

Published in final edited form as:

JAMA Psychiatry. 2014 March ; 71(3): 326–333. doi:10.1001/jamapsychiatry.2013.3935.

Suicide, Fatal Injuries, and Other Causes of Premature Mortality in Patients With Traumatic Brain Injury:

A 41-Year Swedish Population Study

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Abstract

IMPORTANCE—Longer-term mortality in individuals who have survived a traumatic brain injury (TBI) is not known.

OBJECTIVES—To examine the relationship between TBI and premature mortality, particularly by external causes, and determine the role of psychiatric comorbidity.

DESIGN, SETTING, AND PATIENTS—We studied all persons born in 1954 or later in Sweden who received inpatient and outpatient *International Classification of Diseases*-based diagnoses of TBI from 1969 to 2009 (n = 218 300). We compared mortality rates 6 months or more after TBI to general population controls matched on age and sex (n = 2 163 190) and to unaffected siblings of patients with TBI (n = 150 513). Furthermore, we specifically examined external causes of death (suicide, injury, or assault). We conducted sensitivity analyses to investigate whether mortality rates differed by sex, age at death, severity (including concussion), and different follow-up times after diagnosis.

MAIN OUTCOMES AND MEASURES—Adjusted odds ratios (AORs) of premature death by external causes in patients with TBI compared with general population controls.

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Author Contributions:

Dr Fazel had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Analysis and interpretation of data: Fazel, Wolf, Pillas, Lichtenstein.

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Statistical analysis: Fazel, Wolf, Pillas, Lichtenstein.

Obtained funding: Fazel, Lichtenstein, Långström.

Administrative, technical, or material support: Fazel, Wolf, Lichtenstein, Långström.

Study supervision: Fazel.

Additional Contributions: Marcus Boman, MSc, provided data management.

Conflict of Interest Disclosures: None reported.

RESULTS—Among those who survived 6 months after TBI, we found a 3-fold increased odds of mortality (AOR, 3.2; 95% CI, 3.0-3.4) compared with general population controls and an adjusted increased odds of mortality of 2.6 (95% CI, 2.3-2.8) compared with unaffected siblings. Risks of mortality from external causes were elevated, including for suicide (AOR, 3.3; 95% CI, 2.9-3.7), injuries (AOR, 4.3; 95% CI, 3.8-4.8), and assault (AOR, 3.9; 95% CI, 2.7-5.7). Among those with TBI, absolute rates of death were high in those with any psychiatric or substance abuse comorbidity (3.8% died prematurely) and those with solely substance abuse (6.2%) compared with those without comorbidity (0.5%).

CONCLUSIONS AND RELEVANCE—Traumatic brain injury is associated with substantially elevated risks of premature mortality, particularly for suicide, injuries, and assaults, even after adjustment for sociodemographic and familial factors. Current clinical guidelines may need revision to reduce mortality risks beyond the first few months after injury and address high rates of psychiatric comorbidity and substance abuse.

Traumatic brain injury (TBI) is a substantial cause of disability with high societal costs.^{1,2} In the United States, TBI is the primary or secondary diagnosis in more than 2 million emergency department visits annually,³ with 3.2 million persons, or 1% of the population, living with long-term disability from TBI.⁴ In the European Union, there are approximately 1 million hospitalizations after TBIs annually.⁵ The public health burden may increase in coming decades because road traffic collisions, a leading TBI cause and ninth largest contributor to disability-adjusted life-years globally, are estimated to become the third largest contributor by 2030.⁶ In addition, large numbers of veterans have sustained TBIs.^{7,8}

Traumatic brain injury-related mortality is high. In the United States, approximately 50 000 deaths annually are directly related to TBIs.³ There are a number of uncertainties regarding TBI mortality. First, overall standardized mortality ratios vary from 2 to 7.⁹⁻¹¹ Second, although high suicide rates have been reported in TBI patients,^{9,12} little is known about other external causes of death. Third, prior work reached different conclusions about longer-term mortality.^{9,13,14} Fourth, previous studies highlighted physical comorbidity as a mortality risk factor^{9,11} but did not examine associations with psychiatric comorbidity. Tracking mortality trends is important because most patients survive the immediate consequences and associated injuries of TBI and are discharged from the hospital (estimated at 300 000 annually in the United States³).

