in the workplace.

## 2.5 Conclusion

This systematic review of the literature revealed that patients treated under compensation schemes or undergoing litigation consistently have worse outcomes after surgery than non-compensated patients. Of the 211 papers reviewed, 175 reported a worse outcome in compensated patients. Overall, a compensated patient has more than three times the odds of an unsatisfactory outcome compared to a non-compensated patient.

The findings of this review are based on observational data, and confounding and bias (from selection bias, and differences in disease severity and illness reporting) may account for part of the effect. Further prospective research controlling for these factors is required to confirm the findings of this review and to determine the mechanism for any association between compensation status and outcome.

Due to the strength and consistency of the association between compensation status and poor outcome after surgery shown in this systematic review and meta-analysis, the study hypothesis is accepted.

# CHAPTER THREE. THE ASSOCIATION BETWEEN COMPENSATION AND HEALTH OUTCOMES AFTER MAJOR TRAUMA: THE MAJOR TRAUMA OUTCOME STUDY

## 3.1 Introduction

The literature regarding the association between compensation status and health status is discussed in Chapter One, and the systematic review in Chapter Two examines the association between compensation status and outcome after surgery. This chapter aims to explore the association within a defined cohort, while attempting to minimise the biases inherent in studies in which control over the exposure variable is not possible.

The study will examine outcomes in a cohort of consecutive major trauma patients presenting to one major trauma centre in Sydney, and will compare outcomes between compensated and non-compensated patients, allowing for possible confounders and effect modifiers.

This type of study is possible in the state of New South Wales (NSW) because of the laws covering accidental injury. Whereas in some states, and in some other countries (for example, New Zealand) accidents are universally covered by a single compensation system, in New South Wales, accident victims are variably covered depending on the circumstances of the accident.

The workers compensation scheme covers workers injured in the course of their work and it is a condition of employment that all employees are covered by workers' compensation. This compensation scheme also covers workers who are injured travelling to or from work.

For those injured in motor vehicle collisions, a fault-based compensation system exists through compulsory third party insurance of all motor vehicles. This scheme, controlled by the state Motor Accidents Authority (MAA) and provided by private insurance companies, provides compensation to any person involved in a motor vehicle collision who was not at fault. This applies to all passengers and to the vast majority of pedestrians involved in motor vehicle collisions. Those at fault, for example drivers in single vehicle collisions, are not entitled to claim any compensation or to claim for any medical expenses through the third party system. Any medical treatment required must be paid for by the patient, or through Medicare (the public health system).

These systems provide two populations of injured people in NSW: those who are covered by compensation and those who are not covered, allowing the opportunity to compare outcomes between these two groups.
The study hypothesis states that health outcomes will be worse in the compensated group, compared to the non-compensated group. The study hypotheses are further defined later in this section.

Although the effect of compensation on outcome has been widely reported in the literature (this literature is reviewed in Chapter Two), there is little published on any effect on major trauma patients.

A North American study, known as the LEAP study (Lower Extremity Assessment Project) studied consecutive patients presenting to several major trauma centres with severe lower extremity injuries and was mainly concerned with health outcomes after amputation compared to limb salvage but accounted for other predictors of outcome, including psychosocial variables. They found that in multivariate analysis, involvement with the legal system was associated with poor psychosocial outcomes, but did not provide an analysis of the association between this variable and physical outcome.[307]

Studies that have examined the effect of compensation on outcome after trauma have all shown a negative association, but have differed from this study by the inclusion of minor injuries,[105 192 392] or by restricting the outcome to psychiatric variables, such as PTSD.[128 190-192 194 252]

As previously discussed in Chapter One, an association between compensation status and aspects of health are widely reported, but interpretation of the literature on this topic is hampered by selection bias which may result in important differences between the compensated and uncompensated groups leading to possible confounding from physical factors (age, sex, injury severity, past health etc.) and psychosocial factors (cultural, occupational, socio-economic etc.), and measurement bias (difficulties with diagnostic criteria, invalid outcome tools, and conflicting definitions of compensation).

This study examines the effect of compensation status in a well-defined cohort, using validated health outcomes, allowing for possible confounders.

### 3.1.1 Study aims and hypotheses

This study aims to explore the association between compensation-related factors, and other psychosocial variables, with health outcomes after major physical injury, allowing for demographic and injury-related factors.

The primary hypotheses relate to the overall hypothesis of the thesis, that compensation status is a determinant of general health after injury. Specifically,

1. that general physical health, as measured by the physical component summary (PCS) of the SF-36 General Health Survey, will be significantly worse for patients who pursued compensation than for patients who did not pursue compensation, and
2. that general mental health, as measured by the mental component summary (MCS) of the SF-36 General Health Survey, will be significantly worse for patients who pursued compensation than for patients who did not pursue compensation.

These hypotheses have been explored in the previous chapters and are the same major hypotheses of the prospective study detailed in Chapter Four.

The secondary hypotheses pertain to each of the secondary outcome measures, namely

3. that neck pain will be significantly worse for patients pursuing compensation,
4. that back pain will be significantly worse for patients pursuing compensation,
5. that patient satisfaction will be significantly lower for patients pursuing compensation, and
6. that the incidence of Post Traumatic Stress Disorder (PTSD) will be significantly higher in patients claiming compensation.

In those who pursue a claim through compensation, the effect of claim settlement was expected to be significant. Therefore, the group who pursue compensation were divided into those whose claim has settled, and those whose claim has not settled.

The possible effect of compensation type was also explored. This was done by comparing the main outcomes in those claiming under workers compensation (a no-fault scheme), to the main outcomes in those claiming under third party compensation (a fault-based scheme).

## 3.2 Methods

### 3.2.1 General methods

Approval from the Human Research Ethics Committee for Liverpool Hospital and for the University of Sydney was sought and provided. Copies of the approval from the ethics committees are provided in Appendix 2.

In the state of New South Wales (and in most trauma institutions internationally), a major trauma is defined as any patient with an Injury Severity Score (ISS) of greater than 15. The ISS is the standard way of assessing severity of injury in trauma patients and was first introduced by Baker et al in 1974.[244] The ISS is calculated by summing the mathematical squares of the Abbreviated Injury Score[393] (AIS) for the three worst affected body regions. The AIS is a method of scoring injury severity, on a scale of 1 to 5, for each of the five body regions: head, chest, abdomen, pelvis, and extremities. A score of 1 is considered a minor injury (for example, abrasions, minor lacerations) and a score of 5 is the most severe (for example abdominal bleeding necessitating urgent surgery, major open pelvic fracture, or bilateral lung and rib injuries.

To use a clinical example, a person may have a major head injury with intra-cranial bleeding and secondary neurological deficit, a closed tibia fracture, and seat-belt abrasions across the abdomen with no evidence of significant intra-abdominal damage. The head/neck component of the AIS score would be 5, the extremity component would be 3, and the abdominal component would be 1. The ISS is calculated by squaring the highest AIS scores from the three worst affected body regions. Therefore, the ISS for this patient would be $5^2 + 3^2 + 1^2 = 35$. This patient, then, would be classified as “major trauma” (defined as an ISS > 15).

The grading for each of the five body regions is based on guides provided in a manual for researchers using the AIS. In all major trauma centres in NSW, data on all major trauma cases presenting to hospital are collected contemporaneously and kept on a database, which is usually used for research purposes and to provide comparisons between trauma centres. Data are usually coded and entered by one person, the trauma data manager. This is the case at Liverpool Hospital where these data have been collected continuously since 1995.

### 3.2.2 Study population

The study population was identified from the Liverpool Hospital Trauma database and included consecutive adult (18 years and over) patients presenting to Liverpool Hospital (a metropolitan trauma centre) with major trauma after accidental injury. Major trauma was chosen as it is defined at the time of presentation, has well established objective criteria, and it excludes minor injuries, which may include a higher proportion of feigned injuries and conditions diagnosed by subjective criteria, such as whiplash and low back pain.

Children were excluded because of expected difficulties with interpretation of the social parameters (such as education level, income, and occupational factors) and possible difficulties with aspects of consent and the interpretation of the questionnaires by third parties (parents and guardians). Also, some dilution of the effect of compensation was expected as the children may not be the direct recipients of any financial compensation, or may not understand the significance of the compensation. No upper age limit was set.

Patients with non-accidental injury were excluded. Injuries were prospectively coded as accidental or non-accidental in the trauma database; non-accidental injuries were usually either attempted suicides, or assaults. These were excluded for several reasons: they may be the result of mental illness in the patient; they are acts that are under the control of the patient and may therefore reflect a particular personality type and are therefore less “random” than an accident; and they are not usually subject to compensation.

Patient data were retrieved from the trauma database for patients admitted from May 1999 to April 2004, inclusive. This covered a five-year period of between one and six years prior to the mail-out of the questionnaire (May, 2005).

Patients who died in hospital, as recorded in the Trauma Registry database, were excluded. The list of surviving patients from the trauma registry was then screened by the NSW Department of Births, Deaths and Marriages for deaths, before mailing the questionnaire.

### 3.2.3 Medical record data

Data was abstracted from the trauma registry database. Demographic data from the database included name, date of birth, and gender. Identifying data, such as the medical record number, was also abstracted to assist in obtaining contact information from the hospital records.

Injury-related data from the database included the date of injury, the mechanism of injury, the length of stay in the Intensive Care Unit (ICU), and the ISS and AIS (Abbreviated Injury Scale, measured for each body region). The ISS was chosen as the main injury severity factor as it has previously been shown to be a good predictor of outcome after trauma.[244 245 247-249 392 394-399]

The mechanism of injury (MVA or other) was measured as an explanatory variable and was considered important when considering generalisability, as many previous similar studies, and the prospective study in Chapter Four, only include patients from motor vehicle accidents. The presence of a cervical spine fracture, and the presence of a thoraco-lumbar spine fracture, were recorded as possible predictors of neck pain and back pain, respectively.

The AIS allowed determination of whether or not a significant head injury was present. This was recorded because traumatic brain injury may influence a person's ability to participate in the study due to cognitive ability. Therefore, any information regarding the incidence of severe head injury in study participants, compared to those that do not participate, may be useful to assess generalisability of the sample. Also, presence of a severe head injury was tested as a predictor of outcome, as it may be an important confounder of compensation status, independent of ISS.

Presence of a severe head injury, presence of a spine fracture, and length of stay in ICU were recorded as additional measures of injury severity, as the ISS was developed and has been used mainly as a predictor of mortality following trauma, rather than a predictor of general health.

Other data, to determine exclusions, included in-hospital mortality, and whether or not the injury was accidental (this is coded during the initial admission).

### 3.2.4 Questionnaire data

The questionnaire (Appendix 3) provided information regarding all outcome variables, and the explanatory variables not obtained from the trauma database.

The first eleven questions consisted of the SF-36 General Health Survey,[400] a widely used and validated health outcome tool.[400 401] The format used was that described in the SF-36 Manual and Interpretation Guide.[400] Formatting and numbering of the questionnaire were continued in a similar manner after the questions pertaining to the SF-36, to provide continuity.

Questions 12 and 13 measured severity and functional impact of back pain, and questions 14 and 15 measured neck pain in the same manner. These questions were worded and formatted identically to the two questions that contribute to the Bodily Pain index of the SF-36, except that the words "low back" or "neck" were inserted before the word "pain". Adaptations of these SF-36 questions have previously been used and validated in back pain research[402] and they have also been used in neck pain (whiplash) research (C Maher, University of Sydney, personal communication). The result of piloting these questions is provided in Chapter Four in the section on piloting of the questionnaire. The responses were scored and combined in the same manner as the Bodily Pain Score of the SF-36.

Question 16 measured chronic illnesses, by asking participants to list their chronic illnesses (choosing from a list of 12 options).

Question 17 asked patients if they took any regular medications, and provided a yes/no response. The question was meant as a screening tool for chronic disease but data from this question was not used in the analysis, as it was not considered a valid measure. Many patients, for example, may have been on prophylactic medication (statins, aspirin, etc.) unrelated to previous disease, or may have been on medication (such as analgesics) as a result of their injury.

Question 18 asked patients to list the number of times they had visited different health practitioners over the last three months. It was designed to measure health care utilisation, as an alternative outcome, but this data was not used in the analysis as it was not considered a primary health outcome, and therefore not directly relevant to the thesis.

Question 19 asked patients to describe the mechanism of injury. This was initially designed to determine the mechanism of the injury (MVA or other), but these data were available in the trauma database. Therefore, analyses for this variable were based on the database data, as these were considered more reliable (as classification occurred at the time of injury, and by an independent observer).

Questions 20 to 23 provided information that was used to determine eligibility for compensation. Question 20 asked if patients were injured in the course of their work, and all patients responding positively were considered eligible to claim compensation (as workers compensation is compulsory). Question 21 asked if they were involved in a motor vehicle collision, and if they responded positively, questions 22 and 23 asked about whether they were a driver, passenger or pedestrian, and whether they were considered at fault for insurance purposes. These questions were used to determine eligibility to claim under the compulsory third party insurance scheme.

Question 24 asked patients to rank their satisfaction with their progress since the injury on a 4-point Likert scale. This question was validated in the pilot study reported in Chapter Four.

Question 25 asked patients to rank how well they remembered the injury event, and this question was included in the pilot questionnaire as described in Chapter Four. Memory of the event was an outcome variable that was not used in this analysis, as it did not relate to health. However, it was used to test the validity of the exposure variable “head injury”, as a high negative correlation between memory and head injury was expected.

Question 26 asked patients to state their opinion regarding who was at fault for the injury: themselves, someone else, or not known. This question was piloted in the study reported in Chapter Four and was used to measure blame, which has been shown in previous studies to affect outcome after traumatic injury.[110 130 219 297 298 403]

Question 27 asked the highest education level attained by the patient: primary school, secondary school, certificate/diploma, or degree. These groups are the only significant options possible, and a reasonable spread of results was expected based on piloting this question as outlined in Chapter Four.

Question 28 categorised the average annual household income, with $50,000 as the median value, based on figures from the Australian Bureau of Statistics data.[404] This question was also piloted and the results are provided in Chapter Four.

The questions relating to occupation, education level, and income were prefaced by a sentence indicating the period of time to which the questions referred.

Questions 29 and 30 asked patients to give their occupation and the basis on which they were employed (part time, full time, or casual), at the time of injury and at the time of follow-up, respectively. The specific occupation was sought so that it could be ranked for prestige, to be used as an indicator of socio-economic status. This ranking system was developed by Daniel, who published a report entitled Power, Privilege and Prestige: Occupations in Australia[405] that ranked occupations in order of perceived status. The amount of employment (e.g., part time versus full time) was recorded to be used as an alternative outcome, similar to return to work, but was not used as it was not a direct measure of health.

Occupation at follow-up was measured as it may impact on the main outcome measures. Also, it has been suggested that the high unemployment rate in compensated patients may explain the association between compensation and chronic pain.[51 406] Other details regarding occupation (whether casual, part time, or full time) were not used in the analysis as they were not considered to be relevant to the study hypotheses.

Questions 31 and 32 ask whether a claim was made for the injury, and whether the claim has settled, under either the workers compensation system, or under third party insurance or personal litigation, respectively. Patients were also asked to provide the date of settlement. Pursuit of a claim (making a claim) was chosen as the preferred measure of compensation status as it was measurable on all patients and unlikely to be subject to measurement error.

Question 33 asked patients if they had retained the services of a lawyer regarding their injury, and a yes / no response was provided. Legal involvement (presence of a lawyer) has been shown to significantly influence outcome after injury.[61 68 117 176 178 260 304 305 407]

Question 34 was the PTSD Checklist – Civilian version.[408] This tool has been validated as a screening tool for PTSD[408-411] and is used widely. Other, more formal tests for PTSD involve clinician interview or more extensive questionnaires, such as the Minnesota Multiphasic Personality Inventory.[217] The PTSD Checklist was chosen for this questionnaire, as it is brief, suitable for self-reporting, and valid. The PTSD Checklist corresponds better to DSM defined symptoms than other self-report screening tools.[409]

Questions relating to the injury were prefaced by a statement indicating that these questions relate to the participants original injury. In order to avoid confusion about which injury we were referring to, for patients who may have had multiple admissions to Liverpool Hospital for trauma, the year of the injury was specified. This required six different questionnaires to be produced (for the years 1999 to 2004) and the patients were grouped into those calendar years for mailing purposes so that they could be provided with the appropriate questionnaire.

Further information regarding each question is outlined in the following section on explanatory and outcome variables. Questions that were not part of previously validated instruments were validated in a pilot study that was performed for the questionnaire used in Chapter Four. The results of this pilot study are provided in Chapter Four.

### 3.2.5 Mailing of the questionnaire

Addresses were retrieved from the hospital records where possible. If no address was provided in the records, local doctors and treating specialists were contacted, and telephone contact with the patient was made if possible. The White Pages directory was also used to contact patients.

Questionnaires were sent over two consecutive business days in May 2005, along with a cover letter (Appendix 4) and an addressed, reply-paid envelope.

Two weeks after the initial mail-out, reminder letters (Appendix 5) were sent to all non-responders. Four weeks after the initial mail-out, non-responders were sent a second reminder letter (Appendix 6) along with another copy of the questionnaire, and another addressed, reply-paid envelope.
Any remaining non-responders were contacted by telephone when possible and patients were given the opportunity to answer the questionnaire by

telephone if they preferred. A template was provided for telephone transcript (Appendix 7).

Questionnaires that were returned as “Return to Sender” because the person was not living at that address were investigated further by contacting treating doctors and searching the White Pages telephone directory for possible addresses or telephone contact numbers.

Identifying data were kept on all patients until a completed questionnaire was returned. When incomplete questionnaires were received, patients were contacted by telephone and the missing answers were completed by telephone. Once a completed questionnaire was entered, the identifying data were separated from the results database and no further contact was made with the participant.

People refusing to participate were listed as “refusals”. People from whom there was no response after all the mail-outs and the attempted telephone contact were listed as “non-responders”.

People for whom no contact information was available, and those whose questionnaires were returned because they were not known at the address, were classified as “uncontactable” and were excluded from the analysis.

### 3.2.6 Measures

#### 3.2.6.1 Explanatory variables

The explanatory variables were grouped as general, injury severity, socio-economic, and claim-related factors and are summarised in Table 3.1.

Regarding the general factors, age was defined as age at the time of injury, and was measured in years and treated as a continuous variable. It was calculated from the date of birth and the date of injury. Gender was either male or female (dichotomous) and was determined from the trauma database. The time since injury was measured in months, and treated as a continuous variable. It was calculated from the date of injury and the date of receipt of the completed questionnaire.

The other general factor, chronic illness, was measured by counting the number of chronic illnesses listed in the questionnaire. The responses “depression”, “anxiety” and “fibromyalgia” were excluded, as they are diagnoses that were likely to have resulted from the injury, rather than being present prior to the injury. It was recorded as a categorical variable, with categories for 0, 1, 2, or 3 or more chronic illnesses. Although this variable was measured at the time of follow-up, it was used as a measure of pre-existing chronic illness. Therefore, the illnesses listed were all of a chronic nature and were illnesses that were unlikely to have been caused by the injury (e.g., chronic bronchitis, heart disease, diabetes).

Table 3.1. A list of all exposure and outcome variables.

| Exposure variables | Outcome variables |
|---|---|
| ***General factors*** | PCS |
| Age at follow-up | MCS |
| Gender | Neck pain |
| Time since injury | Back pain |
| Number of chronic illness | Patient satisfaction |
| ***Injury severity factors*** | PTSD |
| Injury Severity Score (ISS) | |
| Presence of a severe head injury | |
| Presence of a cervical fracture | |
| Presence of a thoracolumbar spine fracture | |
| Mechanism (MVA or other) | |
| Length of stay in ICU | |
| ***Socioeconomic factors*** | |
| Education level at time of injury | |
| Income at time of injury | |
| Occupation status at time of injury | |
| Employed at injury | |
| Employed at follow-up | |
| ***Compensation-related factors*** | |
| Claim made | |
| Claim settled (if made) | |
| Compensation system | |
| Lawyer involvement | |
| Blame (patient perception of fault) | |
| Time to settlement | |
| Time since settlement | |

Regarding the injury severity factors, the ISS was directly imported from the trauma database as a dimensionless continuous variable ranging from 16 (the cut-off value for inclusion) to 75 (the maximum possible score). ICU stay was recorded in days (patients who did not attend ICU were assigned a value of zero) as a continuous variable. Mechanism of injury and presence of a spinal fracture were abstracted from the trauma database.

Presence or absence of severe head injury was abstracted from the trauma registry, based on whether the AIS for the head was 3 or higher, or less than three. This cut-off value was used because any intracranial pathology is given a minimum value of 3, and values of 1 and 2 include injuries such as scalp lacerations and bruises, which do not necessarily indicate a severe or significant head injury.

Regarding the socio-economic factors, the highest education level and the annual household income were classified into the same four categories as given in the questionnaire. The occupational prestige score was used as a continuous variable, as the raw score provided a number from one to nine, to one decimal place. Employment at the time of injury and time of follow-up were coded as dichotomous variables.

Memory of the event was coded directly from the questionnaire responses. Memory was not used as an outcome variable in this analysis, but was used to validate the use of severe head injury as an explanatory variable.

Eight compensation-related variables were measured and analysed separately to allow discernment of the relative influence of different aspects of the compensation process.

The primary compensation-related variable was pursuit of compensation, a dichotomous variable that was positive if any claim for the injury had been made.

Four of the variables could only be measured for the subgroup of patients who had made claims. Claim type was a dichotomous variable defined as third party or workers compensation. Claim settlement was defined as settled or not settled. Time (from injury) to settlement and time since settlement (from settlement to follow-up) were measured in months as continuous variables, and were only scored for those whose claim has settled. The one to six year follow-up in this study was considered necessary to provide information regarding the effect of length of time (time since injury, time since settlement, or time to settlement) on the outcomes, as settlement for compensation cases may take several years.

Eligibility for compensation, a dichotomous variable, was determined depending on the circumstances around the accident. A person was considered entitled to compensation if they were injured in the course of their work, or were passengers involved in motor vehicle collisions. Drivers considered to be at fault (for insurance purposes) who were not injured in the course of their work were considered non-eligible for compensation. This resulted in a significant number of patients for whom eligibility could not be determined. Also, there may be circumstances under which the criteria to determine eligibility may be wrong. Therefore, pursuit of a claim (not eligibility) was used as the explanatory variable, and any effect was compared to the effect of claim eligibility in a separate analysis.

It is likely that most patients who are severely injured will pursue a claim if they are entitled, so a high correlation between these two variables was expected. The distinction is important, though, as pursuit of a claim implies a conscious decision and may bias the results, whereas entitlement to compensation reflects the circumstances of the injury and is therefore less under the control of the patient.

The participants' opinion regarding who was at fault was classified into three categories: the patient was at fault, someone else was at fault, or they did not know who was at fault. Although this is related to compensation, particularly in the fault-based third party system, it was retained as a separate variable because of the potential to influence outcome, independent of compensation status. This variable was also referred to as "blame".

The final compensation-related variable was use of a lawyer regarding the injury. Although this variable was expected to be strongly associated with pursuit of a compensation claim, each factor was treated as a separate variable in the analysis to determine any differences, confounding or interaction for these two variables.

#### 3.2.6.2 Outcome variables

The primary outcome measures were general physical health and general mental health. These were measured by the physical component summary (PCS) and the mental component summary (MCS) of the SF-36 General Health Survey.[400]

The scores for the eight health domains measured by the SF-36 General Health Survey were calculated by entering the responses into a spreadsheet that provided automatic calculations for each score and scale (the scale converts the score for each domain into a percentage). The PCS and MCS were calculated using the website of the developers of the SF-36 in the United States. The calculator on this website allows entry of the SF-36 scores for each of the eight health domains and calculates the summary scores based on standardised population data from the US from 1998, which have a mean of 50 and a standard deviation of 10. The PCS and MCS scores are provided as continuous variables ranging from 0 to 100 to one decimal place with a normal distribution, with higher numbers indicating better health.

Neck pain and back pain was measured by combining the scores of the two questions (severity and functional impact) pertaining to each of these outcomes. This is the method used to score the Bodily Pain index of the SF-36, and provides continuous scores ranging from 2 to 11, with higher numbers indicating worse pain.

Patient satisfaction with progress since the injury was measured on a four-point Likert scale, and the results were converted to a dichotomous variable, satisfied / dissatisfied.

The final outcome variable used was a screening tool for Post-Traumatic Stress Disorder (PTSD). The tool used is known as the PTSD Checklist (Civilian version) and was produced by the United States Veteran Affairs Department.[408] As the tool consists of 17 questions with responses ranging from 1 to 5, the final score is a continuous variable ranging from 17 to 85, with higher numbers indicating more PTSD-related symptoms.

Although several cut-off values have been suggested (44, 45 and 50) for a diagnosis of PTSD using the PTSD Checklist, a cut-off of 44 is recommended as having greater diagnostic accuracy[408 409 412] and is recommended for civilian populations.[408] Greater accuracy is achieved by combining the total score with the presence of a score of 3 or higher on individual symptoms that meet the DSM criteria. This was not done for this analysis, as PTSD is a secondary outcome, and the aim was to determine associations with PTSD and the severity of PTSD-like symptoms, rather than to accurately determine the incidence of PTSD in this population, as the incidence would have been most affected by change in the diagnostic criteria.

### 3.2.7 Sample size calculation

As the main method of analysis was multiple regression (with general health as the continuous outcome) the sample size calculation was based on having 10 – 20 participants per exposure variable. This study measured approximately 13 exposure parameters and therefore required approximately 200 participants. It was expected that there were approximately 200 patients per year who would satisfy the inclusion criteria, giving a total of 1,000 patients for the five-year period. Previous surveys mailed to trauma patients from Liverpool Hospital achieved a 30% return rate, despite reminder letters, telephone calls and financial incentives (Michael Sugrue, Trauma Department Director, personal communication). Assuming that approximately 600 – 700 patients would remain after removing those with non-accidental injury and those who had died or for whom there was no contact address, a return rate of 30% should have provided the 200 patients required for the statistical calculation. Further, it was expected that some of the exposure variables would be collinear, such as the different measures of injury severity and the different measures of socio-economic status, potentially reducing the number of independent variables required in the final model. Therefore, selection of patients over a five-year period was considered adequate.

### 3.2.8 Statistical analysis

Associations between each explanatory variable and each outcome variable (according to the stated hypotheses) were assessed in univariate (one-way) analyses.

Continuous outcomes (PCS, MCS, neck pain, back pain, and PTSD) and dichotomous explanatory variables (e.g., sex, claim made, use of a lawyer) were assessed by comparing means in the two groups, using a t test. The t test for dichotomous variables was calculated using either the pooled or Satterthwaite methods, depending on the probability of the variances being equal. For explanatory variables with more than two categories (e.g., blame, income, education and chronic illnesses) one-way analysis of variance (ANOVA) was used to assess differences in means (F test for overall association). Association with continuous variables (e.g., age, time since injury) were assessed using the Pearson correlation coefficient.

Associations between PTSD, a dichotomous outcome variable, and continuous variables were assessed by comparing means (using a t test). The association with other dichotomous variables was assessed using the chi-square test. The association with categorical variables was assessed using the chi-square tests for overall association and for trend (Mantel-Haenszel).

Associations between the ordered categorical outcome (patient satisfaction) and continuous variables was assessed using ANOVA (F test for overall association), and the chi-square tests for overall association and for trend (Mantel-Haenszel) was used to assess the association with dichotomous and categorical explanatory variables.

Any variable with a significance level of 0.25 or lower on univariate analysis was included in a multiple regression equation, separately for each outcome.[413] Backward elimination was performed to find the model which best explained the changes in the outcome. Variables were removed sequentially, removing the variable with the least significance at each step. Variables with significance levels of 0.05 or less were retained in the final model. Interaction terms were introduced into the final model and retained if their significance level was less than or equal to 0.01.

For multiple linear regression, the assumptions of linearity and equal variance about the mean were tested by noting observations whose Cook's distance was greater than 1 (indicating undue influence on the regression model), studentised residuals were plotted against the continuous variables in the model (time since injury and ISS), studentised residuals were also plotted against predicted values, and the histogram, boxplot and normal probability plot for the distribution of residuals was examined. The assumption of normality was tested using the Proc Univariate procedure in SAS. A small negative skew in the distribution of the residuals for neck pain and back pain models was accepted as the sample size was large and there were no influential points.[414]

Due the importance of a linear association between continuous predictor variables and dichotomous outcomes in logistic regression, the continuous variables were each divided into five even groups (quintiles) in order of rank, using the Proc Rank statement in SAS, and analysed as categorical variables.

The variables “claim made” and “claim settled” were combined to allow use of data from all participants in one model. These variables were combined to make a 3-part variable, “Claim”. The referent group was “No claim made”, and two dummy variables were created: “Claim made – settled” and “Claim made – not settled”.

As ISS was positively skewed, an alternative variable was created by logarithmic transformation of the ISS value. This new value was shown to be less positively skewed (by histogram, box plot, and stem-and-leaf plot) and was also tested against the outcomes in the analysis. In the univariate analyses, however, these two variables showed similar associations. Therefore, ISS was kept as a continuous variable without transformation.

The distribution of the stay in ICU (measured in days) was highly positively skewed, with 39.4% of subjects not admitted to ICU, and most admissions being for only 1 – 4 days. Although ICU stay was retained as a continuous variable due to the large sample size, ICU stay was also tested as a dichotomous variable (admitted / not admitted). ICU was also recoded as an ordered categorical variable in six groups (0, 1, 2, 3, 4, 5+ days) and tested against each outcome variable. The categorical variable for ICU was not remotely associated ($p<0.25$) with any of the outcome variables except low back pain (F value = 1.70, DF = 5, 349, p = 0.13), however for low back pain, there was no observed trend, with the highest mean scores for low back pain occurring in those whose ICU stay was 0, 3 or 4 days, and the lowest means scores in those whose ICU stay was 1 or 2 days. Consequently, the information in the tables regarding the analysis of ICU stay is restricted to ICU as a dichotomous or continuous variable.

The outcome scores for neck pain and back pain (range 2 to 11) were highly positively skewed. Therefore, an alternative dichotomous variable was created by using a cut-off score of six for “significant” pain. This was based on a reasonably even distribution of responses, and required reasonably high responses. Both outcomes gave similar final models, although with higher significance levels for the continuous outcomes. Although the continuous outcome scores were not normally distributed, they were used in the final model as these models were supported by the alternative (logistic analysis), and because the assumptions for multiple regression were not violated (see above).

To simplify the statistical analysis for the multivariate modelling, and to provide a more easily interpretable outcome, patient satisfaction was dichotomised by combining the first two responses as “satisfied” and the last two responses as “unsatisfied”.

The effects of time to settlement and time since settlement were not explored in the main analysis as data were only available for a small subset of participants (those whose cases had settled). If significant on univariate analysis, the effect of these variables was explored in a separate analysis.

The units of measurement of the exposure and outcome variables are given in Appendix 8. All of the statistical calculations were performed using SAS version 8.2 (Cary, NC, USA).

## 3.3 Results

### 3.3.1 Patient sample

A total of 1156 major trauma patients (ISS 15 or higher) were recorded on the Liverpool hospital trauma database as having been admitted between the dates 1 May 1999 and 30 April 2004 (i.e., one to six years prior to commencing the mail-out of the questionnaire). This number excluded children (ages 17 and under). A flowchart of exclusions and responses is given in Figure 3.1.

One hundred and sixty patients were excluded because they died in hospital, leaving 996.

From the remaining group, 198 were excluded because their injury was classified as non-accidental (164 assaults and 34 cases of self-harm). There were 10 cases that had undetermined accident status. Examination of these 10 cases revealed seven that were suspicious of suicide (six falls from a height and one struck by a train) and these cases were excluded. The remaining three, labelled as motor vehicle accidents, were included in the study.

The names and dates of birth of the remaining patients were screened by the Department of Births, Deaths and Marriages, which reported that 60 of these patients had died since discharge.

Figure 3.1. Summary of study participants.

Major trauma patients admitted: 1156

160 Died in hospital

996

205 Non-accidental injury

791
**Error!**

60 Reported as dead by Births,
Deaths & Marriages

731
**Error!**

3 No contact details provided

Number of questionnaires mailed: 728
**Error!**

10 Reported as dead

718
**Error!**

46 Unable to complete survey

672
**Error!**

90 Not contactable

Number eligible for study: 582
**Error!**

134 No response

448

93 Refusals

Number of questionnaires completed: 355

Of the 731 remaining patients, a contact address was not found for three (two overseas visitors and one labelled “no known address”). Therefore, 728 patients were sent a questionnaire.

Ten further deaths were discovered through responses from the mail-out. Forty-six patients were unable to complete the survey, usually because of language difficulties (20), being overseas for extended periods, dementia or severe brain injury. Ninety patients were not contactable. For these patients, the questionnaires were returned (not known at that address) and further contact by telephone or alternative addresses was not possible. These 146 patients were excluded from the study, leaving 582.

Of those who were sent surveys, 93 patients refused to participate (two of these stated that this was under instructions from their lawyer), 134 did not respond despite three mailings and one attempted telephone call, and 355 questionnaires were returned completed.

If the 134 non-responding subjects were counted as refusals (worst-case scenario), the response rate was 61.0% (355 out of 582). If the 134 non-responding subjects are counted as uncontactable and therefore excluded from the study (best-case scenario), the follow-up rate was 79.2% (355 out of 448).

Seventeen of the completed questionnaires were completed by telephone interview.

A comparison of responders and non-responders is provided in Table 3.2. A comparison with regard to compensation status could not be directly measured, as this was not recorded for non-responding subjects. However, the relative proportions of passengers, pedestrians and drivers were expected to give some information regarding this, as passengers and pedestrians are much more likely to be covered by third party compensation than drivers. Of the 583 subjects eligible for the study, the ratio of drivers/riders to pedestrians/passengers was 2.34 in those who responded, significantly higher than the ratio for non-responders (1.41, $chi^2 = 5.52$, 1 DF, $p = 0.02$).

Table 3.2. A comparison of responding and non-responding patients.

| Variable (value) | Responders | Non-responders | p value |
|---|---|---|---|
| Age (mean in years) | 47.8 | 44.5 | 0.02 |
| Gender (% male) | 72.1 | 69.0 | 0.35 |
| ISS (mean score) | 24.3 | 23.4 | 0.2 |
| Head injury (%) | 51.3 | 54.7 | 0.36 |
| Time since injury (mean months) | 41.0 | 45.2 | 0.001 |

### 3.3.2 Frequency and distribution of responses

Characteristics of the participants are given in Table 3.3. Age was noted to be normally distributed, with a mean age of 47.8 years (SD 18.4) and a median of 47.0 years. The time since injury was also normally distributed.

Table 3.3. Frequency and means of explanatory variables.

| Variable | N | Category | N | % | Mean (range) |
|---|---|---|---|---|---|
| ***General factors*** | | | | | |
| Age | 355 | | | | 47.8 years (19-91) |
| Sex | 355 | Male | 256 | 72.1 | |
| | | Female | 99 | 27.9 | |
| Time since injury | 355 | | | | 41.0 months (12-74) |
| Chronic illnesses | 355 | None | 146 | 41.1 | |
| | | 1 | 106 | 29.9 | |
| | | 2 | 64 | 18.0 | |
| | | 3 or more | 39 | 11.1 | |
| ***Injury severity factors*** | | | | | |
| Injury severity score | 355 | | | | 24.3 (16-66) |
| ICU admission | 355 | No | 140 | 39.4 | |
| | | Yes | 215 | 60.6 | |
| Days in ICU | 355 | | | | 5.50 (0-227) |
| Head injury | 355 | No | 173 | 48.7 | |
| | | Yes | 182 | 51.3 | |
| Mechanism | 355 | MVA | 225 | 63.4 | |
| | | Other | 130 | 36.6 | |
| Neck fracture | 355 | No | 227 | 92.1 | |
| | | Yes | 28 | 7.9 | |
| Back fracture | 355 | No | 217 | 89.3 | |
| | | Yes | 38 | 10.7 | |
| ***Socioeconomic factors*** | | | | | |
| Highest education | 349 | Primary | 30 | 8.6 | |
| | | Secondary | 189 | 54.2 | |
| | | Certificate/Diploma | 100 | 28.7 | |
| | | Bachelor degree | 30 | 8.6 | |
| Annual income | 345 | $0 – 30,000 | 160 | 46.4 | |
| | | $30,000 – 50,000 | 83 | 24.1 | |
| | | $50,000 – 75,000 | 54 | 15.7 | |
| | | $75,000+ | 48 | 13.9 | |
| Occupational status | 235 | | | | 4.9 (2.2-6.6) |
| Employed prior to injury | 355 | No | 108 | 30.4 | |
| | | Yes | 247 | 69.6 | |
| Employed now | 355 | No | 193 | 54.4 | |
| | | Yes | 162 | 45.6 | |
| ***Compensation related factors*** | | | | | |
| Claim pursuit, settlement | 354 | No | 201 | 56.8 | |
| | | Yes, settled | 70 | 19.8 | |
| | | Yes, not settled | 83 | 23.4 | |
| Claim type | 153 | Workers comp. | 48 | 31.4 | |
| | | Third party | 82 | 53.6 | |
| | | Both | 23 | 15.0 | |
| Lawyer used | 353 | No | 225 | 63.7 | |
| | | Yes | 128 | 36.3 | |
| Blame | 352 | Self | 129 | 36.7 | |
| | | Someone else | 113 | 32.1 | |
| | | Don’t know | 110 | 31.3 | |
| Time to settlement | 59 | | | | 32.3 months (4-60) |
| Time since settlement | 59 | | | | 19.2 months (1-65) |

Data regarding the mechanism of injury was available for all patients. Motor vehicle accidents made up the largest group (63.4%), which was made up of 115 car drivers (32.4% of the 355 total responses), 37 car passengers (10.4%), 45 motorcycle drivers (12.7%), one motorcycle passenger (0.3%), and 27 pedestrians or cyclists struck by a motorised vehicle (7.6%).

Falls made up 27.3% of the total, 12.7% from falls less than one metre, and 14.7% from falls of one metre or more. The mechanism of injury was classified as “other” in 9.3% of cases.

The ISS ranged from 16 (the cut-off for major trauma) to 66 and was positively skewed. The mean was 24.3 (SD 9.3) and the median was 21.0.

The validity of ISS was tested by comparing the mean ISS in the group of patients who were excluded because they died while in hospital (during the admission after the accident), to the mean ISS for the survivors. As expected, the mean ISS in the 160 patients who died in hospital was higher than the mean ISS for the 996 survivors (38.8 and 23.7, respectively, $p < 0.001$). The findings are similar if the analysis is restricted to those eligible for the study. The mean ISS in the 116 patients who died in hospital (and were eligible for the study) was 37.8, significantly higher than the mean ISS of 23.7 for the 721 patients eligible to receive the questionnaire who were alive at the time of follow-up ($p < 0.001$).

Severe head injury was present in 51.3% of responding participants, although this was based on the Abbreviated Injury Scale (a score of 3 or higher was considered significant), and was not necessarily related to neurological deficit. This exposure measure was compared with patients' memory of the accident (as measured by four ordered categories), which was expected to be worse for those with a significant head injury. Presence of a head injury was significantly associated with memory of the accident (M-H chi square for trend = 43.93, DF = 1, $p < 0.0001$), with less memory of the accident being associated with presence of a head injury.

Data on occupational status was only available for 235 patients. Due to the nature of the occupational prestige scale,[405] there is no scale for the unemployed, and only 247 subjects were employed at the time of injury. The distribution of occupational status for those employed was close to normal on inspection of the stem-and-leaf plot, the box plot, and the normal probability plot.

Associations between occupational status and other indicators of socio-economic status were highly significant. Each increase in education and income level was associated with increasing status of occupation ($p < 0.0001$ for each).

As occupational status was only able to be calculated for 247 subjects, and was highly correlated with education and income, it was excluded from the multivariate analysis. The author of the occupational prestige scale was contacted (Ann Daniels, personal communication) and stated that education level was closely related to occupation status, and could be used instead of occupation status in any analysis.

All 355 responders provided information regarding employment. Of the 247 (69.6%) responding subjects employed at the time of injury, 46 of these were casual or part time. Of the193 (54.4%) employed at the time of follow-up, 40 of these were casual or part time.

### 3.3.3 Hypothesis 1: Physical health

#### 3.3.3.1 Univariate analysis

In the study population, the mean PCS was 41.9, the median 40.9, and the SD 11.8. The distribution was normal on inspection of the stem-and-leaf plot, the box plot, and the normal probability plot.

The unadjusted association between the explanatory variables and the Physical Component Summary of the SF-36 is given in Table 3.4.

The direction of the findings was such that the PCS score was worse (lower) for those who made a claim, whose claim had not settled, who used a lawyer, who blamed someone else for their injury, and who were unemployed (either prior to the injury or at the time of follow-up).

Table 3.4. Unadjusted association between explanatory variables and PCS.

| Explanatory variable | P value | Category (if applicable) | Mean PCS (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age | 0.0007 | | |
| Sex | 0.02 | Male | 42.8 (41.3 – 44.3) |
| | | Female | 39.5 (37.4 – 41.7) |
| Time since injury | 0.03 | | |
| Chronic illnesses | <0.0001 | None | 45.1 (43.3 – 47.0) |
| | | One | 42.9 (40.7 – 45.0) |
| | | Two | 37.1 (34.3 – 39.9) |
| | | Three or more | 34.6 (31.0 – 38.2) |
| ***Injury severity factors*** | | | |
| ISS | 0.05 | | |
| ICU admission | 0.35 | No | 42.6 (40.7 – 44.6) |
| | | Yes | 41.4 (39.8 – 43.0) |
| Days in ICU | 0.009 | | |
| Head injury | 0.02 | No | 40.4 (38.7 – 42.2) |
| | | Yes | 43.3 (41.5 – 45.0) |
| Mechanism | 0.09 | MVA | 41.1 (39.5 – 42.6) |
| | | Other | 43.3 (41.3 – 45.3) |
| ***Socioeconomic factors*** | | | |
| Education | 0.39 | Primary | 39.1 (34.7 – 43.4) |
| | | Secondary | 41.9 (40.2 – 43.6) |
| | | Certificate / Diploma | 43.0 (40.6 – 45.3) |
| | | Degree | 40.2 (35.9 – 44.4) |
| Income | 0.03 | $0 – 30,000 | 40.4 (38.6 – 42.3) |
| | | $30,001 – 50,000 | 44.8 (42.3 – 47.3) |
| | | $50,001 – 75,000 | 40.3 (37.2 – 43.5) |
| | | Over $75,000 | 43.5 (40.2 – 46.8) |
| Occupation status | 0.23 | | |
| Employed prior | 0.0007 | Employed | 43.2 (41.8 – 44.7) |
| | | Unemployed | 38.6 (36.5 – 40.8) |
| Employed now | <0.0001 | Employed | 46.1 (44.3 – 47.9) |
| | | Unemployed | 38.2 (36.7 – 39.8) |
| ***Claim-related factors*** | | | |
| Claim made | <0.0001 | No | 45.2 (43.4 – 46.9) |
| | | Yes | 37.3 (35.7 – 39.0) |
| Claim settled | 0.007 | No | 35.3 (33.2 – 37.4) |
| | | Yes | 39.7 (37.2 – 42.2) |
| Claim type | 0.20 | Workers compensation | 39.5 (36.2 – 42.9) |
| | | Third party | 37.2 (35.2 – 39.2) |
| Lawyer used | <0.0001 | No | 44.9 (43.3 – 46.5) |
| | | Yes | 36.8 (35.1 – 38.5) |
| Blame | <0.0001 | Self | 45.3 (43.3 – 47.3) |
| | | Someone else | 37.6 (35.6 – 39.6) |
| Time to settlement | 0.57 | | |
| Time since settlement | 0.43 | | |

The direction of association for the general factors was as expected: a higher PCS was associated with being younger, being male, increasing time since the injury, and a lower number of chronic illnesses.

#### 3.3.3.2 Multivariate analysis

Stepwise regression removed the following variables in order, based on least significance in the model: age, prior employment, mechanism, sex, income, days in ICU, and blame. It was not necessary to consider removal of variables with borderline significance as none of the variables removed in the stepwise regression were remotely significant (the lowest significance level was 0.31).

