

Mortality After Burn Injury in Children: A 33-year Population-Based Study

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abstract

OBJECTIVE: To assess the impact of burn injury sustained during childhood on long-term mortality and to quantify any increased risk of death attributable to burn injury.

METHODS: A population-based cohort study of children younger than 15 years hospitalized for burn injury in Western Australia (1980–2012) and a matched noninjured comparison group. Deidentified extraction of linked hospital morbidity and death records for the period 1980–2012 were provided by the Western Australian Data Linkage System. An inception cohort (1980–2012) of burn cases younger than 15 years of age when hospitalized for a first burn injury ($n = 10\,426$) and a frequency matched noninjured comparison cohort ($n = 40\,818$) were identified. Survival analysis was conducted by using the Kaplan-Meier method and Cox proportional hazards regression. Mortality rate ratios and attributable risk percent adjusted for sociodemographic and preexisting health factors were generated.

RESULTS: The median follow-up time for the pediatric burn cohort was 18.1 years after discharge. The adjusted all-cause mortality rate ratios for burn injury was 1.6 (95% confidence interval: 1.3–2.0); children with burn injury had a 1.6 times greater rate of mortality than those with no injury. The index burn injury was estimated to account for 38% (attributable risk percent) of all recorded deaths in the burn injury cohort during the study period.

CONCLUSIONS: Burn injury sustained by children is associated with an increased risk of long-term all-cause mortality. Estimates of the total mortality burden based on in-hospital deaths alone underestimates the true burden from burn injury.

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WHAT'S KNOWN ON THIS SUBJECT: Burns are a leading cause of pediatric emergency department visits and hospitalizations and are often associated with significant long-term physical and psychological consequences and long-term medical and nursing treatments. Little is known of the long-term impacts of burns on mortality.

WHAT THIS STUDY ADDS: Children with burns had a 1.6 times greater rate of long-term mortality than a matched population-based cohort of children with no injury. Total mortality burden based on in-hospital deaths alone underestimates the true burden from both minor and severe burns.

Pediatric burns are a leading cause of emergency department visits and hospitalizations.¹⁻³ For young children, burns are often associated with significant physical and psychological sequelae⁴ and long-term medical and nursing treatments.⁵ Burns in pediatric patients have been shown to have significant impacts on metabolic markers, the heart, and other organs for up to 3 years postburn.⁶ However, to date, limited longitudinal data have been available to explore the impacts of burns on longer-term survival.

In Western Australia, children younger than 15 years of age account for 35% of all burn hospitalizations.^{7,8} Children represent the most vulnerable proportion of the population for whom the burden of burn injury may be experienced during the remainder of their lifetime. Mortality is an unambiguous health index that has been related to injury.^{9,10} However, most mortality data related to burns are reported in terms of inpatient deaths or death within 30 days of admission.^{5,8,11-13} Limited data are available that examine long-term burn-injury-related mortality among pediatric burns patients.

This study used linked statewide health administrative data of all children hospitalized for a burn injury in Western Australia during the period 1980 to 2012 and a population-based random sample of children with no record of injury hospitalization (1) to assess the impact of burn injury on longer-term all-cause mortality and (2) to estimate any increased risk of death attributable to burn injury while adjusting for sociodemographic and preexisting health factors.

METHODS

The Western Australian Population-Based Burn Injury Project (WAPBIP) is a population-based retrospective cohort study using linked health administrative data from the Western

Australian Data Linkage System (WADLS). The WADLS is a validated record linkage system that routinely links administrative health data from core data sets (including Hospital Morbidity Data System [HMDS], Western Australia Death Register) for the entire population of Western Australia of 2.5 million.¹⁴ Project approvals were obtained from the Western Australian Department of Health and the University of Western Australia Human Research Ethics Committees.

A deidentified extraction of all linked hospital morbidity (HMDS) records for all children younger than 15 years of age who were hospitalized in Western Australia with an index burn injury, for the period January 1, 1980, to June 30, 2012, was undertaken by the WADLS. An index burn injury was defined as the first hospitalization with a burn as the principal and/or additional diagnosis using the *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) codes 940-949 and *ICD, 10th Revision, Australian Modification* (ICD-10-AM) codes T20-T31. A population-based comparison cohort was randomly selected from the Western Australian Birth Registrations; any person with an injury hospitalization during the study period was excluded from the population-based noninjury cohort by WADLS. The resultant noninjury comparison cohort was frequency-matched 4:1 on birth year, gender, and year of index burn discharge for the period 1980 to 2012.

Hospital and death data from the Western Australia HMDS and Death Register were linked to each cohort (burn, noninjury) for the period 1980-2012. Admissions data included principal and additional diagnoses, external cause of injury, age, and gender, Aboriginality, index admission and separation dates, total burns surface area percent (TBSA%), geographic location, and indices of economic and social disadvantage

(Socioeconomic Indices for Areas [SEIFA])¹⁵ and remoteness (Accessibility Remoteness Index of Australia [ARIA+]).¹⁶ The death data included date and cause of death.