We used 41 years of high-quality Swedish population registers to address these uncertainties. First, we calculated premature death rates after a TBI, particularly from external causes. After the current emphasis on population-based approaches to reduce mortality and morbidity,¹⁵ we focused on mortality before 56 years of age, when prevention could substantially contribute to public health. Second, we examined the association of psychiatric and substance abuse comorbidity with external causes of death. Traumatic brain injury may increase the risk of mental illness in the medium¹⁶ and longer term,¹⁷ especially depression^{18,19} and substance abuse.^{12,20} Hence, clarifying the contribution of psychiatric disorders may enable more targeted preventive strategies to reduce premature mortality. Third, we investigated whether TBI is an independent risk factor for premature mortality. We did this by controlling for demographic factors, psychiatric comorbidity, and co-

occurring physical injuries but also by comparing individuals with TBI with their unaffected siblings, a powerful approach to adjust for familial (genetic and early environmental) confounding.

Methods

The Regional Ethics Committee at the Karolinska Institutet approved the study. Data were merged and anonymized by an independent government agency, and the code linking the personal identification numbers to the new case numbers was destroyed immediately after merging. Therefore, informed consent was not required.

Study Setting

We linked longitudinal, nationwide population-based registers in Sweden: the National Patient Register (held at the National Board of Health and Welfare), the National Censuses from 1970 and 1990 (Statistics Sweden), the Multi-Generation Register (Statistics Sweden), and the National Cause-of-Death Register (Statistics Sweden). The Multi-Generation Register connects each person born in Sweden in 1933 or later and ever registered as living in Sweden after 1960 to their parents.²¹ For immigrants, similar information exists for those who became citizens of Sweden before 18 years of age. In Sweden, all residents, including immigrants, have a unique personal identifier used in all national registers, thus enabling data linkage. We selected the cohort of individuals born from 1954 to 2009 and followed up from 1969 to 2009 (n = 7 238 800).

Individuals With TBI

We identified TBI patients from the National Patient Register, which includes individuals admitted to any hospital (starting from 1969 and with national coverage from 1973) or having outpatient appointments with specialist physicians (since 2001).²² Cases had at least 1 patient episode (primary, secondary, or additional diagnoses) according to the *International Classification of Diseases (ICD)* using the Centers for Disease Control and Prevention definition (Author Table 1).²³

Four markers of severity were investigated: (1) inpatient status, (2) overnight hospitalization, (3) moderate to severe TBI (based on *ICD* codes (Author Table 1), which may also provide information on clinically distinct subgroups),²⁴ and (4) concussion (n = 333 118, not overlapping with main analyses).

Outcome Measures

Mortality data by *ICD* chapter were retrieved for all individuals who died from 1969 to 2009. The National Cause-of-Death Register is based on death certificates and covers more than 99% of all deaths.²⁵ In line with previous work,²⁶ uncertain suicides were included as suicides because exclusion could underestimate rates.²⁷

To exclude immediate causes of death that could have caused the TBI, main analyses were restricted to deaths at least 6 months after a TBI. This approach enabled investigation of causal pathways from TBI to mortality by minimizing the risk of reverse causality

associated with shorter time frames. The 6-month time frame was conservative; sensitivity analyses were also conducted with TBI patients who survived 1 week, 1 month, 1 year, or 5 years after diagnosis.

Diagnostic Validity

Swedish patient register data have good to excellent validity for a range of conditions: injuries,²⁸ acute stroke,²⁹ Guillain-Barré syndrome,³⁰ bipolar disorder,³¹ and schizophrenia.^{32,33} Overall, positive predictive values for various inpatient register diagnoses are 85% to 95%.³⁴ Little is known about comorbidity, although fair to moderate agreement for comorbid substance abuse was found in schizophrenia ($\kappa = 0.37$, $P < .001$, 68% full agreement).³⁵

Control Populations

For each case, 10 general population controls without TBI were matched individually by birth year and sex. Controls had to be alive at the time of the matching date.

Sibling Control Studies

Using the Multi-Generation Register, we identified individuals with TBI who also had 1 or more full siblings without TBI. Individuals with TBI were compared for risk of premature death with unaffected full siblings of both sexes with adjustments for age and sex in the analyses. Thus, all potential sibling pairs were investigated.

Sociodemographic and Psychiatric Covariates

We used mean lifetime disposable income (divided into thirds), dichotomized into lowest tertile vs top 2 tertiles. Where unavailable, family lifetime disposable income or parents' lifetime disposable income was used. Single marital status was defined as being unmarried at the end of follow-up, and immigrant status as being born outside Sweden. Missing data were not replaced by imputation or other methods. Data were extracted for all cases and controls on all inpatient and outpatient diagnoses with principal or comorbid diagnoses of alcohol or drug abuse or dependence, depression and related mood disorders, and any psychiatric disorder (Author Table 1).

