Increased overall and cause-specific mortality associated with
disability among workers’ compensation claimants with low
back injuries

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Abstract

Background: Mortality tends to be higher among people who do not work than among workers,
but the impact of work-related disability on mortality has not been well studied.

Methods: The vital status through 2015 was ascertained for 14,219 workers with an accepted
workers’ compensation claim in West Virginia for a low back injury in 1998 or 1999. Mortality
among the cohort compared with the West Virginia general population was assessed using
standard life table techniques. Associations of mortality and disability-related factors within the
cohort were evaluated using Cox proportional hazards regression.

Results: Compared to the general population, mortality from accidental poisoning was
significantly elevated among the overall cohort and lost-time claimants. Most deaths from
accidental poisoning in the cohort were due to drug overdoses involving opioids. Mortality
from intentional self-harm was also significantly elevated among lost-time claimants. In internal
analyses, overall mortality and mortality from cancer, heart disease, intentional self-harm, and
drug overdoses involving opioids was significantly associated with lost time. Overall mortality and mortality from drug overdoses involving opioids were also significantly associated with amount of lost time, permanent partial disability, and percent permanent disability. Heart disease mortality was also significantly associated with the amount of lost time.

**Conclusions:** The results suggest that disability itself may impact mortality risks. If confirmed, these results reinforce the importance of return to work and other efforts to reduce disability.

**Keywords**

occupational cohort; mortality; workers’ compensation; work-related disability; opioids

**INTRODUCTION**

The impact of not working on premature mortality has been the subject of an evolving body of literature. Recent studies have reported conflicting results on the effect on overall mortality of unemployment, early retirement, and medically-related disability. Studies examining the contribution of work-related disability specifically on mortality are limited but have reported an excess in premature mortality from workplace injuries and diseases in general.

There have been two broad methodological challenges with much of the prior research. Firstly, mortality among study subjects is usually compared to that of the general population, potentially obscuring or underestimating any difference in mortality because of a healthy worker effect. Secondly, when studying individuals who leave employment on medical grounds, residual confounding may be present, since the disabling medical condition may be the source of an increase in mortality, rather than an effect mediated by the status of disability itself.

We performed a retrospective mortality study using a state-wide workers’ compensation database on a cohort of claimants who had claims accepted for low back strains in calendar years 1998 and 1999. This condition was chosen as low back pain is currently the single greatest cause of years lived with disability (YLD’s) in the United States, with a lifetime prevalence among working Americans estimated to be as high as 63.4%. The primary objectives of the study were to compare the mortality experience of these claimants with that of the general population and compare the mortality experience of claimants with lost work time to those without lost work time injuries. Secondary objectives included analyzing mortality by the weeks of lost work time, whether claimants were permanently disabled, the percent of permanent disability awarded, and whether claimants had surgical treatment.

The outcomes of interest were all-cause mortality, cancer mortality, heart disease mortality, mortality from intentional self-harm, fatal drug overdoses involving opioids, and other analgesic-related mortality. Because fatal drug overdoses involving opioids are not based on underlying cause of death alone and the underlying cause of death for drug overdoses involving opioids is commonly accidental poisoning, mortality from accidental
poisoning was also of *a priori* interest in the analyses comparing the mortality experience of claimants to that of the general population.

**METHODS**

**Cohort description**

The cohort included 14,219 workers with an accepted workers’ compensation claim in the US state of West Virginia for a diagnosis of either lumbar or lumbosacral sprain or strain (International Classification of Diseases, Ninth Revision (ICD-9) codes 847.2 or 846.0) with a date of injury from January 1, 1998 until December 31, 1999. During this time, West Virginia administered a monopolistic workers’ compensation program with no private carriers, and all workers’ compensation claims were administered through the state. Further details about this database have been described elsewhere. One cohort member was excluded from the analysis because the date of birth was unknown.