Aboriginal status was classified by record of Aboriginal or Torres Strait Islander status on any admission record. Age at index was categorized into 5-year age bands. TBSA% was classified by ICD supplementary codes (ICD-9-CM 948; ICD-10-AM T31) and used to categorize burn severity as minor burns (TBSA <20%), severe burns (TBSA ≥20%), and burns for which no TBSA% was coded. A Charlson Comorbidity Index (CCI)¹⁷ was generated using principal and additional diagnosis fields with a 1-year look-back period in the hospital morbidity data¹⁸ and was used to generate “any comorbidity” (yes: CCI >0; no: CCI = 0, or no previous hospitalization). Record of an existing congenital anomaly within the 5-year period before the index burn was identified using principal and additional diagnosis ICD-9-CM 740-759 and ICD-10-AM Q00-G99. Social disadvantage (SEIFA) and remoteness indices were based on geocoded place of residence (ie, census collector district [~200 households], postcode). SEIFA scores were partitioned into quintiles from the most disadvantaged to the least disadvantaged. ARIA+ was used to classify geographic disadvantage/access in terms of physical distance from services by 5 remoteness categories: major cities, inner regional, outer regional, remote, and very remote. Person-years of risk (PY) were calculated from the final discharge date of the index admission burn cases to study end date (death or censored at June 30, 2012).

χ^2 tests and Kruskal-Wallis nonparametric tests were performed with the level of significance set at .05. Survival analysis was conducted by using the Kaplan-Meier method and Cox proportional hazards model. Kaplan-Meier plots of survival

estimates for burn (total), burn severity (minor, severe, burns—no TBSA coded vs noninjury) and gender versus uninjured were generated and log-rank tests were used to assess equality of survivorship. Cox proportional hazard regression was used to estimate the effects of burn injury on long-term survival while adjusting for index age, gender, Aboriginal status, socioeconomic disadvantage, remoteness, index year, any comorbidity, and congenital anomaly. Subcohorts classified by gender and burn severity (TBSA%) were also analyzed by using Cox proportional hazard regression. The hazard ratios (95% confidence intervals [CI]) estimated from the Cox proportional hazards model were used as measures of mortality rate ratios (MRR). Preliminary analyses revealed no evidence of nonproportionality.^{19,20} Attributable risk percent (AR%) was used to estimate the proportion of long-term mortality in which burn injury was a component cause. The AR% was calculated as the adjusted rate ratio minus 1, divided by the adjusted rate ratio, multiplied by

$$100;^{21} \left(AR\% = \left(\frac{adjMRR - 1}{adjMRR} \right) \times 100 \right).$$

Hence, the AR% was used to estimate the percentage of deaths in the burn-injury cohort that were attributable to sustaining a burn injury, after adjusting for known potential confounders. All statistical analyses were performed by using Stata version 12 statistical software (StataCorp LP, College Station, TX).

RESULTS

There were 10 426 children included in the burn injury cohort, and 40 818 persons in the noninjured cohort were selected randomly from an age and gender frequency match to burn cases. The median age for both cohorts was 2 years (interquartile range [IQR]: 1–7). Overall, the burn cohort had significantly higher proportions children who were Aboriginal, lived in areas outside of

the major cities, and were from socially disadvantaged areas compared with the noninjury cohort. Although the proportions of those with any comorbidity and record of a congenital anomaly were low, these were higher for the burn injury cohort (Table 1). Of children with burns, 46.4% (*n* = 4832) had minor burns (<20% TBSA), 1.3% (*n* = 132) severe burns ($\geq 20\%$ TBSA); for 52.4% (*n* = 5462), TBSA% was not coded. Full-thickness burns were sustained by 8.4% (*n* = 876), 45.6% (*n* = 4755) had partial-thickness burns, 13.4% (*n* = 1443) erythema burns, and for 35.3% (*n* = 3673) burn depth was unspecified. Burns sites included head and neck 20.0% (*n* = 2082); 28.1% (*n* = 2925) trunk; 43.5% (*n* = 4528) upper limbs/hands; 35.5% (*n* = 3694) lower limbs/feet; 1.0% (*n* = 100) each to eyes, respiratory tract, and other internal organs; 6.1% (*n* = 636) multiple regions; and 3% (*n* = 306) unspecified site.

Kaplan-Meier survival plots for burn (total) (Fig 1) and by %TBSA burn

severity (Fig 2) versus noninjury showed reduced survival for each of the burn categories compared with the noninjured, with severe burns demonstrating worse survival outcomes. Noninjured females and males had better survival estimates than both burn-injured females and males (Fig 3). Log-rank tests of burns versus no injury showed significant difference between the counts of deaths for each variable assessed (Table 2).