Statistical Analysis

We estimated the risk of premature death after having been diagnosed as having TBI with matched or sibling controls^{24,35} using the clogit command in Stata statistical software, version 12 (StataCorp). The clogit command fits conditional (fixed-effects) logistic regression models to matched case-control groups. Traumatic brain injury patients and controls were followed up from the same time point (ie, from 6 months after TBI). We included 3 confounders (low income, single marital status, and immigrant status) on theoretical grounds based on related work on other neuropsychiatric disorders^{35,36} and tested whether they were each associated (at $P < .05$) with caseness and outcome measures, respectively.³⁷ Separately, we adjusted for emigration rates after TBI. In sensitivity analyses, we stratified by sex, psychiatric comorbidity (lifetime and separately for preexisting and new diagnoses), and co-occurring injuries at the time of TBI (Author Table

1). The STROBE (*Strengthening the Reporting of Observational Studies in Epidemiology*) guidelines were followed (Author Table 2).

Results

Our original sample contained 220 871 TBI patients and 2 189 174 controls. After excluding individuals who died within 6 months of diagnosis, we compared 218 300 TBI patients with 2 163 190 age- and sex-matched general population controls without TBI. Moreover, we identified 150 513 TBI patients who had unaffected siblings ($n = 237\,535$) (also excluding deaths in the first 6 months). Table 1 provides sociodemographic data on TBI patients and both comparison groups. The TBI patients were followed up for a median of 4 years (interquartile range, 2-7 years).

We identified 11 053 premature deaths after TBI. Of these, 2378 patients (21.5%) died 6 months or later after diagnosis. Compared with the matched general population controls, the unadjusted odds ratio (OR) of premature death among TBI patients was 3.6 (95% CI, 3.5-3.8). After adjustment for sociodemographic confounders, the OR was 3.2 (95% CI, 3.0-3.4) (Table 2). After adjusting for emigration after TBI, no differences in mortality risk were found (data not shown). When compared with their unaffected siblings, adjusted odds ratios (AORs) remained significantly increased but attenuated for all-cause mortality (AOR, 2.6; 95% CI, 2.3-2.8), with no significant differences by sex (interaction test: $P = .29$).

External Causes

Risks for all causes of premature death were elevated (eTable 1 in the Supplement). The largest category was from external causes ($n = 1156$; 48.6% of all deaths after 6 months; AOR, 3.8; 95% CI, 3.5-4.1; Table 2), including injuries ($n = 574$; AOR, 4.3; 95% CI, 3.8-4.8), suicide ($n = 522$; AOR, 3.3; 95% CI, 2.9-3.7), and assault ($n = 52$; AOR, 3.9; 95% CI, 2.7-5.7). Within the injury category, the number of deaths from both nonvehicle injuries (AOR, 5.2; 95% CI, 4.5-6.1) and vehicle collisions (AOR, 3.2; 95% CI, 2.7-3.8) was elevated. Within suicides, 31.4% were classified as uncertain suicides with higher odds (AOR, 4.6; 95% CI, 3.7-5.7) compared with certain suicides (AOR, 2.9; 95% CI, 1.5-3.3). As with all-cause mortality, in sibling comparisons, the odds remained significantly elevated but attenuated (Table 2).

Psychiatric Illness

We found increased rates of psychiatric disorders in TBI patients, both before and after TBI and specifically for alcohol and drug use disorders and for depression (Table 3). Overall, 38 374 TBI patients (17.6%) had a lifetime diagnosis of psychiatric comorbidity. Of those with any lifetime psychiatric and substance abuse comorbidity, 1461 (3.8%) died during the follow-up period (6 months after TBI) compared with 917 (0.5%) of those without any psychiatric diagnoses (eFigure 1 in the Supplement). Of those with substance abuse, 6.2% died compared with 0.6% of those without a substance abuse diagnosis. Preexisting psychiatric diagnoses appeared to have a stronger effect on odds of mortality than post-TBI diagnoses (eTable 2 in the Supplement). Compared with those with neither TBI nor lifetime psychiatric diagnoses, odds of premature mortality in patients with both TBI and psychiatric

comorbidity, and specifically substance abuse and depression, were substantially elevated, ranging from 8 to 24 (Table 4). We also investigated these effects using sibling controls and found them to be similar but attenuated (AORs, 5-11; Author Table 3). In addition, after exclusion of cases and controls with psychiatric diagnoses, odds of mortality remained significantly raised in TBI patients.