**Demographic and disability-related factors**

Data on sex and age but not race were available for the cohort. In the analyses, race was assumed to be white for all workers because the general population of West Virginia was estimated to be 96.2% white according to the 2000 census. The following disability-related variables were created from the claims data: lost work time (defined as 4 or more days away from work), number of weeks of lost work time, permanent partial disability status, permanent total disability status, the percent of permanent disability awarded, and surgical treatment. The number of weeks of lost work time was estimated from the disability award start and end dates and censored at the equivalent of 208 weeks (the maximum amount of lost work time awarded). Disability percentages were based on impairment ratings derived from *The American Medical Association Guides to the Evaluation of Permanent Impairment (The AMA Guides)*, fourth edition. Surgical treatment was identified from Current Procedural Terminology (CPT) codes. If a claimant’s data did not include a CPT code for a back surgery, the claimant was assumed not to have had surgical treatment.

**Vital status follow-up**

Vital status was ascertained through December 31, 2015 using linkages to the Social Security Administration, Internal Revenue Service, and National Death Index (NDI). Causes of death were obtained from NDI Plus. Cohort members with a social security number not known to be invalid and not identified as deceased were assumed to be alive as of December 31, 2015 because the sensitivity of the NDI is over 95% when social security numbers are available.

**Analysis**

Descriptive analyses were conducted to summarize data on sex, vital status, and disability-related factors. The mortality experience of the cohort was compared to that in the West Virginia general population using the NIOSH Life Table Analysis System (LTAS.NET). In LTAS.NET, ICD codes for the underlying causes of death were mapped to cause of death categories as described on the NIOSH website (https://www.cdc.gov/niosh/ltas/pdf/)
For each cohort member, person-years-at-risk (PYAR) began on the date of injury associated with the first accepted claim for a low back sprain or strain in 1998 or 1999. PYAR accumulated until the earlier of the date of death or study end date (December 31, 2015). PYAR were stratified by sex and 5-year intervals of age and calendar time and were multiplied by the appropriate cause-specific sex, age, and calendar time mortality rates for whites in West Virginia to calculate the expected number of deaths for that stratum. The expected numbers of deaths were summed across strata to obtain the cause-specific and total expected number of deaths. The standardized mortality ratio (SMR) was calculated as the ratio of the observed to expected number of deaths. Ninety-five percent confidence intervals (CI) were computed for the SMRs assuming a Poisson distribution for observed deaths. These analyses were also conducted restricting the cohort to those having lost-time injuries and those granted some level of permanent disability.

Drug overdoses involving opioids and other analgesic-related deaths are not captured by underlying cause of death alone so SMRs were not calculated for these causes of death. However, these deaths were identified and included in internal analyses if a sufficient number of deaths was observed. Deaths from drug overdoses involving opioids in 1998 were identified by ICD-9 underlying cause of death codes E850-E858, E950.0-E950.5, E962.0, and E980.0-E980.5 in conjunction with ICD-9 multiple cause of death code 965.0. Drug overdoses involving opioids after 1998 were indicated by ICD-10 underlying cause of death code X40-X44, X60-X64, X85, and Y10-Y14 in conjunction with ICD-10 multiple cause of death codes T40.0, T40.1, T40.2, T40.3, T40.4, and T40.6. Other analgesic-related deaths were identified by ICD-9 multiple cause of death codes 965.1, 965.4, 965.5, 965.7, 965.8 and 965.9 and ICD-10 multiple cause of death codes T39.0, T39.1, T39.2, T39.3, and T39.8.

For internal comparisons, Cox proportional hazards regression was used to evaluate the relation between outcomes of a priori interest and disability-related factors, examining one factor at a time. Cox proportional hazards regression involves the formation of risk-sets for each case. Each risk-set contains all cohort members at risk at the age of death of the index case. As a result, a person may appear in multiple risk-sets and a case may appear in risk-sets of other cases. In addition to matching on attained age, risk-sets were also restricted to cohort members of the same sex and birth date (within 5 years) as the case. Cohort members were followed from the date that PYAR began to the date PYAR ended in the life table analyses. Hazard ratios (HRs) were estimated from the maximum partial likelihood, and confidence intervals (CIs) were based on the profile likelihood.