The variables remaining in the final model were: time since injury, chronic illness, ISS, head injury, current employment, claim, and use of a lawyer. The final model used data from 345 subjects and explained 35.5% of the variation in the PCS score. The adjusted $R^2$ values for the initial model (33.3%) and the final model (33.5%) were similar. Interaction terms for each remaining variable with claim were inserted into the final model and were not found to be significant. The direction of the association for each variable was unchanged from the univariate analysis. For the only categorical variable, each increase in chronic illness group (0,1,2,3 or more) was associated with a lower PCS.

The effect estimates and significance levels for the final model are given in Table 3.5. The mean PCS for each group of claim is given in Table 3.6.

Table 3.5. Adjusted (multivariate) association of explanatory variables for PCS in the final model.

| Variable | Sub-group | Δ mean PCS | p value |
|---|---|---|---|
| Time since injury | Per month increase | 0.070 | 0.03 |
| Chronic illnesses | None | * | |
| | 1 | -2.53 | 0.05 |
| | 2 | -6.86 | <0.0001 |
| | 3 or more | -11.30 | <0.0001 |
| | (overall) | | <0.0001 |
| ISS | Per point increase | -0.134 | 0.02 |
| Head injury | Present | 3.16 | 0.003 |
| Employed now | Unemployed | -5.53 | <0.0001 |
| Claim | No claim made | * | |
| | Claim made - settled | -3.99 | 0.02 |
| | Claim made - not settled | -5.18 | 0.006 |
| | (overall) | | 0.01 |
| Lawyer used | Yes | -4.79 | <0.0001 |

* referent group, Δ = change in

Table 3.6. The adjusted mean PCS for each group of claim.

| Claim group | Mean PCS | 95% confidence intervals |
|---|---|---|
| No claim made | 42.0 | 40.2 – 43.7 |
| Claim made – settled | 38.0 | 35.4 – 40.6 |
| Claim made – not settled | 36.8 | 34.0 – 39.6 |

Confounding between use of a lawyer and pursuit of a claim was expected and shown by removing each variable from the final model, which significantly increased the effect estimate and significance of the other.

The final model met the assumptions for multiple regression.

### 3.3.4 Hypothesis 2: Mental health

#### 3.3.4.1 Univariate analysis

Complete SF-36 data for calculation of the MCS was available in 348 of the 355 questionnaires. The mean MCS was 43.3, the median 45.0, and the SD 13.9. Examination of the stem-and-leaf plot, the box plot and the normal probability plot showed a slight negative skew. Transformation of the data was not considered necessary for the analysis because of the large numbers of samples and because the distribution was close to normal.

The unadjusted association between the explanatory variables and the Mental Component Summary of the SF-36 is given in Table 3.7.

#### 3.3.4.2 Multivariate analysis

Stepwise regression removed the following variables in order, based on least significance in the model: time since injury ($p = 0.74$), blame ($p = 0.65$), education ($p = 0.30$), mechanism ($p = 0.14$), and use of a lawyer ($p = 0.08$).

For chronic illness, the mean MCS did not change with each increasing group above the first two groups (zero and one). Therefore, for simplicity, chronic illness was converted to a dichotomous variable. This resulted in a similar model, and no effect estimate changed by more than 10%.

Table 3.7. Unadjusted (univariate) association of explanatory variables for MCS.

| Explanatory variable | P value | Category (if applicable) | Mean MCS (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age | 0.08 | | |
| Sex | 0.73 | Male | 43.4 (41.7 – 45.2) |
| | | Female | 42.9 (40.0 – 45.7) |
| Time since injury | 0.22 | | |
| Chronic illnesses | 0.04 | None | 45.3 (43.0 – 47.5) |
| | | One | 40.9 (38.3 – 43.6) |
| | | Two | 41.3 (37.8 – 44.7) |
| | | Three or more | 45.4 (41.0 – 49.8) |
| ***Injury severity factors*** | | | |
| ISS | 0.50 | | |
| ICU admission | 0.97 | No | 43.2 (40.9 – 45.5) |
| | | Yes | 43.3 (41.4 – 45.3) |
| Days in ICU | 0.95 | | |
| Head injury | 0.63 | No | 43.6 (41.6 – 45.7) |
| | | Yes | 42.9 (40.8 – 45.0) |
| Mechanism | 0.0007 | MVA | 41.5 (39.5 – 43.4) |
| | | Other | 46.5 (44.3 – 48.6) |
| ***Socioeconomic factors*** | | | |
| Education | 0.21 | Primary | 41.9 (36.7 – 47.1) |
| | | Secondary | 42.2 (40.2 – 44.3) |
| | | Certificate / Diploma | 44.0 (41.2 – 46.7) |
| | | Degree | 47.7 (42.7 – 52.7) |
| Income | 0.26 | $0 – 30,000 | 41.8 (39.6 – 44.0) |
| | | $30,001 – 50,000 | 45.0 (42.0 – 48.0) |
| | | $50,001 – 75,000 | 42.6 (38.9 – 46.3) |
| | | Over $75,000 | 45.3 (41.3 – 49.2) |
| Occupation status | 0.03 | | |
| Employed prior | 0.72 | Employed | 43.4 (41.6 – 45.3) |
| | | Unemployed | 42.9 (40.5 – 45.3) |
| Employed now | <0.0001 | Employed | 47.2 (45.2 – 49.3) |
| | | Unemployed | 39.9 (37.9 – 41.8) |
| ***Claim-related factors*** | | | |
| Claim made | <0.0001 | No | 46.7 (44.9 – 48.5) |
| | | Yes | 38.7 (36.4 – 41.1) |
| Claim settled | 0.007 | No | 34.6 (31.4 – 37.7) |
| | | Yes | 43.6 (40.4 – 46.8) |
| Claim type | 0.61 | Workers compensation | 40.5 (36.4 – 44.6) |
| | | Third party | 39.2 (36.0 – 42.4) |
| Lawyer used | <0.0001 | No | 46.5 (44.8 – 48.1) |
| | | Yes | 37.4 (34.8 – 39.9) |
| Blame | 0.001 | Self | 46.4 (44.2 – 48.7) |
| | | Someone else | 40.6 (37.8 – 43.3) |
| Time to settlement | 0.03 | | |
| Time since settlement | 0.44 | | |

Interaction terms in the final model for each variable with claim were not significant. The final model met the assumptions for multiple regression.

The confounding between the variables claim and lawyer with MCS as the dependent variable was similar to that seen with PCS as the dependent variable.

The final model used data from 347 subjects and explained 22.0% of the variation in MCS. The effect estimates and significance levels for the final model are given in Table 3.8. The mean MCS for each group of claim is given in Table 3.9.

Table 3.8. Adjusted (multivariate) association of explanatory variables for MCS in the final model.

| Variable | Sub-group | Δ mean MCS | p value |
|---|---|---|---|
| Age | Per year increase | 0.157 | 0.0001 |
| Chronic illnesses | Present | -4.60 | 0.002 |
| Current employment | Unemployed | -7.79 | <0.0001 |
| Claim | No claim made | * | |
| | Claim made – settled | -2.77 | 0.11 |
| | Claim made - not settled | -10.24 | <0.0001 |
| | (overall) | | <0.0001 |

* referent group, Δ = change in

Table 3.9. The adjusted mean MCS for each group of claim.

| Claim group | Mean MCS | 95% confidence intervals |
|---|---|---|
| ***Final model*** | | |
| No claim made | 46.2 | 44.4 – 47.9 |
| Claim made – settled | 43.4 | 40.5 – 46.4 |
| Claim made – not settled | 36.0 | 33.2 – 38.7 |

### 3.3.5 Hypothesis 3: Neck pain

#### 3.3.5.1 Univariate analysis

A floor effect was noted in the continuous outcome for neck pain, as many subjects scored 2. This occurred when subjects reported no recent pain and no interference with activities due to the pain. Due to the large numbers, however, the variable was treated as a normally distributed continuous variable, and an alternative analysis using neck pain as a dichotomous outcome revealed similar associations.

The univariate analysis of the association between neck pain and the explanatory is given in Table 3.10.

#### 3.3.5.2 Multivariate analysis

Backward stepwise multiple regression was performed for neck pain as a continuous variable. The final model included sex, chronic illness, current

Table 3.10. Unadjusted association of explanatory variables for neck pain.

| Explanatory variable | P value | Category (if applicable) | Mean neck pain (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age | 0.12 | | |
| Sex | 0.007 | Male | 4.0 (3.7 – 4.3) |
| | | Female | 4.8 (4.2 – 5.3) |
| Time since injury | 0.19 | | |
| Chronic illnesses | 0.05 | None | 3.8 (3.4 – 4.2) |
| | | One | 4.3 (3.8 – 4.8) |
| | | Two | 4.5 (3.8 – 5.1) |
| | | Three or more | 4.8 (4.1 – 5.6) |
| ***Injury severity factors*** | | | |
| ISS | 0.07 | | |
| ICU admission | 0.52 | No | 4.3 (3.8 – 4.7) |
| | | Yes | 4.1 (3.8 – 4.4) |
| Days in ICU | 0.44 | | |
| Head injury | 0.08 | No | 4.4 (4.0 – 4.8) |
| | | Yes | 4.0 (3.6 – 4.3) |
| Mechanism | 0.09 | MVA | 4.3 (4.0 – 4.7) |
| | | Other | 3.9 (3.5 – 4.3) |
| Neck fracture | 0.01 | No | 4.1 (3.8 – 4.3) |
| | | Yes | 5.4 (4.2 – 6.5) |
| ***Socioeconomic factors*** | | | |
| Education | 0.002 | Primary | 5.8 (4.9 – 6.7) |
| | | Secondary | 4.2 (3.8 – 4.5) |
| | | Certificate / Diploma | 3.9 (3.4 – 4.4) |
| | | Degree | 3.6 (2.7 – 4.5) |
| Income | 0.23 | $0 – 30,000 | 4.5 (4.1 – 4.9) |
| | | $30,001 – 50,000 | 3.9 (3.4 – 4.5) |
| | | $50,001 – 75,000 | 4.1 (3.5 – 4.8) |
| | | Over $75,000 | 3.7 (3.0 – 4.5) |
| Occupation status | 0.02 | | |
| Employed prior | 0.14 | Employed | 4.0 (3.7 – 4.4) |
| | | Unemployed | 4.5 (4.0 – 5.0) |
| Employed now | <0.0001 | Employed | 3.6 (3.3 – 3.9) |
| | | Unemployed | 4.7 (4.3 – 5.0) |
| ***Claim-related factors*** | | | |
| Claim made | <0.0001 | No | 3.6 (3.3 – 3.9) |
| | | Yes | 4.9 (4.5 – 5.4) |
| Claim settled | 0.03 | No | 5.4 (4.8 – 6.0) |
| | | Yes | 4.4 (3.8 – 5.0) |
| Claim type | 0.17 | Workers compensation | 4.3 (3.7 – 5.1) |
| | | Third party | 5.0 (4.4 – 5.6) |
| Lawyer used | <0.0001 | No | 3.6 (3.3 – 3.9) |
| | | Yes | 5.2 (4.7 – 5.7) |
| Blame | <0.0001 | Self | 3.4 (3.0 – 3.8) |
| | | Someone else | 4.8 (4.3 – 5.3) |
| Time to settlement | 0.29 | | |
| Time since settlement | 0.08 | | |

employment, head injury, neck fracture, the use of a lawyer, and education level. The direction of the association was such that higher neck pain scores were associated with female gender, unemployment, absence of a significant head injury, presence of a neck fracture, the use of a lawyer, and decreasing levels of education. Pursuit or settlement of a claim was not significant after adjusting for these confounders, and was therefore excluded from the model. For chronic illnesses, differences in the effect between groups was minor, therefore this was analysed as a dichotomous variable. This alternative analysis did not change the effect estimates of the other variables by more than 5%.

The final model contained data from 347 subjects and explained 23.9% of the variation in the neck pain score. Interaction terms introduced into the final model were not significant. The final model satisfied the assumptions for multiple regression. The adjusted effect estimates and significance levels are given in Table 3.11. The effect estimate is the change in the mean neck pain score for each variable group.

In a post hoc univariate analysis, the diagnosis of PTSD at the time of follow-up was noted to be strongly associated with neck pain ($p < 0.0001$). Therefore, a separate multivariate analysis was performed including PTSD as an explanatory variable. The final model (Table 3.12) included PTSD, and explained 31.3% of the variation in neck pain.

Table 3.11. Adjusted (multivariate) association between explanatory variables and neck pain.

| Variable | Group | Δ mean score | P value |
|---|---|---|---|
| Sex | Female | +0.67 | 0.01 |
| Head injury | Present | -0.66 | 0.008 |
| Neck fracture | Present | 1.18 | 0.01 |
| Current employment | Unemployed | +0.58 | 0.03 |
| Chronic illnesses | Present | +0.71 | 0.007 |
| Education level | Primary | * | |
| | Secondary | -1.84 | <0.0001 |
| | Diploma/Certificate | -1.74 | 0.0006 |
| | Degree | -2.46 | <0.0001 |
| | (overall) | | 0.0006 |
| Use of a lawyer | | +1.84 | <0.0001 |

*referent group, Δ = change in

Table 3.12. Multivariate association between explanatory variables and neck pain, including PTSD as an explanatory variable.

| Variable | Value | Δ mean score | P value |
|---|---|---|---|
| Age | Per year increase | +0.016 | 0.02 |
| Sex | Female | +0.58 | 0.03 |
| Neck fracture | Yes | +1.24 | 0.004 |
| Education level | Primary | * | |
| | Secondary | -1.69 | 0.0002 |
| | Diploma/Certificate | -1.65 | 0.0005 |
| | Degree | -2.15 | 0.0002 |
| | (overall) | | 0.0008 |
| Use of a lawyer | Yes | +0.95 | 0.0006 |
| PTSD | Yes | +2.03 | <0.0001 |

*referent group, Δ = change in

### 3.3.6 Hypothesis 4: Back pain

#### 3.3.6.1 Univariate analysis

The distribution of the scores for back pain was similar to the distribution for neck pain. Similarly, it was treated as a continuous variable, and later tested as a dichotomous variable, with similar findings. The univariate analysis of explanatory variables against back pain is given in Table 3.13.

#### 3.3.6.2 Multivariate analysis

The model using back pain as a continuous variable was accepted as this was the model chosen a priori and it satisfied the assumptions of multiple regression.

The associations in the final model were such that increasing levels of back pain were associated with current unemployment, using a lawyer, the presence of chronic illnesses, and lower levels of education. due to minor differences in the effect estimates between the groups with 1, 2, and 3 or more chronic illnesses, this was analysed as a dichotomous variable. This did not change the effect estimates of the other variables by more than 5%.

The effect estimates and significance levels for the final model using back pain as a continuous variable are given in Table 3.14. This model used data from 347 patients and explained 17.1% of the variation in back pain.

In a post hoc univariate analysis, the diagnosis of PTSD at the time of follow-up was noted to be strongly associated with back pain ($p < 0.0001$). Therefore, a separate multivariate analysis was performed including PTSD as an explanatory variable. Compared to the final model without PTSD (above), the final model with PTSD also included chronic illness, education level, and use of a lawyer, but current employment was not significant in this model. Presence of a thoracolumbar fracture at the time of injury was significant in this model. The final model (Table 3.15) used data from 347 patients and explained 27.0% of the variation in back pain.

Table 3.13. Unadjusted association of explanatory variables for low back pain.

| Explanatory variable | P value | Category (if applicable) | Mean back pain (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age | 0.13 | | |
| Sex | 0.05 | Male | 5.0 (4.6 – 5.3) |
| | | Female | 5.6 (5.1 – 6.2) |
| Time since injury | 0.08 | | |
| Chronic illnesses | 0.0004 | None | 4.5 (4.0 – 4.9) |
| | | One | 5.3 (4.8 – 5.8) |
| | | Two | 6.1 (5.4 – 6.7) |
| | | Three or more | 5.7 (4.8 – 6.5) |
| ***Injury severity factors*** | | | |
| ISS | 0.26 | | |
| ICU admission | 0.37 | No | 5.3 (4.9 – 5.8) |
| | | Yes | 5.0 (4.7 – 5.4) |
| Days in ICU | 0.46 | | |
| Head injury | 0.39 | No | 5.3 (4.9 – 5.7) |
| | | Yes | 5.0 (4.6 – 5.4) |
| Mechanism | 0.06 | MVA | 5.4 (5.0 – 5.7) |
| | | Other | 4.8 (4.3 – 5.2) |
| Thoracolumbar fracture | 0.18 | No | 5.1 (4.8 – 5.4) |
| | | Yes | 5.7 (4.9 – 6.5) |
| ***Socioeconomic factors*** | | | |
| Education | 0.05 | Primary | 6.5 (5.5 – 7.5) |
| | | Secondary | 5.1 (4.7 – 5.5) |
| | | Certificate / Diploma | 5.0 (4.5 – 5.6) |
| | | Degree | 4.8 (3.8 – 5.8) |
| Income | 0.03 | $0 – 30,000 | 5.6 (5.2 – 6.1) |
| | | $30,001 – 50,000 | 4.9 (4.1 – 5.3) |
| | | $50,001 – 75,000 | 5.1 (4.3 – 5.8) |
| | | Over $75,000 | 4.6 (3.8 – 5.4) |
| Occupation status | 0.19 | | |
| Employed prior | 0.01 | Employed | 4.9 (4.6 – 5.2) |
| | | Unemployed | 5.7 (5.1 – 6.2) |
| Employed now | 0.0001 | Employed | 4.5 (4.2 – 4.9) |
| | | Unemployed | 5.7 (5.2 – 6.1) |
| ***Claim-related factors*** | | | |
| Claim made | <0.0001 | No | 4.5 (4.2 – 4.9) |
| | | Yes | 6.0 (5.5 – 6.4) |
| Claim settled | 0.01 | No | 6.5 (5.9 – 7.1) |
| | | Yes | 5.4 (4.7 – 6.0) |
| Claim type | 0.29 | Workers compensation | 5.5 (4.7 – 6.3) |
| | | Third party | 6.0 (5.4 – 6.6) |
| Lawyer used | <0.0001 | No | 4.6 (4.2 – 4.9) |
| | | Yes | 6.1 (5.7 – 6.6) |
| Blame | <0.0001 | Self | 4.4 (4.0 – 4.8) |
| | | Someone else | 5.9 (5.4 – 6.4) |
| Time to settlement | 0.31 | | |
| Time since settlement | 0.22 | | |

Table 3.14. Adjusted (multivariate) association between explanatory variables and back pain.

| Variable | Group | ∆ mean score | p value |
|---|---|---|---|
| Chronic illnesses | Present | +1.23 | <0.0001 |
| Current employment | Unemployed | +0.65 | 0.03 |
| Education level | Primary | * | |
| | Secondary | -1.46 | 0.005 |
| | Diploma/Cert. | -1.06 | 0.06 |
| | Degree | -1.48 | 0.03 |
| | (overall) | | 0.03 |
| Use of a lawyer | Yes | +1.80 | <0.0001 |

* referent group, ∆ = change in

Table 3.15. Multivariate association between explanatory variables and back pain, including PTSD as an explanatory variable.

| Variable | Group | ∆ mean score | p value |
|---|---|---|---|
| Chronic illnesses | None | * | |
| | 1 | +0.85 | 0.007 |
| | 2 | +1.37 | 0.0003 |
| | 3 or more | +1.38 | 0.003 |
| | (overall) | | 0.0004 |
| Thoracolumbar fracture | Present | +0.90 | 0.03 |
| Education level | Primary | * | |
| | Secondary | -1.66 | 0.0007 |
| | Diploma/Certificate | -1.28 | 0.01 |
| | Degree | -1.67 | 0.008 |
| | (overall) | | 0.007 |
| Lawyer | Lawyer used | +0.93 | 0.003 |
| PTSD | Present | +2.01 | <0.0001 |

* referent group, ∆ = change in

### 3.3.7 Hypothesis 5: Patient satisfaction

#### 3.3.7.1 Univariate analysis

Data regarding patient satisfaction were available on 353 subjects. Of these, 150 (42.5%) were 'very satisfied' with their progress since the injury, 102 (28.9%) were 'somewhat satisfied', 64 (18.1%) were 'somewhat dissatisfied', and 37 (10.5%) were 'very dissatisfied'. For the dichotomised outcome used in the multivariate analysis, 71.4% were satisfied, and 28.6% were dissatisfied.

The univariate associations for patient satisfaction (as a four-part variable) are given in Table 3.16. In this table, the effect estimate is provided using patient satisfaction as a dichotomous variable (satisfied / unsatisfied) to allow easier interpretation of the association. The direction of the association for the categorical variables can be determined from the table. For the continuous variables, the direction of the association was such that patient satisfaction was associated with: lower age, longer time since injury, lower ISS, lower occupational status, and shorter time to settlement. There was no trend seen in the association between satisfaction and time in ICU or time since settlement.

#### 3.3.7.2 Multivariate analysis

The final model, using backward stepwise logistic regression, resulted from the elimination of the following variables (in order): prior employment ($p = 0.61$),

Table 3.16. Univariate analysis of the association between patient satisfaction and exposure variables.

| Explanatory variable | p value | Category (if applicable) | % satisfied |
|---|---|---|---|
| ***General factors*** | | | |
| Age | 0.48 | | |
| Sex | 0.81 | Male | 71.8 |
| | 0.78(MH) | Female | 70.4 |
| Time since injury | 0.02 | | |
| Chronic illnesses | 0.17 | None | 80.1 |
| | 0.003(MH) | 1 | 66.0 |
| | | 2 | 66.7 |
| | | 3 or more | 60.5 |
| ***Injury severity factors*** | | | |
| ISS | 0.81 | | |
| ICU admission | 0.38 | No | 69.8 |
| | 0.81(MH) | Yes | 72.4 |
| Days in ICU | 0.43 | | |
| Head injury | 0.53 | No | 72.7 |
| | 0.93(MH) | Yes | 70.2 |
| Mechanism | 0.003 | MVA | 66.5 |
| | 0.0004 | Other | 79.8 |
| ***Socioeconomic factors*** | | | |
| Education | 0.44 | Primary | 63.3 |
| | 0.96(MH) | Secondary | 71.8 |
| | | Certificate/Diploma | 74.0 |
| | | Degree | 66.7 |
| Income | 0.51 | $0 – 30,000 | 67.5 |
| | 0.37(MH) | $30,001 – 50,000 | 73.5 |
| | | $50,001 – 75,000 | 79.2 |
| | | Over $75,000 | 72.9 |
| Occupation status | 0.63 | | |
| Employed prior | 0.27 | Employed | 74.5 |
| | 0.09(MH) | Unemployed | 64.2 |
| Employed now | <0.0001 | Employed | 82.1 |
| | <0.0001(MH) | Unemployed | 63.3 |
| ***Claim-related factors*** | | | |
| Claim made | <0.0001 | No | 81.0 |
| | <0.0001(MH) | Yes | 58.6 |
| Claim settled | <0.0001 | No | 43.9 |
| | <0.0001(MH) | Yes | 75.7 |
| Claim type | 0.071 | Workers compensation | 68.7 |
| | 0.12(MH) | Third party | 54.9 |
| Lawyer involvement | <0.0001 | No | 79.9 |
| | <0.0001(MH) | Yes | 55.9 |
| Blame | <0.0001 | Self | 83.7 |
| | <0.0001(MH) | Someone else | 58.9 |
| Time to settlement | 0.37 | | |
| Time since settlement | 0.97 | | |

blame (p = 0.40), use of a lawyer (p = 0.22), and time since injury (p = 0.12). The effect estimates and significance levels for the final model are given in Table 3.17. The final model used data from 352 subjects. There were no significant interaction terms. Satisfactory association between predicted and observed responses was noted with 74.8% concordance and a c value of 0.76.

The odds ratios given are the odds of being unsatisfied, so that an odds ratio above one is a poor outcome (indicating dissatisfaction) and an odds ratio less than one indicates satisfaction. The direction of the associations were such that dissatisfaction was associated with being unemployed, being in a motor vehicle accident, having increasing chronic illnesses, and having an unsettled claim (compared to not making a claim). The odds ratio of dissatisfaction in those having a settled claim (compared to not making a claim) was not significant.

Table 3.17. Independent predictors of patient dissatisfaction.

| Variable | Group | Odds ratio (95% CI) | p value |
|---|---|---|---|
| Current employment | Unemployed | 2.27 (1.31 – 3.93) | 0.004 |
| Mechanism | MVA | 1.90 (1.05 – 3.47) | 0.04 |
| Chronic illness | None | 1.00 | |
| | 1 | 2.50 (1.30 – 4.81) | 0.006 |
| | 2 | 2.25 (1.08 – 4.69) | 0.03 |
| | 3 or more | 3.76 (1.55 – 9.13) | 0.004 |
| | (overall) | | 0.009 |
| Claim | | | |
| | No claim made | 1.00 | |
| | Claim settled | 1.27 (0.63 – 2.57) | 0.51 |
| | Claim not settled | 5.41 (2.89 – 10.14) | <0.0001 |
| | (overall) | | <0.0001 |

### 3.3.8 Hypothesis 6: Post-traumatic stress disorder

#### 3.3.8.1 Univariate analysis

The PTSD was scored on all 355 responding subjects. The distribution of the PTSD score, which has a possible range of 17 – 85, was not normal. The most common scores were the lowest, with decreasing frequencies noted with increasing score, and a floor effect was noted due to approximately 10% of subjects scoring the lowest score.

In addition to analysing the PTSD score as a continuous outcome, a dichotomous outcome variable was used based on the recommended cut-off score of 44 to define PTSD, as stated a priori. Using this new variable, 36.3% of the 355 subjects were classed as having PTSD.

The univariate analyses of the explanatory variables against PTSD as a continuous variable are given in Table 3.18. Univariate analysis of PTSD as a dichotomous variable yielded similar results: the only difference at the 0.25 level was for head injury, which was significant at 0.55 in the continuous model and 0.18 in the dichotomous model.

Table 3.18. Univariate (unadjusted) analysis of explanatory variables for PTSD scale as a continuous outcome.

| Explanatory variable | P value | Category (if applicable) | Mean PTSD score |
|---|---|---|---|
| ***General factors*** | | | |
| Age | 0.001 | | |
| Sex | 0.18 | Male | 37.1 (34.9 – 39.3) |
| | | Female | 40.0 (36.3 – 43.7) |
| Time since injury | 0.15 | | |
| Chronic illnesses | 0.14 | None | 36.0 (33.1 – 39.0) |
| | | One | 39.1 (35.7 – 42.6) |
| | | Two | 41.6 (37.2 – 46.0) |
| | | Three or more | 35.4 (29.7 – 41.1) |
| ***Injury severity factors*** | | | |
| ISS | 0.21 | | |
| ICU admission | 0.41 | No | 36.9 (33.8 – 40.1) |
| | | Yes | 38.5 (36.2 – 40.9) |
| Days in ICU | 0.42 | | |
| Head injury | 0.55 | No | 38.5 (35.8 – 41.2) |
| | | Yes | 37.3 (34.7 – 40.0) |
| Mechanism | <0.0001 | MVA | 41.0 (38.5 – 43.5) |
| | | Other | 32.7 (29.9 – 35.2) |
| ***Socioeconomic factors*** | | | |
| Education | 0.78 | Primary | 37.4 (30.8 – 43.9) |
| | | Secondary | 39.0 (36.4 – 41.6) |
| | | Certificate / Diploma | 36.7 (33.1 – 40.3) |
| | | Degree | 37.9 (31.3 – 44.4) |
| Income | 0.83 | $0 – 30,000 | 38.4 (35.6 – 41.2) |
| | | $30,001 – 50,000 | 36.2 (32.3 – 40.1) |
| | | $50,001 – 75,000 | 38.5 (33.6 – 43.3) |
| | | Over $75,000 | 37.6 (32.4 – 42.7) |
| Occupation status | 0.002 | | |
| Employed prior | 0.35 | Employed | 38.5 (36.2 – 40.8) |
| | | Unemployed | 36.5 (33.2 – 39.9) |
| Employed now | 0.0002 | Employed | 34.0 (31.4 – 36.7) |
| | | Unemployed | 41.2 (38.6 – 43.8) |
| ***Claim-related factors*** | | | |
| Claim made | <0.0001 | No | 30.6 (28.6 – 32.6) |
| | | Yes | 47.6 (44.7 – 55.0) |
| Claim settled | <0.0001 | No | 52.9 (49.1 – 56.7) |
| | | Yes | 41.3 (37.2 – 45.5) |
| Claim type | 0.47 | Workers compensation | 44.7 (38.8 – 57.0) |
| | | Third party | 47.1 (43.5 – 56.0) |
| Lawyer used | <0.0001 | No | 30.9 (29.0 – 32.7) |
| | | Yes | 50.5 (47.3 – 53.6) |
| Blame | <0.0001 | Self | 29.3 (27.1 – 31.5) |
| | | Someone else | 45.9 (42.5 – 49.4) |
| Time to settlement | 0.40 | | |
| Time since settlement | 0.52 | | |

### 3.3.8.2 Multivariate analysis

Multivariate analysis was initially performed using the PTSD score as a continuous variable. The mean score was 37.9, the median was 34.0, and the range was from 17 to 85. The distribution was slightly positively skewed.

Backward stepwise regression resulted in a final model with age, chronic illness, current employment, claim, use of a lawyer and blame as the significant variables. Interaction terms in the final model were not significant. The assumptions for multiple regression were not violated. The final model included data from 350 subjects and accounted for 40.7% of the variation in the PTSD score. The results of the multivariate analysis using the PTSD scale as a continuous outcome is given in Table 3.19.

The adjusted mean PTSD scores for each category of blame and claim are given in Table 3.20.

The multivariate analysis was also performed using logistic regression with PTSD as a dichotomous variable (using the cut-off value of 44 for the diagnosis of PTSD). This resulted in the same final model as that given above (Appendix 9).

Table 3.19. Adjusted (multivariate) association for the explanatory variables and PTSD score as a continuous outcome.

| Variable | Group | ∆ mean score | p value |
|---|---|---|---|
| Age | Per year | -0.26 | <0.0001 |
| Chronic illness | None | * | |
| | 1 | 5.20 | 0.007 |
| | 2 | 9.92 | <0.0001 |
| | 3 or more | 8.47 | 0.005 |
| | (overall) | | <0.0001 |
| Current employment | Unemployed | 7.95 | <0.0001 |
| Use of a lawyer | Yes | 10.24 | <0.0001 |
| Claim | No claim made | * | |
| | Claim made - settled | 1.66 | 0.52 |
| | Claim made - not settled | 8.03 | 0.007 |
| | (overall) | | 0.01 |
| Blame for injury | Blame self | * | |
| | Don't know | 5.87 | 0.004 |
| | Blame others | 6.89 | 0.003 |
| | (overall) | | 0.004 |

* referent group, ∆ = change in

Table 3.20. The adjusted mean PTSD scores for each category of claim and blame in the final model.

| Variable | Subgroup | Mean PTSD score | 95% confidence interval |
|---|---|---|---|
| Claim | No claim made | 37.7 | 34.9 – 40.6 |
| | Claim - settled | 39.4 | 35.6 – 43.2 |
| | Claim - not settled | 45.8 | 41.5 – 50.1 |
| Blame | I was at fault | 36.7 | 33.3 – 40.1 |
| | Don't know | 42.6 | 39.5 – 45.7 |
| | Someone else at fault | 43.6 | 40.6 – 46.6 |

### 3.3.9 Compensation: pursuit versus eligibility

As stated a priori, the effect of claim pursuit was compared to the effect of claim entitlement. Pursuit of a claim, and claim entitlement were strongly associated with each other, but differences existed, enabling a comparison of the effect of these two variables on the main outcomes.

Claim eligibility was substituted for claim pursuit in each off the final models and these results are reported in Appendix 10. The effect estimate for both variables was similar, with claim eligibility having a slightly stronger association with poor outcome for each variable except patient satisfaction.

### 3.3.10 Fault-based versus no-fault compensation

The two compensation systems under which subjects in this study were treated were workers compensation and compulsory third party motor vehicle insurance. The former is a no-fault system (covering all entitled subjects regardless of fault) whereas the latter system only covers those who are not at fault, i.e., it is a fault-based system.

Univariate analysis of the variable CLAIMTYPE, a dichotomous variable measuring whether the claim was made under workers compensation or third party, was tested in the univariate analyses and is given in the tables. Type of claim was not significantly associated with any of the outcome variables in the univariate analyses at a level of 0.05 or less.

To further test the variable CLAIMTYPE against each outcome variable, it was placed in the final model for each outcome. The analysis was then restricted to those subjects who had made a claim, and were treated under the workers compensation or third party systems (subjects who were treated under both systems were excluded from this analysis). As the analysis did not include subjects who had not made a claim, the variable CLAIM was restricted to whether or not the claim was settled, and was therefore able to be kept in the model.

CLAIMTYPE was not found to be significantly associated with any of the outcome variables. The direction of the association was also mixed, with third party compensation being slightly beneficial when MCS and PCS were the outcomes used, and the opposite being the case for the other outcome variables. Further, the estimates of effect for CLAIM (whether settled or not) and use of a lawyer did not significantly change when CLAIMTYPE was added to the models. The effect estimates for CLAIMTYPE are given in Table 3.21.

Table 3.21. The association between type of claim (CLAIMTYPE) and the outcome variables.

| Outcome | n | Parameter estimate | S.E. | Test statistic | P value |
|---|---|---|---|---|---|
| PCS | 125 | 1.11 | 1.73 | t = 0.65 | 0.52 |
| MCS | 127 | 1.76 | 2.55 | t = 0.69 | 0.49 |
| Neck pain | 127 | 0.44 | 0.48 | t = 0.91 | 0.37 |
| Back pain | 127 | 0.11 | 0.10 | t = 1.09 | 0.28 |
| Satisfaction | 130 | 0.37 | 0.43 | $chi^2$ = 0.74 | 0.39 |
| PTSD (dichotomous) | 128 | 0.53 | 0.52 | $chi^2$ = 1.02 | 0.31 |
| PTSD (continuous) | 128 | 0.44 | 3.34 | t = 0.13 | 0.90 |

## 3.4 Discussion

### 3.4.1 Summary of main results

Pursuit of a compensation claim was associated with poor physical health one to six years after major accidental trauma in adults, allowing for differences in age, sex, injury severity, chronic illness and socio-economic factors. Having an unsettled claim was associated with a further decrease in physical health, as measured by the SF-36 Physical Component Summary.

For the same cohort, mental health, as measured by both the Mental Component Summary of the SF-36 and the PTSD Scale (Civilian version), was significantly negatively associated with pursuit of a claim only if the claim had not settled, allowing for the other explanatory variables measured. This was also the case for patient satisfaction, for which having an unsettled claim was the strongest predictor of dissatisfaction.

Having an unsettled claim was also associated with increasing low back pain, but no aspects of compensation claim were significantly associated with neck pain following accidental trauma.

There was confounding between the effects of claiming compensation and using a lawyer. Despite this, use of a lawyer was independently significantly associated with poor outcome as measured by PCS, neck pain, back pain and PTSD. Use of a lawyer was also independently associated with poor MCS scores but the p value (0.08) fell outside the level of statistical significance set a priori and this variable was therefore excluded from the final model. Although use of a lawyer was a strongly associated with dissatisfaction in the univariate analysis ($p<0.0001$) it was not significant in the multivariate analysis due to the stronger effect of having an unsettled claim.

Blame, another exposure variable used in this study, was only significantly associated with PTSD in the multivariate analyses. Blaming oneself for the injury had a protective effect on the development of PTSD, and this effect was independent of the other explanatory variables including claim settlement and use of a lawyer.

The size of the negative associations between the main explanatory variables (claim, use of a lawyer, and blame) when significant, were also clinically important as shown in the multivariate analyses.

The associations between the other explanatory variables and each of the outcomes, when present, were in the direction expected except for the presence of a significant head injury. Presence of a significant head injury was significantly associated with higher (better) PCS scores and lower neck pain scores, however this may reflect an increase in other physical injuries in this group of patients, as discussed earlier.

There was no significant effect modification seen in any of the analyses and the models were noted to be stable when tested using different methods (transforming explanatory and outcome variables, and using different methods of analysis).

### 3.4.2 Comments on methods

#### 3.4.2.1 General methods

This study is limited by its retrospective design. Fortunately, however, data pertaining to the injury were largely collected prospectively on the hospital trauma database. The only injury-related information collected retrospectively (in the questionnaire) was the mechanism of injury. Mechanism of injury was not used in the main analysis but was used to determine claim eligibility. Also, mechanism of injury was considered an objective question, unlikely to be influenced by time or other circumstances occurring since the injury.

The socio-economic information and information regarding blame was also collected retrospectively and may be influenced by recall bias. The remaining information was contemporaneous information pertaining to the outcome variables and therefore not influenced by recall bias.

Marital status was not measured in this study, but has varyingly been implicated as a psychosocial predictor of pain and disability in previous studies.[145 255 415 416]

Another limitation of this study is that in testing the effect of time-related factors such as time since injury and settlement, it measured the effect for the whole group at one point in time. Time-sensitive variables such as time since injury would have been more accurately measured by following individual patients over a period of time. Similarly, the effect of settlement may have been more appropriately measured by measuring the outcomes on each patient before and after settlement.

Incomplete follow-up in studies of this nature may lead to selection bias. Those who respond to the survey may not be representative of the population as a whole. This can be minimised by increasing the rate of follow-up, and can be monitored by comparing the study population to the source population. The follow-up rate in this study was higher than expected, based on a previous study from the same institution (M Sugrue, personal communication). Also, being trauma patients, they may not have lived locally, and were mostly male and with a lower than average education level. The last two factors have previously been associated with lack of follow-up after major trauma.[307] Also, the time to follow-up was relatively long (up to six years) so many of the questionnaires mailed out may have gone to addresses that were no longer valid for the intended patients. Despite this, 60.9% of the questionnaires sent were returned completed, and 79.2% of the known contacts responded.

The inclusion of elderly patients may have also influenced the rate of compliance, as several elderly patients were in nursing homes and unable to complete the questionnaire due to dementia.

Comparisons between non-responding and responding subjects showed that non-responders were more likely to be younger (mean age 44.5 years compared to 47.8) and had a longer time since injury (mean time since injury of 45.2 months compared to 41.0). These findings are in the direction expected, as younger patients are less likely to respond to surveys[417-419] and we found that increasing time since injury was associated with increasing difficulty establishing contact. Therefore, many of the non-responders (with longer times since injury) may not have received the questionnaire due to changes in address.

There was no evidence of selection bias for the other variables taken from the trauma database (gender, ISS or presence of a significant head injury). There was some evidence of selection bias in that non-responding patients were more likely to be passengers or pedestrians. The reasons for this are unclear, however, passengers and pedestrians are more likely to be covered by third party compensation, and there are several possible explanations for an under-representation of compensated patients. They may have been less comfortable reporting aspects of the injury and its consequences (due to increased stress, anxiety and other mental health issues, as shown in the study), they may have been more suspicious about the use of the information particularly if they are involved in an active case (as several patients refused to participate on advice from their lawyers), they may have had to report their health more often due to the compensation process (for medical reports) and may therefore be more likely to refuse any further health reporting, or they

may have been more likely to have changed their address secondary to psychosocial circumstances such as job loss or marriage breakdown.
This study also presumes that the accidental injury was a random event, or at least outside the control of the patient. This may not be the case, as has been suggested,[47 332 333 420] and this is supported by findings which show disproportionate representation of certain patient groups in accidents, for example, the higher motor vehicle accident rate amongst young males.

### 3.4.2.2 Outcome variables

All outcome measures used in this study are patient-based. The main outcome measures (PCS and MCS) are calculated from the scores on the SF-36 General Health Survey and have been widely validated as accurate and reliable measures of physical and mental health.

The neck and back pain scores were based on the Bodily Pain index of the SF-36 survey. They do not, however, provide normally distributed scores due to the floor effect, in turn due to the proportion of subjects with no neck pain or back pain. Despite this, the scores were suitable for statistical analysis and the results were confirmed by forming a new dichotomous variable for each outcome. Although these scores are not widely used for back or neck pain they are likely to be valid as they are similar to the previously validated Bodily Pain index of the SF-36.[400]

The patient satisfaction score was developed for this thesis and has not previously been used. Similar scores, usually based on Likert scales,[421 422] usually with a four-point scale (Blount) have been used but usually only to assess patient satisfaction with a treatment process, for example, satisfaction with the treatment given by a particular clinic. The question used in this study attempted to assess each patient's satisfaction with their general progress since the injury, not their satisfaction with any single part of their treatment. This question was piloted as part of the questionnaire in Chapter Four and was found to have good test-retest reliability. Development of a new question for patient satisfaction was necessary, as previous reviews have shown that there is currently no validated outcome tool for this parameter.[423-425]

PTSD was measured by a patient-reported questionnaire, not by structured interview. While this may have influenced the incidence of PTSD, the main analysis for this study was the score on the PTSD scale, and the PTSD scale used has been previously validated.[408-411] By measuring the influence of the explanatory variables on the overall PTSD score in the main analysis, this study avoided any debate regarding the cut-off score and criteria for the diagnosis of PTSD. The validity of the model was supported by a secondary analysis using a cut-off score of 44 to create a dichotomous variable for PTSD, which reached the same final model.

Other factors (stressors) may influence the incidence of PTSD other than the index trauma. This has not been measured but it is hoped that there would not be bias in the incidence of these factors, and that the influence of other factors would only bias the results towards the null due to measurement error.

O'Donnell et al reviewed the literature pertaining to PTSD after injury and made several suggestions to improve the methodology of future studies.[426] Some of the suggestions relate to measuring PTSD in the early phase (the effects of being in hospital, and of taking narcotics), which are not an issue with this study. Their suggestions to measure brain injury as well as injury severity, to allow for time since injury, to use consecutive admissions, and to allow for compensation have been met by this study.

#### 3.4.2.3 Explanatory variables

The participants' general medical condition was difficult to measure. The method chosen, to measure the number of chronic illnesses, fails to take into consideration the severity of any chronic illness and has not been validated as a measure of general medical condition. The fact that this variable was strongly associated with all outcomes (except neck pain) indicates that it was an important predictor variable. Further research to investigate the impact of the type and severity of condition on baseline outcomes is warranted.

The only previously validated measure of injury severity used in this study was ISS. Several adaptations of the ISS have been proposed to increase its ability to predict outcome (usually mortality) in trauma patients; these include the New Injury Severity Score (NISS),[248] the Trauma and Injury Severity Score

(TRISS),[427] and the International Classification of Diseases Injury Severity Score (ICISS).[245 247] The ISS was chosen as it is easy to calculate (and therefore less prone to recording error), it is commonly used in follow-up studies after trauma, and it is routinely calculated in trauma patients at this institution. Also, although evidence exists that the NISS may be a better predictor of some outcomes in certain populations such as paediatric patients[428] or in patients with multiple orthopaedic injuries,[251] in a general trauma population, the NISS and the ISS have been shown to be similar.[250] Also, due to conflicting evidence regarding any superiority of the newer scoring systems, the ISS remains the current gold standard for scoring major trauma patients and is the most widely reported.

There is some evidence that functional recovery after injury may be sensitive to the body region injured[395 429] in that pelvic and extremity trauma may have a greater impact on physical function than injuries to other body regions. We did not test for any association with body region, choosing instead to use the overall ISS in the analysis.

The measurement of significant head injury was based on the scoring system used to measure ISS (the AIS). This measure, as a separate variable, has not previously been used. It was validated by testing it against memory of the event, for which it was strongly associated with poor memory, as expected. Further, removal of head injury from the final models (if present) did not significantly alter the effect or significance of the main predictor, compensation status.

Education level and household income were lower than national averages given by the Australian Bureau of Statistics,[404] indicating that trauma cases may involve patients with lower socio-economic status, or the fact that Liverpool Hospital is located in an area with relatively low household incomes.

Employment was only used as a dichotomous variable, which ignored the differences between part-time work and full time work. However, including part-time work as a separate variable would have made analysis difficult, and would not have taken into account whether or not patients worked part-time because of illness or because of choice.