Sensitivity Analyses

The odds of dying prematurely were higher in women than in men, although the 95% CIs mostly overlapped (Table 5). Differences were found in absolute mortality rates by TBI subtype: 0.9% of TBI patients with a cerebral edema subdiagnosis died of external causes compared with 3.3% with focal TBI and 3.1% with hemorrhagic TBI ($\chi^2_2 = 75.2, P < .001$). No discernible differences were found by age of death using age bands. Odds of premature death were generally higher in those with markers of increased TBI severity for all-cause mortality and injuries but not for suicide. In addition, when stratified by co-occurring injury status, premature mortality remained significantly elevated in TBI without these injuries (Table 5).

Discussion

In this longitudinal study of 218 300 patients with TBI compared with 2 163 190 general population controls, we investigated causes of death during 41 years in the Swedish population. We found that of the 2378 deaths that occurred 6 months or more after diagnosis, approximately half were due to external causes, almost entirely injuries and suicides. We report 3 principal findings. First, after excluding deaths in the first 6 months, there was a 3-fold increase in odds compared with general population controls after adjustment for sociodemographic factors. This increased mortality risk remained for many years after the TBI. Even 5 years after TBI, odds of all-cause mortality and suicide were 3-fold higher than in the general population. Second, we found evidence that suggested that TBI is an independent risk factor for premature mortality. This finding was demonstrated in different ways. We found higher mortality rates in TBI patients compared with general population controls after adjustment for sociodemographic factors. In addition, even when psychiatric comorbidity was excluded in patients and controls, TBI patients had a substantially increased risk of premature death. This increased risk was also found when we excluded patients with co-occurring injuries sustained at the same time as their head injury. The increased mortality risk of TBI patients compared with their unaffected siblings also supported this conclusion, an approach that accounts for residual confounding.

The third principal finding was the strong associations reported between premature deaths and both psychiatric disorder and substance abuse, with 61% of premature deaths in TBI patients having a lifetime psychiatric or substance abuse diagnosis. In terms of absolute rates, we identified high-risk groups, especially those with more severe forms of TBI. Among those with moderate to severe TBI and psychiatric or substance abuse comorbidity, 1 in 12 died prematurely.

Several implications arise from these findings. First, detection and subsequent management of psychiatric comorbidity should be an integral component of any strategy to reduce

premature mortality for TBI patients, particularly from suicide and injuries. A total of 18% of TBI patients had a lifetime diagnosis of a psychiatric disorder or substance abuse; 5% had been diagnosed as having depression. Second, high comorbid substance abuse rates (8%) suggest that integration of health services for head-injured patients with specialist substance abuse treatment services may need consideration. Closer integration of substance abuse, psychiatric, and other relevant services supports the case for centralizing services for head-injured patients.^{16,38} Of 6 relevant clinical guidelines,³⁸ none of the latest updates discuss psychiatric assessment and management, or how and when substance use management should be undertaken, apart from recommending hospital admission in patients with acute intoxication with alcohol and drugs (Author Table 4). One 1998 guideline³⁹ recommends admission if self-harm is suspected, but no information on the assessment of suicide risk is provided. Consideration should be given to mental health assessments and timely liaison with psychiatry in patients at high risk of suicide.⁴⁰ Third, preventing TBI is a priority, and the role of public health interventions to address underlying causes requires attention,⁴¹ including designing roads,⁴² bicycle helmet safety,⁴³ and traffic-law enforcement.⁴⁴

We found both elevated medium- and longer-term risks of premature mortality. Even 5 years after diagnosis, the elevated risks remained for suicide (AOR, 2.7; 95% CI, 2.3-3.2) and injuries (AOR, 4.5; 95% CI, 3.8-5.3). The odds of suicide in the current study are higher than in a 13-year study of 767 head-injured persons from Glasgow, Scotland, although overall death rates were similar.¹⁰ This finding demonstrates the value of population-based studies for rare outcomes. In addition, the fact that overall mortality rates are similar suggests some generalizability of our findings. Overall, the duration and extent to which health services continue to monitor TBI patients after discharge and after immediate treatment warrant attention. Little is known about the nature of services that can best be provided to follow up such patients, and further examination of pathways into crime,²⁴ homelessness,⁴⁵ and institutional care is needed.