To evaluate temporal patterns in risk, time since injury was also considered. First, Kaplan-Meier curves were estimated stratified by claim-type (lost work time vs non-lost work time). These results were plotted with 95% Hall-Wellner bands and differences between strata were tested with the log-rank test. Second, time since injury was modeled using quadratic splines, with knots placed at 5 and 10 years. Separate splines were also estimated for each claim type (lost work time and no lost work time), and the ratio of these two curves was calculated at each time point. These results (and the 95% CIs) are presented as a graph of the hazard ratio (HR) associated with lost work time by time since an injury. Wald based 95% CIs were calculated from the covariance matrix of parameters of the regression.
All descriptive statistics and internal analyses were conducted in SAS 9.4 (SAS Institute Inc., Cary, NC). Sensitivity analyses were conducted restricting the cohort to 1) workers with a single claim in the data, 2) workers for whom the first accepted claim for a diagnosis of low back sprain or strain in 1998 or 1999 was for a low back sprain or strain only, and 3) workers who resided in West Virginia counties in which the population was over 95% white only according to 2000 census data.13

**Human Subjects’ Protection**

The study was approved by the West Virginia University Institutional Review Board. Informed consent was waived for this records-based study.

**RESULTS**

A total of 14,218 workers contributing 233,314 person-years were included in the main life table analysis. Characteristics of the analysis cohort are shown in Table 1. At the study end date, 9.8% of the cohort was deceased. Causes of death were obtained for 1,390 (99.8%) of the 1,393 cohort members known to have died.

Most (81.4%) of the cohort only had a single accepted claim. The first accepted claim for a low back sprain or strain for over half (57.5%) of the cohort was for low back sprain and/or strain only. Over half (58.8%) of the cohort had lost work time, and 28.2% experienced some level of permanent disability. Only 1% received surgical treatment.

Fewer than five non-opioid analgesic-related deaths and 121 deaths from drug overdoses involving opioids occurred in the cohort. Mortality from other causes of death of a priori interest among the cohort compared to the West Virginia general population is shown in Table 2. Of the 119 deaths from accidental poisoning, 109 (91.6%) were due to drug overdoses involving opioids. Overall mortality (SMR=0.92; 95% CI=0.87–0.97) and mortality from cancer (SMR=0.88; 95% CI=0.79–0.98) and heart diseases (SMR=0.80; 95% CI=0.70–0.91) were significantly lower in the cohort than in the West Virginia general population, consistent with a healthy worker effect. However, when analyses were restricted to workers with lost work time or permanent disability, overall mortality was similar to that of the West Virginia population (lost time: SMR=1.04; 95% CI=0.98–1-11; permanent disability: SMR=1.07; 95% CI 0.98–1.16). Mortality from accidental poisoning was significantly elevated in the overall cohort (SMR=1.62; 95% CI=1.34–1.94), workers with lost work time (SMR=2.02; 95%=CI 1.61–2.50), and workers with permanent disability (SMR=2.78; 95% CI=2.08–3.64). Mortality from intentional self-harm was significantly elevated among workers with lost work time (SMR=1.43; 95% CI=1.06–1.90). The SMR for intentional self-harm was similar for workers with permanent disability, but not significantly elevated (SMR=1.41; 95% CI=0.89–2.11).

Adjusted HRs according to disability-related factors are shown in Table 3. Overall mortality and mortality from drug overdoses involving opioids were significantly associated with lost time, amount of lost time, permanent partial disability, and percent permanent disability but not with surgical treatment. Mortality from cancers and intentional self-harm were significantly associated with lost time only. HRs for intentional self-harm according to

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*Am J Ind Med. Author manuscript; available in PMC 2023 February 27.*
amount of lost time, permanent disability, percent permanent disability, and surgical treatment were also elevated, but were not statistically significant. Heart disease mortality was significantly associated with lost time and amount of lost time.

The results of sensitivity analyses are shown in Supplementary Tables S-1 and S-2. Results for mortality compared to the West Virginia population in sensitivity analyses were not substantially different than those in the main analysis. In internal analyses, results of analyses restricting the cohort to those with a single claim were not substantially different than those in the main analyses. In other sensitivity analyses, in which the confidence intervals were wider, the statistical significance of some findings changed, and the change in the magnitude of some HRs was greater.