This study defined the main exposure variable as whether or not compensation was sought, rather than whether or not the subjects were entitled to compensation. Pursuit of a claim was simpler to measure and was available on all subjects. Entitlement to compensation, however, can be considered a more valid way of determining the effect of being treated under a compensation system as it is out of the control of the subject and reflects the randomness of the accidents. For example, whether the subject was on their way to work when they lost control of the vehicle would influence whether or not they were entitled to compensation. Using pursuit of a claim as the exposure variable is less valid as the decision to pursue a claim can be influenced by subject factors such as socio-economic status, injury severity, and psychological factors.

The advantage of using accidental trauma is that it usually occurs in a somewhat random manner. This allows the application of our main exposure variable, compensation status, in an arbitrary (if not random) manner as it depends on circumstances largely out of control of the participants. This is a strength of this study (and the prospective study in Chapter Four) which limits bias due to self-selection for various diagnoses and treatments which is a potential problem in many of the studies included in the meta-analysis in Chapter Two, due, for example, to the subjective nature of the diagnoses. Use of acute trauma also reduces the effect of previous psychological changes from long term involvement in the medical, compensation or legal systems which may bias studies of chronic conditions such as chronic low back pain.

It should be noted that differences exist between compensated and non-compensated patients in this study. Compensated patients are covered for medical expenses through an insurance scheme that exists outside of Medicare, the public health insurance system. Consequently, patients treated within the compensation system are usually treated by specialists (rather than trainee doctors) and have ready access to premium health services such as private rehabilitation, private physiotherapy, other physical therapies (for example, massage therapy), clinical psychology, counselling, and prostheses. This difference, however, would be expected to bias the outcome in favour of compensated patients. Therefore, any decrease in the health outcomes seen in compensated patients would exist despite these potential advantages.

The differences between the effect of claim pursuit and claim entitlement were tested on a subgroup of subjects for which claim entitlement could be reliably measured. This analysis showed no significant difference between these two variables and, in some cases, an increased negative effect when claim entitlement was used. Also, the two variables were highly correlated. This supports the use of claim pursuit as the explanatory variable. The prospective study in Chapter Four will also measure compensation using both methods and provide further insight as to whether this difference is important.

#### 3.4.2.4 Statistical analysis

The statistical analysis is a strength of this study. The sample size was larger than that expected, and robust models could be created for each outcome variable. For each outcome, the final model fitted well and explained a high proportion of the variability in the outcome.

### 3.4.3 Interpretation of the results for each outcome

#### 3.4.3.1 Physical health

As expected, physical health improved with time since injury, and decreased with increasing injury severity and increasing number of chronic illnesses. Poor physical health was also independently associated with unemployment, although this association is likely to be due to the fact that patients with poorer health are less able to work, rather than unemployment causing poor health.

Unexpectedly, presence of significant head injury was associated with a higher mean PCS. This can be explained by the fact that patients were included in this study because their injuries reached a threshold ISS, and the ISS is made up of the AIS scores for each body region, including the head. Therefore, if a patient has a significant head injury (defined as an AIS of 3 or more for the head region), they do not require significant injuries to the other body regions (eg, pelvis and limbs) in order to satisfy the inclusion criteria. Similarly, patients without a significant head injury are more likely to have significant injuries to their limbs and pelvis, in order to satisfy the inclusion criteria. The effect on PCS is likely to be greater for those with limb and pelvic injuries, than those with head injuries, as it reflects physical functions such as walking and carrying, not cognitive ability. The association between poor physical outcome and injuries to the pelvis and limbs (rather than abdomen, chest and head) has been shown previously.[395 429]

The association with compensation, in which those who pursued compensation (and in a separate analysis, those who were entitled to compensation) had poorer physical health after adjusting for other factors, was significant regardless of whether or not the claim had settled. For those whose claims had not settled however, the association was stronger.

It is likely that the involvement with the compensation process led to the decrease in health status. The reverse argument, that those in poorer health were more likely to pursue compensation, is not supported by the fact that the association remained strong (and was, in fact, stronger) when entitlement to compensation was substituted for pursuit of compensation. Furthermore, the association with claiming compensation remained after allowing for predictors of poor health, such as age, comorbidities and injury severity.

The effect of compensation on physical health or general health after major trauma has only been reported in one previous study.[392] Other studies have examined the effect of general factors such as age, sex and injury severity, and some have included socio-economic predictors. None of the previous studies of general health after major trauma[397-399 430] have used the SF-36 as an outcome tool, though several have used other health-related quality of life measures. Although one study[431] used the SF-36 as a measure of outcome after major trauma, this study was of a small group (n = 76) of patients presenting with an ISS of 50 or greater (extreme injury) and did not provide a multivariate analysis to determine predictors of SF-36: it only commented that the SF-36 scores were below average. The previous studies of outcome after major trauma are summarised in Table 3.22. The results of the Major Trauma Outcome Study reported in this chapter are placed in the last row of this table to allow comparison. This study is comparable to the previous studies except for a higher mean age, this is because many of the other studies excluded elderly patients (65 years and over) and because we recorded age at follow-up, not age at injury.

Unfortunately, only one of the other studies measured compensation status, so a comparison of the effect of this variable is limited. This paper, by Glancy et al,[392] showed a negative association between involvement in litigation and

Table 3.22. Summary of previous studies of outcome after major trauma.

See Appendix 6.

References:

MacKenzie et al, 1988[395]
Glancy et al, 1992[392]
Ott et al, 1996[432]
Vazquez et al, 1996[396]
Anke et al, 1997[394]
Braithwaite et al, 1998[429]
Holbrook et al, 1998[430]
Dimopoulou et al, 2004[397]
Meerding et al, 2004[399]
Vles et al, 2005[398]

return to function, which concurs with this study. The study by Glancy et al uses involvement with litigation as the predictor variable, and there may be a difference between this variable and pursuit of compensation (which may not involve litigation). Involvement with litigation may be a closer measure of to use of a lawyer, rather than pursuit of compensation.

Unfortunately, the study by Glancy et al only tested each variable with age and ISS as potential confounders in the multivariate analysis, rather than including all significant variables. Also, the main (and only) outcome variable was "return to function" which was poorly defined as "resumption of a usual level of activity, most often full-time employment …". The problems inherent in using return to work are discussed elsewhere; the problem is exacerbated when using compensation as a predictor, as the factors influencing return to work are entirely different in compensated versus non-compensated patients, as compensated patients may have less financial incentive to return, may wish to avoid returning to the place of injury, and may find it harder to be cleared to return to work because of the complex bureaucratic process involved in returning injured workers to their workplace. The Glancy study can also be criticised for incomplete follow-up (41.9% at 6 months).

The effect of compensation has been more widely tested against other outcome variables, such as pain or psychiatric outcome (see below).

The association between use of a lawyer and poor physical health has previously been reported, but not specifically after major trauma. The use of a lawyer, though, is the result of a conscious decision by the subject, and this decision may be influenced by many factors (such as psychological) not represented in this analysis. It is a less objective explanatory variable than claim entitlement.

Similarly, claim settlement may, to some extent, be under the control of the subject, as subjects who feel that they have suffered more severe injuries may be less likely to settle early. The strength of this association, allowing for injury severity, however, would indicate that it is the patient's perception of their health (rather than the injury severity), which may be worse in patients who prolong settlement. This, however, supports the hypothesis that prolonged exposure to the claim process may increase the reporting of symptoms (and therefore cause poor health).

The associations with use of a lawyer and with compensation claim may be partly explained by symptom exaggeration. Conscious symptom exaggeration is possible, but this would be expected to be minimised by the study design, which involved reassurance that the information would remain anonymous. Symptom exaggeration may be part of the effect of compensation, as involvement with the compensation system can be associated with increased medical involvement.

This is supported by the stronger association with an unsettled claim. In fact, apart from PCS, the other outcome variables were not associated with pursuit of a claim, per se, but with having an unsettled claim. The persistent and current exposure to the compensation and legal system in which patients are expected to repeatedly give accounts of their symptoms may be the mechanism by which reporting of symptoms is increased. To say that the effect on health is due to increased reporting of symptoms, however, is a circular argument. Health, in particular any patient-based measure of health, is based on symptom reporting. In other words, patients who report an increased number or severity of symptoms are, by definition, sicker.

That patients may be (consciously or subconsciously) over-reporting symptoms because of psychosocial aspects of the claims and legal process provides an explanation for the test of biological plausibility. There is no biologically plausible reason why patients who are eligible to claim should have more physical disability (allowing for injury severity), but it is biologically plausible that they may be over-reporting these symptoms.

It is interesting to explore why the associations between the outcomes and use of a lawyer were much stronger than the simple association with claim pursuit.

Use of a lawyer is more often used as a variable than compensation status and has previously been associated with poor outcome after trauma.[61 68 117 305 307 407] The association with use of a lawyer, however, may represent several possible factors. Those who use a lawyer may be more likely to be seeking secondary gain, either financial compensation (hence the confounding with compensation), or retribution against those who caused the injury (hence the confounding with blame).

It is also possible that use of a lawyer may directly influence the outcome in a negative manner. As mentioned previously, involvement with the legal system may mean a long exposure to an adversarial system. Lawyers may also directly influence their patients' condition by suggestion and by coaching.[219 308]

The effect of use of a lawyer and of claim settlement is discussed further in the next sections.

### 3.4.3.2 Mental health

Mental health was poorer in those with chronic illnesses and in the unemployed. These associations were present for most of the outcomes and are not unexpected. The association between MCS and age, however, was unexpected, as mental health improved with increasing age. The published population norms from Australia,[433] however, also show that mean MCS scores increase with age.

Unlike for PCS, the main study hypothesis is not supported by the results for MCS. Although pursuit of (or entitlement to) compensation was associated with lower MCS scores, the difference was not statistically significant. The association between MCS and claim settlement, however, was larger and more highly significant than for PCS.

It appears then, that pursuit of compensation only has a negative effect on MCS while the claim remains unsettled. This is plausible, as the process of claim settlement carries with it uncertainty, conflict and usually financial burden, rather than reward.

As with physical health, mental health has previously been measured in studies of major trauma patients but with other outcome tools. These studies are shown in Table 3.22 and are discussed above under physical health.

#### 3.4.3.3 Neck pain

Neck pain was associated with female gender, unemployment, lower education, presence of chronic illnesses, presence of a neck fracture, absence of head injury, and use of a lawyer. The association between significant head injury and lower neck pain scores may be spurious, as discussed above. The association with unemployment is similar to that found for the other outcome variables and is expected.

Claiming compensation was strongly associated with neck pain in the univariate analysis, but was not significant in the multivariate analysis, due to the stronger effect of use of a lawyer. Therefore, the hypothesis that neck pain will be stronger in those who pursue compensation is rejected. Further, there is no evidence that neck pain is related to claim settlement, which was a strong predictor for many of the other outcome variables.

Neck pain has been extensively studied after motor vehicle trauma, as discussed in Chapter One, but it has not previously been studied after major trauma. Comparisons with the literature pertaining to motor vehicle trauma are reasonable, as motor vehicle trauma made up the majority of cases in this study (63%).

Females have previously been shown to be more likely to develop neck pain after motor vehicle injuries but the mechanism for this is not clear.[102 117 134 158]

The association between lower education level and higher neck pain score is supported by previous studies which have shown a similar association in patients with whiplash injuries[434-436] and in patients presenting to a spine clinic complaining of neck pain.[437]

The lower neck pain scores in patients with higher education levels may be due to better coping skills in this group, or to lower physical demands in the workplace. While it is possible that there may be less financial burden (and therefore less stress) on those with higher education levels, this is not supported by the lack of an association between income level and neck pain. A post hoc analysis was performed to explore possible confounding between education level and income, but income remained a non-significant predictor of neck pain (overall, and for each individual level) when education was removed from the model (p = 0.40 compared to a p value of 0.54 with education in the model). Therefore, the effect of education level is unlikely to be mediated through differences in income.

Ferrari and Russell[110] noted that whiplash was strongly correlated with blame and postulated a biopsychosocial model to explain this association. Blame has also been implicated in chronic neck pain after motor vehicle accidents by others[96 134] and it has been implicated in the development of PTSD (see below). In this analysis, blaming others was associated with increased neck pain, but the association was not strong in the univariate analysis, and was not significant in the multivariate analysis. Radonov, in a study of 117 whiplash patients also found an association with blame but, as with this study, the association was not significant in multivariate analysis.[155]

Even though neck pain is common in the community, and it may be argued that some of the associations (such as sex and education) may be independent of the effect of any previous trauma, the strong association with neck pain and the use of a lawyer relating to the injury would suggest that the index trauma was a significant factor in the development of neck pain. In fact, use of a lawyer was the strongest predictor of neck pain in this study, despite the fact that most of the patients did not have any significant neck trauma (as defined by the presence of a neck fracture).

Although there is supportive evidence of the association between legal involvement and neck pain after injury,[117 306 407] poor methodology in previous studies of neck pain after motor vehicle injury limits the validity of any conclusions regarding associations between neck pain compensation-related factors, as discussed in Chapter One. Apart from differing definitions of the explanatory variables (described variably as involvement in litigation, claiming compensation, having an unsettled claim, or use of a lawyer) and the outcome variable (severity of neck pain, frequency of neck pain, return to work, treatment time, timing of case closure), many of these studies represent retrospective case series. Consequently, reviews of the literature pertaining to neck pain after injury are not conclusive of any association with compensation or with the use of a lawyer.[86 115 116]

The strong association between neck pain and use of a lawyer in this study is in contrast to the lack of association between neck pain and pursuit or settlement of a claim. If the effect of lawyer presence is not mediated through exposure to the claim process and the stress related to having an unsettled claim, then there may be a more direct effect, perhaps by lawyers suggesting the presence of neck pain and its attribution to the injury, thereby raising the expectation of symptoms. This may also heighten patients' awareness of pre-existing neck pain.

It is not possible to make firm conclusions regarding cause and effect in the association between use of a lawyer and neck pain, as the use of a lawyer is the result of a conscious decision by the patient. Therefore, it is possible that patients who developed neck pain may have been more likely to consult a lawyer regarding their case.

The post hoc multivariate analysis of neck pain, which included PTSD as an explanatory variable increased the adjusted $R^2$ from 19.3% to 28.7%, with little change to the other explanatory variables. The strong association with PTSD, independent of other variables, indicates that there may be significant input to the expression of neck pain from psychological factors. This supports the theory that neck pain is due to somatisation: the expression of psychological stress as a physical symptom, usually one that is more socially acceptable,[94] and one that may be expected from such an injury.[96 98 101 111]

#### 3.4.3.4 Back pain

As with neck pain, back pain was associated with chronic illnesses, current unemployment, lower education levels and the use of a lawyer, and not with pursuit of a claim, allowing for these other factors.

As with neck pain, the association between lower education levels and increased back pain has been reported previously in clinic populations,[279] patients with work-related back pain[291 438] and in general populations.[263] Unlike the association with neck pain, the association between education and back pain may be due to differences in income between the education levels. A post hoc analysis showed income to be significantly associated with back pain when education was removed from the model ($p = 0.03$ compared to $p = 0.18$ with education in the model). This provides support for a financial incentive in complaints of back pain. It may also indicate differences in work-related back pain between patients with low education and income, compared to those with high education and income levels.

Most of the literature regarding outcome in patients with trauma related back pain is restricted to work-related back pain. The differences between those studies and the Major Trauma Outcome Study is that work-related back pain may not necessarily be secondary to significant physical trauma, or the trauma may be minor, and also that this study includes patients treated under third party compensation, as well as workers compensation. These issues should be considered when making comparisons.

Unemployment and use of a lawyer may lead to dissatisfaction and psychological stress, which may present as back pain. This is supported by studies showing an association between psychological stress and work-related back pain[67 69 77 292] and back pain in the community.[70 439]

That stress may present as back pain is also supported by the post hoc analysis that included PTSD as an explanatory variable. This analysis increased the adjusted $R^2$ from 15.9% to 25.1% and showed a strong independent association between symptoms of back pain and PTSD at follow-up.

The methodological issues relating to studies of the relationship between psychosocial work characteristics and back pain have been addressed in a review by Davis and Heaney.[78] While they warn against drawing strong causal inferences from the literature due to methodological concerns, they conclude that some psychological factors (particularly job stress and job satisfaction) are consistently related to low back outcomes.

The link between pursuit of compensation and stress has been discussed in previous chapters, and has also been reported specifically in patients complaining of back pain.[67 71 84 380 440]

#### 3.4.3.5 Patient satisfaction

Patient satisfaction with progress has not been previously measured in trauma patients.

It is interesting to note the similarities between the final models for satisfaction and MCS: each outcome was associated with claim, past illness and current employment. For both outcome variables, the association with claim was only significant for an unsettled claim, and this association was very strong for both outcomes. Patient satisfaction may be a measure of mental well-being, as a post hoc analysis showed MCS to be highly correlated with satisfaction. The mean MCS for the satisfied and unsatisfied groups was 47.6 (95%CI 46.0 – 49.1) and 32.2 (95%CI 29.9 – 34.7) respectively ($t = 10.54$, 344 DF, $p < 0.0001$).

It is also interesting to note that use of a lawyer and pursuit of a claim (regardless of settlement) were not associated with patient satisfaction or MCS. This may be because pursuing a claim (and retaining a lawyer) may be seen as a means to achieve (some form of) secondary gain, and are therefore not seen in a negative light. An unsettled claim may represent the failure to achieve the secondary gain (just as settlement represents achievement), which explains the strong negative effect of an unsettled claim.

The hypothesis that patient satisfaction is negatively influenced by pursuit (or entitlement to claim is not supported by the findings of this study.

#### 3.4.3.6 Post-traumatic stress disorder

Previous studies of predictive factors for PTSD post MVA have failed to show any consistent factor as a predictor,[188] which may reflect methodological problems common in such studies.[195 426]

Although other studies have shown that patients pursuing compensation report increased severity and frequency of psychological symptoms,[185 190 441] only a few studies have looked at the relationship between compensation and PTSD.

One of the earliest and most frequently cited articles on the association between psychological stress and compensation is the Milroy lecture for 1961 delivered to Royal College of Physicians by Henry Miller.[236] Reporting on a consecutive series of 200 head-injured patients, Miller noted that there was an inverse relationship of accident neurosis and injury severity, and that accident neurosis was related to social status. In a follow-up of 50 patients after settlement of their claim, Miller noted that psychological symptoms of stress had resolved in all but two patients. Unfortunately, the methodology in Miller's series lacked information regarding the selection criteria or the diagnostic criteria for "accident neurosis", and there was no statistical analysis to support the observations.

Miller's findings supported previous observations, which may be summarised by a paper by Kennedy from 1946 which attributes compensation neurosis to psychological factors, greed, and the involvement of lawyers and the legal system.[20] Again, though, the diagnostic criteria for "compensation neurosis" were not clear, nor was the relationship between compensation neurosis and PTSD, which was not accepted as a diagnosis until many years later.

In one way, however, the findings of the present study reinforce the opinion of Miller and Kennedy, in that claim settlement was strongly (negatively) associated with PTSD. The opinions of Miller and Kennedy have come under considerable criticism since their publication. One of the most published critics of the concept of being "cured by a verdict" is George Mendelson, a psychiatrist from Australia. Mendelson has reviewed the literature regarding what he calls "the compensation effect" pertaining to the field of pain and has concluded that there is little evidence that pain reporting, pain behaviour and psychological illness are increased in compensated patients. He also concludes, however, that compensated patients are less responsive to treatment and that the prevalence of pain was higher in compensation systems that provided pain contingent benefits.[4 48 49] Mendelson's comments, however, are largely restricted to chronic pain, not general health or any specific condition such as PTSD.

The role of compensation in conditions similar to PTSD, such as railway spine, battle fatigue and traumatic neurosis has been discussed in Chapter One. PTSD however, is a well defined (if evolving) diagnosis, which should allow more accurate assessment of possible predicting factors, by minimising measurement bias which may be present in less well-defined conditions. Of the more recent studies which use modern epidemiological methods to test for predictors of PTSD, only a few have included compensation status as a possible predictor.

A study by Bryant and Harvey,[194] from a major trauma centre in a similar socio-economic area of the same city as this study, followed 62% of 171 consecutive motor vehicle accident patients for 2 years, to test the association between compensation and PTSD. They divided their patients into three groups, those who had not initiated a claim, those whose claim had settled, and those whose claim was still pending: identical grouping to this study. They showed that the groups were similar with respect to age, injury severity score and hospitalisation rate, and commented that claim settlement had no influence on the rate of PTSD, as the number of patients satisfying the criteria for PTSD (30%) was the same in the settled and not settled groups.

Interestingly, the rate of PTSD in the group that had not initiated a claim was 0% indicating that pursuit of a claim may be related to the development of PTSD. They hypothesize that the group that had not initiated a claim may have been less severely injured, but the injury severity score was higher in this group, although the difference did not reach statistical significance. The numbers in this study were low (only 13 patients in the non-compensated group, and 93 in the compensated groups) and the study may have been underpowered to detect a difference in the settled and non-settled groups. The authors offer another explanation for the absence of PTSD in the non-compensated group by stating that the process of litigation may have contributed to PTSD.

Blanchard et al,[284] in a U.S. study of 158 adults 1 – 4 months post motor vehicle accident, used similar methods as this study to look for predictors of PTSD, using multiple regression for PTSD as measured by a scale (continuous) and logistic regression for PTSD as a dichotomous variable. They allowed for age, sex, education level, injury severity and employment status, as well as prior psychiatric status, and found that involvement with litigation was the strongest predictor of the development of PTSD. Their definition of litigation, though, was whether or not the subject had contacted a lawyer. Therefore, although the findings concur with this study, they were unable to distinguish any effect of the litigation process from the effect of lawyer involvement.

In a later analysis of the same cohort, Blanchard et al[190] divided patients into non-litigants, litigants with settled claims, and litigants whose claims had not settled: the same groups that were used in this study. At 12 months they reported similar findings to this study, with significantly higher PTSD scores in those whose claims had not settled, and higher (but not statistically significant) PTSD scores in those whose claims had settled, using the non-litigant group as a control. The effect sizes were comparable, with odds ratios for PTSD in the unsettled and settled groups of 2.31 and 2.09, respectively.

In a study from the same institution as the Major Trauma Outcome Study, Silove et al[188] contacted 81% of 102 patients 18 months after admission to hospital after a motor vehicle accident and found that psychiatric morbidity was not predicted by intent to pursue compensation. This study, however, was designed to test the predictive power of screening tools given soon after the injury, and no mention is made of how compensation status was measured, and no statistical analysis of the "intent to seek compensation" is given. Further, information on PTSD was only available for 66 of the original 102 patients, indicating that the study may have been underpowered.

R. A. Mayou of Oxford has published many papers relating to two separate consecutive cohorts of patients who presented to the emergency department after a motor vehicle accident. The earlier, smaller study did not examine compensation as a predictor variable, a priori, but a 5 year follow-up looked at this factor when examining predictors of PTSD.[128] Completed questionnaires were obtained from 111 of the original 188 subjects and the incidence of

PTSD in claimants who had settled was found to be 6%, compared to 40% in claimants who had not settled, equating to an odds ratio of 10.89. However, a statistical analysis was not provided for this association in the paper.

In the later, larger study, 82% of 1,441 consecutive patients were recruited and were followed prospectively at regular intervals, including at the time of presentation when data relating to pre-accident psychological and social variables, and injury severity factors were measured.[130] In contrast to the Major Trauma Outcome Study, patients mainly presented after minor injuries, as only 26% of patients required admission[191] and most patients had an ISS less than 4.[130]

Regarding predictors of PTSD, the results of this larger study were reported at one year[191] and three years.[192] The authors found that pursuit of compensation and having an unsettled claim were both strong predictors of PTSD at one and three years.

The lack of association between pursuit of compensation and PTSD (rather than having an unsettled claim) in the Major Trauma Outcome Study compared to the other studies reported above may be due to the use of claim pursuit and use of a lawyer as separate variables in this study. There was confounding between these two variables for all outcomes, such that each took some of the effect of the other: the studies reported above used either of these variables but not both. Without use of a lawyer in the final model for this study, the significance of the claim variables increased significantly, such that having a settled claim became strongly associated with the PTSD score ($p = 0.006$).

A high correlation was also found between ongoing financial difficulties and PTSD, which may explain some of the association between employment status and PTSD reported in this study. Interpretation of the association with employment is difficult, as it is highly likely that subjects with poorer health (on any scale) are less likely to want to work or to be able to work.

These studies all use motor vehicle trauma as the index injury, regardless of severity, which may be minor, because in some studies the majority of patients did not require admission to hospital. There are no comparative studies which examine PTSD in major trauma patients. This may be considered a strength of this study as it excludes minor injuries which, according to the definition, would be less likely to cause PTSD and may dilute any possible associations.

Comparisons between this study and those studies restricted to motor vehicle trauma should also be made with caution, as a separate analysis of this study restricting the analysis to motor vehicle trauma showed that while all other explanatory variables retained their strength and significance, claim (with or without settlement) has a non-significant and decreased association with the PTSD score (the p value for the overall association is 0.21, compared to 0.012 in the full model). The lack of significance, however, may reflect the lower numbers used in this secondary analysis.

This study did not include many of the psychological variables included in other studies. Notably, it did not account for past exposure to traumatic events and pre-existing post-traumatic reactions. It assumes that any PTSD present is a result of the index trauma, or that if it is not, that these other factors are likely to be evenly distributed between the main exposure groups. The assumption that the PTSD is related to the index injury is supported by the statistical analysis which shows that factors related to the index injury, and the patients circumstances relating to that injury, explains a large proportion (40.7%) of the variation seen in PTSD related symptoms.

Although this study did not account for pre-injury psychological status, measurement of this retrospectively after an injury is likely to be subject to recall bias. Also, it is hoped that the random nature of the accidents and compensation eligibility would minimise any confounding from this variable.

Use of a lawyer was strongly associated with the reporting of PTSD symptoms in this study, and this association was independent of compensation claim status. Previous studies, discussed above, have used pursuit of compensation or litigation as predictor variables. There are no studies that examine the role of retaining the services of a lawyer in the development of PTSD.

Use of a lawyer, however, is a conscious decision by the subject and may be influenced by unknown confounding factors related to the outcome. Having said that, there is some evidence that lawyers may reinforce symptoms and coach subjects regarding expressing the appropriate symptoms to advantage

their case.[219] Also, use of a lawyer may be related to more prolonged and adversarial legal processes, adding to the level of psychological stress.
As with the other outcome variables, however, it is also possible that patients who developed PTSD were more likely to consult a lawyer. Therefore, caution must be taken when making conclusions regarding cause and effect for this variable.

The protective effect of blaming oneself on the reporting of PTSD symptoms found in this study is supported in the literature. Delahunty[296] showed that participants who felt that they were responsible for the injury were less likely to have persistent PTSD after a car accident, compared to those who blamed others for the accident. In a similar study of motor vehicle accident patients with PTSD, Hickling et al[297] found that patients who blamed themselves were less symptomatic and recovered more rapidly than patients who blamed another party. Similarly, in Mayou's series of motor vehicle accident patients from Oxford, "feeling not to blame" was a significant predictor of psychological symptoms up to 3 years after minor motor vehicle trauma.[130] It has been suggested that blaming oneself for an accident is associated with better coping skills.[298]

In contrast, Silove et al, in a follow-up study of patients admitted to the same institution as this study after motor vehicle trauma, found that "feeling personally responsible for the accident" did not predict psychiatric morbidity at 18 months.[188] This study, however, did not provide information regarding the strength or significance of any association and may have been underpowered to detect a significant difference, as only 66 of 102 patients completed the final review.

Although many authors report an increase in PTSD in females [130 191 195 426] Ursano et al[442] followed a cohort of motor vehicle accident victims and found that while acute post-traumatic stress disorder was 8 times more likely in women, there was no difference in chronic PTSD (more than six months). Similarly, Dougall et al l[443] noted that female gender only predicted PTSD within the first 6 months, but not at 12 months post MVA. These studies support the findings of this study, which showed no association between PTSD and gender.

Blanchard et al[190 193 252] showed that AIS (the score used to calculate ISS) predicted PTSD in MVA victims, and Mayou et al and Ehlers et al,[191 192] in their follow-up studies of patients presenting to an emergency department for treatment after a motor vehicle accident also found that PTSD was related to injury severity, however their measurement tool for injury severity (no injury, soft tissue injury, or bone injury) had not been previously used or validated. Other researchers have not found an association between ISS (or stressor severity) and PTSD after motor vehicle trauma[188 443-447] and Delahunty et al[254] noted lower ISS scores in MVA victims who developed PTSD compared to those who did not. In a study from NSW, rates of psychiatric injury after motor vehicle trauma were not associated with increasing ISS, except for the severely injured (highest decile of ISS).[253] There is evidence that while objective measures of injury severity are not associated with the development of PTSD, subjective measures (patient perception) of the seriousness of the injury (the Impact of Event Scale) is strongly correlated with the development of PTSD after physical trauma.[447]

In longitudinal studies, the prevalence of PTSD usually diminishes over time.[426] This study did not show a correlation between time since injury and the reporting of PTSD symptoms. This difference may be because this study is not truly longitudinal (following the same patients over time), or because the study was underpowered, as previous studies have only shown a small effect over time.

In two reviews of PTSD after trauma, the incidence of PTSD after trauma can range from 0 to 100% according to Blaszczynski et al,[195] and from 2 to 42% according to O'Donnell et al,[426] both of whom attributed the wide variation to methodological differences between studies. Although this study provided an alternative analysis (measuring the rate of PTSD), the main analysis used the PTSD scale as a continuous variable to determine predictors of PTSD symptoms; determining the rate of PTSD was not the primary purpose of the study.

In a similar study to this one, Chan et al[189] measured PTSD using the same tool (PTSD Checklist-Civilian version) in an Australian population by a mailed questionnaire nine months after motor vehicle trauma. They showed a 29% incidence of PTSD using a cut-off score of 50 as the criteria for diagnosis. Although the Major Trauma Outcome Study has a longer follow-up, if the data is re-analysed using a cut-off score of 50 for PTSD the incidence (28.2%) is similar. The study by Chan et al had a poor response rate of 13% (391/3088), but their statistical analysis showed similar findings, with no significant association found with employment at the time of injury, time since injury, admission to hospital, or education level. They found an association with use of medications (an alternative measure for chronic illness) which also concurs with this study.

Unfortunately, Chan et al did not measure the effects of claim factors, as all of their patients were treated under third party insurance and no claims had settled. In that case, the rate of PTSD would be expected to be higher in this group. The lower than expected rate of PTSD in the study by Chan et al (given the claim and settlement status) may be due to the inclusion of minor motor vehicle accidents in that study.

### 3.4.4 Generalisability

The education level of participants in this study was lower than expected. Using data from the Australian Bureau of Statistics, based on Australians aged between 15 and 64 in 2001, 14.3% held a bachelor degree or higher, and 34.1% held a certificate or diploma. Although the sample used for this study was older, the corresponding percentages were considerably lower (8.6% and 28.7%, respectively).

The employment level was also lower than expected, with 30.4% of participants being unemployed at the time of injury. This figure is lower if it is restricted to those aged between 18 and 55 (14.5%), but remains above the national average (approximately 5%).

The high proportion of males (72.1%) was also higher than expected from the normal population. Despite the differences between the study population and the normal population, the study population is representative of patients involved in major trauma. The gender ratio, age and injury severity scores are similar to other studies of similar populations (Table 3.22) and these factors are also similar to the proportions seen in the source population, major trauma patients from this hospital, as discussed in section 3.4.2, Comments on Methods.

The trauma population used in this study is not representative of the general population. This may affect the generalisability of the study beyond a major trauma population and the findings may not be applicable to less severe injuries. It may be reasonable, however, to compare the findings of this study to studies restricted to patients injured after motor vehicle trauma, as the mechanism of injury did not have a significant effect on the health outcomes used in this study, only with patient satisfaction.

Generalisation beyond the geographical boundary of the state of New South Wales should also be done with caution because of the different compensation rules that exist in different political regions. However, no significant difference was seen between the two compensation systems used, despite significant differences in their structure.

Generalisability is also restricted by the number of factors measured. Therefore, extrapolation to population groups defined by variables not measured in this study, such as marital status, is not possible.

### 3.4.5 Implications of the results

The association between compensation (either pursuit of compensation at any time or current involvement in the claim process) and aspects of health reported in this study are consistent with previous findings.

The association between involvement in compensation and legal processes, and poor outcomes is strong and implies that the systems used to process claimants may be iatrogenic. Use of lawyers, the adversarial nature of the process, reliance on subjective symptoms for diagnoses, and the bureaucratic complexity are all aspects that must be considered as contributing to this iatrogenic process.

### 3.4.6 Significance to future research

This study shows a strong association between claim-related variables and outcome after major trauma. It has only limited support from the current literature and therefore needs to be supported by further research.

Future research into the association between compensation claim and health after injury should clearly define the injury, aim to control for possible confounders as much as possible, and clearly define the main exposure variable. It may be better to restrict the study population to a particular, clearly defined diagnosis, rather than using ISS, which includes patients with many different injuries.

Ideally, patients would be randomised to receive compensation or not but this would have obvious difficulties. In place of this, similar research could be done where compensated and uncompensated patients exist with the same injury, as with this study. Also, it would be better to follow patients prospectively, as this would give a less biased measure of time-dependent variables such as time to case settlement.

Lawyer involvement would be difficult to randomise, as it is currently subject to the patient's will. It may be possible in future work to compare different systems of compensation that allow different levels of legal involvement. Similarly, it would be helpful to compare systems that differ in case-settlement times. This research has been done previously in before-and-after studies,[21 90 117 124 448] but interpretation of this research has been hampered by the fact that multiple changes to the system were made at the same time, making attribution to any one factor speculative. Comparative studies where small changes have been made to a particular system (for example, rights of access to a lawyer, financial rewards, early settlement) would be more helpful.

It is also clear that psychological factors influence health outcomes. Comprehensive consideration of these factors is necessary to build better predictive models for outcome after injury.

### 3.4.7 Significance of work

Trauma is a major health problem, particularly in people of working age, and this study improves the understanding of predictors of health in patients who have been subjected to major trauma. This study provides information useful to administrators of public health policy, insurance providers, lawyers, and others involved in the compensation industry. It also provides information to guide future research in the same area.

This study may be used to improve patient outcomes after trauma by changing the processes involved for claimants within compensation systems. However, the effect of any changes to a system (such as earlier resolution, less legal involvement, or removing compensation for specific injuries) would need to be closely monitored as part of ongoing research in this area.

### 3.5 Conclusion

Involvement in a compensation claim strongly predicts poor health outcomes, including general health, one to six years after major physical injury. This association is independent of other predictors, and independent of whether or not the services of a lawyer were used. The harmful effect of claiming compensation was stronger if the claim had not settled, particularly for mental health outcomes.

The use of a lawyer also predicted poor outcome for several outcome measures, but the type of compensation system used (third party or workers compensation), the time to settlement and the time since settlement were not significantly associated with any of the outcome measures.

The study hypothesis, that general health outcomes would be poorer in patients pursuing compensation, is accepted.

This study indicates that aspects of the compensation process may be harmful to the health of patients participating in the process.

# CHAPTER FOUR: THE ASSOCIATION BETWEEN COMPENSATION AND OUTCOME AFTER MOTOR VEHICLE INJURIES, A PROSPECTIVE STUDY (MOTOR VEHICLE ACCIDENT OUTCOME STUDY).

## 4.1 Introduction

This chapter explores the role of compensation-related factors in determining outcomes for a specific group of patients, namely those sustaining major fractures as the result of a motor vehicle accident. This study also differs from the Major Trauma Outcome Study reported in Chapter Three by being prospective, as patients were recruited at the time of presentation after their injury and then followed for six months.

The significance of motor vehicle trauma to public health has been studied extensively. Currently, road traffic injuries are the leading cause of death in high-income countries for people aged between 5 and 44 years. For the same age group, road traffic injuries are second only to HIV/AIDS as the leading cause of death worldwide.[449 450] In high-income countries, road trauma lies third, behind depression and alcohol-related disorders among the leading contributors to the burden of disease, and worldwide it lies third behind depression and HIV/AIDS.[449 450]

General health after motor vehicle trauma has been studied previously, but the effect of compensation on this outcome has not been reported. However, the role of compensation in the impact of illness after motor vehicle trauma has been studied for outcomes other than general health, particularly whiplash and psychological illness. The literature regarding this is discussed in the literature review in Chapter One, and in the discussion in Chapter Three.

The diagnosis of illnesses such as whiplash, depression and psychological stress are patient-based, and can therefore be influenced by psychosocial variables (such as compensation-related factors). By using a more objective diagnosis (specific fractures), it is hoped that biases related to the diagnosis being studied can be minimised.

The role of compensation-related factors in healing after fractures has been reported in only a few studies, none of which were specifically related to motor vehicle trauma. Five studies have been found, all of which were included in the systematic review in Chapter Two. In one study of outcome after surgery for a thoraco-lumbar burst fracture in 28 patients, 75% of whom were involved in motor vehicle trauma, compensation status was found to be the only significant predictor of outcome after two years.[364] In one study of outcome after fixation of osteochondral fractures of the knee in 123 patients, compensation status had a significantly negative effect on outcome, along with age and injury related factors.[451] In each of three separate studies of outcome after fracture of the calcaneus, compensation status was significantly associated with poor outcome, along with other anatomic factors.[350 452 453] The findings of these studies concur with the findings of other studies in the systematic review.

The outcome scores used in these studies all take into consideration patient-reported pain, and it is likely that the patients' symptoms contributed most to the poor outcomes seen in compensated patients, as there was no difference in objective outcomes such as bone healing or fracture reduction. This would indicate the outcome rating provided by the patients is influenced by psychological factors.

As mentioned previously, most studies that explore the effect of compensation status use only one criterion to classify compensation status. Although pursuit of a claim is a reasonable measure, some studies use litigation or use of a lawyer as a surrogate for compensation. This makes comparison of studies difficult, and this problem is addressed in this study which, as in Chapter Three, uses several compensation-related variables to measure compensation status. It is also hoped that this will allow better discrimination of the relative contribution to any association from the different aspects of the compensation process.

### 4.1.1 Study hypotheses

It is hypothesised that subjective health outcomes (physical health, mental health, back pain and neck pain) and patient satisfaction in patients with specific fractures from motor vehicle collisions will be associated with compensation status, allowing for demographic, socio-economic and injury factors, such that compensated patients will have poorer health outcomes and less satisfaction. It is also hypothesised that there will be no association between objective outcome (fracture non-union) and compensation status.

The specific hypotheses are similar to the hypotheses stated in Chapter Three and are as follows:

1. Hypothesis 1: General physical health, as measured by the physical component summary (PCS) of the SF-36 General Health Survey, will be significantly worse for patients who pursued compensation than for patients who did not pursue compensation.
2. Hypothesis 2: General mental health, as measured by the mental component summary (MCS) of the SF-36 General Health Survey, will be significantly worse for patients who pursued compensation than for patients who did not pursue compensation.

These are the same major hypotheses as the retrospective study reported in Chapter Three. The following secondary hypotheses test the main exposure variable (pursuit of compensation) against the secondary outcome measures.

3. Hypothesis 3: Neck pain will be significantly worse for patients pursuing compensation.
4. Hypothesis 4: Back pain will be significantly worse for patients pursuing compensation.
5. Hypothesis 5: Patient-rated satisfaction and recovery will be significantly lower for patients pursuing compensation.

In addition to these patient-reported outcomes, the effect of compensation status on the following outcomes will also be explored, and compared to any effect on the above patient-based outcomes.

6. Hypothesis 6: Surgeon-rated satisfaction and recovery will not be significantly associated with compensation status.
7. Hypothesis 7: Fracture-related complications will not be significantly associated with compensation status.

Comparisons between the findings of each chapter will be discussed in the final chapter, Chapter Five.

## 4.2 Materials and Methods

### 4.2.1 Study population

The study population consisted of adult (18 years and over) patients presenting acutely to one of the participating hospitals with specific fractures resulting from motor vehicle collisions. The specific fractures were fractures of the long bones (humerus, radius, ulna, femur or tibia), the pelvis, the patella, the talus, and the calcaneus.

Initially, only motor vehicle occupants with long bone fractures were included as these criteria were simple to apply, considering recruitment was being performed by many orthopaedic registrars, most of whom were not directly connected with the study. The inclusion criteria were expanded during the study to include other fractures (patella, pelvis, talus and calcaneus) and other mechanisms (motor cyclists, and pedestrians and bicyclists struck by motor vehicles), in order to increase the recruitment rate.

Fractures were chosen as the inclusion criteria for several reasons: they are objective diagnoses (not patient-based), the diagnosis can be made reliably, they set a threshold of physical trauma (to exclude patients with no objective signs of physical trauma), and because patients with these particular fractures are usually admitted to hospital (usually to undergo surgery) thus minimising bias due to selective presentation to hospital.

Patients referred for late treatment, such as reconstructive surgery, were excluded. Recruitment was required within 7 days of the injury. Patients with a cognitive inability to consent were excluded. Patients who died or moved overseas before follow-up were excluded. Attempts were made to use a health care interpreter for patients who did not speak English; the questionnaire was not translated. No upper age limit was used.

### 4.2.2 Study location

Fifteen public hospitals were involved in recruitment, namely, Liverpool, Bankstown, Westmead, Nepean, Blacktown, Hornsby, Gosford, Royal North Shore, Royal Prince Alfred, St Vincent's, Prince of Wales, St George, Sutherland, Wollongong, and Canberra. All hospitals were involved in the treatment of patients after motor vehicle trauma, and all hospitals were within approximately two hours drive of the principal institution, Liverpool Hospital. All major trauma centres in Sydney were included.

### 4.2.3 Ethics approval

Human research ethics committee approval was granted from the University of Sydney (supervising institution), South West Sydney Area Health Service (Liverpool and Bankstown Hospital), Western Area Health Service (Nepean Hospital), ACT Health (Canberra Hospital), Western Sydney Area Health Service (Westmead and Blacktown Hospitals), Northern Sydney Area Health Service (Royal North Shore and Hornsby Hospitals), Central Coast Area

Health Service (Gosford Hospital), St Vincent's Hospital, South East Sydney Area Health Service - Eastern Zone (Prince of Wales Hospital), South East Sydney Area Health Service - Southern Zone (St George and Sutherland Hospitals), and Illawarra Area Health Service and Wollongong University (Wollongong Hospital). Approval for the changes in protocol (to change the inclusion criteria to include different fractures and mechanisms) was also granted from each of the committees. Copies of the letters of approval from the ethics committees are in Appendix 11.

#### 4.2.4 Patient recruitment

Recruitment began on 2 August 2004 but was staggered, due to the irregularity of approval from local ethics committees. Recruitment terminated in October 2005, after recruitment of the target population of 300.

Patients were contacted on two occasions: at the time of recruitment and at the time of follow-up, six months later. At the time of recruitment, patients were approached by the orthopaedic registrar at their hospital and invited to participate in the study. They were provided with a patient information sheet and, if they agreed to participate, they were asked to sign a patient consent form and complete an initial questionnaire. At six months post injury, the final questionnaire was mailed to the patients with a cover letter and a reply-paid envelope. Data were also collected from patients' medical records, where necessary. Consent for this was included in the patient consent form.

The treating surgeons were also contacted at the time of follow-up (six months) and asked to complete a 6-month surgeon questionnaire. The patient information sheet, the cover letters, and the patient and surgeon questionnaires are provided in Appendices 12 – 15.

Several steps were taken to improve recruitment during the study. Two weeks prior to the commencement of recruitment, the registrars at each hospital who were participating in the on-call roster were contacted by telephone to inform them about the study and to get contact details (mobile telephone number) for routine contacting during the recruitment phase of the study. They were sent a packet of questionnaires, patient information sheets, patient consent forms and stamped pre-addressed envelopes for return of the questionnaires, along with a cover letter explaining their role.

Close to the time of commencing recruitment, a presentation was given to the orthopaedic departments at Nepean, Liverpool, Westmead, Royal North Shore, St George, Gosford, Canberra and Wollongong Hospitals, to familiarise the consultants and registrars with the project.