Another implication of our findings is that they contribute to the understanding of the possible mechanisms for the increased risks of premature death, particularly suicide, in TBI. We found substantial contribution of psychiatric comorbidities, particularly depression and substance abuse. Those with preexisting psychiatric comorbidities had higher risks of premature mortality. This may be an indicator of severity or further psychiatric comorbidities and needs further examination of possible mechanisms. Previous work suggests that the direct neuropsychological effects of head injury,⁴⁶ possibly mediated by frontal lobe injuries, are important for outcomes. Our data suggested that suicide and injury death rates were higher in those with focal injuries, although we lacked specific information on the location of the sustained lesion. Finally, we did not find support for the view that increased rates of suicide are secondary to sociodemographic factors that are more common in people with TBIs, such as low income and psychosocial deprivation.⁴⁷

Apart from psychiatric comorbidity, we investigated other risk factors for premature mortality. We found higher risks in patients with more severe TBI (as indicated by inpatient hospitalization) and lower risks in those with concussion (reported as an AOR of 2.2 compared with 3.2 for the main sample of TBI patients). Severity has also been reported in previous work to be associated with higher mortality risks.⁹⁻¹¹ In addition, contrasting other

work,^{9,11} we found evidence of higher risks in those with co-occurring injuries sustained at the time of the TBI.

Strengths of this study include the sample size, which is, to our knowledge, the largest cohort of TBI patients followed up for all causes of deaths. In relation to specific causes of mortality, there are 11 times the TBI cases than a recent population-based study.⁹ In relation to suicide outcomes, it is also approximately 10 times larger than a 2001 Danish study on suicide, with 3 times more concussion cases.¹² The use of unaffected sibling controls partly addressed the issue of residual confounding, of particular importance to TBI because personality and genetic factors could confound the associations between TBI and mortality.⁴⁸ In keeping with this, the ORs for different causes of death were somewhat attenuated, suggesting early environmental or genetic confounding of the association between TBI and premature mortality. This could potentially occur through impulsivity, propensity for risk taking, or other unstudied factors that could confound the association. However, there may be other sources of residual confounding that we were not able to assess, including the presence of chronic medical conditions, differential use of medical services, and treatment for preexisting psychiatric conditions. Furthermore, the choice of comparison groups with non-TBI bodily injuries of similar severity may further mitigate mortality ratios, as shown in a study of 1257 patients followed up for 10 years.⁴⁹ Future research should examine possible mediators in the association between TBI and mortality, in particular personality differences and comorbid medical conditions. Personality and preexisting medical comorbidities could be associated with a proportion of TBIs, psychiatric diagnoses, and subsequent death, thus reducing the effects of TBI and psychiatric comorbidity on mortality.

Our findings may be limited by the use of patient registers, which, although validated for a range of physical and psychiatric disorders, have not been validated specifically for TBI. By limiting our sample to patients presenting to inpatient or outpatient services, mortality may have been overestimated because these individuals are likely to represent more severe cases of TBI. However, this approach has the advantage of investigating individuals who access health services and, hence, to whom interventions could potentially be provided. The use of patient registers is also a limitation in how psychiatric comorbidity was ascertained, and it is probable that more severe cases of depression and substance abuse were identified. Because psychiatric diagnoses were ascertained in the same way in cases and controls, it is unlikely to materially alter the odds of premature death. However, absolute rates of psychiatric comorbidity reported here should be interpreted cautiously. At the same time, this is an inevitable limitation of using routinely collected register information, which provides a unique resource for studying uncommon exposures (psychiatric conditions after a TBI) and rare outcomes (deaths from external causes). Nevertheless, our findings would benefit from replication in clinical studies in which information on a wider set of risk factors and confounders could be collected, although such studies would be underpowered for specific causes of death and may have to use other outcomes, including use of medical and social services, suicidal ideation,⁵⁰ and self-reported injuries.

Conclusions

Traumatic brain injury is associated with substantially elevated risks of premature mortality, particularly for suicide, injuries, and assaults, even after adjustment for sociodemographic and familial factors. Current clinical guidelines may need revision to reduce mortality risks beyond the first few months after injury and address high rates of psychiatric comorbidity and substance abuse.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding/Support: This study was funded by grant 095806 from the Wellcome Trust (Dr Fazel and Mr Wolf), the Swedish Prison and Probation Service (Dr Långström), and the Swedish Research Council (Drs Lichtenstein and Långström).

Role of the Sponsor: The funding sources had no role in the design and conduct of the study; collection, management, analysis, or interpretation of the data; preparation, review, or approval of the manuscript; and the decision to submit the manuscript for publication.