In analyses that looked at risk of mortality by time since injury for the entire cohort, adjusted HRs did not vary substantially for most outcomes of a priori interest (data not shown). For drug overdose deaths involving opioids, risk increased over the first four to five years after injury and then remain elevated throughout follow-up (Figure S-1). Similar trends were observed for intentional self-harm, but the magnitude of the increase in risk over the first four to five years after injury was smaller. Trends in HR associated with lost work time by time since which assumes the hazard ratio is constant over-time. A p-value testing whether the hazard is proportional over time is included (Figure S-2). For the outcomes of mortality from all causes and opioids, the hazard ratio does not appear to be constant over time. The hazard of a lost-time claim for all deaths appears to be elevated about 2 years after injury and the hazard of a lost-time claim for opioid deaths appears to be elevated 5–10 years after injury.

Inspection of the Kaplan-Meier curves (Figure S-3) shows how survival rates begin to diverge when lost-time to no lost-time members of the cohort are compared. For overall mortality, this starts at about three years after injury, for cancer-related at six years, and for heart disease-related mortality at eight years.

Separate SMR’s and HR’s for men and women are reported in Supplementary Tables 3 and 4. SMR’s and HR’s for all deaths were generally similar for men and women, although HR’s for intentional self-harm and drug overdoses involving opioids were higher among men. However, the sample size for some of these subgroups, especially among women, was small and the confidence intervals for these specific causes overlap between men and women.

**DISCUSSION**

Statistically significant elevated HRs for overall deaths as well as those specifically from cancer, heart disease, intentional self-harm, and drug overdoses involving opioids were found when lost-time claimants were compared to workers who did not lose time from work because of a low back strain. We found the highest elevations in HRs for deaths from drug overdoses involving opioids, which was elevated by all measures of disability. If confirmed, these findings point to a largely unrecognized and substantial source of mortality from occupational injuries. Advantages of our study included a large sample size, complete capture of all accepted workers’ compensation claims for the population under
study, information on specific causes of death, and an internal control group consisting of workers with claims contemporaneously accepted for the same ICD-9 codes for low back pain but without associated work-related disability.

Our study adds to a growing body of literature on the adverse health effects of a departure from the workplace. However, we are aware of only two other studies reporting an increase in overall mortality associated with disability which is specifically work-related\(^7\),\(^8\) while this is the first study to our knowledge to report such an association for work-related low back injuries.

A challenge and source of controversy in this area of research has been to distinguish between an impact on mortality mediated by the status of disability itself as opposed to an effect from the disabling medical condition.\(^19\),\(^20\) Under the latter explanation, the observed increased mortality among those with disability reflects a selection bias among those granted disability which persists in many studies despite control measures. Recent studies illustrate these competing viewpoints. Wallman et al. studied medical disability and mortality in five population-based cohorts with a total of 6,887 subjects.\(^5\) Musculoskeletal disease was by far the leading cause of disability, accounting for 47.9% of the cases among women and 39.1% among men. Mortality was associated with disability, with greater effects seen the younger the age at which disability was granted. This effect was not felt to be explained by the medical condition which was the source of the disability, other underlying diseases, or differences in a variety of other factors including age, marital status, level of education, or smoking. The investigators proposed as one possible explanation for their findings that the disability pension process itself accounted for the increase in mortality, possibly through a loss of social status and the social network of work resulting in a bereavement-type response. On the other hand, Hult et al. found no increase in mortality among a cohort of 24,369 male Swedish construction workers after excluding 364,763 subjects in an attempt to control for the increased mortality risk from the disabling condition directly.\(^21\) More recently, Olsson et al. argued that, if the disability status itself accounts for the increased mortality, the effect should accrue over time.\(^9\) Therefore, complete correction for selection bias and residual confounders would mean that there would be no difference in mortality between those granted disability and those employed at the time the disability is first granted with a subsequent rise in mortality over time with disability. In their Swedish population-based prospective cohort study of more than 5 million subjects, they reported elevated mortality hazard rates on the first day of disability which persisted even among those without recent hospitalizations. They concluded that residual confounding may explain previous studies reporting an association between disability and increased mortality. We believe our study results are compatible with the notion that the observed increase in mortality is mediated primarily by the status of disability itself, and we offer several lines of evidence in support of this view.