The registrars were telephoned regularly (approximately twice per week) throughout the recruitment phase so that close monitoring could be made of the progress of the study and to maintain active recruiting. Regular newsletters (approximately every three months) were circulated by mail and email to the registrars and consultants at each of the study hospitals to inform them of the progress of the study, of any changes to the inclusion criteria, and to maintain awareness of the study.

Questionnaires were mailed to patients six months after the injury. Non-responding patients were sent a reminder letter between two and four weeks after the initial mailing, and patients who did not respond to the reminder letter were contacted by telephone, where possible. Patients who were not contactable were traced through the hospital and treating doctors to check contact details.

### 4.2.5 Piloting of the questionnaire

#### 4.2.5.1 Methods

The Initial Patient Questionnaire and the Six Month Patient questionnaires were piloted to validate new questions, such as job satisfaction and patient satisfaction, and to uncover any objections or difficulties patients might have with the questionnaire. Both questionnaires were piloted simultaneously on 55 trauma patients selected from one public and one private fracture clinic. All patients were post-trauma patients with fractures, but not all patients were involved in motor vehicle accidents.

The patients were asked for comments regarding the questionnaire. Specifically, the patients were asked whether they understood the instructions and the questions, and they were asked to write down any comments regarding the instructions. They were also asked to identify any questions that they found confusing or difficult to answer.

Two other questionnaires were attached to the pilot questionnaires. These were to be used as gold standards for some of the questions that had not been previously validated. The additional questionnaires were the Oswestry Disability Questionnaire[454] and the Neck Disability Index.[455] These questionnaires are commonly used and have previously been validated for patients with back pain and disability, and neck pain and disability, respectively.[402 424 454 455] These surveys were used to establish criterion validity of the low back pain and disability questions, and the neck pain and disability questions.

Test-retest reliability was also examined. Patients who completed the pilot questionnaires were posted a repeat questionnaire at a period of one to two weeks after first completing the questionnaires. The repeat questionnaire contained the Initial Patient Questionnaire and the Six Month Patient Questionnaire only.

The job satisfaction question was tested separately for criterion validity by administration of the job satisfaction question and a previously validated job satisfaction questionnaire, the Job Satisfaction Survey.[456] This was distributed to 50 people, through personal contacts. The subjects were mainly doctors, nurses and hospital clerical staff.

### 4.2.5.2 Results

For the pilot study, 55 patients completed the Initial Patient Questionnaire, the Six Month Patient Questionnaire, the Oswestry Disability Questionnaire and the Neck Disability Index. These patients also provided comments on the questionnaires. Thirty (55%) of the 55 initial respondents returned the second mailing of the Initial Patient Questionnaire and the Six Month Patient Questionnaire.

The results of the pilot study are listed in the order that they appear in the Initial Patient Questionnaire and the Six Month Patient Questionnaire. The SF36 questions were not analysed as these questions have been previously validated. Also, some questions in the pilot study that were not used in the main study are not reported.

***Question: Highest education level.***

This question has four parts and follows a pattern previously used with the patient rating their highest education level at either primary school, secondary (high) school, certificate or diploma, or bachelor degree or higher. The responses were not evenly spread with 64%(35/55) respondents selecting option 2 (secondary school). Agreement on retesting was 86% (25/29). The unweighted kappa value was 0.69 (95% CI 0.42 - 0.97). The weighted kappa value was 0.79 (95% CI 0.57 - 1.00).

***Question: How would you rate your job satisfaction.***

The job satisfaction question was trialed on the trauma population (55 patients). The criterion validity was tested on a separate population of 50 people, mostly with a medical, nursing or administrative background.

22% of respondents were not employed (option 5) and the majority of respondents were satisfied with their job (41% very satisfied and 32% somewhat satisfied). The test-retest reliability for this same population (n=27) showed agreement in 85% (23/27). The unweighted kappa value was 0.80 (95% CI 0.61 - 0.98). The weighted kappa value was 0.87 (95% CI 0.73 - 1.01).

Correlation with the Job Satisfaction Survey (JSS) score is shown in Table 4.1. This demonstrates positive correlation between JSS score and job satisfaction as rated on the initial patient questionnaire. It shows, however, a floor effect in the JSS score such that it was not sensitive to the lowest score in the initial patient questionnaire ("very dissatisfied"). This indicates that the question used in the initial patient questionnaire may be able to detect a broader range of job satisfaction.

Table 4.1. Mean JSS (job satisfaction survey) scores for each response.

| Response | Mean JSS score | 95% confidence interval |
|---|---|---|
| Very satisfied | 157 | 104 – 210 |
| Somewhat satisfied | 128 | 96 – 161 |
| Somewhat dissatisfied | 105 | 54 – 157 |
| Very dissatisfied | 109 | 90 – 128 |

### *Question: Average yearly household income*

This question is based around the median household income (including investments and spouse earnings) supplied by the Australian Bureau of Statistics.[404] The median household income at the time of formulation of the questionnaire was approximately $49,000 per year. For simplicity, and allowing for inflation, a midpoint of $50,000 was chosen. As the distribution is gathered around this median point, cut off points of $30,000 on the lower side and $75,000 on the highest side were chosen.

The spread of these responses was satisfactory, although lower than the average expected with responses 1 and 2 (incomes of $50,000 or less) making up 74% of the total. The test-retest reliability showed 83% agreement (20/24). The unweighted kappa value was 0.72 (95% CI 0.47 - 0.98). The weighted kappa value was 0.77 (95% CI 0.53 - 1.01).

### *Question: How would you rate your general recovery from the injury?*

The proportion of responses 'very satisfied', 'somewhat satisfied', 'somewhat dissatisfied', and 'very dissatisfied', was 16%, 35%, 41% and 8%, respectively. Test-retest reliability revealed 63% agreement (17/27). The unweighted kappa value was 0.46 (95% CI 0.21 - 0.72). The weighted kappa value was 0.57 (95% CI 0.35 - 0.78).

The relatively poor correlation for this question may be explained by the fact that the patients interviewed were post-trauma patients with fractures. Consequently, they were recovering from their injuries and they would have expected to record higher rating of recovery in the second questionnaire, which was the case.

***Question: Regarding your injury, who do you feel was at fault?***

The three possible responses were evenly distributed, 36% selecting “I was at fault”, 34% selecting “I don’t know”, and 30% selecting “someone else”. Test-retest reliability showed agreement in 59% (16/27), a unweighted kappa value of 0.39 (95% CI 0.12 - 0.65) and a weighted kappa value of 0.38 (95% CI 0.10-0.67). This question showed poorer test-retest reliability, compared to the other questions. There was no pattern seen in the change between the initial and repeat responses. Patients’ perception of fault was not expected to significantly change between the two questionnaires. This may reflect a lack of sensitivity of this question to detect a patients’ perception of fault. Alternatively, it may reflect fluctuations in patients’ perception of fault. The question was not changed for the final questionnaire as the responses were evenly spread and the wording was considered satisfactory.

***Question: How much low back pain have you had in the past four weeks?***

The distribution of responses was skewed because 47% of respondents indicated that they had no back pain. This may have been due to the fact that none of the patients had back injuries, as these are usually treated in another clinic. This question was an adaptation of one of the pain questions from the SF36, with identical responses. Agreement was 59% (17/29). The unweighted kappa value was 0.34 (95% CI 0.12-0.57). The weighted kappa value was 0.59 (0.40-0.78). Criterion validity was tested against the Oswestry Disability Index. The mean Oswestry score increased with each successive response category, and analysis of variance showed a strong association between the two scores ($F = 41.99$, 1,50 DF, $p<0.0001$).

***Question: During the past four weeks, how much did low back pain interfere with your normal work (including both work outside the home and housework)?***

53% of participants had no disability from back pain (option one), but the distribution of the other four options was evenly distributed (15%, 11%, 11%, and 11%). This question was also adapted from the SF36 survey, with identical responses. Test-retest reliability showed an agreement of 52% (15/29). Back pain disability showed good correlation with the Oswestry Disability Index ($F = 41.19$, 1,50 DF, $p<0.0001$).

***Question: How much neck pain have you had in the past four weeks?***

64% of participants had no neck pain (option 1). Test-retest agreement was 59% (17/29). This question showed satisfactory correlation with a neck disability index (NDI), with increasing NDI scores for each successive response category ($F= 11.37$, 1,48 DF, $p=0.002$).

***Question: During the past four weeks, how much did neck pain interfere with your normal work (including both work outside the home and housework)?***

69% of participants had no disability from neck pain (option one). Agreement was 72% (21/29). The unweighted kappa value was 0.44 (0.15-0.73). The weighted kappa value was 0.51 (0.20-0.81). This question showed good correlation with the NDI, with increasing NDI scores for each successive response category ($F= 8.44$, 1,48 DF, $p=0.006$). Some of the variants in the test-retest scores for the questions regarding neck pain and back pain may be due to fluctuations in the degree of pain. All of the questions referred to the previous four weeks, so some variation was expected as the questionnaires were answered one to two weeks apart.

***Question: Are you satisfied with your progress since the injury?***

64% (34/53) patients responded with 'yes'. Test-retest reliability showed agreement in 82% (23/28). The kappa value was 0.60 (95% CI 0.29-0.91). This question was changed to improve the sensitivity, by converting the response options to a four-point Likert scale for the final version of the questionnaire. Criterion validity could not be assessed for this question. Although validated questionnaires regarding patient satisfaction exist, they usually are designed to measure patient satisfaction with treatment, rather than satisfaction with their condition. Three reviews were found addressing this measurement issue[423-425] but none of the reviews found a validated instrument. One review noted that most studies measuring patient satisfaction used a single question with a four-point scale.[424] This question was similar to the first question in the Six Month Patient Questionnaire regarding patients' perception of their general recovery from the injury. As expected, the trend for correlation between the four-point ratings of recovery and satisfaction was significant (chi 2 7.21, 1 DF, P = 0.007).

***Question: Have you retained the services of a lawyer regarding your injury?***

36% (19/53) patients responded positively. Test-retest reliability showed 90% agreement (26/29), and a kappa value of 0.79 (95% CI 0.56-1.01).

#### 4.2.5.3 Summary

Minor amendments were made to the questionnaires after piloting, as outlined above. Overall, the questionnaires showed a good distribution of responses and satisfactory test-retest reliability and, where possible, good criterion or construct reliability. Some variance in the responses was expected due to the time difference between completion of the questionnaires. Of concern, however, was the relatively poor reliability of the question regarding blame.

### 4.2.6 Measures

The explanatory variables were grouped as general, injury-related, socio-economic, and claim-related. All of the general, injury-related and socio-economic factors were recorded at the time of the injury from hospital records and the initial patient questionnaire (Appendix 13). The other claim-related variables and all outcome variables were recorded from the 6-month patient and surgeon questionnaires (Appendices 14, 15). The explanatory and outcome variables recorded are summarised in Table 4.2.

Table 4.2. The explanatory and outcome variables measured.

| Explanatory variables | Outcome variables |
|---|---|
| ***General factors*** | PCS |
| Age | MCS |
| Gender | Neck pain |
| Hospital | Back pain |
| Country of birth | Patient-rated satisfaction |
| Chronic illnesses | Patient-rated recovery |
| Job satisfaction | Complications |
| ***Injury factors*** | Fracture non-union |
| Mechanism of injury | |
| Number of fractures | |
| Other body regions injured | |
| ***Socioeconomic factors*** | |
| Highest education level | |
| Annual household income | |
| Currently employed | |
| ***Claim-related factors*** | |
| History of compensation claims | |
| Claim made | |
| Claim type | |
| Lawyer involvement | |
| Blame (patient perception of fault) | |

The mechanism of injury was classified as driver, passenger, motorcycle rider, or pedestrian/bicycle rider. The number of fractures present was a count of the number of bones fractured, and was retrieved from the treating doctors at the time of injury. The presence and number of any other body regions injured (excluding fractures) was similarly recorded.

The socio-economic factors measured were education, income and whether or not the patient was currently employed. Highest education level was categorised as primary school, secondary school, diploma or certificate, and bachelor degree or higher. Total annual household income was categorised in the same way as in Chapter Three: $0 – $30,000, $30,001 – $50,000, $50,001 - $75,000, and over $75,000. Employment was a dichotomous variable that was considered positive if an occupation was listed on the initial patient questionnaire. No consideration was given to the type of occupation or to whether the employment was full time or part time.

The claim-related variables measured were: previous claim, claim pursued, compensation system, use of a lawyer, and blame. Previous claim (a dichotomous variable) was determined from the initial patient questionnaire and was considered positive if the patient had made any previous claim for compensation (under any system). The remaining claim-related variables were measured in the same way as in Chapter Three. Claim pursuit was considered positive if the patient had made a claim for compensation related to the injury. The compensation system was categorised as workers compensation, third party or other. Use of a lawyer was considered positive if the patient had consulted a lawyer regarding their injury. Claim eligibility was not measured in this study as there was insufficient information to accurately determine this variable for the majority of patients.

PCS and MCS were calculated from the SF-36 scores, as described in Chapter Three. Neck pain and back pain were measured by repeating the two

SF-36 questions pertaining to bodily pain, and inserting the word “neck” or “low back” before the word “pain”, as described in Chapter Three. Patient and surgeon satisfaction was measured for two parameters: satisfaction with progress, and satisfaction with recovery. Each parameter was scored on a four point Likert scale, and the questions were worded similarly in the patient and surgeon quesitonnaires, to allow a comparison between patient and surgeon ratings. Fracture non-union was a dichotomous variable recorded as positive if any fracture had not healed at the 6-month follow-up (as recorded by the treating surgeons, who were asked specifically if each fracture had united).

Complications were listed in the free text response in the 6-month surgeon questionnaire. However, the presence of complications was not used as an objective outcome measure due to the open nature of the recording of complications, which allowed many complications to be listed which were based on patient complaints (e.g., pain and stiffness). Therefore, this outcome was dichotomised and considered positive if any complication was recorded, including non-union of a fracture (which was determined from a separate question).

The presence of fracture non-union was used as an objective outcome measure, as this diagnosis was provided by the treating surgeon, and is usually based on radiographic findings. This was used both as a possible explanatory variable in the analysis for each patient-based outcome, as well as a separate outcome variable.

### 4.2.7 Sample size calculation

It was initially estimated that there would be 12 explanatory variables for each outcome. Allowing 20 patients for each variable, a target of 240 patients was set. Allowing for 20% loss to follow-up, a recruitment target of 300 was made.

### 4.2.8 Statistical methods

The explanatory and outcome variables were coded and all data entered onto a spreadsheet, then imported and analysed using SAS (Cary, NC, USA). Some recoding of variables was performed during the analysis. Treating hospital was converted to a dichotomous variable (Liverpool versus all others) because of the large number of different hospitals, and the high number of patients recruited at Liverpool Hospital. Similarly, country of birth was dichotomised in to Australia and others, as the majority of patients were born in Australia, and there were a large number of other countries of birth, making statistical analysis difficult.

The number of chronic illnesses was less than in Chapter Three, with the majority of patients having none. Chronic illness was therefore dichotomised into ‘none’ and ‘one or more’.

Job satisfaction was kept as a four-part variable for the univariate analysis, and dichotomised if necessary for statistical purposes.

Mechanism of injury was divided into three parts: car, motorcycle, and pedestrian and cyclist. This was based on previous studies showing a prognostic difference between these groups,[133] and the finding that car drivers and passengers scored similarly for each outcome (therefore grouping them together as car occupants). The number of fractures was dichotomised into one versus two or more, and the number of other body regions was dichotomised into none versus one or more.

The socio-economic factors were kept in their original format, as in Chapter Three.

The claim-related factors were not changed for the statistical analysis and were analysed as in Chapter Three, with the addition of any history of claims, as a dichotomous variable. Claim type included a third category in this study: 'other'.

One-way analyses were performed for all explanatory variables against all outcome variables using Pearson's correlation coefficient when comparing two continuous variables, student's t test when comparing a dichotomous variable to a continuous variable, one-way analysis of variance when testing a categorical against a continuous variable, and the chi square test when comparing categorical variables.

A multivariate analysis was performed for each outcome using explanatory variables with significance level of 0.25 or less in the univariate analysis.

Multiple linear regression was used for continuous outcome variables, and logistic regression for dichotomous outcome variables. Categorical outcome variables (such as patient and surgeon satisfaction ratings) were converted to dichotomous by simple conversion of the four-point Likert scale score.

## 4.3 Results

### 4.3.1 Patient sample

As per protocol, recruitment ceased after 300 patients were entered onto the study, but due to delays in posting responses and in communication, 306 patients were initially entered onto the study. Of the 306 patients initially entered, 232 completed follow-up questionnaires were received. There were 5 exclusions due to moving overseas or death, resulting in a follow-up rate of 77.1%. A flow chart of the patient recruitment and follow-up is given in Figure 4.1. The earliest response was at six months, and no further attempts at contact were made (and no responses accepted) after 12 months from the date of the injury.

For the 232 patients with completed follow-up, a surgeon response was only available for 141 (60.8%). Of the 91 who did not respond, 28 were unable to respond as they had not seen the patient post-injury, and 63 did not respond and no reason was given.

Characteristics of the patients are given in Table 4.3. Comparisons of patient characteristics between responding and non-responding patients at six months, and between patients for whom a six month questionnaire was or was not received by their surgeon, are given in Tables 4.4 and 4.5, respectively.

Figure 4.1. Summary of patient recruitment and follow-up.

306

1 Died

305

4 Moved overseas

301

**Error!**

13 Withdrew consent

288

**Error!**

15 Contacted but did not respond

273

**Error!**

41 No contact acknowledged

232

Table 4.3. Characteristics of the participants.

| Variable | Category | N | % | Mean (range) |
|---|---|---|---|---|
| ***General factors*** | | | | |
| Age (years) | | 232 | | 37.9 (18 - 85) |
| Gender | Male | 232 | 72.4 | |
| | Female | | 27.6 | |
| Hospital | Liverpool | 232 | 39.7 | |
| | Other | | 60.3 | |
| Country of birth | Australia | 232 | 73.7 | |
| | Other | | 26.3 | |
| Chronic illnesses | None | 232 | 64.2 | |
| | One or more | | 35.8 | |
| Job satisfaction (if employed) | Very satisfied | 191 | 59.2 | |
| | Somewhat satisfied | | 36.1 | |
| | Somewhat dissatisfied | | 3.7 | |
| | Very dissatisfied | | 1.6 | |
| ***Injury factors*** | | | | |
| Mechanism | Car driver | 221 | 35.8 | |
| | Car passenger | | 5.9 | |
| | Motor cycle rider | | 44.8 | |
| | Pedestrian / cyclist | | 13.6 | |
| Number of fractures | One | 226 | 43.8 | |
| | Two or more | | 56.2 | |
| Other body regions injured | No | 223 | 56.1 | |
| | Yes | | 44.0 | |
| ***Socioeconomic factors*** | | | | |
| Highest education | Primary | 232 | 2.6 | |
| | Secondary | | 53.9 | |
| | Certificate/Diploma | | 29.3 | |
| | Bachelor degree | | 14.2 | |
| Annual income | $0 – 30,000 | 228 | 30.3 | |
| | $30,000 – 50,000 | | 29.8 | |
| | $50,000 – 75,000 | | 21.9 | |
| | $75,000+ | | 18.0 | |
| Employed at time of injury | No | 232 | 21.1 | |
| | Yes | | 78.9 | |
| ***Claim-related factors*** | | | | |
| Previous claim made | No | 230 | 74.8 | |
| | Yes | | 25.2 | |
| Claim made | No | 231 | 44.2 | |
| | Yes | | 55.8 | |
| Claim type (if made) | Workers comp. | 130 | 33.1 | |
| | Third party | | 56.9 | |
| | Other | | 10.0 | |
| Lawyer used | No | 226 | 57.5 | |
| | Yes | | 42.5 | |
| Blame | Self | 232 | 34.1 | |
| | Someone else | | 47.8 | |
| | Don't know | | 18.1 | |

Table 4.4. A comparison of patients who did or did not respond to the follow-up questionnaire.

| Variable | Responders | Non-responders | p value |
|---|---|---|---|
| Age (mean years) | 37.9 | 34.3 | 0.10 |
| Gender (% male) | 81.9 | 72.4 | 0.10 |
| Country of birth (% Australia) | 73.7 | 76.8 | 0.60 |
| Education (% primary/secondary) | 56.5 | 66.7 | 0.13 |
| Chronic illnesses (% present) | 35.8 | 34.8 | 0.88 |
| Hospital (% Liverpool) | 39.7 | 33.8 | 0.37 |
| Number of fractures (% > 1) | 56.2 | 44.4 | 0.10 |
| Income (% 0 - $50,000) | 60.1 | 76.6 | 0.02 |
| Job satisfaction (% satisfied) | 94.8 | 92.3 | 0.49 |
| Mechanism (% car occupants) | 41.6 | 32.3 | 0.18 |
| Employment (% employed) | 78.9 | 75.4 | 0.54 |
| Other injuries present (% present) | 44.0 | 37.7 | 0.38 |

Table 4.5. Comparison of patients for whom a questionnaire was or was not received from their surgeon.

| Variable | Responders | Non-responders | p value |
|---|---|---|---|
| Age (mean years) | 37.9 | 38.0 | 0.99 |
| Gender (% male) | 71.2 | 74.4 | 0.60 |
| Country of birth (% Australia) | 71.9 | 76.7 | 0.42 |
| Education (% primary/secondary) | 56.2 | 57.0 | 0.90 |
| Chronic illnesses (% present) | 34.9 | 37.2 | 0.73 |
| Hospital (% Liverpool) | 48.6 | 24.4 | 0.0003 |
| Number of fractures (% > 1) | 60.1 | 49.4 | 0.17 |
| Income (% 0 - $50,000) | 65.5 | 50.6 | 0.03 |
| Job satisfaction (% satisfied) | 95.7 | 93.3 | 0.47 |
| Mechanism (% car occupants) | 46.0 | 34.2 | 0.08 |
| Employment (% employed) | 76.7 | 82.6 | 0.29 |
| Other injuries present | 44.0 | 43.9 | 0.99 |
| Blame (%blame self) | 45.5 | 39.5 | 0.43 |
| Claim (%claim made) | 57.2 | 53.5 | 0.58 |
| Use of a lawyer (% yes) | 45.8 | 37.4 | 0.22 |
| Claim type (% workers compensation) | 18.6 | 18.6 | 0.95 |

### 4.3.2 Hypothesis 1: Physical health

The PCS could be calculated for all patients. The mean score was 39.2, median 37.4, standard deviation 11.0, and scores ranged from 15.9 to 65.8. The univariate (unadjusted) associations between the explanatory variables and PCS are given in Table 4.6.

Backward stepwise regression removed the following variables in order (p values in brackets): treating hospital (0.98), current employment (0.81), blame (0.42), chronic illnesses(0.36), other injuries (0.27), mechanism (0.23), income (0.13), and claim pursuit (0.28).

The final model is given in Table 4.7. It included data from 220 patients and explained 30.5% of the variation in PCS. Interaction terms were tested in the final model and found to be not significant.

To test for confounding between the use of a lawyer and pursuit of a claim, claim pursuit was substituted for use of a lawyer in the final model, resulting in a highly significant effect for claim pursuit (effect estimate: -5.39, $p < 0.0001$).

The effect of fracture non-union was tested separately, in the sub group of 141 patients for whom surgeon follow-up was available. Fracture union was removed from the model with a p value of 0.22, resulting in no change in the final model.

Table 4.6. Unadjusted association between explanatory variables and PCS.

| Variable | p value | Category (if applicable) | Mean PCS (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age (years) | <0.0001 | | |
| Gender | 0.0003 | Male | 40.8 (39.1 – 42.4) |
| | | Female | 34.9 (32.3 – 37.6) |
| Hospital | 0.16 | Liverpool | 40.4 (38.3 – 42.5) |
| | | Other | 38.3 (36.4 – 40.3) |
| Country of birth | 0.41 | Australia | 39.5 (37.8 – 41.2) |
| | | Other | 38.2 (35.6 – 40.7) |
| Chronic illnesses | 0.06 | None | 40.2 (38.5 – 41.9) |
| | | One or more | 37.4 (34.8 – 39.9) |
| Job satisfaction (if employed) | 0.46 | Very satisfied | 39.8 (37.8 – 41.9) |
| | | Somewhat satisfied | 40.3 (37.7 – 42.9) |
| | | Somewhat dissatisfied | 43.9 (35.7 – 52.1) |
| | | Very dissatisfied | 48.6 (36.0 – 61.1) |
| ***Injury factors*** | | | |
| Mechanism | 0.003 | Car driver/passenger | 42.2 (39.5 – 44.9) |
| | | Motorcycle | 48.1 (45.5 – 50.7) |
| | | Pedestrian / cyclist | 41.5 (36.7 – 46.2) |
| Number of fractures | 0.004 | One | 47.6 (45.1 – 50.2) |
| | | Two or more | 42.4 (40.0 – 44.8) |
| Other body regions injured | 0.008 | No | 46.7 (44.4 – 49.1) |
| | | Yes | 41.9 (39.1 – 44.6) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.95 | Primary | 36.9 (28.0 – 45.8) |
| | | Secondary | 39.1 (37.1 – 41.0) |
| | | Certificate/Diploma | 39.3 (36.7 – 41.9) |
| | | Bachelor degree | 39.7 (35.9 – 43.5) |
| Annual income | 0.12 | $0 – 30,000 | 37.3 (34.7 – 39.9) |
| | | $30,000 – 50,000 | 38.4 (35.8 – 41.0) |
| | | $50,000 – 75,000 | 40.4 (37.4 – 43.4) |
| | | $75,000+ | 42.0 (38.7 – 45.4) |
| Employed (at injury) | 0.02 | No | 35.9 (32.8 – 38.9) |
| | | Yes | 40.0 (38.5 – 41.6) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.49 | No | 39.4 (37.7 – 41.0) |
| | | Yes | 38.2 (35.3 – 41.1) |
| Claim made | <0.0001 | No | 42.7 (40.4 – 45.0) |
| | | Yes | 36.4 (34.7 – 38.0) |
| Claim type (if made) | 0.09 | Workers comp. | 38.7 (35.9 – 41.5) |
| | | Third party | 34.8 (32.6 – 36.9) |
| | | Other | 37.6 (32.5 – 42.8) |
| Lawyer used | <0.0001 | No | 42.5 (40.5 – 44.6) |
| | | Yes | 34.7 (33.2 – 36.3) |
| Blame | <0.0001 | Self | 43.4 (41.1 – 45.6) |
| | | Someone else | 35.2 (33.3 – 37.1) |
| | | Don’t know | 41.8 (38.6 – 44.9) |
| Fracture union | 0.11 | No | 34.6 (29.7 – 39.5) |
| | | Yes | 38.9 (37.0 – 40.9) |

Table 4.7. Adjusted (multivariate) associations between explanatory variables and PCS.

| Variable | Group | ∆ mean PCS | p value |
|---|---|---|---|
| Age | Per year | -0.18 | <0.0001 |
| Gender | Male | +3.73 | 0.01 |
| Number of fractures | More than one | -3.75 | 0.005 |
| Use of a lawyer | Yes | -7.63 | <0.0001 |

∆ = change in

### 4.3.3 Hypothesis 2: Mental health

The MCS could be calculated for all patients. The mean score was 44.7, median 46.3, standard deviation 13.7, and scores ranged from 13.8 to 71.6. The univariate (unadjusted) associations between the explanatory variables and MCS are given in Table 4.8.

Stepwise regression removed the following variables in order (p values in brackets): gender (0.95), mechanism (0.83), claim pursuit (0.96), employment (0.96), blame (0.87), number of fractures (0.63), country of birth (0.39), and presence of other, non-orthopaedic, injuries (0.16). The final model (Table 4.9) used data from 222 patients and explained 23.1% of the variation in MCS.

Table 4.8. Unadjusted associations between explanatory variables and MCS.

| Variable | p value | Category (if applicable) | Mean MCS (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age (years) | 0.009 | | |
| Gender | 0.002 | Male | 46.4 (44.4 – 48.5) |
| | | Female | 40.3 (37.0 – 43.6) |
| Hospital | 0.27 | Liverpool | 46.0 (43.3 – 48.6) |
| | | Other | 43.9 (41.5 – 46.3) |
| Country of birth | 0.003 | Australia | 46.3 (44.3 – 48.3) |
| | | Other | 40.3 (36.9 – 43.8) |
| Chronic illnesses | 0.78 | None | 44.5 (42.2 – 46.8) |
| | | One or more | 45.1 (42.2 – 48.0) |
| Job satisfaction (if employed) | 0.39 | Very satisfied | 46.6 (44.1 – 49.1) |
| | | Somewhat satisfied | 45.2 (42.0 – 48.4) |
| | | Somewhat dissatisfied | 54.5 (44.3 – 64.6) |
| | | Very dissatisfied | 47.2 (31.7 – 62.8) |
| ***Injury factors*** | | | |
| Mechanism | 0.003 | Car driver/passenger | 42.2 (39.5 – 44.9) |
| | | Motorcycle | 48.1 (45.5 – 50.7) |
| | | Pedestrian / cyclist | 41.5 (36.7 – 46.2) |
| Number of fractures | 0.004 | One | 47.6 (45.1 – 50.2) |
| | | Two or more | 42.4 (40.0 – 44.8) |
| Other body regions injured | 0.008 | No | 46.7 (44.4 – 49.1) |
| | | Yes | 41.9 (39.1 – 44.6) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.31 | Primary | 37.5 (26.5 – 48.5) |
| | | Secondary | 45.3 (42.9 – 47.7) |
| | | Certificate/Diploma | 43.2 (40.0 – 46.5) |
| | | Bachelor degree | 47.0 (42.3 – 51.7) |
| Annual income | <0.0001 | $0 – 30,000 | 37.9 (34.9 – 40.9) |
| | | $30,000 – 50,000 | 45.3 (42.2 – 48.3) |
| | | $50,000 – 75,000 | 47.4 (43.9 – 51.0) |
| | | $75,000+ | 51.5 (47.6 – 55.5) |
| Employed (at injury) | 0.003 | No | 39.6 (35.8 – 43.3) |
| | | Yes | 46.1 (44.1 – 48.1) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.86 | No | 44.5 (42.4 – 46.6) |
| | | Yes | 44.9 (41.4 – 48.4) |
| Claim made | 0.008 | No | 47.4 (44.8 – 50.1) |
| | | Yes | 42.6 (40.2 – 45.0) |
| Claim type (if made) | 0.003 | Workers comp. | 47.1 (43.1 – 51.0) |
| | | Third party | 39.1 (36.1 – 42.1) |
| | | Other | 47.6 (40.4 – 54.8) |
| Lawyer used | <0.0001 | No | 48.0 (45.8 – 50.1) |
| | | Yes | 40.2 (37.4 – 42.9) |
| Blame | 0.003 | Self | 47.9 (45.0 – 50.9) |
| | | Someone else | 41.5 (39.0 – 44.0) |
| | | Don't know | 47.2 (43.1 – 51.2) |
| Fracture union | 0.28 | No | 39.9 (33.4 – 46.4) |
| | | Yes | 43.7 (41.2 – 46.3) |

Table 4.9. Adjusted (multivariate) associations between explanatory variables and MCS.

| Variable | Group | Δ mean MCS | p value |
|---|---|---|---|
| Age | Per year | -0.11 | 0.03 |
| Use of a lawyer | Yes | -7.68 | <0.0001 |
| Annual household income | $0 – $30,000 | * | |
| | $30,001 – $50,000 | +6.79 | 0.001 |
| | $50,001 – $75,000 | +9.53 | <0.0001 |
| | Over $75,000 | +12.55 | <0.0001 |

*referent group, Δ = change in

As the type of claim (third party or workers compensation) was a significant factor on univariate analysis, an alternative analysis was performed combining claim pursuit and claim type into one variable. For both PCS and MCS as an outcome, this alternative analysis resulted in the same final model, except that claim pursuit was included as a significant predictor. However, there was no significant difference in the estimates according to type of claim.

### 4.3.4 Hypothesis 3: Neck pain

A combined score for neck pain, using data from the two questions regarding pain severity and effect of pain on function, was calculated for all patients. The mean score was 3.34, standard deviation 2.16, the median was 2.00, and the range was from 2 to 11. The unadjusted association between each explanatory variable and neck pain is given in Table 4.10.

Stepwise regression removed the following variables in order (p values in brackets): blame (0.82), treating hospital (0.78), mechanism (0.66), country of birth (0.66), employment (0.67), number of fractures (0.46), presence of other injuries (0.25), age (0.23), income (0.14), and claim pursuit (0.08). The final model for neck pain is given in Table 4.11. It included data from 226 patients and accounted for 18.3% of the variation in the neck pain score. Interaction terms in the final model were not significant.

Substitution of claim pursuit for use of a lawyer in the final model showed this to be highly significant ($p = 0.001$) suggesting confounding between these two variables.

The effect of fracture union was tested separately and found to be not significant in multivariate analysis ($p = 0.16$). However, inclusion of fracture union in the modelling process resulted in a different final model (Table 4.12) which included data on 227 patients and explained 21.5% of the variation in the neck pain score. The final model is similar, except that use of a lawyer was excluded ($p = 0.48$) and claim pursuit was retained, with the same effect estimate as use of a lawyer.

Table 4.10. Unadjusted association between explanatory variables and neck pain as a continuous variable.

| Variable | p value | Category (if applicable) | Mean score (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age (years) | 0.003 | | |
| Gender | 0.0004 | Male | 3.0 (2.7 – 3.2) |
| | | Female | 4.3 (3.6 – 5.0) |
| Hospital | 0.19 | Liverpool | 3.1 (2.7 – 3.5) |
| | | Other | 3.5 (3.1 – 3.9) |
| Country of birth | 0.03 | Australia | 3.1 (2.8 – 3.4) |
| | | Other | 4.0 (3.3 – 4.7) |
| Chronic illnesses | 0.77 | None | 3.3 (3.0 – 3.7) |
| | | One or more | 3.4 (2.9 – 3.9) |
| Job satisfaction | 0.73 | Very satisfied | 3.3 (2.9 – 3.7) |
| (if employed) | | Somewhat satisfied | 3.1 (2.6 – 3.6) |
| | | Somewhat dissatisfied | 2.7 (1.2 – 4.2) |
| | | Very dissatisfied | 4.0 (1.7 – 6.3) |
| ***Injury factors*** | | | |
| Mechanism | 0.0008 | Car driver/passenger | 3.9 (3.5 – 4.3) |
| | | Motorcycle | 2.7 (2.3 – 3.2) |
| | | Pedestrian / cyclist | 3.7 (2.9 – 4.4) |
| Number of fractures | 0.004 | One | 2.9 (2.6 – 3.2) |
| | | Two or more | 3.7 (3.3 – 4.1) |
| Other body regions injured | 0.05 | No | 3.1 (2.8 – 3.5) |
| | | Yes | 3.7 (3.2 – 4.2) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.004 | Primary | 6.5 (4.8 – 8.2) |
| | | Secondary | 3.3 (2.9 – 3.6) |
| | | Certificate/Diploma | 3.3 (2.8 – 3.8) |
| | | Bachelor degree | 3.1 (2.4 – 3.8) |
| Annual income | 0.002 | $0 – 30,000 | 4.1 (3.6 – 4.6) |
| | | $30,000 – 50,000 | 3.2 (2.7 – 3.7) |
| | | $50,000 – 75,000 | 3.0 (2.4 – 3.6) |
| | | $75,000+ | 2.7 (2.0 – 3.3) |
| Employed (at injury) | 0.03 | No | 4.1 (3.3 – 4.8) |
| | | Yes | 3.1 (2.9 – 3.4) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.97 | No | 3.3 (3.0 – 3.7) |
| | | Yes | 3.4 (2.8 – 3.9) |
| Claim made | 0.0002 | No | 2.8 (2.5 – 3.1) |
| | | Yes | 3.8 (3.4 – 4.2) |
| Claim type (if made) | 0.89 | Workers comp. | 3.6 (2.9 – 4.4) |
| | | Third party | 3.9 (3.3 – 4.4) |
| | | Other | 3.8 (2.4 – 5.1) |
| Lawyer used | 0.003 | No | 3.0 (2.7 – 3.3) |
| | | Yes | 3.8 (3.3 – 4.3) |
| Blame | 0.008 | Self | 2.9 (2.5 – 3.4) |
| | | Someone else | 3.8 (3.4 – 4.2) |
| | | Don’t know | 2.9 (2.3 – 3.6) |
| Fracture union | 0.23 | No | 4.1 (3.0 – 5.1) |
| | | Yes | 3.4 (3.0 – 3.8) |

Table 4.11. Adjusted (multivariate) associations between explanatory variables and neck pain score.

| Variable | Group | Δ mean score | p value |
|---|---|---|---|
| Gender | Female | +1.32 | <0.0001 |
| Use of a lawyer | Yes | +0.94 | 0.0004 |
| Education level | Primary | * | |
| | Secondary | -3.03 | 0.0002 |
| | Diploma/certificate | -2.94 | 0.0005 |
| | Degree | -3.72 | <0.0001 |
| | (overall) | | 0.0004 |

*referent group, Δ = change in

Table 4.12. Adjusted (multivariate) associations between explanatory variables (including fracture union) and neck pain score.

| Variable | Group | Δ mean score | p value |
|---|---|---|---|
| Gender | Female | +1.07 | <0.0001 |
| Claim pursuit | Yes | +0.94 | 0.0006 |
| Education level | Primary | * | |
| | Secondary | -2.91 | 0.0005 |
| | Diploma/certificate | -2.87 | 0.0008 |
| | Degree | -3.02 | 0.0009 |
| | (overall) | | 0.006 |
| Annual household income | $0 - $30,000 | * | |
| | $30,001 - $50,000 | -0.55 | 0.12 |
| | $50,001 - $75,000 | -0.75 | 0.05 |
| | Over $75,000 | -1.25 | 0.003 |
| | (overall) | | 0.03 |

*referent group, Δ = change in

### 4.3.5 Hypothesis 4: Back pain

Data regarding back pain was available on all patients. The mean back pain score was 4.16, the standard deviation 2.54, the median 3.0, and the range was from 2 to 11. The distribution was positively skewed. The unadjusted associations are given in Table 4.13.

Backward stepwise regression removed the following variables in order (p values in brackets): blame (1.00), mechanism (0.94), country of birth (0.84), claim (0.60), income (0.41), age (0.16), and number of fractures (0.16). The final model (Table 4.14) incorporated data from 226 patients and explained 14.6% of the variation in the back pain score. Interaction terms in the final model were not significant.

Table 4.13. The unadjusted associations between the explanatory variables and back pain score.

| Variable | p value | Category (if applicable) | Mean score (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Age (years) | 0.003 | | |
| Gender | 0.004 | Male | 3.9 (3.5 – 4.2) |
| | | Female | 4.9 (4.2 – 5.6) |
| Hospital | 0.61 | Liverpool | 4.1 (3.5 – 4.6) |
| | | Other | 4.2 (3.8 – 4.7) |
| Country of birth | 0.05 | Australia | 4.0 (3.6 – 4.3) |
| | | Other | 4.7 (4.0 – 5.4) |
| Chronic illnesses | 0.66 | None | 4.2 (3.8 – 4.6) |
| | | One or more | 4.1 (3.5 – 4.6) |
| Job satisfaction (if employed) | 0.88 | Very satisfied | 3.9 (3.4 – 4.3) |
| | | Somewhat satisfied | 3.9 (3.3 – 4.4) |
| | | Somewhat dissatisfied | 3.1 (1.4 – 4.9) |
| | | Very dissatisfied | 4.0 (1.3 – 6.7) |
| ***Injury factors*** | | | |
| Mechanism | 0.06 | Car driver/passenger | 4.5 (3.9 – 5.0) |
| | | Motorcycle | 3.7 (3.2 – 4.2) |
| | | Pedestrian / cyclist | 4.5 (3.6 – 5.4) |
| Number of fractures | 0.003 | One | 3.6 (3.2 – 4.0) |
| | | Two or more | 4.6 (4.1 – 5.0) |
| Other body regions injured | 0.31 | No | 4.0 (3.5 – 4.4) |
| | | Yes | 4.3 (3.8 – 4.8) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.10 | Primary | 5.7 (3.6 – 7.7) |
| | | Secondary | 4.1 (3.7 – 4.6) |
| | | Certificate/Diploma | 4.5 (3.9 – 5.1) |
| | | Bachelor degree | 3.4 (2.5 – 4.3) |
| Annual income | 0.001 | $0 – 30,000 | 5.0 (4.4 – 5.6) |
| | | $30,000 – 50,000 | 4.3 (3.7 – 4.9) |
| | | $50,000 – 75,000 | 3.7 (3.0 – 4.4) |
| | | $75,000+ | 3.2 (2.4 – 3.9) |
| Employed (at injury) | 0.0004 | No | 5.3 (4.5 – 6.1) |
| | | Yes | 3.9 (3.5 – 4.2) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.66 | No | 4.1 (3.7 – 4.5) |
| | | Yes | 4.3 (3.6 – 5.0) |
| Claim made | 0.08 | No | 3.8 (3.4 – 4.3) |
| | | Yes | 4.4 (3.9 – 4.9) |
| Claim type (if made) | 0.12 | Workers comp. | 3.7 (2.9 – 4.5) |
| | | Third party | 4.8 (4.1 – 5.4) |
| | | Other | 4.5 (3.0 – 6.0) |
| Lawyer used | 0.002 | No | 3.7 (3.3 – 4.1) |
| | | Yes | 4.8 (4.2 – 5.4) |
| Blame | 0.03 | Self | 3.6 (3.1 – 4.2) |
| | | Someone else | 4.6 (4.2 – 5.1) |
| | | Don’t know | 3.9 (3.1 – 4.7) |
| Fracture union | 0.59 | No | 4.8 (3.6 – 6.0) |
| | | Yes | 4.4 (3.9 – 4.9) |

Table 4.14. Adjusted (multivariate) associations between explanatory variables and back pain score.

| Variable | Group | $\Delta$ mean score | p value |
|---|---|---|---|
| Gender | Female | +0.80 | 0.04 |
| Employed at time of injury | Yes | -1.04 | 0.01 |
| Use of a lawyer | Yes | +1.15 | 0.0005 |
| Education level | Primary | * | |
| | Secondary | -1.45 | 0.15 |
| | Diploma/certificate | -0.92 | 0.37 |
| | Degree | -2.46 | 0.02 |
| | (overall) | | 0.01 |

*referent group, $\Delta$ = change in

### 4.3.6 Hypothesis 5: Patient-rated satisfaction and recovery

Patient satisfaction data was complete and the results are shown in Table 4.15.

Table 4.15. Frequency of patient responses regarding satisfaction with progress since the injury.

| Response | Frequency |
|---|---|
| Very satisfied | 57 (24.6%) |
| Somewhat satisfied | 116 (50.0%) |
| Somewhat dissatisfied | 35 (15.1%) |
| Very dissatisfied | 24 (10.4%) |

The univariate analyses for categorical variables were performed using patient satisfaction as a four-part variable, but odds ratios for a poor outcome (using a dichotomous outcome) are provided in the table for easier interpretation. For age (the only continuous variable) the t test was significant (p = 0.07), with a mean age of 36.4 for satisfied patients, and 42.4 for dissatisfied patients. The univariate associations for the categorical variables are provided in Table 4.16.

Backward stepwise logistic regression removed all variables except blame. Consequently, the final model is the same as the univariate association for blame. The variables for claim pursuit or use of a lawyer (significant on univariate analysis) were not significant when they were in the same model as blame ($p = 0.25$ and $0.83$, respectively).

In separate subgroup analyses, claim type was not significant ($p = 0.32$), and nor was fracture union, although there was a trend towards lower patient satisfaction when fracture non-union was present ($p = 0.08$).