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Table 1
Baseline Sociodemographic Characteristics for Individuals With Inpatient and Outpatient ICD-Based Diagnoses of TBI During 41 Years and Comparison Groups^a

Characteristic	Patients With TBI (n = 218 300)	General Population Controls (n = 2 163 190)	Patients With TBI With Unaffected Siblings (n = 150 513)	Unaffected Sibling Controls (n = 237 535)
Male, No. (%)	151 362 (69.3)	1 496 995 (69.2)	104 803 (69.6)	118 697 (50.0)
Single marital status, No. (%)	175 677 (80.5)	1 647 260 (76.1)	126 344 (83.9)	188 479 (79.3)
Immigrant status, No. (%)	2653 (1.2)	37 945 (1.8)	597 (0.4)	850 (0.4)
Individual disposable income, in thousands, mean (SD), \$k	1060 (932)	1113 (1101)	1039 (967)	1019 (812)
Age at diagnosis, median (IQR), y	18.6 (6.0-30.3)	NA	17.0 (5.6-27.3)	NA
Age at death, median (IQR), y	40.6 (29.2-47.9)	38.1 (25.9-47.9)	39.4 (27.9-47.1)	36.7 (23.9-46.0)

Abbreviations: IQR, interquartile range; NA, not applicable; TBI, traumatic brain injury.

^aData on marital status were not available for 494 individuals with TBI, 29 045 matched population controls, 211 TBI patients with sibling controls, and 257 unaffected sibling controls. Data on individual income were not available for 75 823 individuals with TBI, 795 625 matched population controls, 56 365 TBI patients with sibling controls, and 83 849 unaffected sibling controls.

Table 2
Mortality Estimates at Least 6 Months After a Diagnosis of Traumatic Brain Injury in Patients Compared With Population Controls and Unaffected Siblings

Cause of Death	Adjusted Odds Ratios (95% CIs) ^a	
	Mortality Compared With Population Controls	Mortality Compared With Unaffected Sibling Controls
All-cause mortality	3.2 (3.0-3.4)	2.6 (2.3-2.8)
External cause	3.8 (3.5-4.1)	2.7 (2.4-3.1)
All injuries	4.3 (3.8-4.8)	3.0 (2.5-3.7)
Vehicle	3.2 (2.7-3.8)	2.5 (1.9-3.4)
Other	5.2 (4.5-6.1)	3.4 (2.6-4.5)
Suicide	3.3 (2.9-3.7)	2.3 (1.9-2.9)
Assault	3.9 (2.7-5.7)	2.7 (1.3-5.4)

^a Adjusted odds ratios of external deaths compared with population controls (matched for age [birth year] and sex and adjusted for income, marital, and immigration status) or unaffected sibling controls (adjusted for age and sex).

Table 3
Rates of Preexisting, New, and Lifetime Psychiatric Morbidity in Individuals With TBI
During 41 Years and Population Controls^a

Diagnosis	No. (%)	
	Patients With TBI (n = 218 300)	Controls (n = 2 163 190)
Preexisting diagnoses		
Any psychiatric diagnosis	20 378 (9.3)	83 454 (3.9)
Substance abuse	9704 (4.4)	19 685 (0.9)
Alcohol	7137 (3.3)	13 720 (0.6)
Other drugs	4775 (2.2)	8488 (0.4)
Depression	4333 (2.0)	17 454 (0.8)
New diagnoses		
Any psychiatric diagnosis	17 996 (8.2)	99 787 (4.6)
Substance abuse	8129 (3.7)	23 532 (1.1)
Alcohol	6665 (3.1)	16 462 (0.8)
Other drugs	3601 (1.6)	10 604 (0.5)
Depression	5503 (2.5)	29 644 (1.4)
Lifetime diagnoses		
Any psychiatric diagnosis	38 374 (17.6)	183 241 (8.5)
Substance abuse	17 833 (8.2)	43 217 (2.0)
Alcohol	13 802 (6.3)	30 182 (1.4)
Other drugs	8376 (3.8)	19 092 (0.9)
Depression	9836 (4.5)	47 098 (2.2)

Abbreviation: TBI, traumatic brain injury.

^a Any psychiatric disorder includes substance abuse diagnoses. New diagnoses refers to any psychiatric diagnoses assigned after TBI.