Firstly, our study compared those with the same injury, differing only in whether or not they experienced disability from that injury. Nevertheless, our results could be influenced by several confounders, which we are not able to control for in our study, such as smoking, obesity, and depression which are risk factors for disability and increase mortality from specific causes such as cancer, heart disease, and intentional self-harm.\(^22\),\(^23\) On the other
hand, the follow-up period of our study occurred during a time of increased physician prescribing of opioids and the predictors of disability from low back pain have been found to be predominantly psychosocial variables and not perceived health status. The results of sensitivity analyses limited to those with claims for low back strain only, one claim only, and no surgery all yielded similar results. Interestingly, we did indeed observe a selection bias when the mortality of our overall study cohort was compared to that of the general population. SMR’s for all deaths, cancers, and heart disease for the overall cohort are below unity, consistent with a healthy worker effect. The results from the analysis restricted to those with disability in this comparison were close to the null, suggesting that the effect on mortality from disability was roughly comparable in magnitude to that of the healthy worker effect (Table 2). Mortality from intentional self-harm was elevated among all groups, achieving statistical significance only among claimants with lost-time injuries. The highest elevations in SMRs were observed for accidental poisonings, which were statistically significant among all three groups. The date of injury was used as a surrogate of the claim date when calculating these SMRs. Since the interval between these dates is typically small because of the need to secure claim benefits, this would lead to a slight underestimation in the SMRs.

Secondly, an increase in mortality has been reported in two studies as a generalized phenomenon from work-related diseases and injuries. Scott-Marshall et al. performed a similar retrospective cohort study as ours, comparing workers’ compensation claimants in the Canadian province of Ontario who received a permanent impairment from any cause to controls residing in the same province matched using a complex formula which included age, sex, and earnings. They reported an HR of 1.29 (95% CI 1.13 – 1.45) for women and 1.22 (95% CI 1.15 – 1.28) for men for overall mortality and impairment. Younger age of onset of the impairment was associated with higher mortality. Survival curves showed a divergence at approximately 13 years from the date of injury for women and 15 years for men. A higher impairment (defined as ≥10% by The AMA Guides, edition not specified) compared to a lower impairment was associated with mortality among men but not women. Specific causes of death were not reported. Boden et al. compared the mortality experience of lost-time injuries (defined as greater than 7 days off work) to no lost time injuries among New Mexico workers’ compensation claimants from 1994–2000. All diseases and injuries were used in this study. After excluding death cases, they reported HRs for lost-time injuries of 1.24 for women (95% CI 1.15 – 1.35) and 1.21 (95% CI 1.15 – 1.27) for men. Specific causes of death were not reported in this study. The observation that increased mortality is associated more generally with work-related disability from all causes is again supportive of the disability itself being the primary driver of this effect.

Thirdly, while it may be assumed that the vast majority of cases of low back pain are uncomplicated and would not increase the risk of death, some studies have reported an elevation in mortality. However, most did not address the level of disability or employment status with many performed in geriatric populations unlikely to include large numbers of actively working subjects. Interestingly, one study of Danish twins 70 years of age or older reported an all-cause mortality HR of 1.13 (95% CI 1.06 – 1.21) which became non-significant after adjustment for functional ability and depressive symptoms. Similarly, in a recent study of women over 65 years of age, those with frequent persistent back
pain were found to have an adjusted all-cause mortality HR of 1.24 [95% CI, 1.11–1.39], a cancer-specific mortality HR of 1.33 [95% CI, 1.03–1.71], and a cardiovascular disease-specific mortality HR of 1.34 [95% CI, 1.12–1.62].\textsuperscript{26} A mediation analyses reported that 98% of the observed all-cause mortality elevation was explained by measures of disability, such as activities of daily living (ADL). Again, these findings suggest that the level of function accounts for those studies reporting an elevation in mortality from low back pain, either in whole or in part.