Table 4.16. Univariate (unadjusted) associations between explanatory variables and patient satisfaction.

| Variable | p value for overall $chi^2$ (MH) | Category | OR (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Gender | 0.07 (0.04) | Female | 1.00 |
| | | Male | 0.44 (0.23 – 0.82) |
| Hospital | 0.26 (0.21) | Other | 1.00 |
| | | Liverpool | 0.75 (0.39 – 1.45) |
| Country of birth | 0.33 (0.30) | Other | 1.00 |
| | | Australia | 0.75 (0.39 – 1.45) |
| Chronic illnesses | 0.73 (0.72) | None | 1.00 |
| | | One or more | 1.09 (0.59 – 2.02) |
| Job satisfaction (if employed) | 0.03 (0.58) | Very satisfied | 0.76 (0.07 – 8.64) |
| | | Somewhat satisfied | 0.42 (0.04 – 5.03) |
| | | Somewhat dissatisfied | 0.33 (0.01 – 8.18) |
| | | Very dissatisfied | 1.00 |
| ***Injury factors*** | | | |
| Mechanism | 0.28 (0.71) | Car occupant | 1.00 |
| | | Motorcycle | 0.72 (0.36 – 1.41) |
| | | Pedestrian / cyclist | 2.17 (0.92 – 5.12) |
| Number of fractures | 0.81 (0.66) | One | 1.00 |
| | | Two or more | 1.14 (0.62 – 2.09) |
| Other injuries | 0.67 (0.67) | No | 1.00 |
| | | Yes | 1.05 (0.58 – 1.92) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.35 (0.17) | Primary | 1.00 |
| | | Secondary | 0.75 (0.13 – 4.27) |
| | | Certificate/Diploma | 0.72 (0.12 – 4.28) |
| | | Bachelor degree | 0.36 (0.05 – 2.50) |
| Annual income | 0.05 (0.01) | $0 – 30,000 | 1.00 |
| | | $30,000 – 50,000 | 0.47 (0.22 – 0.99) |
| | | $50,000 – 75,000 | 0.41 (0.18 – 0.96) |
| | | $75,000+ | 0.34 (0.13 – 0.88) |
| Employed (at injury) | 0.27 (0.06) | No | 1.00 |
| | | Yes | 0.63 (0.32 – 1.26) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.90 (0.48) | No | 1.00 |
| | | Yes | 1.28 (0.66 – 2.49) |
| Claim made | 0.02 (0.004) | No | 1.00 |
| | | Yes | 2.69 (1.41 – 5.13) |
| Claim type (if made) | 0.50 (0.11) | Workers comp. | 1.00 |
| | | Third party | 1.77 (0.77 – 4.06) |
| Lawyer used | 0.05 (0.008) | No | 1.00 |
| | | Yes | 1.74 (0.96 – 3.16) |
| Blame | 0.001 (0.0001) | Self | 1.00 |
| | | Don’t know | 1.24 (0.44 – 3.47) |
| | | Someone else | 3.62 (1.72 – 7.62) |
| Fracture union | 0.21 (0.26) | No | 1.00 |
| | | Yes | 0.52 (0.19 – 1.44) |

MH = Mantel-Haenszel test for trend, OR = odds ratio for patient dissatisfaction

Data on patient-rated recovery were complete. The responses are provided in Table 4.17and the univariate analyses for the categorical variables are given in Table 4.18. As with the other analyses of satisfaction and recovery, the p values are given for the four-part outcome variable, and the odds ratios and multivariate analyses use the dichotomised outcomes.

The mean age in patients who felt they had recovered (responses 1 and 2) was 34.2 (95% CI 31.2 – 37.1) compared to 40.9 (95% CI 38.0 – 43.9) in those who felt that they had not significantly recovered (responses 3 and 4). This difference was statistically significant ($p = 0.002$).

Table 4.17. Frequency of patient responses regarding general recovery from the injury.

| Response | Frequency |
|---|---|
| Back to normal | 18 (7.8%) |
| Minor problems only | 85 (36.6%) |
| Significant problems remain | 117 (50.4%) |
| No significant recovery has occurred since the injury | 12 (5.2%) |

The final model for patient-rated recovery (Table 4.19) used data from 226 patients. Satisfactory association between predicted and observed responses was noted with 68.3% concordance and a c value of 0.75.

Substituting claim pursuit for lawyer in the final model resulted in claim becoming significant ($p = 0.03$)

Table 4.18. Univariate (unadjusted) associations between explanatory variables and patient-rated recovery.

| Variable | p value for overall $chi^2$ (MH) | Category | OR (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Gender | 0.01 (0.004) | Female | 1.00 |
| | | Male | 0.35 (0.18 – 0.65) |
| Hospital | 0.13 (0.37) | Other | 1.00 |
| | | Liverpool | 0.74 (0.44 – 1.25) |
| Country of birth | 0.01 (0.11) | Other | 1.00 |
| | | Australia | 0.76 (0.42 – 1.37) |
| Chronic illnesses | 0.97 (0.70) | None | 1.00 |
| | | One or more | 1.07 (0.62 – 1.83) |
| Job satisfaction (if employed) | 0.02 (0.05) | Very satisfied | 2.61 (0.23 – 29.6) |
| | | Somewhat satisfied | 2.31 (0.20 – 26.7) |
| | | Somewhat dissatisfied | 0.33 (0.01 – 8.18) |
| | | Very dissatisfied | 1.00 |
| ***Injury factors*** | | | |
| Mechanism | 0.08 (0.36) | Car occupant | 1.00 |
| | | Motorcycle | 0.73 (0.41 – 1.29) |
| | | Pedestrian / cyclist | 2.21 (0.89 – 5.48) |
| Number of fractures | 0.003 (0.0006) | One | 1.00 |
| | | Two or more | 2.29 (1.34 – 3.93) |
| Other injuries | 0.635 (0.22) | No | 1.00 |
| | | Yes | 1.38 (0.81 – 2.36) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.66 (0.73) | Primary | 1.00 |
| | | Secondary | 1.19 (0.23 – 6.14) |
| | | Certificate/Diploma | 1.35 (0.25 – 7.15) |
| | | Bachelor degree | 1.36 (0.24 – 7.75) |
| Annual income | 0.008 (0.007) | $0 – 30,000 | 1.00 |
| | | $30,000 – 50,000 | 0.72 (0.36 – 1.43) |
| | | $50,000 – 75,000 | 0.57 (0.27 – 1.19) |
| | | $75,000+ | 0.54 (0.25 – 1.19) |
| Employed (at injury) | 0.25 (0.18) | No | 1.00 |
| | | Yes | 0.83 (0.44 – 1.58) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.83 (0.83) | No | 1.00 |
| | | Yes | 1.15 (0.63 – 2.10) |
| Claim made | <0.0001 (0.0001) | No | 1.00 |
| | | Yes | 3.89 (2.24 – 6.74) |
| Claim type (if made) | 0.32 (0.09) | Workers comp. | 1.00 |
| | | Third party | 1.89 (0.85 – 4.23) |
| Lawyer used | <0.0001 (<0.0001) | No | 1.00 |
| | | Yes | 3.67 (2.08 – 6.49) |
| Blame | <0.0001 (<0.0001) | Self | 1.00 |
| | | Don't know | 0.82 (0.38 – 1.77) |
| | | Someone else | 4.15 (2.24 – 7.70) |
| Fracture union | 0.27 (0.08) | No | 1.00 |
| | | Yes | 0.34 (0.11 – 1.09) |

MH = Mantel-Haenszel test for trend, OR = odds ratio for poor patient recovery

In a separate analysis, claim type was found to be not significant (p = 0.66). In another separate analysis (n = 140) the presence of fracture non-union was found to be significantly associated with poor patient recovery (p = 0.04).

Table 4.19. Adjusted (multivariate) associations between explanatory variables and patient-rated recovery.

| Variable | Group | OR (95% CI) | p value |
|---|---|---|---|
| Gender | Male | 0.31 (0.15 – 0.61) | 0.04 |
| Use of a lawyer | Yes | 2.13 (1.01 – 4.47) | 0.05 |
| Blame | Self | 1.00 | |
| | Don’t know | 0.77 (0.34 – 1.79) | 0.55 |
| | Someone else | 2.86 (1.29 – 6.34) | 0.01 |
| | (overall) | | 0.006 |

OR = odds ratio for poor recovery

The mean SF-35 summary scores were compared to categories of patient-rated satisfaction and recovery, to test the association between these scores and measures of health. A strong association was seen for each variable, as shown in Table 4.20.

Table 4.20. Associations between the SF-36 summary scores and patient-rated satisfaction and recovery.

| Outcome | Group | Mean PCS (95% CI)* | Mean MCS (95% CI)* |
|---|---|---|---|
| Satisfaction | Satisfied | 41.6 (39.9 – 43.2) | 47.6 (45.7 – 49.6) |
| | Dissatisfied | 32.2 (30.3 – 34.1) | 36.2 (32.8 – 39.6) |
| Recovery | Good | 47.7 (46.0 – 49.5) | 50.7 (48.4 – 53.0) |
| | Poor | 32.3 (31.1 – 33.6) | 40.0 (37.6 – 42.3) |

* the p value for each association was <0.0001.

### 4.3.7 Hypothesis 6: Surgeon-rated satisfaction and recovery

Surgeon-rated satisfaction with patient progress, and surgeon-rated patient recovery were categorised as four-part variables. However, due to only one surgeon choosing the worst ranking for satisfaction, and only two surgeons choosing the worst ranking for recovery, these variables were both dichotomised into ‘good’ and ‘poor’. As the number of samples in the ‘poor’ group was low, some four-part explanatory variables (education and job satisfaction) were also dichotomised and this was performed by combining responses from the first two and last two responses, separately.

Surgeon questionnaires were only requested if a completed patient questionnaire was received, however the response rate for the surgeon questionnaire was poor (141/232, 60.8%). In 28 cases however, the treating surgeons was not able to respond because the patient had not attended for follow-up.

Surgeons rated their satisfaction with the patient’s progress since the injury as good in 124 cases (88.0%) and poor in 17 (12.1%). Surgeons rated patient recovery as good in 94 cases (66.7%) and poor in 47 (33.3%).

The univariate associations between the categorical explanatory variables and surgeon-rated satisfaction and recovery are given in Tables 4.21 and 4.22, respectively. Patients for whom surgeon satisfaction was poor were older than those who were rated good (mean ages 43.3 and 37.3, respectively, $t = -1.41$, 139 DF, $p = 0.16$). Similarly, patients whose recovery was rated as poor were older than those rated as good (mean ages 40.2 and 37.0, respectively, $t = 1.09$, 139 DF, $p = 0.28$).

For surgeon satisfaction, logistic regression removed the following variables in order (p values in brackets): blame (0.64), claim (0.10), age (0.13), and number of fracture (0.08). The final model, using data from 140 patients, contained fracture non-union as the only significant variable. The presence of non-union predicted surgeon dissatisfaction (OR 9.09, 95% CI 2.91 – 28.6).

For surgeons' rating of recovery, logistic regression removed variables in the following order (p values in brackets): treating hospital (0.98), blame (0.92), gender (0.83), number of fractures (0.70), use of a lawyer (0.54), income (0.41), mechanism (0.20), claim pursuit (0.09), and presence of other injuries (0.10). The only remaining variable in the model was the presence of fracture non-union, for which the odds ratio of poor recovery was 10.4 (95% CI 3.23 – 33.3)

The associations between surgeon-rated satisfaction and recovery, and the PCS and MCS scores are given in Table 4.23.

Table 4.21. Univariate (unadjusted) associations between explanatory variables and surgeon-rated satisfaction.

| Variable | p value for overall $chi^2$ (MH) | Category | OR (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Gender | 0.68 | Female | 1.00 |
| | | Male | 1.28 (0.39 – 4.19) |
| Hospital | 0.87 | Other | 1.00 |
| | | Liverpool | 0.92 (0.33 – 2.53) |
| Country of birth | 0.97 | Other | 1.00 |
| | | Australia | 0.98 (0.32 – 2.99) |
| Chronic illnesses | 0.51 | None | 1.00 |
| | | One or more | 1.42 (0.50 – 3.99) |
| Job satisfaction | 0.41 | Satisfied | 1.00 |
| | | Dissatisfied | Not estimatable |
| ***Injury factors*** | | | |
| Mechanism | 0.52 (0.88) | Car occupant | 1.00 |
| | | Motorcycle | 0.57 (0.18 – 1.81) |
| | | Pedestrian / cyclist | 1.24 (0.30 – 5.19) |
| Number of fractures | 0.24 | One | 1.00 |
| | | Two or more | 0.55 (0.20 – 1.51) |
| Other injuries | 0.70 | No | 1.00 |
| | | Yes | 0.82 (0.29 – 2.28) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.51 | Certificate or higher | 1.00 |
| | | Primary / secondary | 0.71 (0.26 – 1.96) |
| Annual income | 0.99 (0.89) | $0 – 30,000 | 1.00 |
| | | $30,000 – 50,000 | 1.00 (0.28 – 3.53) |
| | | $50,000 – 75,000 | 1.10 (0.28 – 4.28) |
| | | $75,000+ | 0.80 (0.15 – 4.33) |
| Employed (at injury) | 0.93 | No | 1.00 |
| | | Yes | 0.95 (0.29 – 3.14) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.96 | No | 1.00 |
| | | Yes | 1.03 (0.31 – 3.42) |
| Claim made | 0.09 | No | 1.00 |
| | | Yes | 2.81 (0.87 – 9.09) |
| Claim type (if made) | 0.99 | Workers comp. | 1.00 |
| | | Third party | 0.99 (0.26 – 3.79) |
| Lawyer used | 0.26 | No | 1.00 |
| | | Yes | 1.81 (0.64 – 5.06) |
| Blame | 0.23 (0.17) | Self | 1.00 |
| | | Don't know | 0.54 (0.06 – 5.12) |
| | | Someone else | 2.12 (0.64 – 7.00) |
| Fracture union | <0.0001 | No | 1.00 |
| | | Yes | 0.11 (0.04 – 0.34) |

MH = Mantel-Haenszel test for trend, OR = odds ratio for surgeon dissatisfaction

Table 4.22. Univariate (unadjusted) associations between explanatory variables and surgeon-rated recovery.

| Variable | p value for overall chi$^2$ (MH) | Category | OR (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Gender | 0.23 | Female | 1.00 |
| | | Male | 0.63 (0.29 – 1.35) |
| Hospital | 0.08 | Other | 1.00 |
| | | Liverpool | 0.52 (0.26 – 1.07) |
| Country of birth | 0.60 | Other | 1.00 |
| | | Australia | 0.82 (0.38 – 1.75) |
| Chronic illnesses | 0.45 | None | 1.00 |
| | | One or more | 0.75 (0.35 – 1.59) |
| Job satisfaction | 0.52 | Satisfied | 1.00 |
| | | Dissatisfied | 2.09 (0.23 – 19.3) |
| ***Injury factors*** | | | |
| Mechanism | 0.13 (0.07) | Car occupant | 1.00 |
| | | Motorcycle | 0.48 (0.22 – 1.06) |
| | | Pedestrian / cyclist | 0.44 (0.13 – 1.52) |
| Number of fractures | 0.05 | One | 1.00 |
| | | Two or more | 2.13 (1.00 – 4.57) |
| Other injuries | 0.02 | No | 1.00 |
| | | Yes | 2.40 (1.16 – 4.98) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.29 (0.28) | Primary | 1.00 |
| | | Secondary | 0.16 (0.02 – 1.65) |
| | | Certificate/Diploma | 0.17 (0.02 – 1.80) |
| | | Bachelor degree | 0.11 (0.01 – 1.33) |
| Annual income | 0.23 (0.09) | $0 – 30,000 | 1.00 |
| | | $30,000 – 50,000 | 1.01 (0.43 – 2.37) |
| | | $50,000 – 75,000 | 0.40 (0.14 – 1.14) |
| | | $75,000+ | 0.53 (0.16 – 1.69) |
| Employed (at injury) | 0.78 | No | 1.00 |
| | | Yes | 1.13 (0.49 – 2.64) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.35 | No | 1.00 |
| | | Yes | 1.47 (0.65 – 3.32) |
| Claim made | 0.05 | No | 1.00 |
| | | Yes | 2.09 (1.00 – 4.36) |
| Claim type (if made) | 0.35 | Workers comp. | 1.00 |
| | | Third party | 1.62 (0.59 – 4.45) |
| Lawyer used | 0.04 | No | 1.00 |
| | | Yes | 2.17 (1.06 – 4.47) |
| Blame | 0.35 (0.15) | Self | 1.00 |
| | | Don't know | 1.46 (0.48 – 4.47) |
| | | Someone else | 1.82 (0.81 – 4.07) |
| Fracture union | <0.0001 | No | 1.00 |
| | | Yes | 0.10 (0.03 – 0.31) |

MH = Mantel-Haenszel test for trend, OR = odds ratio for poor recovery

Table 4.23. Associations between the SF-36 summary scores and surgeon-rated satisfaction and recovery.

| Outcome | Group | Mean PCS (95% CI)* | Mean MCS (95% CI)** |
|---|---|---|---|
| Satisfaction | Satisfied | 39.4 (37.5 – 41.3) | 43.8 (41.2 – 46.3) |
| | Dissatisfied | 30.1 (26.5 – 33.6) | 39.8 (32.6 – 47.0) |
| Recovery | Good | 41.7 (39.5 – 43.9) | 45.7 (42.9 – 48.4) |
| | Poor | 31.5 (29.3 – 33.7) | 38.5 (34.2 – 42.9) |

* p value for each association with PCS was <0.0001
** for satisfaction and MCS, p = 0.3; for recovery and MCS, p = 0.005.

#### 4.3.8 Hypothesis 7: Fracture-related complications

Complications were present in 60 (42.6%) of 141 patients for whom the surgeon response was available. As described in the methods, the presence of complications was not used in the analysis as an objective measure of outcome because of the unstructured nature of the recording of complications. This allowed a large number of complications to be recorded that were considered subjective (dependent on patient complaints), for example, 24 (40%) of the complications were persistent pain and/or stiffness in the injured region. Apart from fracture non-union, the other complications that may be considered objective occurred in low numbers (nerve injury or irritation in 8 patients, infection in 4 patients, and residual deformity in 4 patients). Therefore, the presence of one or more fracture non-unions was used as the objective measure of outcome.

Data regarding fracture union was available for 140 patients: 19 (13.6%) had fractures that had not united according to their treating surgeon. Univariate analysis of each categorical explanatory variable and fracture union is given in Table 4.24. Age was not significantly associated with fracture union ($p = 0.89$). Due to the low number of fracture non-unions, and therefore small numbers in some cells, job satisfaction and education were dichotomised.

Multivariate analysis showed that none of the explanatory variables were significantly associated with fracture union.

Table 4.24. Unadjusted association between explanatory variables and fracture union (absence of non-union).

| Variable | p value for overall $chi^2$ (MH) | Category | OR (95% CI) |
|---|---|---|---|
| ***General factors*** | | | |
| Gender | 0.22 | Female | 1.00 |
| | | Male | 0.44 (0.12 – 1.61) |
| Hospital | 0.25 | Other | 1.00 |
| | | Liverpool | 1.80 (0.66 – 4.89) |
| Country of birth | 0.82 | Other | 1.00 |
| | | Australia | 0.88 (0.29 – 2.62) |
| Chronic illnesses | 0.75 | None | 1.00 |
| | | One or more | 0.85 (0.31 – 2.32) |
| Job satisfaction | 0.98 | Satisfied | 1.00 |
| | | Dissatisfied | Not estimatable |
| ***Injury factors*** | | | |
| Mechanism | 0.93 (0.70) | Car occupant | 1.00 |
| | | Motorcycle | 1.19 (0.41 – 3.44) |
| | | Pedestrian / cyclist | 1.30 (0.25 – 6.67) |
| Number of fractures | 0.19 | One | 1.00 |
| | | Two or more | 0.49 (0.16 – 1.44) |
| Other injuries | 0.48 | No | 1.00 |
| | | Yes | 0.71 (0.27 – 1.87) |
| ***Socioeconomic factors*** | | | |
| Highest education | 0.88 | Certificate or higher | 1.00 |
| | | Primary / secondary | 1.08 (0.41 – 2.85) |
| Annual income | 0.28 (0.77) | $0 – 30,000 | 1.00 |
| | | $30,000 – 50,000 | 0.32 (0.09 – 1.12) |
| | | $50,000 – 75,000 | 0.56 (0.13 – 2.42) |
| | | $75,000+ | 0.80 (0.13 – 4.76) |
| Employed (at injury) | 0.43 | No | 1.00 |
| | | Yes | 0.60 (0.16 – 2.19) |
| ***Claim-related factors*** | | | |
| Previous claim made | 0.87 | No | 1.00 |
| | | Yes | 1.10 (0.34 – 3.59) |
| Claim made | 0.25 | No | 1.00 |
| | | Yes | 0.55 (0.19 – 1.53) |
| Claim type (if made) | 0.98 | Workers comp. | 1.00 |
| | | Third party | 0.98 (0.26 – 3.75) |
| Lawyer used | 0.25 | No | 1.00 |
| | | Yes | 0.56 (0.21 – 1.50) |
| Blame | 0.98 (0.87) | Self | 1.00 |
| | | Don't know | 0.88 (0.20 – 3.91) |
| | | Someone else | 0.91 (0.31 – 2.69) |

MH = Mantel-Haenszel test for trend, OR = odds ratio for fracture union.

## 4.5 Discussion

### 4.4.1 Summary of main results

Retaining the services of a lawyer regarding an accident was strongly negatively associated with physical and mental health in patients six months after sustaining a fracture in a motor vehicle accident. Pursuit of a compensation claim was not significantly associated with general health in the same group, however there was confounding between the use of a lawyer and pursuing a claim. Both of these variables were strongly associated with physical and mental health on univariate analysis, but the effect of use of a lawyer was stronger and accounted for the effect of claim pursuit on multivariate analysis. When claim pursuit was substituted for use of a lawyer in the final models (for PCS and MCS), it was highly significant.

The type of claim (workers compensation or third party compensation) was not significantly associated with the general health outcomes. When the type of claim was included in a separate analysis, a similar final model was reached, only with claim pursuit instead of use of a lawyer, again indicating confounding between these two variables.

Apart from the age of the patient, general health was not associated with any demographic or socio-economic factors on multivariate analysis. Regarding injury severity, having more than one fracture was negatively associated with physical health, but none of the injury severity factors were associated with mental health.

As with general health, the final models for neck pain, back pain, and patient satisfaction contained only a few variables. The models for neck pain and back pain were similar, with female sex, lower education levels, and use of a lawyer being associated with increasing pain. As with the previous models, claim pursuit was only significant if use of a lawyer was excluded from the model, due to confounding between these two variables.

Patient satisfaction with progress was only associated with blame (where lower satisfaction was associated with blaming others) on multivariate analysis whereas patient-rated recovery was only associated with blame, male sex, and use of a lawyer.

Although 13.6% of patients had fractures that had not united at follow-up, fracture non-union was not significantly associated with any of the explanatory variables. While fracture non-union may have been expected to be associated with injury-severity factors, it was not expected to be associated with the demographic, socio-economic or compensation-related variables

### 4.4.2 Comments on methods

#### 4.4.2.1 Study population

Confining the population to patients with certain fractures, and to motor vehicle accidents, diminishes the effect of variation in the type of injury on the outcomes. It also decreases the chance of missed patients, because patients with injuries satisfying the inclusion criteria would normally be admitted to hospital. Some variation was expected in the types of treatment provided, and in the outcomes for the different fractures included in the study, and this may have caused some bias in the results. The sample size, however, did not allow discrimination of the effects for each fracture type.

No record of refusals was kept, and considerable variation in response between hospitals was found. This was felt to be due to differences in the attitude of the recruiting orthopaedic registrars at each institution, as the recruitment rate changed for each hospital, as new registrars rotated through every six months. It is likely that there were differences in the approach of the registrars to potential patients, and that this explained the differences in the recruitment rates. Although each hospital employed several orthopaedic registrars, the job of patient recruitment was usually left to only one, thus increasing the variation between hospitals due to differences in the attitude of each registrar to the study.

Follow-up was aimed at a single time-point, six months, but difficulties in contacting some patients, and late returns from some patients, resulted in some follow-up times as long as 12 months. Although this may introduce some bias, differences in follow-up time were not considered significant enough to justify inclusion of this factor as another explanatory variable.

While the patient response rate was close to the rate expected, the surgeon response rate was poor. This was partly due to poor responses from the surgeons, and partly due to patients not seeing their treating doctors after the initial post-injury period. The poor response rate from the surgeons affected the validity of the results that relied on these responses, in particular, the complication rate and the measures of surgeon and patient satisfaction. It did not, however, affect the main outcome measures, which were patient-based. Nor did it affect the main explanatory variables, which were measured at the time of injury, and in the six-month patient questionnaire.

A comparison of responding to non-responding patients showed that non-responding patients were more likely to be younger, female, and have a lower education level. These associations were not significant, but may be explained by confounding from the only significant factor associated with a lack of response: a low income level. The poorer response in patients with low income may be due to difficulties contacting this group of patients (possibly due to address changes), or because this group were less likely to respond when contacted.

In contrast to predictors of patient response, the only significant patient factors associated with lack of surgeon response were higher income and attending a hospital other than Liverpool Hospital. The strong association between surgeon response and attendance at Liverpool Hospital can be explained by Liverpool Hospital being the coordinating centre for the study, consequently all of the orthopaedic registrars and consultants were familiar with the study, patients and surgeons could be tracked more easily, and it was easier to return the forms as the surgeons worked in the same building that was used as the study centre. The less significant association with income is probably due to confounding from this factor, as Liverpool is in a relatively low socio-economic area.

Although the high response rate in the patient follow-up is expected to diminish any selection bias, the low response rate for surgeons means that selection bias is more likely. However, apart from the treating hospital and the income level, there was no significant difference between responders and non-responders for any other variables, particularly the main explanatory variables regarding blame, claim pursuit, claim type, and use of a lawyer.

#### 4.4.2.2 Explanatory variables

The majority of the explanatory variables used were trialed in a pilot study of the questionnaire, and the validity of these variables is discussed in the methods section of this chapter and in Chapter Three. Variables that were not used in the study in Chapter Three are discussed below.

Contraction of the treating hospital into a dichotomous variable was justified because the variable was not significantly associated with any of the outcomes, even though the contraction would have resulted in some loss of information. Similarly, the country of birth was converted to a dichotomous variable, but was not found to be a significant factor in any of the analyses.

The responses to the question on job satisfaction were biased towards the first two responses (indicating satisfaction), but a valid statistical association was still possible for the major outcome variables. The validity of this question is discussed above under Methods.

When measuring the mechanism of injury, pedestrians and bicyclists were grouped together. This was necessary because of the low numbers in these groups, but was considered reasonable, as pedestrians and bicyclists would be expected to be subjected to similar forces, and the mechanism of injury (direct impact on the body) is similar.

The measures of injury severity (number of fractures and presence of other injuries) were considered appropriate for this population, as an increasing number of fractures implies an increasing energy of impact. The presence of fracture non-union was also used as a measure of injury severity, as this has previously been shown to be strongly related to high energy injuries.[457-459]

Regarding the type of claim pursued, analysis was confined to comparing patients claiming under workers compensation, to those claiming under third party insurance. While this decreased the number of patients included in the analysis, it allowed a valid comparison of the two main compensation systems: one fault-based and one no-fault system. Including the small number of patients claiming under “other” compensation schemes would have made analysis more difficult and would not have offered any further information without knowing what the “other” schemes were.

The poor surgeon response resulted in difficulties with the multivariate analyse for these outcomes. This was overcome by grouping several of the four-part variables as dichotomous, although this resulted in some loss of definition regarding the effect of different levels of the variables.

#### 4.4.2.3 Outcome variables

The main outcomes, PCS and MCS, are widely used measures of general health and, along with neck pain, back pain and patient satisfaction, have been discussed in Chapter Three.

Patient assessment of recovery after the injury was expected to provide a more objective opinion of the patients’ health status at the time, rather than “satisfaction with progress” which was expected to be influenced by the processes related to their injury. Patient satisfaction and patient-rated recovery were highly associated with the PCS and MCS scores, indicating that both measures are strongly influenced by health status. However, whereas the PCS was associated with gender and injury severity (number of fractures), patient satisfaction and patient-rated recovery were not associated with gender or injury severity. Interestingly, both patient satisfaction and patient-rated recovery were strongly associated with blaming others for the injury, a factor that was not associated with PCS and MCS on multivariate analysis.

The presence of complications and fracture union were included, not as predictors of other outcomes (although this was examined to a limited extent), but as objective measures of outcome that would provide a comparison for the patient-based outcomes. The presence of complications, however, was non-specific and was influenced by patient complaints (and was therefore less objective). This is supported by an observation of the responses for this outcome, which shows that the most common complication was complaints of pain and stiffness in the affected joints, rather than infections or the need for further surgery. The presence of fracture union (or non-union) was considered to be more objective, and this may explain the lack of association between fracture union and any of the possible predictors, which are mainly demographic and socio-economic factors.

#### 4.4.2.4 Statistical analysis

The statistical analysis was similar to that performed in Chapter Three. The assumptions of the models were satisfied, and meaningful associations for patient-based outcomes could be derived. The low surgeon response rate, however, made modelling for surgeon outcomes, and use of the presence of complications as a predictor, difficult.

### 4.4.3 Interpretation of the results

#### 4.4.3.1 Physical health

In the multivariate analysis, physical health was associated with age, gender, number of fractures, and use of a lawyer. The decrease in the PCS score with increasing age, and the higher scores in males, are consistent with Australian population norms.[433] Also, the lower mean PCS in patients with more than one fracture is biologically plausible and likely to be a true effect. It also validates the use of this variable as a measure of injury severity.

Physical health was not significantly associated with claim pursuit on multivariate analysis despite being strongly associated in the univariate analysis, and therefore the main hypothesis is rejected. However, physical health was strongly associated with the use of a lawyer, and it is the confounding between these two variables that led to claim pursuit being rejected from the final model, as claim pursuit is strongly associated with PCS if use of a lawyer is removed from the model, and claim pursuit and use of a lawyer were associated with each other (76.5% agreement).

Although there was confounding between these two variables, both of which were strongly associated with PCS in the univariate analysis, there was some evidence of an independent effect of claim pursuit, as this was the final predictor removed in the modelling process. Therefore, it is possible that the effect of claim pursuit may have been statistically significant (allowing for legal involvement) if the sample size was larger, as was seen in Chapter Three.

Although determinants of general health after motor vehicle accidents have been previously studied,[159 189 399 460] these studies have not examined the influence of compensation-related factors. The majority of the previous studies on the effect of compensation-related factors on outcome after motor vehicle accidents have used psychological outcomes (e.g., PTSD) or neck pain as their main outcome, rather than measures of general health. These studies are discussed elsewhere.

#### 4.4.3.2 Mental health

As with PCS, claim pursuit was significantly associated with MCS in the univariate analysis, this association was not significant on multivariate analysis, and a strong association was seen between use of a lawyer and MCS. Claim pursuit became significant if use of a lawyer was removed from the model, indicating confounding between these variables, but it appears that the effect of claim pursuit seen in the univariate analysis was due to this confounding and, therefore, the hypothesis that MCS is related to claim pursuit is rejected.

However, the strong association between MCS and the use of a lawyer indicates that MCS is related to at least one aspect of the compensation process. The stronger association seen with legal involvement, rather than claim pursuit, still indicates that factors related to compensation may influence the outcome. It appears that legal involvement is a more important predictor than claim pursuit; this is discussed below under Implication of the Results (4.5.5).

Previous studies of outcome after motor vehicle accidents have concentrated on psychological outcomes such as PTSD, rather than measures of general health. Therefore, there are no comparable studies on which to comment regarding this outcome.

#### 4.4.3.3 Neck pain

Claim pursuit was the variable with the strongest association with neck pain in the univariate analysis. In the multivariate analysis, claim pursuit was associated (independently of lawyer involvement and the other variables in the final model) with a lower neck pain score but was discarded from the final model with a p value of 0.08. It is likely that the effect of claim pursuit may have been significant if the sample size was larger.

The effect of claim pursuit is supported by the alternative analysis using fracture union in the model. In this model, claim pursuit was significant and use of a lawyer was discarded as not significant. This indicates confounding between these variables, as seen in other analyses in Chapter Three.

The hypothesis relating claim pursuit to neck pain is rejected, and the first model is accepted, because although inclusion of fracture union resulted in a stronger final model, the modelling process was considered less stable, as the sample size was significantly reduced (140) when fracture union was included in the analysis. However, the significant association between legal involvement and neck pain indicates that some aspect of the claim process may be significant.

The increase in reported neck pain associated with female gender, lower education level, and use of a lawyer found in this study was also found in the study in Chapter Three and, as discussed in that chapter, these findings are supported by previous studies, most of which are studies of neck pain specifically after motor vehicle accidents.

Although previous research has shown differences in neck pain between groups treated in a fault-based versus a no-fault compensation system,[117 461] there was no difference found between these two systems in this study. This may reflect differences in methodology, as the previous reports both explored differences before and after legislative change within one system, whereas this study explored differences between two different systems simultaneously. The previous reports, however, were larger, indicating that this study may have been underpowered to detect a difference between the two systems, but this is unlikely, as the type of claim (fault-based versus no-fault) was not associated with neck pain in the univariate analysis ($p = 0.9$).

Although this study did not specifically include patients who complained of neck pain or had neck injuries, neck pain is common after motor vehicle accidents, and therefore the inclusion criteria and study design are considered reasonable to allow conclusions regarding possible predictors of neck pain. The tool used to measure neck pain (and back pain) in this study has not been previously used, but it has face validity and was shown to be reliable during the pilot study of the questionnaire.

No control group was used to examine the magnitude of neck pain in patients not involved in motor vehicle accidents, but this is not expected to affect the validity of the study, as the contribution to neck pain from underlying or pre-existing symptoms would only bias the effect of any possible predictor towards the null. That significant predictors were found diminishes this criticism.

This study has advantages over previous studies of the effect of compensation on neck pain after motor vehicle accidents, as previous studies have been limited by only including legal involvement or litigation as the compensation-related predictor (rather than claim pursuit),[93 125 306 407 462 463] or by only including claimants in the study population.[107 117 124 138 462 464]

Another advantage of this study over previous research is the large number of potential predictors and confounders used, particularly with respect to compensation-related factors. The methodology and results of studies of neck pain after motor vehicle accidents are discussed in more detail in Chapters One and Three.

#### 4.4.3.4 Back pain

As with the previous outcomes, claim pursuit was not associated with back pain, but use of a lawyer was a strong predictor. Therefore, the hypothesis regarding the effect of claim pursuit on back pain is rejected.

Unlike neck pain, the predictors of back pain after motor vehicle accidents have not been widely studied. In a similar study to the whiplash study performed by the same authors,[117] Cassidy et al demonstrated a decrease in the incidence of claims for back pain after the compensation system was changed from a tort to a no-fault system, eliminating payment for pain and suffering.[68] The primary outcome in the study by Cassidy et al was time to case closure, and this was also increased by lawyer involvement and female gender, both of which were also significant in this study. Unfortunately, the study by Cassidy et al only included claimants, and it can be criticised for using the time to case closure as the main outcome, rather than the incidence or severity of back pain.

The predictors of back pain in this study were similar to those for neck pain (gender, use of a lawyer, and education level). This is consistent with two large surveys of motor vehicle accident claimants that showed a high incidence of back pain in patients complaining of whiplash after motor vehicle accidents.[159 465] Similarities in predictors for back pain and neck pain after motor vehicle accidents is also implied by the parallel studies performed by Cassidy et al, referred to earlier, in which the incidence of neck pain and back pain claims fell after a change in the compensation system in Saskatchewan, Canada.[68 117]

This study only included patients with fractures of the extremities, and did not take into consideration any back injury. This lack of specificity for back injuries would be expected to limit the power to detect predictors of back pain. This means that the study may not have measured injury severity accurately enough when back pain was used as the outcome. However, the possible lack of representation of patients with back injuries would be expected to bias the association between any possible predictors and back pain towards having no effect. The presence then, of significant predictors in the final model, indicates that these factors are strong predictors that are probably unrelated to the nature of the accident.

#### 4.4.3.5 Patient-rated satisfaction and recovery

The results of this study highlight the importance of blame in how patients perceive their progress and recovery after an injury.

Interestingly, blame was the strongest predictor of these two outcomes, and it is likely that confounding from this variable resulted in the loss of statistical significance for claim pursuit that was seen in the univariate analysis. As claim pursuit was not significant in either model, the hypothesis regarding the effect of claim pursuit on patient-rated satisfaction and recovery is rejected.

The associations between the presence of fracture non-union and poor ratings of satisfaction and recovery indicate that these ratings are influenced by the presence of complications, but to a lesser extent than blame.

There is no relevant literature regarding these outcome variables as they were only developed for this study. While this represents a weakness of this study, by not allowing direct comparison with similar studies, it does broaden the scope of the outcomes used.

Although the patient ratings of satisfaction with progress and recovery were highly correlated with the main outcome (the SF-36 summary scores), blame was not a significant predictor of these scores. This indicates that whereas blame may not significantly predict general health, it is a strong predictor of how happy patients are with their condition (regardless of how good or bad their condition may be).

The role of blame in patient perception of outcome after injury has been studied previously, consistently showing an association between blaming others (compared to blaming oneself) and a poor psychological outcome.[296-298 466] In that patient satisfaction may be considered a psychological outcome, this study supports these previous investigations.

#### 4.4.3.6 Surgeon-rated satisfaction and recovery

As expected, surgeon ratings of satisfaction and recovery were not associated with claim pursuit, so this hypothesis is accepted. The only significant predictor of these ratings was the presence of fracture non-union, indicating that ratings given by surgeons are more likely to be based on objective outcomes than ratings given by patients. The surgeons' perception of pain and disability were not recorded. Although these variables may have influenced surgeon satisfaction, they were not considered valid measures of patient-reported health.

As with patient-rated satisfaction and recovery, there is no directly comparable literature regarding this outcome. While the lack of a standard for this outcome may be a criticism, it was included only as a comparator to patient ratings.

#### 4.4.3.7 Fracture-related complications

As expected, fracture non-union was not predicted by any of the variables measured in this study. Therefore the hypothesis stating that objective fracture-related outcomes would not be associated with claim pursuit is accepted.

Factors influencing the rates of non-union after fractures received in motor vehicle accidents have been studied previously, however, the role of compensation related factors has not. However, as with surgeon-rated satisfaction, this outcome was only used in this study as an objective outcome measure to compare to the patient-based main outcome variables. The lack of association between this objective measure of outcome and the predictors of the patient-based outcomes highlights the subjective nature of these outcomes, and the importance of psychosocial factors. It also addresses the concern that the differences seen in the patient-based outcomes were due to differences in objective outcomes (such as fracture non-union).

The smaller sample size in the analysis of fracture union decreases the power of that analysis to find significant predictors, but the lack of any significant association on univariate analysis indicates that no strong predictor would have been found, even with a larger sample size.

### 4.4.4 Generalisability

By restricting the inclusion criteria to certain fractures and to motor vehicle trauma, the influence of the type of injury on any outcome has been controlled. However, narrowing the inclusion criteria affects generalisability, and it may be that the associations shown in this study are not applicable to other injuries, for example, head injuries or workplace (industrial) injuries. The similarity of the findings to those found in Chapter Three, which included a broad range of injuries, indicates that these results may be generalisable to populations with other injuries.

Similarly, although the results may not be valid for patients with longer follow-up, the similarity with the findings in Chapter Three would indicate that the results might not be time-sensitive.

Extrapolation of the results of this study outside the state of New South Wales should take into account the jurisdictional differences in compensation and insurance systems.

### 4.4.5 Implications of the results

Regarding the effect of claiming compensation on trauma outcomes, while this study did not show a significant association, it is likely that aspects of the process are still significant. Significant confounding was noted between lawyer use and claiming compensation, and although it appeared that each variable had an independent effect on several of the outcomes, the sample size may not have been large enough to find a significant independent effect of claiming compensation, which was found in the study reported in Chapter Three.

The consistent association between the use of a lawyer and poor outcome implies that some aspect of the legal involvement may be causing increased symptom reporting in patients, regardless of whether or not they pursued compensation. Although the association may be due to confounding from factors such as injury severity and presence of complications, the most objective measures of these factors (the number of fractures, and the presence of fracture non-union at six months) did not support this. In fact, there was a trend towards an association between lawyer use and fracture union, rather than non-union.

The possible mechanisms of this association have been discussed in Chapter Three, and include: the direction of patients by lawyers, regarding symptom reporting, the need to repeatedly report and consider their symptoms (for medical reports), and a higher likelihood of involvement in an adversarial environment.

The negative association between blaming others (compared to blaming oneself) and patient-ratings of satisfaction and recovery demonstrate the importance of such psychological factors in these outcomes, and that, while blame may not influence measures of general health (PCS and MCS), it may be important to the patients' well being. The association between blame and outcome after injury has been reported previously and the mechanism is likely to be related to the psychology of victimisation, retribution, and coping mechanisms.[72 110 295-297] The association, however, is also related to compensation, particularly in the fault-based, third party system, and confounding between these two variables is likely to explain why blame became a less significant predictor on multivariate analysis for most outcomes.

The difference between patient satisfaction and general health (although both were strongly associated) implies that studies relying on measures of general health after trauma may be too narrowly focussed if their aim is to measure patient wellbeing.

### 4.4.6 Significance to future research

Use of a lawyer, pursuit of a claim, and blame were all strongly associated with aspects of health after fracture, but due to confounding between these variables, the true effect of each was difficult to determine, as each one of the variables was significantly associated with at least one outcome, depending on the analysis performed. Future research should aim to distinguish between these variables by having a larger sample size.

Although the use of a lawyer was associated with poor outcome, this study provides no information regarding what aspect of the legal involvement may be responsible for this effect. For example, legal involvement may have increased the time involved in the claim process, symptoms may have changed after legal contact, and differing degrees of legal involvement may also influence the effect of this variable. Also, other aspects of the claim process should be considered in order to further define which aspects of the claim process best predict outcome. These other factors include the (financial) size of the claim and settlement, the timing of settlement, differences between insurance companies (e.g., liability acceptance policies, and claim processing procedures), and the degree of dispute or conflict present in the process. Other social factors, such as marital status, should also be considered. Future, larger studies may be able to include these other variables.

The association between blame and some of the outcomes indicates the importance of this psychological factor, and future research may need to include other psychological factors in trauma outcomes. Furthermore, the importance of patient satisfaction should be explored further, as this or similar factors may be important to patients after traumatic injuries. In particular, the relative importance of such factors and measures of general health could be explored.

In this study, different predictors were noted for patient-based reporting of satisfaction and recovery compared to surgeon-based reporting. Also, surgeons rated the results more positively than patients. While this difference was not explored as it lies outside the scope of this thesis, it may be useful to explore this difference further in future research.

Road trauma is a significant cause of the burden of disease, particularly in the developed world and particularly in young people. The role of factors that strongly predict poor outcome after road trauma should be studied further so that the morbidity associated with these injuries can be reduced.

#### 4.4.7 Significance of work

This study provides useful information regarding predictors of health after road trauma. It implicates factors associated with blame and the compensation process in the poor outcomes often seen after these injuries, and reinforces other research in highlighting the importance of these factors.

The findings indicate that consideration should be given to compensation-related factors when assessing the outcome after road trauma. They also provide information that may lead to correcting factors within the compensation process that may lead to poor outcomes in this population. These factors may include the role of fault in determining compensation, and the adversarial nature of the compensation process.

## 4.5 Conclusion

In patients sustaining a major fracture in a motor vehicle accident, pursuit of compensation, per se, was not associated with poor general health after six months, whereas the use of a lawyer was a strong negative predictor of health for the same group. However, separation of these two variables may explain the association between compensation and poor outcome reported in other studies, as these two variables are highly correlated.

The study hypothesis, that general health would be significantly poorer in patients pursuing compensation is rejected.

# CHAPTER FIVE. SUMMARY AND CONCLUSIONS

## 5.1 A comparison of the three main studies

The systematic review reported in Chapter Two provided a detailed summary of published studies pertaining to the effect of compensation status on outcome after surgery. This is related to outcome after injury, as the patients in these studies were undergoing surgery for injury. Apart from including a mixture of compensation types and surgical conditions, the review also highlighted problems with the previous studies in that the definition of injury was not clear. Outcomes also varied between studies, and these differences produced difficulties in providing summary estimates for the studies as a whole. These same difficulties exist when attempting to compare the systematic review (Chapter Two) to the two other studies included in this thesis (Chapters Three and Four). Indeed, the methodological issues noted in the studies included in the systematic review, as well as in the studies discussed in the literature review in Chapter One, formed the basis for the design of the studies in Chapters Three and Four.