Table 4
Absolute Rates and Relative Odds of Premature Death at Least 6 Months After a
Diagnosis of TBI Compared With Population Controls^a

Diagnostic Group	All-Cause Mortality		Suicide		Fatal Injuries	
	No. (%)	AOR (95% CI)	No. (%)	AOR (95% CI)	No. (%)	AOR (95% CI)
Any psychiatric disorder						
No TBI, no psychiatric disorder	4332 (0.2)	1 [Reference]	606 (0.03)	1 [Reference]	872 (0.04)	1 [Reference]
No TBI, psychiatric disorder	2337 (1.3)	4.5 (4.2-4.8)	792 (0.4)	10.9 (9.5-12.6)	341 (0.2)	3.6 (2.0-4.2)
TBI, no psychiatric disorder	917 (0.5)	2.2 (2.0-2.4)	141 (0.1)	2.4 (1.9-2.9)	277 (0.2)	3.1 (2.7-3.7)
TBI, psychiatric disorder	1461 (3.8)	11.0 (10.1-12.0)	381 (1.0)	19.3 (15.6-23.8)	297 (0.8)	13.1 (10.7-16.2)
Depression						
No TBI, no depression	5963 (0.3)	1 [Reference]	1044 (0.05)	1 [Reference]	1127 (0.1)	1 [Reference]
No TBI, depression	706 (1.5)	3.4 (3.1-3.8)	354 (0.8)	12.2 (10.1-14.7)	86 (0.2)	2.8 (2.1-3.6)
TBI, no depression	1999 (1.0)	3.0 (2.9-3.3)	373 (0.2)	3.1 (2.7-3.6)	508 (0.2)	4.2 (3.7-4.8)
TBI, depression	379 (3.9)	8.2 (7.0-9.5)	149 (1.5)	17.6 (13.0-23.7)	66 (0.7)	8.5 (5.9-12.2)
Substance abuse						
No TBI, no substance abuse	5375 (0.3)	1 [Reference]	963 (0.05)	1 [Reference]	941 (0.04)	1 [Reference]
No TBI, substance abuse	1294 (3.0)	6.7 (6.2-7.4)	435 (1.0)	10.8 (9.1-12.8)	272 (0.6)	8.6 (7.0-10.6)
TBI, no substance abuse	1278 (0.6)	2.4 (2.2-2.6)	231 (0.1)	2.4 (2.0-2.8)	316 (0.2)	3.2 (2.8-3.7)
TBI, substance abuse	1100 (6.2)	12.5 (11.3-13.9)	291 (1.6)	15.6 (12.5-19.4)	258 (1.4)	24.2 (18.5-31.7)

Abbreviations: AOR, adjusted odds ratio; TBI, traumatic brain injury.

^a Any psychiatric disorder also included substance abuse. The AORs are odds ratios for deaths during follow-up in TBI patients compared with unrelated population controls, stratified by psychiatric diagnoses and adjusted for income, marital status, and immigration status. The suicide category included both certain and undetermined deaths.

Table 5
Absolute Rates and Relative Odds of Death at Least 6 Months After TBI Compared With Population Controls^a