Further, we found that mortality increased with the amount of disability, as measured by both the duration of lost-time from work and the amount of the permanent disability award. Inspection of the survival curves indicates greater divergence in mortality over time, starting at approximately 3 years after the date of injury, when those with lost-time low back injuries are compared to those without lost-time (Figure S-3). This accrual of mortality with time from the date of injury is as expected if the increased mortality is mediated by the status of disability rather than a direct effect from injury.

The mechanism by which work-related disability may increase mortality is not known, although factors such as loss of social support, decreased self-worth, reduced or lost income, and reduced physical activity levels may all mediate such a potential relationship. Although psychosocial variables appear to be more predictive, some studies have reported an association between measures of poor self-reported health and well-being with chronic disability from low back pain.\textsuperscript{27} Intriguingly, one longitudinal study of chronic low back pain in a primary care setting found that impaired fasting glucose tolerance and a higher BMI were predictive of disability one year later.\textsuperscript{28} We also found an association between work-related disability and fatal drug overdoses involving opioids. Applebaum \textit{et al.} recently reported the results of a follow-up study from the New Mexico workers’ compensation cohort which found an HR of 2.63 (95% CI = 1.91–3.64) for combined drug-related and suicide mortality among women and an HR of 1.42 (95% CI = 1.13–1.79) among men comparing lost-time to no lost-time injuries. Circulatory disease mortality was also elevated for men with an HR = 1.25 (95% CI = 1.05–1.50).\textsuperscript{29} In contrast, we did not observe significantly different HR’s between women and men, either by lost-time injury or other measures of disability (Table S-3). An increase in opioid-related overdose deaths has also been associated with disability in several previous studies, including those who are unemployed,\textsuperscript{30} with physical disabilities,\textsuperscript{30} and Medicare beneficiaries under 65 years of age likely receiving Social Security Disability.\textsuperscript{31} The basis for the relationship between disability and opioid-related overdose is also not well understood, and there are conflicting viewpoints on whether the use of opioids for patients with chronic low back leads to disability or vice versa. King \textit{et al.} noted that, from 1997–2013, the drug poisoning overdose mortality rate and the percentage of awards for Social Security Disability to workers with a musculoskeletal diagnosis both increased in the United States, even though the percentage of adults reporting persistent low back pain over the previous 3 months remained stable during this time.\textsuperscript{32} They hypothesized that this may reflect a “medicalization of social support” in which individuals were incentivized to seek out opioid prescriptions for established conditions in order to demonstrate a higher level of impairment needed to qualify for disability.
On the other hand, several longitudinal studies in different countries have found that an early prescription for opioids for low back pain is associated with a reduced likelihood of return to work and an increased risk of subsequent disability.\textsuperscript{33–36} These studies report that those prescribed opioids early after a low back strain differed significantly from those who were not prescribed opioids in several baseline measures of characteristics including greater self-reported disability, distress, fear of movement, catastrophizing, and lower self-efficacy. Such psychological factors therefore represent shared predictive factors for both opioid use and disability among patients with low back pain.\textsuperscript{22} Pre-injury use of opioids has also been associated with an increased risk of a workers’ compensation claim.\textsuperscript{37} In our study, we do not have data to determine whether opioids were initiated before or after the date of injury.

In the United States, a cross-sectional study based on data from the National Survey on Drug Use and Health found that disability related to ADL’s, but not higher-level instrumental ADL’s, was correlated with opioid misuse.\textsuperscript{38} The correlation between opioid misuse and ADL’s was attenuated when self-reported health status measures were included in the regression model. Opioid misuse was also associated with a variety of measures of social engagement, including unemployment. Our study is not able to distinguish between the source of opioids, whether legally prescribed or obtained illicitly. A cross-sectional study based on the US National Health and Nutrition Examination Survey (NHANES) found that a diagnosis of chronic low back pain was associated with higher odds of using illicit drugs including marijuana, cocaine, heroin, and methamphetamine.\textsuperscript{39} Prescription opioid analgesic use was also found to be more common among those with a history of illicit drug use.