With these limitations in mind, there are still similarities in the findings of the three main studies in this thesis. The main similarity is the consistent finding that factors related to the compensation process, in particular claim pursuit and lawyer involvement, are associated with poor outcome after injury, whether it be measured after treatment for the injury, short term, or long term, and regardless of the type of outcome measured.

Another consistent finding was that there was no evidence that the type of claim influenced the effect. Analysis of this variable was restricted to the sub group of claimants, therefore reducing the sample size, but both of the surveys included significant proportions of workers compensation and third party patients, so that any difference, if present, should have been detected. These results indicate that the claim process contributes to poor outcomes independent of type of compensation system.

In both studies, the PCS was associated with a measure of injury severity, whereas the MCS was not. This association with PCS is expected, but the lack of association between injury severity and MCS, as well as the other non-physical outcomes (PTSD and patient satisfaction) is of interest. Most of the effort in treating trauma patients is directed toward restoration of physical function. The assumption when restoring function is that improvements in physical function will cause improvements in mental well-being. While this may be the case, it appears that there are stronger predictors of mental health that are not affected by physical factors.

Female gender, use of a lawyer, and lower education levels were predictive of neck pain and back pain in both studies. Consistency between these two studies, along with support from previous studies, strengthens the findings. Whether lower education level and female gender influence reporting directly, or whether confounding exists (due, for example, to differences in work conditions) cannot be determined from this study, although both studies allowed for many possible confounders.

Similarly, patients with neck pain or back pain may be more likely to use a lawyer. However, complaints of neck or back pain were not associated with any of the indicators of injury severity, and use of a lawyer was highly associated with claim pursuit. Further, the possibility that patients with greater injuries were more likely to pursue claims is also not supported, as (in the Major Trauma Outcome Study) claim entitlement was a stronger predictor of general health than claim pursuit, indicating that the effect on outcome is not influenced by any factors related to the conscious decision to pursue a claim.

It is also of interest to note that none of the patients in the studies were selected on the basis of having a neck or back injury, and there is evidence that those who complain of neck and back pain after an accident are more likely to complain of other, more systemic complaints.[159 465] The association between legal involvement and neck and back symptoms may, therefore, be associated with increased symptom reporting in general, rather than physical injury to the spine. This may also explain the association between legal involvement and the other patient-based outcomes. The possible explanations for the association between increased symptom reporting and legal involvement include: encouraging the reporting of symptoms to maximise settlements, the need to repeatedly report symptoms for medicolegal reports and consultations, and increased and extended exposure to an adversarial environment. It is also likely that the increased symptom reporting in these patients is not consciously controlled and may represent somatisation: the presentation of physical symptoms (in the form of common and definable conditions, such as whiplash) to legitimise or express psychological stress or discomfort. Put in other words, we may be seeing the expression of an unpleasant experience (the compensation process) as pain (which is, in any case, defined as an unpleasant experience). The form that the symptoms take may be influenced by such factors as social acceptability, symptom expectation, or suggestions from doctors or lawyers.

The differences between the two clinical studies provide more information regarding the role of compensation-related factors in determining outcome after injury. In particular, the main difference between the two studies was that claim pursuit was associated with many of the outcomes in the Major Trauma Outcome Study, whereas the use of a lawyer was more strongly associated with the same outcomes in the Motor Vehicle Accident Outcome Study. Dissection of these differences shows that the associations with PCS were similar, where there was an independent association with claim pursuit and use of a lawyer, but that the association with use of a lawyer was stronger. In the second study, claim pursuit did not reach statistical significance but the effect was similar and may have reached significance with a similar sample size. The main differences between the studies was in the predictors of MCS, where the Major Trauma Outcome Study found an association with claim settlement, and the Motor Vehicle Accident Outcome Study found an association with use of a lawyer, claim pursuit not being significant. This can be explained by the difference in the variables used, as the former study divided claim pursuit into those that had settled, and those that had not. While this was not practical in the latter study, as the follow-up period was too short to allow settlement, it indicates, as suggested before, that the claim process may be the main predictor of the poor outcome, not whether or not a claim was made. A settled claim is less likely to be a disputed claim and may also be associated with less legal involvement. It is possible that legal involvement and having an unsettled claim are measuring the same thing: prolonged involvement with a stressful, adversarial dispute. The fact that these variables (an unsettled claim and legal involvement) have stronger effects on measures of pain and mental health, than on physical health, supports the hypothesis that these factors act at a psychological level.

Other differences between the two studies may explain the differences in significant predictors found, particularly the confounding between the use of a lawyer and involvement in the claims process. The Major Trauma Outcome Study had much longer follow-up, and therefore the patients were more likely to have already been involved in the claims process, whereas those in the second study would have only had limited involvement in the claims process. As legal involvement was a much stronger predictor than claim pursuit in the second study, it may be that legal involvement is a significant factor leading to poor outcome, and may have led to the poor outcome seen with claim pursuit in the Major Trauma Outcome Study. This can be explained if the involvement of a lawyer increases the likelihood of becoming involved in the claims process, or having a longer or a more adversarial involvement.

Although there is confounding between the use of a lawyer and involvement in the claims process, at least for some major outcomes, both variables have an independent effect. The third variable that confounded with these was blame.

In both studies, blame was significant for all outcomes on univariate analysis but was discarded from the models on multivariate analysis. Blame was a strong predictor of patient-rated satisfaction and recovery in the Motor Vehicle Accident Outcome Study, and was a significant predictor of PTSD in the Major Trauma Outcome Study. However, in all other analyses, it was not significant due to confounding from claim pursuit and use of a lawyer. Although patients who blame others may be more likely to pursue claims and use lawyers, the effect of these latter two factors appears to be much stronger for the major outcomes measured.

There are two other differences between the two main studies that deserve comment. The presence of chronic illnesses was a significant predictor of most outcomes in the first study, but not significant in the other. This is likely to be due to the difference in follow-up time between the two studies. The Major Trauma Outcome Study had much longer follow-up and the effect of chronic illnesses would be expected to become more significant with time. Also, the Major Trauma Outcome Study had a higher mean age (47 versus 38 years), so that chronic illnesses were more common, and were therefore more likely to reach significance for any association.

Current employment was a significant predictor in the first study but not in the second. Again, this is likely to be due to the difference in follow-up times, as most of the patients in the second study were likely to be still in employment, although possibly on sick leave.

When comparing the two main clinical studies, apart from differences that may be due to differences in methodology, the findings were similar in that involvement in the claims process, whether through legal involvement, claim pursuit, or ongoing settlement processes, was a strong and significant predictor of all major outcomes, and, although there was confounding between the compensation-related factors, in many cases these factors had an effect that was independent of the others.

The presence of an independent effect for the claim-related variables in some of the final models is evidence against collinearity, but in models that contained only one compensation-related variable, the level of confounding raises issues of collinearity between these variables. Collinearity was not thought to be present, however, as the variance inflation factors for all variables in all models were less than 2.5, considerably lower than 10 which is considered evidence of collinearity. The differences between the compensation-related variables in the studies are shown in Appendix 16.

## 5.2 Summary

Three separate studies were performed to explore the role of compensation in determining outcomes after injury. In each study, the systematic review, the retrospective study of major trauma patients, and the prospective study of patients with fractures resulting from motor vehicle accidents, consistent and strong associations were shown between compensation-related factors and poor outcomes such as general health, pain, PTSD, and surgery-specific scores. The possible mechanisms for these associations include: the expression of psychological stresses incurred as part of the compensation process as symptoms (such as pain and loss of function), increased exposure to lawyers and doctors who may reinforce or guide symptom reporting, or other psychological mechanisms, perhaps relating to blame. Alternatively, the effect may not be true, it may be due to confounding from other factors not measured in these studies, or due to sampling biases within the studies. However, the consistency and strength of the findings, as well as the allowance for multiple possible confounders, minimises this possibility.

The findings of these studies add to the body of knowledge regarding the role of compensation in influencing outcomes after injury. The findings represent new knowledge as they were the result of well-controlled studies, they used validated general health outcomes, and they explored aspects of the compensation process in more detail than previous studies.

Modifications of current compensation systems could be made, based on finding from this research. Possible changes to the claims process based on this research would include: the limitation of legal involvement (both in the form of lawyer presence, and use of the courts); limiting or removing compensation for specific conditions such as non-specific neck pain; shortening the time to settlement; and removing areas of contention by having more rigid guidelines and limiting the ability to challenge decisions.

Further research in this area should be directed at the mechanisms by which the association may be mediated. For example, any modifications made to the claims process should be studied to monitor their effect on patient outcomes.

This research also highlights the importance of psychological factors in determining outcome after injury and it is hoped that this study will increase the awareness of these issues in trauma patients. Further research exploring the psychological aspects of the compensation process may be helpful in predicting outcomes in injured patients. Also, more attention to psychological well being after injury may allow the identification of patients at risk of poor outcomes, and this may allow earlier and more effective interventions.

Improving the education of injured patients regarding aspects of the claims process may also improve outcomes, by adjusting expectations and minimising the stress associated with dealing with an unfamiliar and complex system. Apart from health care workers, this research is also applicable to any individuals involved in the administration of compensation systems, and the insurance industry. Increasing the awareness of potential problems associated with the claims process among those involved with managing injured patients may also lead to better patient (and claim) management.

## 5.3 Conclusion

This research indicates that aspects of the compensation and legal systems may be harmful to injured people, and further elucidation of these aspects may result in systems that do not add to the harm already present from the injury, while still managing them fairly and appropriately.

## REFERENCES


1. Marcus EH. Compensation payments - blessing or curse? ***Medical Trial Technique Quarterly*** 1983;29:319-23.

2. Hadler NM. If you have to prove you are ill, you can't get well. The object lesson of fibromyalgia. ***Spine*** 1996;21(20):2397-400.

3. ***The Macquarie Dictionary***. 4th ed. Sydney: Macquarie Library, 2005.

4. Mendelson G. Chronic pain, compensation and clinical knowledge. ***Theoretical Medicine*** 1991;12(3):227-46.

5. ***Diagnostic and statistical manual of mental disorders***. 4th ed. Washington, DC: American Psychiatric Association, 1994.

6. Tennant C. Psychological trauma: psychiatry and the law in conflict. ***Australian and New Zealand Journal of Psychiatry*** 2004;38:344-7.

7. Summerfield D. The invention of post-traumatic stress disorder and the social usefulness of a psychiatric category. ***BMJ*** 2001;322(7278):95-98.

8. Marx RG, Jones EC, Angel M, Wickiewicz TL, Warren RF. Beliefs and attitudes of members of the American Academy of Orthopaedic Surgeons regarding the treatment of anterior cruciate ligament injury. ***Arthroscopy*** 2003;19(7):762-70.

9. Workcover NSW Annual Report 2004/2005. Sydney: Workcover NSW, 2005.

10. The Motor Accidents Authority of NSW Annual Report 2004-05. Sydney: NSW Government, 2005.

11. Moyes G. Personal injury compensation legislation. In: 1 GPSCN, editor. Sydney: New South Wales Parliament, 2005.

12. Callinan R. The Future of the New South Wales Workers' Compensation Scheme: Parliament of New South Wales, 2001.

13. Hadler NM. ***Occupational musculoskeletal disorders***. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.

14. Compensable injuries and health outcomes. Sydney: The Australasian Faculty of Occupational Medicine and The Royal Australasian College of Physicians, 2001:40.

15. Lafosse FGL. Histoire de la cicatrisation, de ses modes de formation, et des considerations pathologiques et therapeutiques qui en decoulent. Montpellier, 1836.

16. Beecher HK. Relationship of significance of wound to pain experienced. ***JAMA*** 1956;161(17):1609-13.

17. Strong CA. The psychology of pain. ***The Psychological Review*** 1895;2(4):329-47.

18. Millen FJ. Post-traumatic neurosis in industry. ***Industrial Medicine & Surgery*** 1966;35(11):929-35.

19. Trimble MR. ***Post-traumatic neurosis: from railway spine to the whiplash***. New York: John Wiley & Sons, 1981.

20. Kennedy F. The mind of the injured worker. Its effect on disability periods. ***Compensation Medicine*** 1946;1:19-24.

21. Eisenberg L. Disease and illness. Distinctions between professional and popular ideas of sickness. ***Culture, Medicine & Psychiatry*** 1977;1(1):9-23.

22. Summerfield D. ICD and DSM are contemporary cultural documents. ***BMJ*** 2002;324(7342):914.

23. Anonymous. Railway injuries: a medico-legal subject. ***The Lancet*** 1861:255-6.

24. Cohen ML, Quinter JL. The derailment of railway spine: a timely lesson for post-traumatic fibromyalgia syndrome. ***Pain Reviews*** 1996;3:181-202.

25. Harrington R. The railway accident: trains, trauma and technological crisis in nineteenth century Britain. York: University of York, Institute of Railway Studies, 2004.

26. Gowers WR. ***A manual of diseases of the nervous system***. 2nd ed. London: J & A Churchill, 1892.

27. Dembe AE. ***Occupation and disease. How social factors affect the conception of work-related disorders***. New Haven: Yale University Press, 1996.

28. Ireland DC. Repetition strain injury: the Australian experience--1992 update. ***Journal of Hand Surgery - American Volume*** 1995;20(3 Pt 2):S53-6.

29. Figlio K. How does illness mediate social relations? Workmen's compensation and medico-legal practices, 1890-1940. In: Wright P, Treacher A, editors. ***The problem of knowledge Examining the social construction of medicine***. Edinburgh: Edinburgh University Press, 1982:173-224.

30. Hadler NM. The disabling backache. An international perspective. ***Spine*** 1995;20(6):640-9.

31. Lucire Y. ***Constructing RSI. Belief and desire.*** Sydney: University of New South Wales Press Ltd, 2003.

32. Bell DS. "Repetition strain injury": an iatrogenic epidemic of simulated injury. ***Medical Journal of Australia*** 1989;151(5):280-4.

33. Ferguson DA. "RSI": putting the epidemic to rest. ***Medical Journal of Australia*** 1987;147(5):213-4.

34. Cleland LG. "RSI": a model of social iatrogenesis. ***Medical Journal of Australia*** 1987;147(5):236, 238-9.

35. Cohen ML, Arroyo JF, Champion GD, Browne CD. In search of the pathogenesis of refractory cervicobrachial pain syndrome. A deconstruction of the RSI phenomenon. ***Medical Journal of Australia*** 1992;156(6):432-6.

36. Gun RT. The incidence and distribution of RSI in South Australia 1980-81 to 1986-87. ***Medical Journal of Australia*** 1990;153(7):376-80.

37. Hayes JG. RSI, whiplash and cervicobrachial pain. ***Medical Journal of Australia*** 1992;157(3):212.

38. Mee B. Insurance claim evidence for growth of RSI. ***Occupational Health*** 1989;41(5):138.

39. Reid J, Reynolds L. Requiem for RSI: the explanation and control of an occupational epidemic. ***Medical Anthropology Quarterly*** 1990;4:162-90.

40. Lucire Y. Social iatrogenesis of the Australian disease 'RSI'. ***Community Health Studies*** 1988;12(2):146-50.

41. Quintner JL. The Australian RSI debate: stereotyping and medicine. ***Disability & Rehabilitation*** 1995;17(5):256-62.

42. Lam SJ. Repetitive strain injury (RSI) or cumulative trauma disorder (CTD) as legal and clinical entities. The need for a total reappraisal of this concept as being occupationally caused and therefore compensatable. ***Medicine, Science & the Law*** 1995;35(4):279-86.

43. Quintner J. The "RSI" syndrome in historical perspective. ***Occasional Papers on Medical History Australia*** 1991;5:49-55.

44. Willis E. RSI as a social process. ***Community Health Studies*** 1986;10(2):210-9.

45. Horin A. Hi-tech epidemic. Victims of a bright new technology that maims. ***The National Times*** 1984 Octboer 12;15-18.

46. Walker J. Tenosynovitis. A crippling new epidemic in industry. ***New Doctor*** 1979;13:19-21.

47. Chapman SL, Brena SF. Pain and litigation. In: Wall PD, Melzack R, editors. ***Textbook of pain***. 2nd ed. Edinburgh: Churchill Livingstone, 1989:1032-41.

48. Mendelson G. Chronic pain and compensation: a review. ***Journal of Pain and Symptom Management*** 1986;1(3):135-44.

49. Mendelson G. Chronic pain and compensation issues. In: Wall PD, Melzack R, editors. ***Textbook of pain***. 3rd ed. Edinburgh: Churchill Livingstone, 1994:1387-1400.

50. Dworkin RH. Compensation in chronic pain patients: cause or consequence? ***Pain*** 1990;43(3):387-8.

51. Dworkin RH, Handlin DS, Richlin DM, Brand L, Vannucci C. Unraveling the effects of compensation, litigation, and employment on treatment response in chronic pain. ***Pain*** 1985;23(1):49-59.

52. Talo S, Hendler N, Brodie J. Effects of active and completed litigation on treatment results: workers' compensation patients compared with other litigation patients. ***Journal of Occupational Medicine*** 1989;31(3):265-9.

53. Waddell G. Pain as a biological and social phenomenon. ***World Congress on Medicine and Health*** 2000.

54. Andersson GB. Epidemiologic aspects on low-back pain in industry. ***Spine*** 1981;6(1):53-60.

55. Deyo RA, Cherkin D, Conrad D, Volinn E. Cost, controversy, crisis: low back pain and the health of the public. ***Annual Review of Public Health*** 1991;12:141-56.

56. Nachemson AL, Jonsson EE. ***Neck and back pain: the scientific evidence of causes, diagnosis, and treatment***. Philadelphia: Lippincott Williams and Wilkins, 2000.

57. Frymoyer JW. Predicting disability from low back pain. ***Clinical Orthopaedics & Related Research*** 1992(279):101-9.

58. Waddell G. Biopsychosocial analysis of low back pain. ***Baillieres Clinical Rheumatology*** 1992;6(3):523-57.

59. Vallfors B. Acute, subacute and chronic low back pain: clinical symptoms, absenteeism and working environment. ***Scandinavian Journal of Rehabilitation Medicine - Supplementum*** 1985;11:1-98.

60. Spengler DM, Bigos SJ, Martin NA, Zeh J, Fisher L, Nachemson A. Back injuries in industry: a retrospective study. I. Overview and cost analysis. ***Spine*** 1986;11(3):241-5.

61. Haddad GH. Analysis of 2932 workers' compensation back injury cases. The impact on the cost to the system. ***Spine*** 1987;12(8):765-9.

62. Webster BS, Snook SH. The cost of compensable low back pain. ***Journal of Occupational Medicine*** 1990;32(1):13-5.

63. Walker BF, Muller R, Grant WD. Low back pain in Australian adults: prevalence and associated disability. ***Journal of Manipulative & Physiological Therapeutics*** 2004;27(4):238-44.

64. Walker BF, Muller R, Grant WD. Low back pain in Australian adults: the economic burden. ***Asia-Pacific Journal of Public Health*** 2003;15(2):79-87.

65. Johnson WG, Baldwin ML, Butler RJ. Back pain and work disability: the need for a new paradigm. ***Industrial Relations*** 1998;37(1):9-34.

66. Waddell G, Aylward M, Sawney P. ***Back pain, incapacity for work and social security benefits: an international literature review and analysis.*** London: Royal Society of Medicine Press, 2002.

67. Bigos SJ, Battie MC, Spengler DM, Fisher LD, Fordyce WE, Hansson T, et al. A longitudinal, prospective study of industrial back injury reporting. ***Clinical Orthopaedics & Related Research*** 1992(279):21-34.

68. Cassidy JD, Carroll L, Cote P, Berglund A, Nygren A. Low back pain after traffic collisions: a population-based cohort study. ***Spine*** 2003;28(10):1002-9.

69. Holmstrom EB, Lindell J, Moritz U. Low back and neck/shoulder pain in construction workers: occupational workload and psychosocial risk factors. Part 2: Relationship to neck and shoulder pain. ***Spine*** 1992;17(6):672-7.

70. Bergenudd H, Nilsson B. Back pain in middle age; occupational workload and psychologic factors: an epidemiologic survey. ***Spine*** 1988;13(1):58-60.

71. Greenough CG, Fraser RD. The effects of compensation on recovery from low-back injury. ***Spine*** 1989;14(9):947-55.

72. Brewin CR, Robson MJ, Shapiro DA. Social and psychological determinants of recovery from industrial injuries. ***Injury*** 1983;14(5):451-5.

73. Hoogendoorn WE, van Poppel MN, Bongers PM, Koes BW, Bouter LM. Systematic review of psychosocial factors at work and private life as risk factors for back pain. ***Spine*** 2000;25(16):2114-25.

74. Croft PR, Papageorgiou AC, Thomas E, Macfarlane GJ, Silman AJ. Short-term physical risk factors for new episodes of low back pain. Prospective evidence from the South Manchester Back Pain Study. ***Spine*** 1999;24(15):1556-61.

75. Muller CF, Monrad T, Biering-Sorensen F, Darre E, Deis A, Kryger P. The influence of previous low back trouble, general health, and working conditions on future sick-listing because of low back trouble. A 15-year follow-up study of risk indicators for self-reported sick-listing caused by low back trouble. ***Spine*** 1999;24(15):1562-70.

76. Papageorgiou AC, Macfarlane GJ, Thomas E, Croft PR, Jayson MI, Silman AJ. Psychosocial factors in the workplace--do they predict new episodes of low back pain? Evidence from the South Manchester Back Pain Study. ***Spine*** 1997;22(10):1137-42.

77. Bigos SJ, Battie MC, Spengler DM, Fisher LD, Fordyce WE, Hansson TH, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. ***Spine*** 1991;16(1):1-6.

78. Davis KG, Heaney CA. The relationship between psychosocial work characteristics and low back pain: underlying methodological issues. ***Clinical Biomechanics*** 2000;15(6):389-406.

79. Hemingway H, Shipley MJ, Stansfeld S, Marmot M. Sickness absence from back pain, psychosocial work characteristics and employment grade among office workers. ***Scandinavian Journal of Work, Environment & Health*** 1997;23(2):121-9.

80. Greenough CG. Recovery form low back injury. ***Acta Orthopaedica Scandinavica*** 1993;64(Supplementum 254):1-64.

81. Hadler NM, Carey TS, Garrett J. The influence of indemnification by workers' compensation insurance on recovery from acute backache. North Carolina Back Pain Project. ***Spine*** 1995;20(24):2710-5.

82. McNaughton HK, Sims A, Taylor WJ. Prognosis for people with back pain under a No-fault 24-hour-cover compensation scheme. ***Spine*** 2000;25(10):1254-8.

83. Chapman SL, Brena SF. Learned helplessness and responses to nerve blocks in chronic low back pain patients. ***Pain*** 1982;14(4):355-64.

84. Guest GH, Drummond PD. Effect of compensation on emotional state and disability in chronic back pain. ***Pain*** 1992;48(2):125-30.

85. Kopec JA, Sayre EC. Work-related psychosocial factors and chronic pain: a prospective cohort study in Canadian workers. ***Journal of Occupational & Environmental Medicine*** 2004;46(12):1263-71.

86. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its management. ***Spine*** 1995;20(8 Suppl):1S-73S.

87. Pennie B, Agambar L. Patterns of injury and recovery in whiplash. ***Injury*** 1991;22(1):57-9.

88. Quinlan KP, Annest JL, Myers B, Ryan G, Hill H. Neck strains and sprains among motor vehicle occupants-United States, 2000. ***Accident Analysis & Prevention*** 2004;36(1):21-7.

89. MAA Whiplash Guidelines welcomed by health and insurance industries. ***InforMAAtion***: Motor Accidents Authority, 2002.

90. Whiplash and the NSW Motor Accidents Scheme statistical information paper number 7. Sydney: Motor Accidents Authority of NSW, 1999.

91. Anderson L. Motor Accident Insurance Commission Annual Report 2004-05. Brisbane: Motor Accident Insurance Commission, 2005.

92. Report on personal injury compensation legislation. Sydney: New South Wales Parliament, Legislative Council, General purpose standing committee No. 1, 2005.

93. Joslin CC, Khan SN, Bannister GC. Long-term disability after neck injury. a comparative study. ***Journal of Bone & Joint Surgery - British Volume*** 2004;86(7):1032-4.

94. Malleson A. ***Whiplash and other useful illnesses***. Montreal: McGill-Queen's University Press, 2002.

95. Schrader H, Obelieniene D, Bovim G, Surkiene D, Mickeviciene D, Miseviciene I, et al. Natural evolution of late whiplash syndrome outside the medicolegal context. ***Lancet*** 1996;347(9010):1207-11.

96. Obelieniene D, Schrader H, Bovim G, Miseviciene I, Sand T. Pain after whiplash: a prospective controlled inception cohort study. ***Journal of Neurology, Neurosurgery & Psychiatry*** 1999;66(3):279-83.

97. Ferrari R, Obelieniene D, Russell AS, Darlington P, Gervais R, Green P. Symptom expectation after minor head injury. A comparative study between

Canada and Lithuania. ***Clinical Neurology & Neurosurgery*** 2001;103(3):184-90.

98. Partheni M, Constantoyannis C, Ferrari R, Nikiforidis G, Voulgaris S, Papadakis N. A prospective cohort study of the outcome of acute whiplash injury in Greece. ***Clinical & Experimental Rheumatology*** 2000;18(1):67-70.

99. Bonk AD, Ferrari R, Giebel GD, Edelmann M, Huser R. Prospective, randomized, controlled study of activity versus collar, and the natural history for whiplash injury, in Germany. ***Journal of Musculoskeletal Pain*** 2000;8(1/2):123-132.

100. Barnsley L, Lord S, Bogduk N. Whiplash injury. ***Pain*** 1994;58(3):283-307.

101. Balla JI. The late whiplash syndrome: a study of an illness in Australia and Singapore. ***Culture, Medicine & Psychiatry*** 1982;6(2):191-210.

102. Guez M, Hildingsson C, Nilsson M, Toolanen G. The prevalence of neck pain: a population-based study from northern Sweden. ***Acta Orthopaedica Scandinavica*** 2002;73(4):455-9.

103. Brison RJ, Hartling L, Pickett W. A prospective study of acceleration-extension injuries following rear-end motor vehicle collisions. ***Journal of Musculoskeletal Pain*** 2000;8(1/2):97-113.

104. Hodgson SP, Grundy M. Neck sprains after car accidents. ***BMJ*** 1989;298(6685):1452.

105. Mayou R, Bryant B. Outcome of 'whiplash' neck injury. ***Injury*** 1996;27(9):617-23.

106. Mayou R, Radanov BP. Whiplash neck injury. ***Journal of Psychosomatic Research*** 1996;40(5):461-74.

107. Parmar HV, Raymakers R. Neck injuries from rear impact road traffic accidents: prognosis in persons seeking compensation. ***Injury*** 1993;24(2):75-8.

108. Berglund A, Alfredsson L, Cassidy JD, Jensen I, Nygren A. The association between exposure to a rear-end collision and future neck or shoulder pain: a cohort study. ***Journal of Clinical Epidemiology*** 2000;53(11):1089-94.

109. Ferrari R, Russell AS. Epidemiology of whiplash: an international dilemma. ***Annals of the Rheumatic Diseases*** 1999;58(1):1-5.

110. Ferrari R, Russell AS. Why blame is a factor in recovery from whiplash injury. ***Medical Hypotheses*** 2001;56(3):372-5.

111. Ferrari R, Kwan O, Russell AS, Pearce JM, Schrader H. The best approach to the problem of whiplash? One ticket to Lithuania, please. ***Clinical and Experimental Rheumatology*** 1999;17(3):321-6.

112. Kwan O, Friel J. A new strategy to support the "chronic injury" model of whiplash: ignore Lithuania. ***Medical Science Monitor*** 2002;8(2):LE3-5.

113. Nygren A, Berglund A, von Koch M. Neck-and-shoulder pain, an increasing problem. Strategies for using insurance material to follow trends. ***Scandinavian Journal of Rehabilitation Medicine - Supplementum*** 1995;32:107-12.

114. Mills H, Horne G. Whiplash--manmade disease? ***New Zealand Medical Journal*** 1986;99(802):373-4.

115. Cote P, Cassidy JD, Carroll L, Frank JW, Bombardier C. A systematic review of the prognosis of acute whiplash and a new conceptual framework to synthesize the literature. ***Spine*** 2001;26(19):E445-58.

116. Scholten-Peeters GG, Verhagen AP, Bekkering GE, van der Windt DA, Barnsley L, Oostendorp RA, et al. Prognostic factors of whiplash-associated disorders: a systematic review of prospective cohort studies. ***Pain*** 2003;104(1-2):303-22.

117. Cassidy JD, Carroll LJ, Cote P, Lemstra M, Berglund A, Nygren A. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. ***New England Journal of Medicine*** 2000;342(16):1179-86.

118. Russell RS. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims. ***New England Journal of Medicine*** 2000;343(15):1119-20.

119. Clionsky M. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims. ***New England Journal of Medicine*** 2000;343(15):1119; author reply 1120.

120. Merskey H, Teasell RW. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims. ***New England Journal of Medicine*** 2000;343(15):1119; author reply 1120.

121. Freeman MD, Rossignol AM. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims. ***New England Journal of Medicine*** 2000;343(15):1118-9; author reply 1120.

122. Reilly PA, Travers R, Littlejohn GO. Epidemiology of soft tissue rheumatism: the influence of the law. ***Journal of Rheumatology*** 1991;18(10):1448-9.

123. Shapiro AP, Roth RS. The effect of litigation on recovery from whiplash. ***Spine: State of the Art Reviews*** 1993;7(3):531-56.

124. Cameron I, Johnson S. Legislative change improved health outcomes for people with whiplash: pre and post comparison. ***CTP Claims Management Symposium***. Darling Harbour, Sydney: Motor Accidents Authority, 2005.

125. Kasch H, Bach FW, Jensen TS. Handicap after acute whiplash injury: a 1-year prospective study of risk factors. ***Neurology*** 2001;56(12):1637-43.

126. Hohl M. Soft-tissue injuries of the neck in automobile accidents. Factors influencing prognosis. ***Journal of Bone & Joint Surgery - American Volume*** 1974;56(8):1675-82.

127. Mayou R, Bryant B, Duthie R. Psychiatric consequences of road traffic accidents. ***BMJ*** 1993;307(6905):647-51.

128. Mayou R, Tyndel S, Bryant B. Long-term outcome of motor vehicle accident injury. ***Psychosomatic Medicine*** 1997;59(6):578-84.

129. Norris SH, Watt I. The prognosis of neck injuries resulting from rear-end vehicle collisions. ***Journal of Bone & Joint Surgery - British Volume*** 1983;65(5):608-11.

130. Mayou R, Bryant B. Outcome 3 years after a road traffic accident. ***Psychological Medicine*** 2002;32(4):671-5.

131. Mayou R. Medico-legal aspects of road traffic accidents. ***Journal of Psychosomatic Research*** 1995;39(6):789-98.

132. Bryant B, Mayou R, Lloyd-Bostock S. Compensation claims following road accidents: a six-year follow-up study. ***Medicine, Science & the Law*** 1997;37(4):326-36.

133. Mayou R, Bryant B. Consequences of road traffic accidents for different types of road user. ***Injury*** 2003;34(3):197-202.

134. Mayou R, Bryant B. Psychiatry of whiplash neck injury. ***British Journal of Psychiatry*** 2002;180:441-8.

135. Mayou RA, Black J, Bryant B. Unconsciousness, amnesia and psychiatric symptoms following road traffic accident injury. ***British Journal of Psychiatry*** 2000;177:540-5.

136. Mayou R, Bryant B, Ehlers A. Prediction of psychological outcomes one year after a motor vehicle accident. ***American Journal of Psychiatry*** 2001;158(8):1231-8.

137. Bjorgen IA. Late whiplash syndrome. ***Lancet*** 1996;348(9020):124; author reply 125-6.

138. Holm L, Cassidy JD, Sjogren Y, Nygren A. Impairment and work disability due to whiplash injury following traffic collisions. An analysis of insurance material from the Swedish Road Traffic Injury Commission. ***Scandinavian Journal of Public Health*** 1999;27(2):116-23.

139. Cote P, Cassidy JD, Carroll L. The Saskatchewan Health and Back Pain Survey. The prevalence of neck pain and related disability in Saskatchewan adults. ***Spine*** 1998;23(15):1689-98.

140. Cassou B, Derriennic F, Monfort C, Norton J, Touranchet A. Chronic neck and shoulder pain, age, and working conditions: longitudinal results from a large random sample in France. ***Occupational & Environmental Medicine*** 2002;59(8):537-44.

141. Webb R, Brammah T, Lunt M, Urwin M, Allison T, Symmons D. Prevalence and predictors of intense, chronic, and disabling neck and back pain in the UK general population. ***Spine*** 2003;28(11):1195-202.

142. Carroll LJ, Cassidy JD, Cote P. The Saskatchewan Health and Back Pain Survey: the prevalence and factors associated with depressive symptomatology in Saskatchewan adults. ***Canadian Journal of Public Health Revue Canadienne de Sante Publique*** 2000;91(6):459-64.

143. Palmer KT, Walker-Bone K, Griffin MJ, Syddall H, Pannett B, Coggon D, et al. Prevalence and occupational associations of neck pain in the British population. ***Scandinavian Journal of Work, Environment & Health*** 2001;27(1):49-56.

144. Gordon SJ, Trott P, Grimmer KA. Waking cervical pain and stiffness, headache, scapular or arm pain: gender and age effects. ***Australian Journal of Physiotherapy*** 2002;48(1):9-15.

145. Guez M, Hildingsson C, Stegmayr B, Toolanen G. Chronic neck pain of traumatic and non-traumatic origin: a population-based study. ***Acta Orthopaedica Scandinavica*** 2003;74(5):576-9.

146. Awerbuch MS. Whiplash in Australia: illness or injury? ***Medical Journal of Australia*** 1992;157(3):193-6.

147. Macnab I. Chapter 19. The whiplash syndrome. ***Clinical Neurosurgery*** 1973;20:232-41.

148. Bosworth DM. Whiplash - an unacceptable medical term. ***Journal of Bone and Joint Surgery*** 1959;41A:16.

149. Bogduk N. Management of chronic low back pain. ***Medical Journal of Australia*** 2004;180:79-83.

150. Berry H. Chronic whiplash syndrome as a functional disorder. ***Archives of Neurology*** 2000;57(4):592-4.

151. Alpar EK, Onuoha G, Killampalli VV, Waters R. Management of chronic pain in whiplash injury. ***Journal of Bone & Joint Surgery - British Volume*** 2002;84(6):807-11.

152. Gargan MF. What is the evidence for an organic lesion in whiplash injury? ***Journal of Psychosomatic Research*** 1995;39(6):777-81.

153. Shaffer JW, Nussbaum K, Little JM. MMPI profiles of disability insurance claimants. ***American Journal of Psychiatry*** 1972;129(4):403-7.

154. Lees-Haley PR, English LT, Glenn WJ. A Fake Bad Scale on the MMPI-2 for personal injury claimants. ***Psychological Reports*** 1991;68(1):203-10.

155. Radanov BP, Sturzenegger M. Predicting recovery from common whiplash. ***European Neurology*** 1996;36(1):48-51.

156. Sterling M, Kenardy J, Jull G, Vicenzino B. The development of psychological changes following whiplash injury. ***Pain*** 2003;106(3):481-9.

157. Sterling M, Jull G, Vicenzino B, Kenardy J, Darnell R. Physical and psychological factors predict outcome following whiplash injury. ***Pain*** 2005;114(1-2):141-8.

158. Leclerc A, Niedhammer I, Landre MF, Ozguler A, Etore P, Pietri-Taleb F. One-year predictive factors for various aspects of neck disorders. ***Spine*** 1999;24(14):1455-62.

159. Berglund A, Alfredsson L, Jensen I, Cassidy JD, Nygren A. The association between exposure to a rear-end collision and future health complaints. ***Journal of Clinical Epidemiology*** 2001;54(8):851-6.

160. Hadler NM. Workers with disabling back pain. ***New England Journal of Medicine*** 1997;337(5):341-3.

161. Eriksen W, Natvig B, Knardahl S, Bruusgaard D. Job characteristics as predictors of neck pain. A 4-year prospective study. ***Journal of Occupational & Environmental Medicine*** 1999;41(10):893-902.

162. Kivimaki M, Vahtera J, Ferrie JE, Hemingway H, Pentti J. Organisational downsizing and musculoskeletal problems in employees: a prospective study. ***Occupational & Environmental Medicine*** 2001;58(12):811-7.

163. Linton SJ, Kamwendo K. Risk factors in the psychosocial work environment for neck and shoulder pain in secretaries. ***Journal of Occupational Medicine*** 1989;31(7):609-13.

164. Ring D, Guss D, Malhotra L, Jupiter JB. Idiopathic arm pain. ***Journal of Bone & Joint Surgery - American Volume*** 2004;86-A(7):1387-91.

165. Nathan PA, Meadows KD, Istvan JA. Predictors of carpal tunnel syndrome: an 11-year study of industrial workers. ***Journal of Hand Surgery - American Volume*** 2002;27(4):644-51.

166. Nathan PA, Keniston RC, Myers LD, Meadows KD. Longitudinal study of median nerve sensory conduction in industry: relationship to age, gender, hand dominance, occupational hand use, and clinical diagnosis. ***Journal of Hand Surgery - American Volume*** 1992;17(5):850-7.

167. Falkiner S, Myers S. When exactly can carpal tunnel syndrome be considered work-related? ***ANZ Journal of Surgery*** 2002;72(3):204-9.

168. Stapleton MJ. Occupation and carpal tunnel syndrome. ***Australian and New Zealand Journal of Surgery*** 2006;76:494-6.

169. Elmaraghy MW, Hurst LN. Single-portal endoscopic carpal tunnel release: Agee carpal tunnel release system. ***Annals of Plastic Surgery*** 1996;36(3):286-291.

170. Concannon MJ, Brownfield ML, Puckett CL. The incidence of recurrence after endoscopic carpal tunnel release. ***Plastic & Reconstructive Surgery*** 2000;105(5):1662-5.

171. Davies BW, Pennington GA, Fritz AM. Two-portal endoscopic carpal tunnel release: an outcome analysis of 333 hands. ***Annals of Plastic Surgery*** 1998;40(5):542-8.

172. Higgs PE, Edwards D, Martin DS, Weeks PM. Carpal tunnel surgery outcomes in workers: effect of workers' compensation status. ***Journal of Hand Surgery - American Volume*** 1995;20(3):354-60.

173. al-Qattan MM, Bowen V, Manktelow RT. Factors associated with poor outcome following primary carpal tunnel release in non-diabetic patients. ***Journal of Hand Surgery - British Volume*** 1994;19(5):622-5.

174. Hallock GG, Lutz DA. Prospective comparison of minimal incision "open" and two-portal endoscopic carpal tunnel release. ***Plastic & Reconstructive Surgery*** 1995;96(4):941-7.

175. Lee H, Jackson TA, Wray RC, Jr. Carpal tunnel release through a limited skin incision under direct visualization using a new instrument, the carposcope. ***Plastic & Reconstructive Surgery*** 1996;98(2):313-320.

176. Olney JR, Quenzer DE, Makowsky M. Contested claims in carpal tunnel surgery: outcome study of worker's compensation factors. ***Iowa Orthopaedic Journal*** 1999;19:111-21.

177. Herbert R, Janeway K, Schechter C. Carpal tunnel syndrome and workers' compensation among an occupational clinic population in New York State. ***American Journal of Industrial Medicine*** 1999;35(4):335-42.

178. Katz JN, Losina E, Amick BC, 3rd, Fossel AH, Bessette L, Keller RB. Predictors of outcomes of carpal tunnel release. ***Arthritis & Rheumatism*** 2001;44(5):1184-93.

179. Shuford H, Restrepo T. Carpal tunnel claims rank second among major lost time diagnoses. ***NCCI Research Brief***: NCCI, 2005.

180. Proud G, Burke F, Lawson IJ, McGeoch KL, Miles JN. Cold provocation testing and hand-arm vibration syndrome--an audit of the results of the Department of Trade and Industry scheme for the evaluation of miners. ***British Journal of Surgery*** 2003;90(9):1076-9.

181. Falkiner S. Diagnosis and treatment of hand-arm vibration syndrome and its relationship to carpal tunnel syndrome. ***Australian Family Physician*** 2003;32(7):530-4.

182. Bongers PM, Kremer AM, ter Laak J. Are psychosocial factors, risk factors for symptoms and signs of the shoulder, elbow, or hand/wrist?: A review of the epidemiological literature. ***American Journal of Industrial Medicine*** 2002;41(5):315-42.

183. Binder LM, Rohling ML. Money matters: a meta-analytic review of the effects of financial incentives on recovery after closed-head injury. ***American Journal of Psychiatry*** 1996;153(1):7-10.

184. Philpott T. Vet Stress Might Rise With Review of 72,000 PTSD Cases. In: Military.com, editor, 2005:documents rise in PTSD.

185. Frueh BC, Elhai JD, Gold PB, Monnier J, Magruder KM, Keane TM, et al. Disability compensation seeking among veterans evaluated for posttraumatic stress disorder. ***Psychiatric Services*** 2003;54(1):84-91.

186. DeViva JC, Bloem WD. Symptom exaggeration and compensation seeking among combat veterans with posttraumatic stress disorder. ***Journal of Traumatic Stress*** 2003;16(5):503-7.

187. Sayer NA, Thuras P. The influence of patients' compensation-seeking status on the perceptions of veterans affairs clinicians. ***Psychiatric Services*** 2002;53(2):210-2.

188. Silove D, Blaszczynski A, Manicavasager V, Tyndall K, Petridis A, Hillman K. Capacity of screening questionnaires to predict psychiatric morbidity 18 months after motor vehicle accidents. ***Journal of Nervous & Mental Disease*** 2003;191(9):604-10.

189. Chan AO, Air TM, McFarlane AC. Posttraumatic stress disorder and its impact on the economic and health costs of motor vehicle accidents in South australia. ***Journal of Clinical Psychiatry*** 2003;64(2):175-81.

190. Blanchard EB, Hickling EJ, Taylor AE, Buckley TC, Loos WR, Walsh J. Effects of litigation settlements on posttraumatic stress symptoms in motor vehicle accident victims. ***Journal of Traumatic Stress*** 1998;11(2):337-54.

191. Ehlers A, Mayou RA, Bryant B. Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. ***Journal of Abnormal Psychology*** 1998;107(3):508-19.

192. Mayou RA, Ehlers A, Bryant B. Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. ***Behaviour Research & Therapy*** 2002;40(6):665-75.

193. Blanchard EB, Hickling EJ, Taylor AE, Loos WR, Forneris CA, Jaccard J. Who develops PTSD from motor vehicle accidents? ***Behaviour Research & Therapy*** 1996;34(1):1-10.

194. Bryant RA, Harvey AG. The influence of litigation on maintenance of posttraumatic stress disorder. ***Journal of Nervous & Mental Disease*** 2003;191(3):191-3.

195. Blaszczynski A, Gordon K, Silove D, Sloane D, Hillman K, Panasetis P. Psychiatric morbidity following motor vehicle accidents: a review of methodological issues. ***Comprehensive Psychiatry*** 1998;39(3):111-21.

196. Matthews LR. Work potential of road accident survivors with post-traumatic stress disorder. ***Behaviour Research & Therapy*** 2005;43(4):475-83.

197. MacKenzie EJ, Morris JA, Jr., Jurkovich GJ, Yasui Y, Cushing BM, Burgess AR, et al. Return to work following injury: the role of economic, social, and job-related factors. ***American Journal of Public Health*** 1998;88(11):1630-7.

198. Bosse MJ, MacKenzie EJ, Kellam JF, Burgess AR, Webb LX, Swiontkowski MF, et al. An analysis of outcomes of reconstruction or amputation after leg-threatening injuries. ***New England Journal of Medicine*** 2002;347(24):1924-31.