Variable	All-Cause Mortality		Suicide		Fatal Injuries	
	No. (%)	AOR (95% CI)	No. (%)	AOR (95% CI)	No. (%)	AOR (95% CI)
No TBI	6669 (0.3)	1 [Reference]	1398 (0.1)	1 [Reference]	1213 (0.1)	1 [Reference]
TBI						
All TBI	2378 (1.1)	3.2 (3.0-3.4)	522 (0.2)	3.3 (2.9-3.7)	574 (0.3)	4.3 (3.8-4.8)
Sex						
Male	1891 (1.2)	3.1 (3.0-3.3)	422 (0.3)	3.0 (2.7-3.4)	497 (0.3)	4.1 (3.6-4.7)
Female	487 (0.7)	3.4 (3.0-3.8)	100 (0.1)	4.5 (3.5-5.9)	77 (0.1)	5.9 (4.3-8.0)
Subtype						
Cerebral edema	60 (0.9)	3.6 (2.6-4.9)	10 (0.2)	3.4 (1.6-7.1)	20 (0.3)	8.1 (3.9-16.6)
Focal	49 (3.3)	5.8 (3.9-8.5)	11 (0.7)	5.1 (2.2-12.1)	14 (0.9)	14.3 (5.7-36.1)
Hemorrhagic	116 (3.1)	6.8 (5.3-8.8)	13 (0.4)	3.6 (1.8-7.1)	20 (0.5)	6.2 (3.4-11.4)
Age at death, y						
0-14	72 (0.03)	1.2 (0.8-1.6)	4 (0.00)	2.1 (0.5-10.0)	17 (0.01)	1.2 (0.6-2.4)
15-24	308 (0.1)	2.1 (1.8-2.5)	83 (0.04)	1.8 (1.3-2.5)	129 (0.1)	2.6 (2.0-3.4)
25-34	514 (0.2)	3.2 (2.9-3.7)	159 (0.1)	3.0 (2.4-3.8)	149 (0.1)	4.2 (3.3-5.4)
35-44	673 (0.3)	3.8 (3.4-4.2)	156 (0.1)	3.5 (2.5-4.9)	156 (0.1)	6.5 (5.2-8.2)
45-56	811 (0.4)	3.5 (3.2-3.8)	120 (0.1)	4.0 (3.3-4.9)	123 (0.1)	6.5 (5.1-8.2)
Severity						
Moderate-severe TBI	842 (3.5)	4.5 (4.1-5.0)	153 (0.6)	3.2 (2.5-4.0)	225 (0.9)	7.7 (6.2-9.6)
Length of stay						
Overnight stay	1326 (3.1)	3.6 (3.4-3.9)	275 (0.7)	3.2 (2.7-3.8)	329 (0.8)	5.4 (4.5-6.3)
No overnight stay	1052 (0.6)	2.9 (2.7-3.1)	247 (0.1)	3.3 (2.8-3.8)	245 (0.1)	3.6 (3.1-4.2)
Co-occurring injuries						
Yes	512 (2.1)	3.6 (3.2-4.0)	135 (0.6)	4.3 (3.4-5.5)	129 (0.5)	5.5 (4.2-7.2)
No	1866 (1.0)	3.1 (2.9-3.3)	387 (0.2)	3.0 (2.7-3.4)	445 (0.2)	4.1 (3.6-4.6)
Source						
Inpatient	1541 (3.0)	3.8 (3.5-4.0)	320 (0.6)	3.4 (2.9-4.0)	390 (0.8)	5.6 (4.8-6.6)
Outpatient	837 (0.5)	2.6 (2.4-2.8)	202 (0.1)	3.1 (2.6-3.7)	184 (0.1)	3.1 (2.6-3.7)
Time of diagnosis						
Before 2001	1229 (4.5)	3.5 (3.3-3.8)	258 (1.0)	3.1 (2.6-3.7)	322 (1.2)	5.5 (4.6-6.5)
2001 or later	1149 (0.6)	3.0 (2.8-3.2)	264 (0.1)	3.4 (2.9-3.9)	252 (0.1)	3.5 (3.0-4.1)
Timing of death						
1 wk	2993 (1.4)	3.5 (3.3-3.7)	635 (0.3)	3.5 (3.2-3.9)	921 (0.4)	5.3 (4.8-5.8)
1 mo	2691 (1.2)	3.3 (3.2-3.5)	600 (0.3)	3.3 (3.1-3.6)	691 (0.3)	4.7 (4.2-5.2)
6 mo	2378 (1.1)	3.2 (3.0-3.4)	522 (0.2)	3.3 (2.9-3.7)	574 (0.3)	4.3 (3.8-4.8)
12 mo	2123 (1.0)	3.1 (2.9-3.2)	454 (0.2)	3.1 (2.7-3.5)	513 (0.2)	4.2 (3.7-4.7)
5 y	1080 (0.5)	3.1 (2.8-3.3)	214 (0.1)	2.7 (2.3-3.2)	262 (0.1)	4.5 (3.8-5.3)
Concussion						

Variable	All-Cause Mortality		Suicide		Fatal Injuries	
	No. (%)	AOR (95% CI)	No. (%)	AOR (95% CI)	No. (%)	AOR (95% CI)
No concussion	26 332 (0.8)	1 [Reference]	5962 (0.2)	1 [Reference]	5249 (0.2)	1 [Reference]
Concussion						
Men	4817 (2.4)	2.2 (2.1-2.3)	1320 (0.7)	2.4 (2.2-2.5)	1203 (0.6)	2.5 (2.3-2.7)
Women	1293 (1.0)	2.0 (1.8-2.1)	344 (0.3)	3.2 (2.8-3.7)	186 (0.1)	2.6 (2.1-3.1)

Abbreviations: AOR, adjusted odds ratio; TBI, traumatic brain injury.

^aThe AORs reflect odds of mortality in individuals with TBI compared with general population controls (matched for age and sex and adjusted for income, marital, and immigration statuses). Results were stratified by sex, TBI diagnostic subtype, age band, and timing of death. The suicide category included both certain and undetermined deaths.