Since the risk of death from both drug overdose involving opioids and intentional self-harm increased over the first four to five years after injury and then remained elevated throughout follow-up in our study, we believe that, similar to our findings for overall mortality, that it is the disability status which is the potential antecedent risk factor for these specific causes of death.

In summary, we report an increase in overall mortality and deaths from cancer, heart disease, intentional self-harm and opioid overdoses associated with disability from work-related low back strains. These results add to a growing body of literature pointing to the status of disability itself as the primary cause of both an overall decline in health and an increased risk of death from opioid overdose. Case and Deaton have drawn broad attention to an increase in overall mortality as well as deaths from drug overdoses, suicides, and alcohol-related liver mortality (so called “deaths of despair”) in the United States, particularly among whites with lower levels of education.\textsuperscript{40} They have proposed a model of “cumulative disadvantage” in which declining labor market participation triggers a cascade of negative social effects. It seems likely that this association is mediated by multiple factors. Some of these can be regarded as endogenous to the injured worker, may be present at the time of the initial injury, and would include factors such as catastrophizing, unfavorable self-perceptions of health and psychological status. Such variables may represent risks for both long-term disability and increased mortality. Additional exogenous risk factors from the disability status itself could accrue over time from being disabled, such as reduced physical activity, low self-worth and social isolation.
If confirmed, these results reinforce the need to see disability from work as an urgent and growing public health problem and the importance of prioritizing return to work efforts through aggressive and comprehensive rehabilitation efforts. Since opioids have been found to be ineffective in treating patients with chronic low back pain, our findings add to the reasons why these therapies are contraindicated in this setting. Further research should investigate other common non-fatal causes of work-related disability to determine if similar associations with mortality exist. In addition, prospective cohort studies designed to specifically address the complex factors impacting the association between work-related disability and mortality would both clarify the nature of this relationship and identify modifiable risk factors for targeted prevention.

**Supplementary Material**

Refer to Web version on PubMed Central for supplementary material.

**ACKNOWLEDGMENTS**

The authors acknowledge and thank the West Virginia Offices of the Insurance Commissioner for providing workers’ compensation data and the National Death Index for providing death data for this study. This study was funded by the intramural research program of the National Institute for Occupational Safety and Health.

**REFERENCES**


Table 1:
Characteristics of the study population.

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<tr>
<th></th>
<th>N (%)</th>
<th>Median (Interquartile range)</th>
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<tbody>
<tr>
<td>Number of workers in analysis</td>
<td>14,218</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>8,948 (62.9%)</td>
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<tr>
<td>Female</td>
<td>5,270 (37.1%)</td>
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<tr>
<td>Vital status (as of 12/31/2015)</td>
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<tr>
<td>Alive</td>
<td>12,825 (90.2%)</td>
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<tr>
<td>Deceased</td>
<td>1,393 (9.8%)</td>
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<tr>
<td>Age at injury</td>
<td></td>
<td>37.7 (29.1–45.8)</td>
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<tr>
<td>Lost work time(^a)</td>
<td>8,365 (58.8%)</td>
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<tr>
<td>Number of lost work days(^a) (among those with lost work time)</td>
<td>112 (14–328)</td>
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<tr>
<td>Permanent disability(^a)</td>
<td></td>
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<tr>
<td>Partial</td>
<td>3,944 (27.7%)</td>
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<tr>
<td>Total</td>
<td>69 (0.5%)</td>
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<tr>
<td>Percent permanent disability(^a) (among those with permanent disability)</td>
<td>10 (6–16)</td>
<td></td>
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<tr>
<td>Surgical treatment</td>
<td>146 (1.0%)</td>
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<tr>
<td>Single claim</td>
<td>11,571 (81.4%)</td>
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<tr>
<td>First claim for low back sprain/strain only(^b)</td>
<td>8,175 (57.5%)</td>
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<tr>
<td>Over 95% of county white(^c)</td>
<td>7,766 (54.6%)</td>
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</table>

\(^a\)Lost work time missing for 1,310 cohort members, number of lost work days missing for 107 cohort members with lost work time, permanent disability status missing for 1,395 cohort members, percent permanent disability missing for 50 cohort members with permanent disability, and over 95% of county white in 2000 census missing for 1,335 cohort members.