199. Bergner M, Bobbitt RA, Carter WB, Gilson BS. The Sickness Impact Profile: development and final revision of a health status measure. ***Medical Care*** 1981;19(8):787-805.

200. Spigelman AD, Dwyer P. Is there a link between work-related stress and colorectal cancer? ***Medical Journal of Australia*** 2004;180:339-340.

201. Nisselle P. Stress, colon cancer and worker's compensation. ***Medical Journal of Australia*** 2004;180:341-342.

202. Collie J. ***Malingering and feigned sickness***. London: Edward Arnold, 1913.

203. Waddell G, McCulloch JA, Kummel E, Venner RM. Nonorganic physical signs in low-back pain. ***Spine*** 1980;5(2):117-25.

204. Pearce JM. Aspects of the failed back syndrome: role of litigation. ***Spinal Cord*** 2000;38(2):63-70.

205. Doxey NC, Dzioba RB, Mitson GL, Lacroix JM. Predictors of outcome in back surgery candidates. ***Journal of Clinical Psychology*** 1988;44(4):611-22.

206. Dzioba RB, Doxey NC. A prospective investigation into the orthopaedic and psychologic predictors of outcome of first lumbar surgery following industrial injury. ***Spine*** 1984;9(6):614-23.

207. Kiester PD, Duke AD. Is it malingering, or is it 'real'? Eight signs that point to nonorganic back pain. ***Postgraduate Medicine*** 1999;106(7):77-80, 83-4.

208. Liar, liar, pants on fire: Differences between genuine and faked facial expressions of acute pain. 23rd Annual Scientific Meeting of the American Pain Society; 2004; Vancouver, BC, Canada.

209. Iverson GL, King RJ, Scott JG, Adams RL. Cognitive complaints in litigating patients with head injuries or chronic pain. ***Journal of Forensic Psychology*** 2001;2(1):19-30.

210. Rohling ML, Binder LM, Langhinrichsen-Rohling J. Money matters: A meta-analytic review of the association between financial compensation and the experience and treatment of chronic pain. ***Health Psychology*** 1995;14(6):537-47.

211. Leavitt F, Garron DC, McNeill TW, Whisler WW. Organic status, psychological disturbance, and pain report characteristics in low-back-pain patients on compensation. ***Spine*** 1982;7(4):398-402.

212. Barsky AJ, 3rd. Patients who amplify bodily sensations. ***Annals of Internal Medicine*** 1979;91(1):63-70.

213. Melzack R, Katz J, Jeans ME. The role of compensation in chronic pain: analysis using a new method of scoring the McGill Pain Questionnaire. ***Pain*** 1985;23(2):101-12.

214. Mendelson G. Compensation, pain complaints, and psychological disturbance. ***Pain*** 1984;20(2):169-77.

215. Lees-Haley PR, Williams CW, Zasler ND, Marguilies S, English LT, Stevens KB. Response bias in plaintiffs' histories. ***Brain Injury*** 1997;11(11):791-9.

216. Lees-Haley PR, Williams CW, English LT. Response bias in self-reported history of plaintiffs compared with nonlitigating patients. ***Psychological Reports*** 1996;79(3 Pt 1):811-8.

217. Butcher JN, Graham JR, Tellegen A, Dahlstrom WG, Kaemmer B. ***Manual for the administartion and scoring of the MMPI-s***. Minneapolis: University of Minnesota Press, 2001.

218. Green P, Rohling ML, Lees-Haley PR, Allen LM, 3rd. Effort has a greater effect on test scores than severe brain injury in compensation claimants. ***Brain Injury*** 2001;15(12):1045-60.

219. Lees-Haley PR. MMPI-2 base rates for 492 personal injury plaintiffs: implications and challenges for forensic assessment. ***Journal of Clinical Psychology*** 1997;53(7):745-55.

220. Lees-Haley PR. Efficacy of MMPI-2 validity scales and MCMI-II modifier scales for detecting spurious PTSD claims: F, F-K, Fake Bad Scale, ego strength, subtle-obvious subscales, DIS, and DEB. ***Journal of Clinical Psychology*** 1992;48(5):681-9.

221. Grillo J, Brown RS, Hilsabeck R, Price JR, Lees-Haley PR. Raising doubts about claims of malingering: implications of relationships between MCMI-II and MMPI-2 performances. ***Journal of Clinical Psychology*** 1994;50(4):651-5.

222. Lees-Haley PR, Fox DD. MMPI subtle-obvious scales and malingering: clinical versus simulated scores. ***Psychological Reports*** 1990;66(3 Pt 1):907-11.

223. Lees-Haley PR. Commentary on "The detection of feigned uncoached and coached posttraumatic stress disorder with the MMPI-2 in a sample of workplace accident victims". ***International Journal of Forensic Psychology*** 2004;1(2):94-8.

224. Clayer JR, Bookless C, Ross MW. Neurosis and conscious symptom exaggeration: its differentiation by the Illness Behaviour Questionnaire. ***Journal of Psychosomatic Research*** 1984;28(3):237-41.

225. Pilowsky I, Spence ND. Illness behaviour syndromes associated with intractable pain. ***Pain*** 1976;2(1):61-71.

226. Zusman J, Simon J. Differences in repeated psychiatric examinations of litigants to a lawsuit. ***American Journal of Psychiatry*** 1983;140(10):1300-4.

227. Kucharski LT, Johnsen D. A comparison of simulation and known groups in the detection of malingering on the MMPI-2. ***Journal of Forensic Sciences*** 2002;47(5):1078-82.

228. Bury AS, Bagby RM. The detection of feigned uncoached and coached posttraumatic stress disorder with the MMPI-2 in a sample of workplace accident victims. ***Psychological Assessment*** 2002;14(4):472-84.

229. Storm J, Graham JR. Detection of coached general malingering on the MMPI-2. ***Psychological Assessment*** 2000;12(2):158-65.

230. Mendelson G. Measurement of conscious symptom exaggeration by questionnaire: a clinical study. ***Journal of Psychosomatic Research*** 1987;31(6):703-11.

231. Binder LM. Assessment of malingering after mild head trauma with the Portland Digit Recognition Test. ***Journal of Clinical & Experimental Neuropsychology*** 1993;15(2):170-82.

232. Millis SR. The recognition memory test in the detection of malingered and exaggerated memory deficits. ***The Clinical Neuropsychologist*** 1992;6(4):406-14.

233. Fishbain DA, Cutler R, Rosomoff HL, Rosomoff RS. Chronic pain disability exaggeration/malingering and submaximal effort research. ***Clinical Journal of Pain*** 1999;15(4):244-74.

234. Leavitt F, Sweet JJ. Characteristics and frequency of malingering among patients with low back pain. ***Pain*** 1986;25(3):357-64.

235. Miller H. Accident neurosis. Lecture 2. ***British Medical Journal*** 1961;1:992-8.

236. Miller H. Accident Neurosis. Lecture 1. ***British Medical Journal*** 1961;1:919-25.

237. Gotten N. Survey of one hundred cases of whiplash injury after settlement of litigation. ***JAMA*** 1956;162(9):865-7.

238. Mendelson G. Not "cured by a verdict". Effect of legal settlement on compensation claimants. ***Medical Journal of Australia*** 1982;2(3):132-4.

239. Balla JI, Moraitis S. Knights in armour. A follow-up study of injuries after legal settlement. ***Medical Journal of Australia*** 1970;2(8):355-61.

240. Sprehe DJ. Workers' compensation. A psychiatric follow-up study. ***International Journal of Law & Psychiatry*** 1984;7(2):165-78.

241. Tarsh MJ, Royston C. A follow-up study of accident neurosis. ***British Journal of Psychiatry*** 1985;146:18-25.

242. Mendelson G. The effects of compensation and litigation on disability following compensable injuries. ***The American Journal of Forensic Psychiatry*** 1983;4:97-112.

243. Mendelson G. 'Compensation neurosis' revisited: outcome studies of the effects of litigation. ***Journal of Psychosomatic Research*** 1995;39(6):695-706.

244. Baker SP, O'Neill B, Haddon W, Jr., Long WB. The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. ***Journal of Trauma-Injury Infection & Critical Care*** 1974;14(3):187-96.

245. Rutledge R, Fakhry S, Rutherford E, Muakkassa F, Meyer A. Comparison of APACHE II, Trauma Score, and Injury Severity Score as predictors of outcome in critically injured trauma patients. ***American Journal of Surgery*** 1993;166(3):244-7.

246. Rutledge R, Hoyt DB, Eastman AB, Sise MJ, Velky T, Canty T, et al. Comparison of the Injury Severity Score and ICD-9 diagnosis codes as predictors of outcome in injury: analysis of 44,032 patients. ***Journal of Trauma-Injury Infection & Critical Care*** 1997;42(3):477-87; discussion 487-9.

247. Osler T, Rutledge R, Deis J, Bedrick E. ICISS: an international classification of disease-9 based injury severity score. ***Journal of Trauma-Injury Infection & Critical Care*** 1996;41(3):380-6; discussion 386-8.

248. Osler T, Baker SP, Long W. A modification of the injury severity score that both improves accuracy and simplifies scoring. ***Journal of Trauma-Injury Infection & Critical Care*** 1997;43(6):922-5; discussion 925-6.

249. Lavoie A, Moore L, LeSage N, Liberman M, Sampalis JS. The New Injury Severity Score: a more accurate predictor of in-hospital mortality than the Injury Severity Score. ***Journal of Trauma-Injury Infection & Critical Care*** 2004;56(6):1312-20.

250. Tay SY, Sloan EP, Zun L, Zaret P. Comparison of the New Injury Severity Score and the Injury Severity Score. ***Journal of Trauma-Injury Infection & Critical Care*** 2004;56(1):162-4.

251. Balogh ZJ, Varga E, Tomka J, Suveges G, Toth L, Simonka JA. The new injury severity score is a better predictor of extended hospitalization and intensive care unit admission than the injury severity score in patients with multiple orthopaedic injuries. ***Journal of Orthopaedic Trauma*** 2003;17(7):508-12.

252. Blanchard EB, Hickling EJ, Mitnick N, Taylor AE, Loos WR, Buckley TC. The impact of severity of physical injury and perception of life threat in the development of post-traumatic stress disorder in motor vehicle accident victims. ***Behaviour Research & Therapy*** 1995;33(5):529-34.

253. Large MM. Relationship between compensation claims for psychiatric injury and severity of physical injuries from motor vehicle accidents. ***Medical Journal of Australia*** 2001;175(3):129-32.

254. Delahanty DL, Raimonde AJ, Spoonster E, Cullado M. Injury severity, prior trauma history, urinary cortisol levels, and acute PTSD in motor vehicle accident victims. ***Journal of Anxiety Disorders*** 2003;17(2):149-64.

255. Stansfeld SA, Fuhrer R, Shipley MJ, Marmot MG. Psychological distress as a risk factor for coronary heart disease in the Whitehall II Study. ***International Journal of Epidemiology*** 2002;31(1):248-55.

256. Bosma H, Marmot MG, Hemingway H, Nicholson AC, Brunner E, Stansfeld SA. Low job control and risk of coronary heart disease in Whitehall II (prospective cohort) study. ***BMJ*** 1997;314(7080):558-65.

257. Macleod J, Davey Smith G. Psychosocial factors and public health: a suitable case for treatment? ***Journal of Epidemiology and Community Health*** 2003;57(8):565-70.

258. Macleod J, Davey Smith G, Heslop P, Metcalfe C, Carroll D, Hart C. Psychological stress and cardiovascular disease: empirical demonstration of bias in a prospective observational study of Scottish men. ***BMJ*** 2002;324(7348):1247-51.

259. Fishbain DA, Rosomoff HL, Cutler RB, Rosomoff RS. Secondary gain concept: a review of the scientific evidence. ***Clinical Journal of Pain*** 1995;11(1):6-21.

260. Worrall JD, Butler RJ. Benefits and claim duration. In: Worral JD, Appel D, editors. ***Workers' compensation benefits: adequacy, equity, and efficiency***. Ithaca: ILR Press, 1985:57-70.

261. Carron H, DeGood DE, Tait R. A comparison of low back pain patients in the United States and New Zealand: psychosocial and economic factors affecting severity of disability. ***Pain*** 1985;21(1):77-89.

262. Barondess JA. Disease and illness--a crucial distinction. ***American Journal of Medicine*** 1979;66(3):375-6.

263. Neuhauser H, Ellert U, Ziese T. Chronic back pain in the general population in Germany 2002/2003: prevalence and highly affected population groups. ***Gesundheitswesen*** 2005;67(10):685-93.

264. Enterline PE. Sick absence in certain Western countries. ***Industrial Medicine & Surgery*** 1964;33(10):738-41.

265. Ruser JW. Workers' compensation insurance, experience-rating, and occupational injuries. ***Rand Journal of Economics*** 1985;16(4):487-503.

266. Worrall JD, Appel D. The impact of workers' compensation benefits on low-back claims. In: Hadler NM, editor. ***Clinical concepts in regional musculoskeletal illness***. Orlando: Grune and Stratton, 1987:281-97.

267. Worrall JD, Appel D. The wage replacement rate and benefit utilization in workers' compensation insurance. ***Journal of Risk Insurance*** 1982;49:361-71.

268. Bednar JM, Baesher-Griffith P, Osterman AL. Workers compensation. Effect of state law on treatment cost and work status. ***Clinical Orthopaedics & Related Research*** 1998(351):74-7.

269. Young JN, Shaffrey CI, Laws ER, Jr., Lovell LR. Lumbar disc surgery in a fixed compensation population: a model for influence of secondary gain on surgical outcome. ***Surgical Neurology*** 1997;48(6):552-8; discussion 558-9.

270. The Accident Compenstion Commission 1987 - 1988 annual report. Melbourne: The Accident Compensation Commission, 1988.

271. Insurance claim propensity and claim size for injured road users. 2005 National Claims Symposium; 2005 15-16 June; Sydney.

272. Leigh JP. Analysis of workers' compensation using data on individuals. ***Industrial Relations*** 1985;24(2):247-55.

273. Bell C. Affection of voluntary nerves. ***The Nervous System of the Human Body***. London: Henry Renshaw, 1854:436-7.

274. Reynolds JR. Paralysis, and other disorders of motion and sensation, dependent on an idea. ***BMJ*** 1869:483-5.

275. St. Clare W. An epidemic of hysterics. In: Hunter R, McAlpine I, editors. ***300 years of psychiatry***. Oxford: Oxford University Press, 1964.

276. Ferrari R, Shorter E. From railway spine to whiplash - the recycling of nervous irritation. ***Med Sci Monit*** 2003;9(11):HY27-37.

277. Hadler NM. Fibromyalgia, chronic fatigue, and other iatrogenic diagnostic algorithms. Do some labels escalate illness in vulnerable patients? ***Postgraduate Medicine*** 1997;102(2):161-2.

278. Ferrari R, Kwan O. The no-fault flavor of disability syndromes. ***Medical Hypotheses*** 2001;56(1):77-84.

279. Deyo RA, Diehl AK. Psychosocial predictors of disability in patients with low back pain. ***Journal of Rheumatology*** 1988;15(10):1557-64.

280. Schlesinger EB. Factors contributing to failure in surgery of the low back syndrome. ***Journal of Trauma*** 1962;2:162-73.

281. Fredrickson BE, Trief PM, VanBeveren P, Yuan HA, Baum G. Rehabilitation of the patient with chronic back pain. A search for outcome predictors. ***Spine*** 1988;13(3):351-3.

282. Blanchard EB, Hickling EJ, Taylor AE, Loos WR, Gerardi RJ. Psychological morbidity associated with motor vehicle accidents. ***Behaviour Research & Therapy*** 1994;32(3):283-90.

283. Harvey AG, Bryant RA. Predictors of acute stress following motor vehicle accidents. ***Journal of Traumatic Stress*** 1999;12(3):519-25.

284. Blanchard EB, Hickling EJ, Taylor AE, Loos W. Psychiatric morbidity associated with motor vehicle accidents. ***Journal of Nervous & Mental Disease*** 1995;183(8):495-504.

285. van der Giezen AM, Bouter LM, Nijhuis FJ. Prediction of return-to-work of low back pain patients sicklisted for 3-4 months. ***Pain*** 2000;87(3):285-94.

286. Borg V, Kristensen TS, Burr H. Work environment and changes in self-rated health: a five year follow-up study. ***Stress Medicine*** 2000;16:37-47.

287. Vahtera J, Kivimaki M, Pentti J. Effect of organisational downsizing on health of employees. ***Lancet*** 1997;350(9085):1124-8.

288. Cheng Y, Kawachi I, Coakley EH, Schwartz J, Colditz G. Association between psychosocial work characteristics and health functioning in American women: prospective study. ***BMJ*** 2000;320(7247):1432-6.

289. Pransky G, Benjamin K, Hill-Fotouhi C, Fletcher KE, Himmelstein J. Occupational upper extremity conditions: a detailed analysis of work-related outcomes. ***Journal of Occupational Rehabilitation*** 2002;12(3):131-8.

290. U.S. Department of Health and Human Sciences. Musculoskeletal disorders and workplace factors. A critical review of epidemiological evidence for work-related musculoskeletal disorders of the neck, upper extremity and low back. In: Bernard BP, editor. Cincinnati: Centres fo Disease Control and Prevention, National Institute for Occupational Safety and Helath, 1997.

291. Deyo RA, Tsui-Wu YJ. Functional disability due to back pain. A population-based study indicating the importance of socioeconomic factors. ***Arthritis & Rheumatism*** 1987;30(11):1247-53.

292. Magora A. Investigation of the relation between low back pain and occupation. V. Psychological aspects. ***Scandinavian Journal of Rehabilitation Medicine*** 1973;5(4):191-6.

293. Beals RK. Compensation and recovery from injury. ***The Western Journal of Medicine*** 1984;140(2):233-237.

294. Derebery VJ, Tullis WH. Delayed recovery in the patient with a work compensable injury. ***Journal of Occupational Medicine*** 1983;25(11):829-35.

295. Shaver KG, Drown D. On causality, responsibility, and self-blame: a theoretical note. ***Journal of Personality & Social Psychology*** 1986;50(4):697-702.

296. Delahanty DL, Herberman HB, Craig KJ, Hayward MC, Fullerton CS, Ursano RJ, et al. Acute and chronic distress and posttraumatic stress disorder as a function of responsibility for serious motor vehicle accidents. ***Journal of Consulting & Clinical Psychology*** 1997;65(4):560-7.

297. Hickling EJ, Blanchard EB, Buckley TC, Taylor AE. Effects of attribution of responsibility for motor vehicle accidents on severity of PTSD symptoms, ways of coping, and recovery over six months. ***Journal of Traumatic Stress*** 1999;12(2):345-53.

298. Bulman RJ, Wortman CB. Attributions of blame and coping in the "real world": severe accident victims react to their lot. ***Journal of Personality & Social Psychology*** 1977;35(5):351-63.

299. Williams CW, Lees-Haley PR, Brown RS. Human response to traumatic events: an integration of counterfactual thinking, hindsight bias, and attribution theory. ***Psychological Reports*** 1993;72(2):483-94.

300. Turnquist DC, Harvey JH, Andersen BL. Attributions and adjustment to life-threatening illness. ***British Journal of Clinical Psychology*** 1988;27(Pt 1):55-65.

301. Dansak DA. On the tertiary gain of illness. ***Comprehensive Psychiatry*** 1973;14(6):523-34.

302. Weintraub MI. Chronic pain in litigation. What is the relationship? ***Neurologic Clinics*** 1995;13(2):341-9.

303. Taylor VM, Deyo RA, Ciol M, Farrar EL, Lawrence MS, Shonnard NH, et al. Patient-oriented outcomes from low back surgery: a community-based study. ***Spine*** 2000;25(19):2445-52.

304. Klekamp J, McCarty E, Spengler DM. Results of elective lumbar discectomy for patients involved in the workers' compensation system. ***Journal of Spinal Disorders*** 1998;11(4):277-82.

305. Dichraff RM. When the injured worker retains an attorney: the relationship between attorney involvement and case outcome. ***AAOHN Journal*** 1993;41(10):491-8.

306. Risk factors for prolonged disability after whiplash, a prospective study. 63rd Annual Scientific Meeting of the Australian Orthopaedic Association; 2003; Adelaide, Australia. Austrailan Orthopaedic Association.

307. MacKenzie EJ, Bosse MJ, Pollak AN, Webb LX, Swiontkowski MF, Kellam JF, et al. Long-term persistence of disability following severe lower-limb trauma. Results of a seven-year follow-up. ***Journal of Bone & Joint Surgery - American Volume*** 2005;87(8):1801-9.

308. Lees-Haley PR. Attorneys influence expert evidence in forensic psychological and neuropsychological cases. ***Assessment*** 1997;4(4):321-324.

309. Brown JR. Factors contributing to the development of low back pain in industrial workers. ***American Industrial Hygiene Association Journal*** 1975;36(1):26-31.

310. Hadler NM. Point of view. ***Spine*** 1994;19(10):1116.

311. Kwan O, Ferrari R, Friel J. Tertiary gain and disability syndromes. ***Medical Hypotheses*** 2001;57(4):459-64.

312. Cherington M, Cherington C. Thoracic outlet syndrome: reimbursement patterns and patient profiles. ***Neurology*** 1992;42(5):943-5.

313. Johnson WG, Baldwin ML, Burton JF, Jr. Why is the treatment of work-related injuries so costly? New evidence from California. ***Inquiry*** 1996;33(1):53-65.

314. Loeser JD, Sullivan M. Doctors, diagnosis, and disability: a disastrous diversion. ***Clinical Orthopaedics & Related Research*** 1997(336):61-6.

315. Jarvik JG, Hollingworth W, Martin B, Emerson SS, Gray DT, Overman S, et al. Rapid magnetic resonance imaging vs radiographs for patients with low back pain: a randomized controlled trial. ***JAMA*** 2003;289(21):2810-8.

316. Verrilli D, Welch HG. The impact of diagnostic testing on therapeutic interventions. ***JAMA*** 1996;275(15):1189-91.

317. Lurie JD, Birkmeyer NJ, Weinstein JN. Rates of advanced spinal imaging and spine surgery. ***Spine*** 2003;28(6):616-20.

318. Illich I. ***Limits to Medicine. Medical Nemesis: the Expropriation of Health***. London: Marion Boyars, 1995.

319. Illich I. The medicalization of life. ***Journal of Medical Ethics*** 1975;1(2):73-7.

320. Illich I. Clinical damage, medical monopoly, the expropriation of health: three dimensions of iatrogenic tort. ***Journal of Medical Ethics*** 1975;1(2):78-80.

321. Johanson R, Newburn M, Macfarlane A. Has the medicalisation of childbirth gone too far? ***BMJ*** 2002;324(7342):892-5.

322. Ebrahim S. The medicalisation of old age. ***BMJ*** 2002;324(7342):861-3.

323. Clark D. Between hope and acceptance: the medicalisation of dying. ***BMJ*** 2002;324(7342):905-7.

324. Double D. The limits of psychiatry. ***BMJ*** 2002;324(7342):900-4.

325. Hadler NM. Regional back pain: predicament at home, nemesis at work. ***Journal of Occupational & Environmental Medicine*** 1996;38(10):973-8.

326. Miller P, Kendrick D, Bentley E, Fielding K. Cost-effectiveness of lumbar spine radiography in primary care patients with low back pain. ***Spine*** 2002;27(20):2291-7.

327. Kendrick D, Fielding K, Bentley E, Kerslake R, Miller P, Pringle M. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. ***BMJ*** 2001;322(7283):400-5.

328. Borenstein DG, O'Mara JW, Jr., Boden SD, Lauerman WC, Jacobson A, Platenberg C, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects : a seven-year follow-up study. ***Journal of Bone & Joint Surgery - American Volume*** 2001;83-A(9):1306-11.

329. Ireland DC. Australian repetition strain injury phenomenon. ***Clinical Orthopaedics & Related Research*** 1998(351):63-73.

330. Marmot M, Wilkinson RG, Eds. ***Social determinants of health***. Oxford: Oxford University Press, 1999.

331. Sen A. Mortality as an indicator of economic success and failure. ***The Economic Journal*** 1998;108:1-25.

332. Behan RC, Hirschfeld AH. The accident process II. Toward more rational treatment of industrial injuries. ***JAMA*** 1963;186(4):300-6.

333. Weinstein MR. The concept of the disability process. ***Psychosomatics*** 1978;19(2):94-7.

334. CONSORT. www.consort-statement.org, accessed 20 June 2006.

335. Stroup DF, Berlin JA, Morton SC, Olkin I, Williamson GD, Rennie D, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. ***JAMA*** 2000;283(15):2008-12.

336. Greenough CG, Fraser RD. Assessment of outcome in patients with low-back pain. ***Spine*** 1992;17(1):36-41.

337. Harris WH. Traumatic arthritis of the hip after dislocation and acetabular fractures: treatment by mold arthroplasty. An end-result study using a new method of result evaluation. ***Journal of Bone & Joint Surgery - American Volume*** 1969;51(4):737-55.

338. Ware JE, Jr., Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. ***Medical Care*** 1992;30(6):473-83.

339. Enterline PE. Social causes of sick absence. ***Archives of Environmental Health*** 1966;12(4):467-73.

340. Baldwin ML, Johnson WG, Butler RJ. The error of using returns-to-work to measure the outcomes of health care. ***American Journal of Industrial Medicine*** 1996;29(6):632-41.

341. Cochrane Review Manager Software: The Cochrane Collaboration. http://www.cochrane.org/software/revman.htm. Accessed 1 July 2003.

342. Spielgelhalter DJ, Abrams KR, Myles PJ. Evidence Synthesis. ***Bayesian Approaches to Clinical Trials and Health-Care Evaluation***. Hoboken, NJ: John Wiley and Sons Ltd, 2004:267-303.

343. Winbugs. Cambridge, UK: MRC Biostatistics Unit, http://www.mrc-bsu.cam.ac.uk/bugs/. Accessed, 2004.

344. Higgins JPT, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. ***British Medical Journal*** 2003;327:557-60.

345. Wetzel FT, Hoffman MA, Arcieri RR. Freeze-dried fibular allograft in anterior spinal surgery: cervical and lumbar applications. ***Yale Journal of Biology & Medicine*** 1993;66(3):263-75.

346. Hanley EN, Shapiro DE. The development of low-back pain after excision of a lumbar disc. ***Journal of Bone and Joint Surgery Series A*** 1989;71(5):719-721.

347. Agazzi S, Reverdin A, May D. Posterior lumbar interbody fusion with cages: an independent review of 71 cases. ***Journal of Neurosurgery*** 1999;91(2 Suppl):186-92.

348. Hanley EN, Jr., Levy JA. Surgical treatment of isthmic lumbosacral spondylolisthesis. Analysis of variables influencing results. ***Spine*** 1989;14(1):48-50.

349. Hattrup SJ, Cofield RH, Weaver AL. Anterior shoulder reconstruction: prognostic variables. ***Journal of Shoulder & Elbow Surgery*** 2001;10(6):508-13.

350. Buckley R, Tough S, McCormack R, Pate G, Leighton R, Petrie D, et al. Operative compared with nonoperative treatment of displaced intra-articular calcaneal fractures: a prospective, randomized, controlled multicenter trial. ***Journal of Bone & Joint Surgery - American Volume*** 2002;84-A(10):1733-44.

351. Little DG, MacDonald D. The use of the percentage change in Oswestry Disability Index score as an outcome measure in lumbar spinal surgery. ***Spine*** 1994;19(19):2139-43.

352. Strasberg SR, Novak CB, Mackinnon SE, Murray JF. Subjective and employment outcome following secondary carpal tunnel surgery. ***Annals of Plastic Surgery*** 1994;32(5):485-9.

353. Novak CB, van Vliet D, Mackinnon SE. Subjective outcome following surgical management of upper extremity neuromas. ***Journal of Hand Surgery - American Volume*** 1995;20(2):221-6.

354. Mont MA, Antonaides S, Krackow KA, Hungerford DS. Total knee arthroplasty after failed high tibial osteotomy. A comparison with a matched group. ***Clinical Orthopaedics & Related Research*** 1994(299):125-30.

355. Asch HL, Lewis PJ, Moreland DB, Egnatchik JG, Yu YJ, Clabeaux DE, et al. Prospective multiple outcomes study of outpatient lumbar microdiscectomy: should 75 to 80% success rates be the norm? ***Journal of Neurosurgery*** 2002;96(1 Suppl):34-44.

356. Atlas SJ, Chang Y, Kammann E, Keller RB, Deyo RA, Singer DE. Long-term disability and return to work among patients who have a herniated lumbar disc: the effect of disability compensation. ***Journal of Bone & Joint Surgery - American Volume*** 2000;82(1):4-15.

357. MacDermid JC, Donner A, Richards RS, Roth JH. Patient versus injury factors as predictors of pain and disability six months after a distal radius fracture. ***Journal of Clinical Epidemiology*** 2002;55(9):849-854.

358. Quigley MR, Bost J, Maroon JC, Elrifai A, Panahandeh M. Outcome after microdiscectomy: results of a prospective single institutional study. ***Surgical Neurology*** 1998;49(3):263-7; discussion 267-8.

359. Pfeiffer M, Griss P, Haake M, Kienapfel H, Billion M. Standardized evaluation of long-term results after anterior lumbar interbody fusion. ***European Spine Journal*** 1996;5(5):299-307.

360. Shinners TJ, Noordsij PG, Orwin JF. Arthroscopically assisted mini-open rotator cuff repair. ***Arthroscopy*** 2002;18(1):21-6.

361. Hulsizer DL, Staebler MP, Weiss AP, Akelman E. The results of revision carpal tunnel release following previous open versus endoscopic surgery.[comment]. ***Journal of Hand Surgery - American Volume*** 1998;23(5):865-9.

362. Arcand MA, O'Rourke P, Zeman CA, Burkhead WZ, Jr. Revision surgery after failed subacromial decompression. ***International Orthopaedics*** 2000;24(2):61-4.

363. Vaccaro AR, Ring D, Scuderi G, Cohen DS, Garfin SR. Predictors of outcome in patients with chronic back pain and low-grade spondylolisthesis. ***Spine*** 1997;22(17):2030-4; discussion 2035.

364. Sanderson PL, Fraser RD, Hall DJ, Cain CM, Osti OL, Potter GR. Short segment fixation of thoracolumbar burst fractures without fusion. ***European Spine Journal*** 1999;8(6):495-500.

365. Martin LF, Tan TL, Holmes PA, Becker DA, Horn J, Mann LD, et al. Preoperative insurance status influences postoperative complication rates for gastric bypass. ***American Journal of Surgery*** 1991;161(6):625-34.

366. Katz JN, Keller RB, Simmons BP, Rogers WD, Bessette L, Fossel AH, et al. Maine Carpal Tunnel Study: outcomes of operative and nonoperative therapy for carpal tunnel syndrome in a community-based cohort.[erratum

appears in J Hand Surg [Am] 1999 Jan;24(1):201]. ***Journal of Hand Surgery - American Volume*** 1998;23(4):697-710.

367. Salcedo-Wasicek MC, Thirlby RC. Postoperative course after inguinal herniorrhaphy. A case-controlled comparison of patients receiving workers' compensation vs patients with commercial insurance. ***Archives of Surgery*** 1995;130(1):29-32.

368. Katz JN, Keller RB, Simmons BP, Rogers WD, Bessette L, Fossel AH, et al. Maine Carpal Tunnel Study: outcomes of operative and nonoperative therapy for carpal tunnel syndrome in a community-based cohort. ***Journal of Hand Surgery - American Volume*** 1998;23(4):697-710.

369. Rish BL. A critique of the surgical management of lumbar disc disease in a private neurosurgical practice. ***Spine*** 1984;9(5):500-4.

370. Tregonning GD, Transfeldt EE, McCulloch JA, Macnab I, Nachemson A. Chymopapain versus conventional surgery for lumbar disc herniation. 10-year results of treatment. ***Journal of Bone & Joint Surgery - British Volume*** 1991;73(3):481-6.

371. Greenough CG, Taylor LJ, Fraser RD. Anterior lumbar fusion. A comparison of noncompensation patients with compensation patients. ***Clinical Orthopaedics & Related Research*** 1994(300):30-7.

372. Greenough CG, Peterson MD, Hadlow S, Fraser RD. Instrumented posterolateral lumbar fusion. Results and comparison with anterior interbody fusion. ***Spine*** 1998;23(4):479-86.

373. Javid MJ. Signs and symptoms after chemonucleolysis. A detailed evaluation of 214 worker's compensation and noncompensation patients. ***Spine*** 1988;13(12):1428-37.

374. Javid MJ. A 1- to 4-year follow-up review of treatment of sciatica using chemonucleolysis or laminectomy. ***Journal of Neurosurgery*** 1992;76(2):184-90.

375. MacKay MA, Fischgrund JS, Herkowitz HN, Kurz LT, Hecht B, Schwartz M. The effect of interposition membrane on the outcome of lumbar laminectomy and discectomy. ***Spine*** 1995;20(16):1793-6.

376. Spangehl MJ, Hawkins RH, McCormack RG, Loomer RL. Arthroscopic versus open acromioplasty: a prospective, randomized, blinded study. ***Journal of Shoulder & Elbow Surgery*** 2002;11(2):101-7.

377. Jerosch J, Castro WH, Halm H, Muller-Silvergieter G. Long-term results following percutaneous facet coagulation. ***Zeitschrift fur Orthopadie und Ihre Grenzgebiete*** 1993;131(3):241-7.

378. Klassen TP, Wiebe N, Russell K, Stevens K, Hartling L, Craig WR, et al. Abstracts of randomized controlled trials presented at the society for pediatric

research meeting: an example of publication bias.[comment]. ***Archives of Pediatrics & Adolescent Medicine*** 2002;156(5):474-9.

379. Callaham ML, Wears RL, Weber EJ, Barton C, Young G. Positive-outcome bias and other limitations in the outcome of research abstracts submitted to a scientific meeting.[erratum appears in JAMA 1998 Oct 14;280(14):1232]. ***JAMA*** 1998;280(3):254-7.

380. Cassisi JE, Sypert GW, Lagana L, Friedman EM, Robinson ME. Pain, disability, and psychological functioning in chronic low back pain subgroups: myofascial versus herniated disc syndrome. ***Neurosurgery*** 1993;33(3):379-85; discussion 385-6.

381. Winspur I. Arm pain without physical findings: medicine vs. the law? ***Journal of Hand Surgery - British Volume*** 2001;26(5):409-13.

382. Schrader H, Obelieniene D, Bovim G, Surkiene D, Mickeviciene D, Miseviciene I, et al. Natural evolution of late whiplash syndrome outside the medicolegal context.[comment]. ***Lancet*** 1996;347(9010):1207-11.

383. Cherington M, Cherington C. Thoracic outlet syndrome: reimbursement patterns and patient profiles.[see comment]. ***Neurology*** 1992;42(5):943-5.

384. McAuley L, Pham B, Tugwell P, Moher D. Does the inclusion of grey literature influence estimates of intervention effectiveness reported in meta-analyses? ***Lancet*** 2000;356(9237):1228-31.

385. Egger M, Juni P, Bartlett C, Holenstein F, Sterne J. How important are comprehensive literature searches and the assessment of trial quality in systematic reviews? Empirical study. ***Health Technology Assessment (Winchester, England)*** 2003;7(1):1-76.

386. Butler RJ. Wage and injury rate response to shifting levels of workers' compensation. In: Worral JD, editor. ***Safety in the workforce: incentives and disincentives in workers' compensation.*** Ithaca: ILR Press, 1983:61-86.

387. Worrall JD, Butler RJ. Benefits and claim duration. In: Worral JD, Appel D, editors. ***Workers' compensation benefits: adequacy, equity, and efficiency.*** Ithaca: ILR Press, 1985:57-70.

388. Worrall JD, Appel D. The impact of workers' compensation benefits on low-back claims. In: Hadler NM, editor. ***Clinical concepts in regional musculoskeletal illness.*** Orlando: Grune and Stratton, 1987:281-97.

389. Weighill VE. 'Compensation neurosis': a review of the literature. ***Journal of Psychosomatic Research*** 1983;27(2):97-104.

390. Martin RD. Secondary gain, everybody's rationalization. ***Journal of Occupational Medicine*** 1974;16(12):800-1.

391. Bigos SJ, Baker R, Lee S, Vaegter K. Back injury compensation: overcoming an adversarial system. ***The Journal of Musculoskeletal Medicine*** 1994;11(6):17-24.

392. Glancy KE, Glancy CJ, Lucke JF, Mahurin K, Rhodes M, Tinkoff GH. A study of recovery in trauma patients. ***Journal of Trauma Injury Infection & Critical Care*** 1992;33(4):602-9.

393. Committee on Medical Aspects of Automotive Safety. Rating the severity of tissue damage. I. The Abbreviated Scale. ***JAMA*** 1971;215:277-80.

394. Anke AG, Stanghelle JK, Finset A, Roaldsen KS, Pillgram-Larsen J, Fugl-Meyer AR. Long-term prevalence of impairments and disabilities after multiple trauma. ***Journal of Trauma Injury Infection & Critical Care*** 1997;42(1):54-61.

395. MacKenzie EJ, Siegel JH, Shapiro S, Moody M, Smith RT. Functional recovery and medical costs of trauma: an analysis by type and severity of injury. ***Journal of Trauma Injury Infection & Critical Care*** 1988;28(3):281-97.

396. Vazquez MG, Rivera FR, Perez AA, Gonzalez CA, Fernandez ME, Navarrete NP. Analysis of quality of life in polytraumatized patients two years after discharge from an intensive care unit. ***Journal of Trauma Injury Infection & Critical Care*** 1996;41(2):326-32.

397. Dimopoulou I, Anthi A, Mastora Z, Theodorakopoulou M, Konstandinidis A, Evangelou E, et al. Health-related quality of life and disability in survivors of multiple trauma one year after intensive care unit discharge. ***American Journal of Physical Medicine & Rehabilitation*** 2004;83(3):171-6.

398. Vles WJ, Steyerberg EW, Essink-Bot ML, van Beeck EF, Meeuwis JD, Leenen LP. Prevalence and determinants of disabilities and return to work after major trauma. ***Journal of Trauma Injury Infection & Critical Care*** 2005;58(1):126-35.

399. Meerding WJ, Looman CW, Essink-Bot ML, Toet H, Mulder S, van Beeck EF. Distribution and determinants of health and work status in a comprehensive population of injury patients. ***Journal of Trauma Injury Infection & Critical Care*** 2004;56(1):150-61.

400. Ware JE. ***SF-36 Health Survey. Manual and interpretation guide***. Boston: The Health Institute, New England Medical Center, 1993.

401. Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. ***Journal of Health & Social Behavior*** 1997;38(1):21-37.

402. Deyo RA, Battie M, Beurskens AJ, Bombardier C, Croft P, Koes B, et al. Outcome measures for low back pain research. A proposal for standardized use. ***Spine*** 1998;23(18):2003-13.

403. DeGood DE, Kiernan B. Perception of fault in patients with chronic pain. ***Pain*** 1996;64(1):153-9.

404. Household income and income distribution, Australia. Canberra: Australian Bureau of Statistics, 2003.

405. Daniel A. ***Power, privilege and prestige. Occupations in Australia.*** Melbourne: Longman Cheshire Pty Ltd, 1983.

406. Sanderson PL, Todd BD, Holt GR, Getty CJ. Compensation, work status, and disability in low back pain patients. ***Spine*** 1995;20(5):554-6.

407. Gun R, Osti OL, O'Riordan A, Mpelasoka F, Eckerwall CG, Smyth JF. Risk factors for prolonged disability after whiplash injury: a prospective study. ***Spine*** 2005;30(4):386-91.

408. Blanchard EB, Jones-Alexander J, Buckley TC, Forneris CA. Psychometric properties of the PTSD Checklist (PCL). ***Behaviour Research & Therapy*** 1996;34(8):669-73.

409. Ruggiero KJ, Del Ben K, Scotti JR, Rabalais AE. Psychometric properties of the PTSD Checklist-Civilian Version. ***Journal of Traumatic Stress*** 2003;16(5):495-502.

410. Andrykowski MA, Cordova MJ, Studts JL, Miller TW. Posttraumatic stress disorder after treatment for breast cancer: prevalence of diagnosis and use of the PTSD Checklist-Civilian Version (PCL-C) as a screening instrument. ***Journal of Consulting & Clinical Psychology*** 1998;66(3):586-90.

411. DuHamel KN, Ostrof J, Ashman T, Winkel G, Mundy EA, Keane TM, et al. Construct validity of the posttraumatic stress disorder checklist in cancer survivors: analyses based on two samples. ***Psychological Assessment*** 2004;16(3):255-66.

412. Ventureyra VA, Yao SN, Cottraux J, Note I, De Mey-Guillard C. The validation of the Posttraumatic Stress Disorder Checklist Scale in posttraumatic stress disorder and nonclinical subjects. ***Psychotherapy & Psychosomatics*** 2002;71(1):47-53.

413. Hosmer DW, Lemeshow S. ***Applied logistic regression***. New York: John Wiley and Sons, 1989.

414. Katz MH. ***Multivariable analysis: a practical guide for clinicians***. Cambridge: Cambridge University Press, 1999.

415. The impact of patient demographics on standardized outcome measures for the SF-36. 66th Annual Meeting of the American Academy of Orthopaedic Surgeons; 1999 February 4-8, 1999; Anaheim, California, U.S.A. American Academy of Orthopaedic Surgeons.

416. Proctor T, Gatchel RJ, Robinson RC. Psychosocial factors and risk of pain and disability. ***Occupational Medicine*** 2000;15(4):803-12.

417. Renfroe EG, Heywood G, Foreman L, Schron E, Powell J, Baessler C, et al. The end-of-study patient survey: methods influencing response rate in the AVID Trial. ***Controlled Clinical Trials*** 2002;23(5):521-33.

418. Burg JA, Allred SL, Sapp JH, 2nd. The potential for bias due to attrition in the National Exposure Registry: an examination of reasons for nonresponse, nonrespondent characteristics, and the response rate. ***Toxicology & Industrial Health*** 1997;13(1):1-13.

419. Marti B. Bernese runner study '84: methodology, response rate and comparison between respondents and nonrespondents. ***Sozial und Praventivmedizin*** 1984;29(4-5):213-4.

420. Hirschfeld AH, Behan RC. The accident process I. Etiological considerations of industrial injuries. ***JAMA*** 1963;186(3):193-9.

421. Degl' Innocenti A, Guyatt GH, Wiklund I, Heels-Ansdell D, Armstrong D, Fallone CA, et al. The influence of demographic factors and health-related quality of life on treatment satisfaction in patients with gastroesophageal reflux disease treated with esomeprazole. ***Health & Quality of Life Outcomes*** 2005;3(1):4.

422. White B. Measuring patient satisfaction: how to do it and why to bother. ***Family Practice Management*** 1999;6(1):40-4.

423. Ali RM, Boachie-Adjei O, Rawlins BA. Functional and radiographic outcomes after surgery for adult scoliosis using third-generation instrumentation techniques. ***Spine*** 2003;28(11):1163-9; discussion 1169-70.

424. Blount KJ, Krompinger WJ, Maljanian R, Browner BD. Moving toward a standard for spinal fusion outcomes assessment. ***Journal of Spinal Disorders & Techniques*** 2002;15(1):16-23.

425. Kvien TK, Heiberg T. Patient perspective in outcome assessments--perceptions or something more? ***Journal of Rheumatology*** 2003;30(4):873-6.

426. O'Donnell ML, Creamer M, Bryant RA, Schnyder U, Shalev A. Posttraumatic disorders following injury: an empirical and methodological review. ***Clinical Psychology Review*** 2003;23(4):587-603.

427. Boyd CR, Tolson MA, Copes WS. Evaluating trauma care: the TRISS method. Trauma Score and the Injury Severity Score. ***Journal of Trauma-Injury Infection & Critical Care*** 1987;27(4):370-8.

428. Sullivan MJ, Stanish W, Waite H, Sullivan M, Tripp DA. Catastrophizing, pain, and disability in patients with soft-tissue injuries. ***Pain*** 1998;77(3):253-60.

429. Braithwaite IJ, Boot DA, Patterson M, Robinson A. Disability after severe injury: five year follow up of a large cohort. ***Injury*** 1998;29(1):55-9.

430. Holbrook TL, Anderson JP, Sieber WJ, Browner D, Hoyt DB. Outcome after major trauma: 12-month and 18-month follow-up results from the Trauma Recovery Project. ***Journal of Trauma Injury Infection & Critical Care*** 1999;46(5):765-71.

431. Brenneman FD, Boulanger BR, McLellan BA, Culhane JP, Redelmeier DA. Acute and long-term outcomes of extremely injured blunt trauma victims. ***Journal of Trauma Injury Infection & Critical Care*** 1995;39(2):320-4.

432. Ott R, Holzer U, Spitzenpfeil E, Kastl S, Rupprecht H, Hennig FF. Quality of life after survival of severe trauma. ***Unfallchirurg*** 1996;99(4):267-74.

433. National health survey SF-36 population norms. Canberra: Australian Bureau of Statistics, 1997.

434. Hendriks EJ, Scholten-Peeters GG, van der Windt DA, Neeleman-van der Steen CW, Oostendorp RA, Verhagen AP. Prognostic factors for poor recovery in acute whiplash patients. ***Pain*** 2005;114(3):408-16.

435. Ottosson C, Nyren O, Johansson SE, Ponzer S. Outcome after minor traffic accidents: a follow-up study of orthopedic patients in an inner-city area emergency room. ***Journal of Trauma Injury Infection & Critical Care*** 2005;58(3):553-60.

436. Miettinen T, Airaksinen O, Lindgren KA, Leino E. Whiplash injuries in Finland--the possibility of some sociodemographic and psychosocial factors to predict the outcome after one year. ***Disability & Rehabilitation*** 2004;26(23):1367-72.

437. Luo X, Edwards CL, Richardson W, Hey L. Relationships of clinical, psychologic, and individual factors with the functional status of neck pain patients. ***Value in Health*** 2004;7(1):61-9.

438. Perper JA, Kuller LH, Shim YK. Vehicular crashes and alcohol involvement in drivers at fault, and related fatalities. ***American Journal of Forensic Medicine & Pathology*** 1993;14(3):177-84.

439. Kopec JA, Sayre EC, Esdaile JM. Predictors of back pain in a general population cohort. ***Spine*** 2004;29(1):70-7; discussion 77-8.

440. Woodyard JE. Injury, compensation claims and prognosis: Part II. ***Journal of the Society of Occupational Medicine*** 1980;30(2):57-60.

441. Bryant RA, Harvey AG. Avoidant coping style and post-traumatic stress following motor vehicle accidents. ***Behaviour Research & Therapy*** 1995;33(6):631-5.

442. Ursano RJ, Fullerton CS, Epstein RS, Crowley B, Kao TC, Vance K, et al. Acute and chronic posttraumatic stress disorder in motor vehicle accident victims. ***American Journal of Psychiatry*** 1999;156(4):589-95.

443. Dougall AL, Ursano RJ, Posluszny DM, Fullerton CS, Baum A. Predictors of posttraumatic stress among victims of motor vehicle accidents. ***Psychosomatic Medicine*** 2001;63(3):402-11.

444. Silove D, Brooks R, Steel Z, Blaszczynski A, Hillman K, Tyndall K. Can structured interviews for post-traumatic stress disorder assist clinical decision-making after motor vehicle accidents? An exploratory analysis. ***Comprehensive Psychiatry*** 2005.

445. Davidson JR, Stein DJ, Shalev AY, Yehuda R. Posttraumatic stress disorder: acquisition, recognition, course, and treatment. ***Journal of Neuropsychiatry & Clinical Neurosciences*** 2004;16(2):135-47.

446. Zatzick DF, Kang SM, Muller HG, Russo JE, Rivara FP, Katon W, et al. Predicting posttraumatic distress in hospitalized trauma survivors with acute injuries. ***American Journal of Psychiatry*** 2002;159(6):941-6.

447. Feinstein A, Dolan R. Predictors of post-traumatic stress disorder following physical trauma: an examination of the stressor criterion. ***Psychological Medicine*** 1991;21(1):85-91.

448. PricewaterhouseCoopers. Health, return to work, social and financial outcomes associated with different compensation pathways in NSW: quantitative survey of claimants. Sydney: NSW WorkCover, 2003:159.

449. Peden M, McGee K, Krug EE. Injury: a leading cause of the global burden of disease, 2000. Geneva: World Health Organization, 2002.

450. Peden M, McGee K, Sharma GE. The injury chart book: a graphical overview of the global burden of injuries. Geneva: World Health Organization, 2002.

451. Ghazavi MT, Pritzker KP, Davis AM, Gross AE. Fresh osteochondral allografts for post-traumatic osteochondral defects of the knee. ***Journal of Bone & Joint Surgery - British Volume*** 1997;79(6):1008-13.

452. Hutchinson F, 3rd, Huebner MK. Treatment of os calcis fractures by open reduction and internal fixation. ***Foot & Ankle International*** 1994;15(5):225-32.

453. Geel CW, Flemister AS, Jr. Standardized treatment of intra-articular calcaneal fractures using an oblique lateral incision and no bone graft. ***Journal of Trauma-Injury Infection & Critical Care*** 2001;50(6):1083-9.

454. Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry low back pain disability questionnaire. ***Physiotherapy*** 1980;66(8):271-3.

455. Vernon H, Mior S. The Neck Disability Index: a study of reliability and validity. ***Journal of Manipulative & Physiological Therapeutics*** 1991;14(7):409-15.

456. Spector PE. Job Satisfaction Survey: http//:chuma.cas.usf.edu/~spector/scales/jssovr.html. Accessed 31 May 2004.

457. Bhandari M, Tornetta P, 3rd, Sprague S, Najibi S, Petrisor B, Griffith L, et al. Predictors of reoperation following operative management of fractures of the tibial shaft. ***Journal of Orthopaedic Trauma*** 2003;17(5):353-61.

458. Harris I, Lyons M. Reoperation rate in diaphyseal tibia fractures. ***ANZ Journal of Surgery*** 2005;75(12):1041-4.

459. Karladani AH, Granhed H, Karrholm J, Styf J. The influence of fracture etiology and type on fracture healing: a review of 104 consecutive tibial shaft fractures. ***Archives of Orthopaedic & Trauma Surgery*** 2001;121(6):325-8.

460. Jeavons S. Predicting who suffers psychological trauma in the first year after a road accident. ***Behaviour Research & Therapy*** 2000;38(5):499-508.

461. Singh-Manoux A. Psychosocial factors and public health. ***Journal of Epidemiology & Community Health*** 2003;57(8):553-6; discussion 554-5.

462. Swartzman LC, Teasell RW, Shapiro AP, McDermid AJ. The effect of litigation status on adjustment to whiplash injury. ***Spine*** 1996;21(1):53-8.

463. Maimaris C, Barnes MR, Allen MJ. 'Whiplash injuries' of the neck: a retrospective study. ***Injury*** 1988;19(6):393-6.

464. Hodgson SP, Grundy M. Whiplash injuries: their long-term prognosis and its relationship to compensation. ***Neuro-orthopaedics*** 1989;7:88-91.

465. Ferrari R, Russell AS, Carroll LJ, Cassidy JD. A re-examination of the whiplash associated disorders (WAD) as a systemic illness. ***Annals of the Rheumatic Diseases*** 2005;64(9):1337-42.

466. Holt PL. Stressful life events preceding road traffic accidents. ***Injury*** 1981;13(2):111-5.

# APPENDICES

## Appendix 1. References for articles included in the meta-analysis in Chapter Two.


Agazzi S, Reverdin A, May D. Posterior lumbar interbody fusion with cages: an independent review of 71 cases. ***J. Neurosurg.*** 1999;91(2 Suppl):186-92.

al-Qattan MM, Bowen V, Manktelow RT. Factors associated with poor outcome following primary carpal tunnel release in non-diabetic patients. ***J. Hand Surg. (Br).*** 1994;19(5):622-5.

Albert TJ, Balderston RA, Heller JG, et al. Upper lumbar disc herniations. ***J. Spinal Disord.*** 1993;6(4):351-9.

Albert TJ, Pinto M, Denis F. Management of symptomatic lumbar pseudarthrosis with anteroposterior fusion. A functional and radiographic outcome study. ***Spine*** 2000;25(1):123-9; discussion 30.

Antoniou J, Duckworth DT, Harryman DT, 2nd. Capsulolabral augmentation for the the management of posteroinferior instability of the shoulder. ***J. Bone Joint Surg. Am.*** 2000;82(9):1220-30.

Arcand MA, O'Rourke P, Zeman CA, Burkhead WZ, Jr. Revision surgery after failed subacromial decompression. ***Int. Orthop.*** 2000;24(2):61-4.

Aronson N, Filtzer DL, Bagan M. Anterior cervical fusion by the Smith-Robinson approach. ***J. Neurosurg.*** 1968;29(4):396-404.

Asch HL, Lewis PJ, Moreland DB, et al. Prospective multiple outcomes study of outpatient lumbar microdiscectomy: should 75 to 80% success rates be the norm? ***J. Neurosurg.*** 2002;96(1 Suppl):34-44.

Atlas SJ, Chang Y, Kammann E, Keller RB, Deyo RA, Singer DE. Long-term disability and return to work among patients who have a herniated lumbar disc: the effect of disability compensation. ***J. Bone Joint Surg. Am.*** 2000;82(1):4-15.

Barrett GR, Rook RT, Nash CR, Coggin MR. The effect of Workers' Compensation on clinical outcomes of arthroscopic-assisted autogenous patellar tendon anterior cruciate ligament reconstruction in an acute population. ***Arthroscopy*** 2001;17(2):132-7.

Biondi J, Greenberg BJ. Redecompression and fusion in failed back syndrome patients. ***J. Spinal Disord.*** 1990;3(4):362-9.

Branch CL, Branch CL, Jr. Posterior lumbar interbody fusion with the keystone graft: technique and results. ***Surg. Neurol.*** 1987;27(5):449-54.

Bray RC, Roth JH, Jacobsen RP. Arthroscopic lateral release for anterior knee pain: a study comparing patients who are claiming worker's compensation with those who are not. ***Arthroscopy*** 1987;3(4):237-47.

Buckley R, Tough S, McCormack R, et al. Operative compared with nonoperative treatment of displaced intra-articular calcaneal fractures: a prospective, randomized, controlled multicenter trial. ***J. Bone Joint Surg. Am.*** 2002;84-A(10):1733-44.

Burke FD. Lumbar disc surgery. A review of a series of patients. ***Br. J. Clin. Pract.*** 1976;30(2):29-31.

Campbell E, Whitfield RD. Certain reasons for failure following disk operations. ***N. Y. State J. Med.*** 1947;47:2569-73.

Christie WR, Sprague NF, 3rd, Kim L. Arthroscopic evaluation and treatment of the symptomatic previously operated knee. ***Arthroscopy*** 1988;4(3):194-8.

Concannon MJ, Brownfield ML, Puckett CL. The incidence of recurrence after endoscopic carpal tunnel release. ***Plast. Reconstr. Surg.*** 2000;105(5):1662-5.

Connor PM, Yamaguchi K, Pollock RG, Flatow EL, Bigliani LU. Comparison of arthroscopic and open revision decompression for failed anterior acromioplasty. ***Orthopedics*** 2000;23(6):549-54.

Davies BW, Pennington GA, Fritz AM. Two-portal endoscopic carpal tunnel release: an outcome analysis of 333 hands***. Ann. Plast. Surg.*** 1998;40(5):542-8.

Davis GW, Onik G, Helms C. Automated percutaneous discectomy. ***Spine*** 1991;16(3):359-63.

Dellon AL, Mackinnon SE. Treatment of the painful neuroma by neuroma resection and muscle implantation. ***Plast. Reconstr. Surg.*** 1986;77(3):427-38.

DePalma AF, Rothman RH, Levitt RL, Hammond NL, 3rd. The natural history of severe cervical disc degeneration. ***Acta Orthop. Scand.*** 1972;43(5):392-6.

Deshmukh SC, Givissis P, Belloso D, Stanley JK, Trail IA. Blatt's capsulodesis for chronic scapholunate dissociation. ***J. Hand Surg. (Br).*** 1999;24(2):215-20.

Ellman H, Kay SP. Arthroscopic subacromial decompression for chronic impingement. Two- to five-year results. ***J. Bone Joint Surg. Br.*** 1991;73(3):395-398.

Finnegan WJ, Fenlin JM, Marvel JP, Nardini RJ, Rothman RH. Results of surgical intervention in the symptomatic multiply-operated back patient. Analysis of sixty-seven cases followed for three to seven years. ***J. Bone Joint Surg. Am.*** 1979;61(7):1077-82.

Flemister AS, Jr., Infante AF, Sanders RW, Walling AK. Subtalar arthrodesis for complications of intra-articular calcaneal fractures. ***Foot Ankle Int.*** 2000;21(5):392-9.

Friedman MJ, Brna JA, Gallick GS, et al. Failed arthroscopic meniscectomy: prognostic factors for repeat arthroscopic examination. ***Arthroscopy*** 1987;3(2):99-105.

Frieman BG, Fenlin JM, Jr. Anterior acromioplasty: effect of litigation and workers' compensation. ***J. Shoulder Elbow Surg.*** 1995;4(3):175-81.

Gartsman GM. Arthroscopic acromioplasty for lesions of the rotator cuff. ***J. Bone Joint Surg. Am.*** 1990;72(2):169-80.

Geel CW, Flemister AS, Jr. Standardized treatment of intra-articular calcaneal fractures using an oblique lateral incision and no bone graft. ***J. Trauma*** 2001;50(6):1083-9.

Ghazavi MT, Pritzker KP, Davis AM, Gross AE. Fresh osteochondral allografts for post-traumatic osteochondral defects of the knee. ***J. Bone Joint Surg. Br.*** 1997;79(6):1008-13.

Glowacki KA, Weiss AP. Anterior intramuscular transposition of the ulnar nerve for cubital tunnel syndrome. ***J. Shoulder Elbow Surg.*** 1997;6(2):89-96.

Graham CE. Chemonucleolysis: a double blind study comparing chemonucleolysis with intra discal hydrocortisone: in the treatment of backache and sciatica. ***Clin. Orthop.*** 1976(117):179-92.

Green RM, McNamara J, Ouriel K. Long-term follow-up after thoracic outlet decompression: an analysis of factors determining outcome. ***J. Vasc. Surg.*** 1991;14(6):739-45; discussion 45-6.

Greenough CG, Taylor LJ, Fraser RD. Anterior lumbar fusion. A comparison of noncompensation patients with compensation patients. ***Clin. Orthop.*** 1994(300):30-7.

Greenough CG, Peterson MD, Hadlow S, Fraser RD. Instrumented posterolateral lumbar fusion. Results and comparison with anterior interbody fusion. ***Spine*** 1998;23(4):479-86.

Greenwood J. A study of the causes of failure in the herniated intervertebral disc operation. ***J. Neurosurg.*** 1952;9:15-20.

Gurdjian ES, Webster JE, Ostrowski AZ, Hardy WG, Lindner DW, Thomas LM. Herniated lumbar intervertebral discs - an analysis of 1176 operated cases. ***J. Trauma*** 1961;1:158-76.

Hallock GG, Lutz DA. Prospective comparison of minimal incision "open" and two-portal endoscopic carpal tunnel release. ***Plastic Reconstr. Surg.*** 1995;96(4):941-7.

Hanel DP, Lederman ES. Index transposition after resection of the long finger ray. ***J. Hand Surg. (Am).*** 1993;18(2):271-7.

Hanley EN, Jr., Levy JA. Surgical treatment of isthmic lumbosacral spondylolisthesis. Analysis of variables influencing results. ***Spine*** 1989;14(1):48-50.

Hanley EN, Shapiro DE. The development of low-back pain after excision of a lumbar disc. ***J. Bone Joint Surg. Am.*** 1989;71(5):719-21.

Hawkins RJ, Brock RM, Abrams JS, Hobeika P. Acromioplasty for impingement with an intact rotator cuff. ***J. Bone Joint Surg. Br.*** 1988;70(5):795-7.

Hawkins RJ, Chris T, Bokor D, Kiefer G. Failed anterior acromioplasty. A review of 51 cases. ***Clin. Orthop.*** 1989(243):106-11.

Hawkins RJ, Morin WD, Bonutti PM. Surgical treatment of full-thickness rotator cuff tears in patients 40 years of age or younger. ***J. Shoulder Elbow Surg.*** 1999;8(3):259-65.

Hawkins RJ, Plancher KD, Saddemi SR, Brezenoff LS, Moor JT. Arthroscopic subacromial decompression. ***J. Shoulder Elbow Surg.*** 2001;10(3):225-30.

Herron L, Turner J, Weiner P. Does the MMPI predict chemonucleolysis outcome? ***Spine*** 1988;13(1):84-8.

Herron LD, Turner JA, Novell LA, Kreif SL. Patient selection for lumbar discectomy with a revised objective rating system. ***Clin. Orthop.*** 1996(325):148-55.

Higgs PE, Edwards D, Martin DS, Weeks PM. Carpal tunnel surgery outcomes in workers: effect of workers' compensation status. ***J. Hand Surg. (Am).*** 1995;20(3):354-60.

Hill GM, Ellis EA. Chemonucleolysis as an alternative to laminectomy for the herniated lumbar disc. Experience with patients in a private orthopedic practice. ***Clin. Orthop.*** 1987(225):229-33.

Hostin E, Mont MA, Mayerson JA, Jones LC, Hungerford DS. Total hip arthroplasty in patients receiving Workers' Compensation. ***Clin. Orthop.*** 2000(379):161-8.

Hudgins WR. Compensation and success of lumbar disc surgery. ***Tex. Med.*** 1974;70(10):62-5.

Hulsizer DL, Staebler MP, Weiss AP, Akelman E. The results of revision carpal tunnel release following previous open versus endoscopic surgery. ***J. Hand Surg. (Am).*** 1998;23(5):865-9.

Hutchinson F, 3rd, Huebner MK. Treatment of os calcis fractures by open reduction and internal fixation***. Foot Ankle Int.*** 1994;15(5):225-32.

Iskandar BJ, Fulmer BB, Hadley MN, Oakes WJ. Congenital tethered spinal cord syndrome in adults. ***J. Neurosurg.*** 1998;88(6):958-61.

Javid MJ. Treatment of herniated lumbar disk syndrome with chymopapain. ***JAMA*** 1980;243(20):2043-8.

Javid MJ. Efficacy of chymopapain chemonucleolysis. A long-term review of 105 patients. ***J. Neurosurg.*** 1985;62(5):662-6.

Javid MJ. Signs and symptoms after chemonucleolysis. A detailed evaluation of 214 worker's compensation and noncompensation patients. ***Spine*** 1988;13(12):1428-37.

Javid MJ. A 1- to 4-year follow-up review of treatment of sciatica using chemonucleolysis or laminectomy. ***J. Neurosurg.*** 1992;76(2):184-90.

Jebson PJ, Engber WD. Radial tunnel syndrome: long-term results of surgical decompression. ***J. Hand Surg. (Am).*** 1997;22(5):889-96.

Jebson PJ, Hayes EP, Engber WD. Proximal row carpectomy: a minimum 10-year follow-up study. ***J. Hand Surg. (Am).*** Jul 2003;28(4):561-569.

Jerosch J, Castro WH, Halm H, Muller-Silvergieter G. Long-term results following percutaneous facet coagulation. ***Z. Orthop. Ihre Grenzgeb.*** 1993;131(3):241-7.

Johannsen HV, Andersen NH, Sojbjerg JO, Sneppen O. Arthroscopic subacromial decompression. ***Ugeskr. Laeger*** 1997;159(2):166-70.

Katz JN, Keller RB, Simmons BP, et al. Maine Carpal Tunnel Study: outcomes of operative and nonoperative therapy for carpal tunnel syndrome in a community-based cohort. ***J. Hand Surg. (Am).*** 998;23(4):697-710.

Klekamp J, McCarty E, Spengler DM. Results of elective lumbar discectomy for patients involved in the workers' compensation system. ***J. Spinal Disord.*** 1998;11(4):277-82.

Knox BD, Chapman TM. Anterior lumbar interbody fusion for discogram concordant pain. ***J. Spinal Disord.*** 1993;6(3):242-4.

Kucharzyk DW. A controlled prospective outcome study of implantable electrical stimulation with spinal instrumentation in a high-risk spinal fusion population. ***Spine*** 1999;24(5):465-8.

LaMont RL, Morawa LG, Pederson HE. Comparison of disk excision and combined disk excision and spinal fusion for lumbar disk ruptures. ***Clin. Orthop.*** 1976(121):212-6.

Lauerman WC, Bradford DS, Ogilvie JW, Transfeldt EE. Results of lumbar pseudarthrosis repair. ***J. Spinal Disord.*** 1992;5(2):149-57.

Law JD, Lehman RA, Kirsch WM. Reoperation after lumbar intervertebral disc surgery. ***J. Neurosurg.*** 1978;48(2):259-63.

Lee H, Jackson TA, Wray RC, Jr. Carpal tunnel release through a limited skin incision under direct visualization using a new instrument, the carposcope. ***Plastic Reconstr. Surg.*** 1996;98(2):313-20.

Lew SM, Mehalic TF, Fagone KL. Transforaminal percutaneous endoscopic discectomy in the treatment of far-lateral and foraminal lumbar disc herniations. ***J. Neurosurg.*** 2001;94(2 Suppl):216-20.

Lin SS, Bono CM, Treuting R, Shereff MJ. Limited intertarsal arthrodesis using bone grafting and pin fixation. ***Foot Ankle Int.*** 2000;21(9):742-8.

Lonner JH, Hershman S, Mont M, Lotke PA. Total knee arthroplasty in patients 40 years of age and younger with osteoarthritis. ***Clin. Orthop.*** 2000(380):85-90.

Maciunas RJ, Onofrio BM. The long-term results of chymopapain. Ten-year follow-up of 268 patients after chemonucleolysis. ***Clin. Orthop.*** 1986(206):37-41.

MacKay MA, Fischgrund JS, Herkowitz HN, Kurz LT, Hecht B, Schwartz M. The effect of interposition membrane on the outcome of lumbar laminectomy and discectomy***. Spine*** 1995;20(16):1793-6.

Madan SS, Boeree NR. Comparison of instrumented anterior interbody fusion with instrumented circumferential lumbar fusion. ***Eur. Spine J.*** Dec 2003;12(6):567-575.

Marks RA. Transcutaneous lumbar diskectomy for internal disk derangement: a new indication. ***South. Med. J.*** 2000;93(9):885-90.

Misamore GW, Ziegler DW, Rushton IJ. Repair of the rotator cuff: A comparison of results in two populations of patients. ***J. Bone Joint Surg. Am.*** 1995;77(9):1335-9.

Mobbs RJ, Gollapudi PR, Chandran NK. Outcome following anterior cervical discectomy in compensation patients. ***J. Clin. Neuroscience*** 2001;8(2):124-5.

Mont MA, Antonaides S, Krackow KA, Hungerford DS. Total knee arthroplasty after failed high tibial osteotomy. A comparison with a matched group. ***Clin. Orthop.*** 1994(299):125-30.

Mont MA, Mayerson JA, Krackow KA, Hungerford DS. Total knee arthroplasty in patients receiving Workers' Compensation. ***J. Bone Joint Surg. Am.*** 1998;80(9):1285-90.

Moro JK, Werier J, MacDermid JC, Patterson SD, King GJ. Arthroplasty with a metal radial head for unreconstructible fractures of the radial head. ***J. Bone Joint Surg. Am.*** 2001;83-A(8):1201-11.

Myerson MS, McGarvey WC, Henderson MR, Hakim J. Morbidity after crush injuries to the foot. ***J. Orthop. Trauma*** 1994;8(4):343-9.

Noerdlinger MA, Cole BJ, Stewart M, Post M. Results of pectoralis major transfer with fascia lata autograft augmentation for scapula winging. ***J. Shoulder Elbow Surg.*** 2002;11(4):345-50.

Novak CB, van Vliet D, Mackinnon SE. Subjective outcome following surgical management of upper extremity neuromas. ***J. Hand Surg. (Am).*** 1995;20(2):221-6.

Novak CB, Mackinnon SE, Stuebe AM. Patient self-reported outcome after ulnar nerve transposition. ***Ann. Plast. Surg.*** 2002;48(3):274-80.

Ogilvie-Harris DJ, Wiley AM, Sattarian J. Failed acromioplasty for impingement syndrome. ***J. Bone Joint Surg. Br.*** 1990;72(6):1070-2.

Olsewski JM, Depew AD. Arthroscopic subacromial decompression and rotator cuff debridement for stage II and stage III impingement. ***Arthroscopy*** 1994;10(1):61-8.

Parker LM, Murrell SE, Boden SD, Horton WC. The outcome of posterolateral fusion in highly selected patients with discogenic low back pain. ***Spine*** 1996;21(16):1909-16; discussion 16-7.

Paulos LE, Franklin JL. Arthroscopic shoulder decompression development and application. A five year experience. ***Am. J. Sports Med.*** 1990;18(3):235-44.

Paulos LE, Kody MH. Arthroscopically enhanced "miniapproach" to rotator cuff repair. ***Am. J. Sports Med.*** 1994;22(1):19-25.

Penta M, Fraser RD. Anterior lumbar interbody fusion. A minimum 10-year follow-up. ***Spine*** 1997;22(20):2429-34.

Polk DL. Percutaneous discectomy. ***J. Okla. State Med. Assoc.*** 1994;87(1):16-9.

Raaf J. Some observations regarding 905 patients operated upon for protruded lumbar intervertebral disc. ***Am. J. Surg.*** 1959;97:388-99.

Ricciardi JE, Pflueger PC, Isaza JE, Whitecloud TS, 3rd. Transpedicular fixation for the treatment of isthmic spondylolisthesis in adults. ***Spine*** 1995;20(17):1917-22.

Rish BL. A critique of the surgical management of lumbar disc disease in a private neurosurgical practice. ***Spine*** 1984;9(5):500-4.

Ritts GD, Wood MB, Linscheid RL. Radial tunnel syndrome. A ten-year surgical experience. ***Clin. Orthop.*** 1987(219):201-5.

Rotman MB, Manske PR, Pruitt DL, Szerzinski J. Scaphocapitolunate arthrodesis. ***J. Hand Surg. (Am).*** 1993;18(1):26-33.

Schaffer JL, Kambin P. Percutaneous posterolateral lumbar discectomy and decompression with a 6.9-millimeter cannula. Analysis of operative failures and complications. ***J. Bone Joint Surg. Am.*** 1991;73(6):822-831.

Schnee CL, Freese A, Ansell LV. Outcome analysis for adults with spondylolisthesis treated with posterolateral fusion and transpedicular screw fixation. ***J. Neurosurg.*** 1997;86(1):56-63.

Shinners TJ, Noordsij PG, Orwin JF. Arthroscopically assisted mini-open rotator cuff repair. ***Arthroscopy*** 2002;18(1):21-6.

Siambanes D, Miz GS. Treatment of symptomatic anterior cervical nonunion using the Rogers interspinous wiring technique. ***Am. J. Orthop.*** 1998;27(12):792-6.

Silvers HR. Microsurgical versus standard lumbar discectomy. ***Neurosurgery*** 1988;22(5):837-41.

Silvers HR. Lumbar percutaneous facet rhizotomy.[erratum appears in Spine 1990 Sep;15(9):984]. ***Spine.*** 1990;15(1):36-40.

Silvers HR, Lewis PJ, Asch HL. Decompressive lumbar laminectomy for spinal stenosis. ***J. Neurosurg.*** 1993;78(5):695-701.

Silvers HR, Lewis PJ, Asch HL, Clabeaux DE. Lumbar diskectomy for recurrent disk herniation. ***J. Spinal Disord.*** 1994;7(5):408-19.

Slepian A. Lumbar disk surgery. Long follow-up results from three neurosurgeons. ***N. Y. State J. Med.*** 1966;66(9):1063-8.

Slosar PJ, Reynolds JB, Schofferman J, Goldthwaite N, White AH, Keaney D. Patient satisfaction after circumferential lumbar fusion. ***Spine*** 2000;25(6):722-6.

Slotman GJ, Stein SC. Laminectomy compared with laparoscopic diskectomy and outpatient laparoscopic diskectomy for herniated L5-S1 intervertebral disks. ***J. Laparoendosc. Adv. Surg. Tech. A*** 1998;8(5):261-7.

Snyder GM, Bernhardt M. Anterior cervical fractional interspace decompression for treatment of cervical radiculopathy. A review of the first 66 cases. ***Clin. Orthop.*** 1989(246):92-9.

Sotereanos DG, Varitimidis SE, Giannakopoulos PN, Westkaemper JG. Results of surgical treatment for radial tunnel syndrome. ***J. Hand Surg. (Am).*** 1999;24(3):566-70.

Spangehl MJ, Hawkins RH, McCormack RG, Loomer RL. Arthroscopic versus open acromioplasty: a prospective, randomized, blinded study. ***J. Shoulder Elbow Surg.*** 2002;11(2):101-7.

Stauffer RN, Coventry MB. Posterolateral lumbar-spine fusion. Analysis of Mayo Clinic series. ***J. Bone Joint Surg. Am.*** 1972;54(6):1195-204.

Straub TA. Endoscopic carpal tunnel release: a prospective analysis of factors associated with unsatisfactory results. ***Arthroscopy*** 1999;15(3):269-74.

Strefling MS, Ness DA. Lumbar laminectomy. Experience in 228 consecutive cases. ***Minn. Med.*** 1984;67(3):143-7.

Strickland JW, Idler RS, Lourie GM, Plancher KD. The hypothenar fat pad flap for management of recalcitrant carpal tunnel syndrome. ***J. Hand Surg. (Am).*** 1996;21(5):840-8.

Sutton JC, Jr. Chemonucleolysis in the management of lumbar disc disease. A minimum six-year follow-up evaluation. ***Clin. Orthop.*** 1986(206):56-60.

Taylor VM, Deyo RA, Ciol M, Kreuter W. Surgical treatment of patients with back problems covered by workers compensation versus those with other sources of payment. ***Spine*** 1996;21(19):2255-9.

Taylor VM, Deyo RA, Ciol M, et al. Patient-oriented outcomes from low back surgery: a community-based study. ***Spine*** 2000;25(19):2445-52.

Tomaras CR, Blacklock JB, Parker WD, Harper RL. Outpatient surgical treatment of cervical radiculopathy. ***J. Neurosurg.*** 1997;87(1):41-3.

Tregonning GD, Transfeldt EE, McCulloch JA, Macnab I, Nachemson A. Chymopapain versus conventional surgery for lumbar disc herniation. 10-year results of treatment. ***J. Bone Joint Surg. Br.*** 1991;73(3):481-6.

Tribus CB, Corteen DP, Zdeblick TA. The efficacy of anterior cervical plating in the management of symptomatic pseudoarthrosis of the cervical spine. ***Spine*** 1999;24(9):860-4.

Vaccaro AR, Ring D, Scuderi G, Cohen DS, Garfin SR. Predictors of outcome in patients with chronic back pain and low-grade spondylolisthesis. ***Spine*** 1997;22(17):2030-4; discussion 5.

Weir BK, Jacobs GA. Reoperation rate following lumbar discectomy. An analysis of 662 lumbar discectomies. ***Spine*** 1980;5(4):366-70.

Westkaemper JG, Mitsionis G, Giannakopoulos PN, Sotereanos DG. Wrist arthroscopy for the treatment of ligament and triangular fibrocartilage complex injuries. ***Arthroscopy*** 1998;14(5):479-83.

White AA, 3rd, Southwick WO, DePonte RJ. Cervical spine fusions--psychological and social considerations. ***Arch. Surg.*** 1973;106(2):150-152.

Zucherman J, Hsu K, White A, Wynne G. Early results of spinal fusion using variable spine plating system. ***Spine.*** 1988;13(5):570-579.

## Appendix 2. Coding of explanatory and outcome variables in the Major Trauma Outcome Study.

Measurement units and categories for outcome variables in the MTOS.

| Outcome variable | Units / categories |
|---|---|
| Physical component summary of SF-36 | Score from 0 – 100 (to 1 DP) |
| Mental component summary of SF-36 | Score from 0 – 100 (to 1 DP) |
| Back pain | Integer from 2 - 11 |
| Neck pain | Integer from 2 - 11 |
| Patient satisfaction | Very satisfied |
| | Somewhat satisfied |
| | Somewhat dissatisfied |
| | Very dissatisfied |
| Post-traumatic stress disorder (Scale) | Integer from 17 – 85 |
| Post-traumatic stress disorder (dichotomous) | No (score less than 44) |
| | Yes (score 44 or higher) |

DP = decimal place

Units and categories of explanatory variables for the MTOS.

| Exposure variable | Code | Units / categories |
|---|---|---|
| ***General factors*** | | |
| Age at follow up | AGE | Years |
| Gender | SEX | Male |
| | | Female |
| Time since injury | TIME | Months (12 – 72) |
| Past medical history | PASTILL | No. chronic diseases = 0 |
| | | No. chronic diseases = 1 |
| | | No. chronic diseases = 2 |
| | | No. chronic diseases = 3+ |
| ***Injury severity factors*** | | |
| ISS (Injury Severity Score) | ISS | Integers from 16 - 75 |
| Head injury | HEADINJ | No: AIS (head) of 0, 1 or 2 |
| | | Yes: AIS (head) of 3, 4 or 5 |
| Mechanism | MVA | No (any other mechanism) |
| | | Yes |
| Length of stay in ICU | ICUDAYS | Days |
| ICU admission | ICUCAT | No |
| | | Yes |
| ***Socioeconomic factors*** | | |
| Education (highest level) | EDUCATE | Primary school |
| | | Secondary school |
| | | Certificate or diploma |
| | | Degree |
| Income (annual household) | INCOME | $0 – 30,000 |
| | | $30,001 – 50,000 |
| | | $50,001 – 75,000 |
| | | Over $75,000 |
| Occupational prestige | PRESTIGE | Prestige scale 1.0 – 9.0 |
| Employed at injury | EMPLPRIOR | Yes |
| | | No |
| Employed at follow-up | EMPLNOW | Yes |
| | | No |
| ***Claim-related factors*** | | |
| Claim made, settlement | CLAIM | No claim made |
| | | Claim made, not settled |
| | | Claim made, settled |
| Compensation system | CLAIMTYPE | Workers compensation |
| | | Third party |
| | | Both |
| Lawyer involvement | LAWYER | No |
| | | Yes |
| Blame | FAULTOWN | Self |
| | | Someone else |
| | | Don't know |
| Time to settlement | TTOSETTLE | Months |
| Time since settlement | TSNCESETTL | Months |

**Appendix 3. Final model for the multivariate analysis using PTSD as a dichotomous outcome in the MTOS.**

| Variable | Odds ratio | 95% CI | Wald $chi^2$ (df) | P value |
|---|---|---|---|---|
| Age (years) | | | | |
| 1st quintile (19 – 28) | 3.82 | 1.36 – 10.78 | 6.42 (1) | 0.011 |
| 2nd quintile (29 – 41) | 6.57 | 2.46 – 17.59 | 14.06 (1) | 0.0002 |
| 3rd quintile (42 – 51) | 3.21 | 1.25 – 8.26 | 5.88 (1) | 0.015 |
| 4th quintile (52 – 66) | 1.71 | 0.71 – 4.14 | 1.42 (1) | 0.23 |
| 5th quintile (67 – 91) | 1.00* | | | |
| (overall association) | | | 15.79 (4) | 0.0033 |
| Chronic illness | | | | |
| None | 1.00* | | | |
| 1 | 1.99 | 0.98 – 4.01 | 3.65 (1) | 0.056 |
| 2 | 4.77 | 2.13 – 10.71 | 14.37 (1) | 0.0002 |
| 3 or more | 3.62 | 1.28 – 10.26 | 5.86 (1) | 0.015 |
| (overall association) | | | 15.04 (3) | 0.0018 |
| Current employment | 4.09 | 2.22 – 7.53 | 20.37 (1) | <0.0001 |
| Use of a lawyer | 3.51 | 1.60 – 7.73 | 10.63 (1) | 0.0011 |
| Claim | | | | |
| No claim made | 1.00* | | | |
| Claim settled | 1.04 | 0.44 – 2.45 | 0.0087 (1) | 0.93 |
| Claim not settled | 2.79 | 1.06 – 7.37 | 4.29 (1) | 0.038 |
| (overall) | | | 6.58 (2) | 0.037 |
| Blame for injury | | | | |
| Blame self | 1.00* | | | |
| Don’t know | 2.32 | 1.02 – 5.26 | 4.03 (1) | 0.045 |
| Blame others | 2.33 | 1.14 – 4.76 | 5.34 (1) | 0.021 |
| (overall) | | | 5.89 (3) | 0.053 |

* referent group

**Appendix 4. Significance of claim eligibility (CLAIMENT) when substituted for claim pursuit (CLAIMMADE) in the multivariate analyses of the MTOS.**

Comparison of entitlement to claim (CLAIMENT) and pursuit of claim (CLAIMMADE) when replacing claim in the final model for PCS.

| Variable used | Parameter estimate | Standard error | T value | P value | Adjusted $R^2$ for model |
|---|---|---|---|---|---|
| CLAIMENT | -5.03 | 1.65 | -3.04 | 0.0027 | 0.404 |
| CLAIMMADE | -4.44 | 1.55 | -2.86 | 0.0045 | 0.336 |

Comparison of entitlement to claim (CLAIMENT) and pursuit of claim (CLAIMMADE) when replacing claim in the final model for MCS.

| Variable used | Parameter estimate | Standard error | T value | P value | Adjusted $R^2$ for model |
|---|---|---|---|---|---|
| CLAIMENT | -3.59 | 2.30 | -1.56 | 0.12 | 0.252 |
| CLAIMMADE | -3.05 | 1.97 | -1.55 | 0.12 | 0.203 |

Comparison of estimates of effect for CLAIMENT and CLAIMMADE in the final model using neck pain as the outcome variable.

| Variable used | Parameter estimate | Standard error | T value | P value | Adjusted $R^2$ for model |
|---|---|---|---|---|---|
| CLAIMENT | 0.54 | 0.45 | 1.21 | 0.34 | 0.197 |
| CLAIMMADE | 0.35 | 0.37 | 0.96 | 0.34 | 0.193 |

Comparison of estimates of effect for CLAIMENT and CLAIMMADE in the final model using back pain as the outcome variable.

| Variable used | Parameter estimate | Standard error | T value | P value | Adjusted $R^2$ for model |
|---|---|---|---|---|---|
| CLAIMENT | 1.30 | 0.48 | 2.68 | 0.008 | 0.204 |
| CLAIMMADE | 0.66 | 0.41 | 1.61 | 0.11 | 0.155 |

Comparison of estimates of effect for CLAIMENT and CLAIMMADE in final model for patient satisfaction.

| Variable used | n | Odds ratio | 95% CI | Wald $chi^2$ | P value |
|---|---|---|---|---|---|
| CLAIMENT | 178 | 2.63 | 1.14 – 6.08 | 5.16 | 0.023 |
| CLAIMMADE | 352 | 3.43 | 2.05 – 5.73 | 22.1 | <0.0001 |

Comparison of estimates of effect for CLAIMENT and CLAIMMADE in final model for PTSD as a continuous variable.

| Variable used | Parameter estimate | Standard error | T value | P value | Adjusted $R^2$ for model |
|---|---|---|---|---|---|
| CLAIMENT | 3.86 | 2.96 | 1.30 | 0.20 | 0.423 |
| CLAIMMADE | 3.62 | 2.47 | 1.47 | 0.14 | 0.379 |

Comparison of estimates of effect for CLAIMENT and CLAIMMADE in final model for PTSD as a dichotomous variable.

| Variable used | n | Odds ratio | 95% CI | Wald $chi^2$ | P value |
|---|---|---|---|---|---|
| CLAIMENT | 177 | 1.24 | 0.38 – 4.11 | 0.13 | 0.72 |
| CLAIMMADE | 350 | 1.44 | 0.65 – 3.20 | 0.80 | 0.37 |

### Appendix 5. Associations between the compensation-related variables in Chapters 3 and 4.

Association between compensation status and use of a lawyer in the Major Trauma Outcome Study (Chapter 3).

| | Lawyer used | |
|---|---|---|
| Claim pursued | Yes | No |
| Yes | 116 | 35 |
| No | 12 | 189 |

Association between compensation status and blame in the Major Trauma Outcome Study (Chapter 3).

| | Blame | |
|---|---|---|
| Claim pursued | Others | Self |
| Yes | 91 | 12 |
| No | 22 | 117 |

Association between blame and use of a lawyer in the Major Trauma Outcome Study (Chapter 3).

| | Blame | |
|---|---|---|
| Lawyer used | Others | Self |
| Yes | 73 | 10 |
| No | 39 | 119 |

Association between compensation status and use of a lawyer in the Motor Vehicle Accident Outcome Study (Chapter 4).

| | Lawyer used | |
|---|---|---|
| Claim pursued | Yes | No |
| Yes | 83 | 40 |
| No | 13 | 83 |

Association between compensation status and blame in the Motor Vehicle Accident Outcome Study (Chapter 4).

| | Blame | |
|---|---|---|
| Claim pursued | Others | Self |
| Yes | 94 | 18 |
| No | 17 | 61 |

Association between blame and use of a lawyer in the Motor Vehicle Accident Outcome Study (Chapter 4).

| | Blame | |
|---|---|---|
| Lawyer used | Others | Self |
| Yes | 80 | 6 |
| No | 28 | 71 |

**Appendix 6: Table 3.22. A summary of previous studies that have analysed predictors of outcome after major trauma.**

| Study | Inclusion criteria | n | Time to follow up | Follow-up | Mean age | Mean ISS | % male | Compen-sation measured | SE factors measured | Outcome | Significant predictors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| MacKenzie et al, 1988[395] | All trauma admissions | 479 | 1 y | 80% | 25 | NS | 78 | No | Yes | PDS, NLTCS | ISS, education, income, social support |
| Glancy et al, 1992[392] | All trauma admissions | 441 | 6 m | 42% | 33 | 9 | NS | Yes | Yes | Return to function | Age, ISS, litigation, income replacement, education |
| Ott et al, 1996[432] | Severe trauma | 73 | 1-13 y | 90% | 35 | NS | 73 | No | No | ALOS | Age, injury severity, time since injury, head injury, extremity injury |
| Vazquez et al, 1996[396] | ICU admission | 351 | 2 y | 95% | 31 | 24 | 80 | No | No | QOL | Age, ISS |
| Anke et al, 1997[394] | ISS>15 | 69 | 3 y | 84% | 33 | 25 | 70 | No | Yes | Cognitive and physical impairment | Age, ISS, job status |
| Braithwaite et al, 1998[429] | ISS>15 | 158 | 5 y | 75% | 37 | 27 | NS | No | No | Bull disability score | Extremity injury (no other variables analysed) |
| Holbrook et al, 1999[430] | Admission> 24 hours | 780 | 18 m | 74% | 36 | 13 | 70 | No | Yes | QWB | Age, days in ICU, education |
| Dimopoulou et al, 2004[397] | ICU admission | 87 | 1 y | 74% | 31 | 22 | 85 | No | No | NHP | ISS, head injury |
| Meerding et al, 2004[399] | Accidental injury | 2702 | 9 m | 39% | 25-44 | N/A | 55 | No | Yes | EQ-5D | Age, sex, LOS, ISS, education |
| Vles et al, 2005[398] | ISS>15 | 166 | 1-6 y | 85% | 33 | 23 | 81 | No | No | EQ-5D | Sex, ISS |
| MTOS, 2005 | ISS>15 | 351 | 1-6 y | 61% | 48 | 24 | 72 | Yes | Yes | SF-36 | See tables for PCS and MCS multivariate analysis |

SE = Socioeconomic, NS = not stated, PDS = Post-census Disability Survey, NLTCS = National Long-term Care Survey, ALOS = Aachen Longtime Outcome Score, QOL = Quality of Life, QWB = Quality of Well-Being Scale, NHP = Nottingham Health Profile, EQ-5D = EuroQOL, 5 dimension measure of general health, MTOS = Major Trauma Outcome Study, m = month, y = year, > = greater than.