\(^b\)First claim for low back sprain or strain was for low back sprain and/or strain only

\(^c\)Over 95% of the claimant county was white only according to the 2000 census
Table 2:

Mortality among workers with a claim for low back sprain or strain (1998–2015, West Virginia Referent Rates).^a^  

<table>
<thead>
<tr>
<th></th>
<th>Overall cohort (N=14,218)</th>
<th>Cohort members with lost work time (N=8,365)</th>
<th>Cohort members with permanent disability^b^ (N=4,013)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OBS</td>
<td>SMR</td>
<td>95% CI</td>
</tr>
<tr>
<td>All deaths</td>
<td>1,393</td>
<td>0.92</td>
<td>0.87–0.97</td>
</tr>
<tr>
<td>All cancers</td>
<td>353</td>
<td>0.88</td>
<td>0.79–0.98</td>
</tr>
<tr>
<td>Heart diseases</td>
<td>239</td>
<td>0.80</td>
<td>0.70–0.91</td>
</tr>
<tr>
<td>Intentional self-harm</td>
<td>65</td>
<td>1.14</td>
<td>0.88–1.45</td>
</tr>
<tr>
<td>Accidental poisoning</td>
<td>119</td>
<td>1.62</td>
<td>1.34–1.94</td>
</tr>
</tbody>
</table>

OBS, observed number of deaths; SMR, standardized mortality ratio; CI, confidence interval


^b^Permanent partial or permanent total disability
Table 3:

Adjusted hazard ratios (HRs) for selected outcomes according to disability-related factors.\(^a\)

<table>
<thead>
<tr>
<th></th>
<th>All deaths</th>
<th>All cancers</th>
<th>Heart diseases</th>
<th>Intentional self-harm</th>
<th>Drug overdoses involving opioids</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
</tr>
<tr>
<td>Lost time</td>
<td>1.44 1.27–1.63</td>
<td>1.41 1.10–1.81</td>
<td>1.42 1.06–1.92</td>
<td>1.85 1.02–3.37</td>
<td>1.89 1.22–2.93</td>
</tr>
<tr>
<td>Weeks of lost time (per 100 weeks)</td>
<td>1.27 1.14–1.41</td>
<td>1.05 0.84–1.32</td>
<td>1.35 1.06–1.71</td>
<td>1.53 0.94–2.50</td>
<td>1.73 1.23–2.44</td>
</tr>
<tr>
<td>Permanent partial disability (vs. no disability)</td>
<td>1.25 1.12–1.40</td>
<td>0.98 0.78–1.23</td>
<td>1.25 0.95–1.63</td>
<td>1.28 0.75–2.18</td>
<td>2.31 1.59–3.37</td>
</tr>
<tr>
<td>Permanent total disability (vs. no disability)</td>
<td>1.11 0.61–2.03</td>
<td>1.37 0.51–3.71</td>
<td>1.38 0.43–4.50</td>
<td>3.36 0.46–24.82</td>
<td>3.16 0.43–23.04</td>
</tr>
<tr>
<td>Percent permanent disability (per 10%)</td>
<td>1.10 1.03–1.17</td>
<td>0.97 0.85–1.11</td>
<td>1.13 0.98–1.31</td>
<td>1.27 0.96–1.68</td>
<td>1.40 1.14–1.71</td>
</tr>
<tr>
<td>Surgical treatment</td>
<td>0.90 0.55–1.48</td>
<td>0.40 0.10–1.61</td>
<td>0.92 0.29–2.87</td>
<td>1.46 0.20–10.56</td>
<td>0 NC</td>
</tr>
</tbody>
</table>

HR, hazard ratio; CI, confidence interval; NC, not calculated as no cases exposed

\(^{a}\)Models were adjusted for age (time scale) and for sex and birth date (±5 years) by matching. The number of observed cases in these analyses ranged from 60 to 1295, depending on the death category and disability-related factor in the model.

\(^{b}\)Permanent partial or permanent total disability