In this pediatric burn cohort, 0.1% (*n* = 8) died in hospital. During the follow-up period after discharge, an additional 1.5% (*n* = 156) of the burn cohort died of which 68% were male with 0.6% (*n* = 248) deaths in the noninjured cohort died of which 77% were male. There was no significant difference (*P* = .09) in the median (IQR) age at death between the burn and uninjured (19 [13–25] vs 22 [14–30] years). There was no statistically significant difference (*P* = .30) in median [IQR] time to death (years) between the burn and

TABLE 1 Baseline Demographic and Preexisting Health Status Factors for Children Younger Than 15 Years at First Burn Injury Hospitalization and Frequency Matched Noninjury Cohort, Western Australia, 1980–2012

Characteristics	Burn Injury, <i>n</i> (%)	Noninjury, <i>n</i> (%)	<i>P</i>
Total	10 426 (100)	40 818 (100)	
Demographic			
Male	6455 (61.9)	25 020 (61.4)	.27
Age group (y)			
<5	6788 (65.1)	27 060 (66.3)	.037
5–9	1803 (17.3)	6966 (17.1)	
10–14	1835 (17.6)	6792 (16.6)	
Aboriginality (yes/no)	2055 (19.7)	1 974 (4.8)	<.001
Social disadvantage quintiles ^a			
Quintile 1 (most disadvantaged)	2368 (22.9)	5197 (12.8)	<.001
Quintile 2	3402 (33.0)	10 371 (25.6)	
Quintile 3	2058 (19.9)	7360 (18.1)	
Quintile 4	1264 (12.3)	6880 (17.0)	
Quintile 5 (least disadvantaged)	1229 (11.9)	10 789 (26.6)	
Remoteness ^b			
Major city	5448 (52.6)	27 283 (67.4)	<.001
Inner regional	1067 (10.3)	4267 (10.5)	
Outer regional	1649 (15.9)	4691 (11.6)	
Remote	1206 (11.7)	2640 (6.5)	
Very remote	983 (9.5)	1610 (4.0)	
Health status			
Any comorbidity (yes/no)	106 (1.0)	197 (0.5)	<.001
Record congenital anomaly (yes/no)	430 (4.1)	1227 (3.0)	<.001

^a SEIFA socioeconomic disadvantage quintiles.

^b ARIA+ remoteness classification.

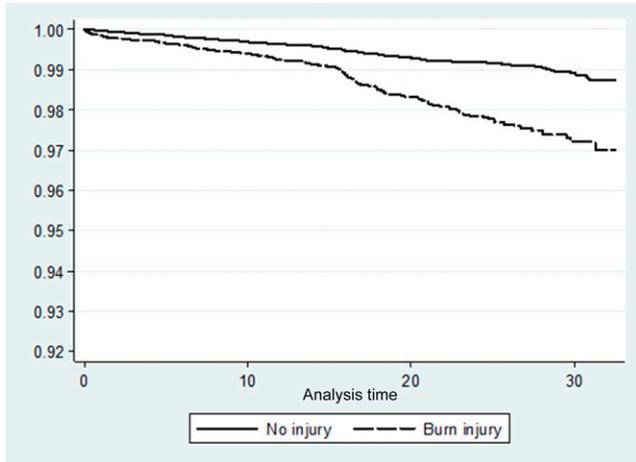


FIGURE 1 Kaplan-Meier estimates of the survivor function for burn injury and matched noninjury cohorts with follow-up from date of index discharge for the period 1980 to 2012.

noninjured cohorts (15 [6–18] vs 12 [5–17] years). No statistically significant difference in the cause of death proportions between burn and noninjured cohorts was found ($P = .245$). Refer to Fig 4 for the distribution of causes of death.

The burn injury cohort had a median follow-up time of 18.1 years (IQR; min–max: 10.3–25.6; 0.05–32.5) for a total of 184 309 PYs. The noninjury cohort had a median follow-up time of 17.9 (IQR; min–max: 10.2–25.5; 0.04–32.5) for a total of 710 657 PYs. For the study period, the burn injury (total) cohort had an all-cause

mortality rate of 8.5 per 10 000 PYs compared with 3.5 per 10 000 PYs in the noninjured cohort, giving an unadjusted MRR of 2.5 (95% CI: 2.0–3.0). After adjustment for sociodemographic (index age, gender, Aboriginal status, economic and social disadvantage, remoteness, index admission year) and preexisting health status factors (any comorbidity, congenital anomaly), the overall long-term adjusted MRR for burn injury was 1.6 (95% CI: 1.3–2.0); those with burn injury had a 1.6 times greater rate of mortality than those with no injury. After

adjustment for known confounders, the percentage of deaths attributable to burn injury among children younger than 15 years of age hospitalized for a first burn was estimated to be 38% (AR%), accounting for 59 deaths in the burn injury cohort over the 33-year study period.

Gender-specific mortality rates for the study period estimated the all-cause mortality rate for females with burn injury was 7.2 per 10 000 PYs compared with 2.0 per 10 000 PYs for noninjured females, giving an unadjusted MRR of 3.6 (95% CI: 2.4–5.2). After adjusting for sociodemographic and previous health status factors, the adjusted MRR for females was 2.3 (95% CI: 1.5–3.5). The all-cause mortality rate for males with burn injury was 9.2 per 10 000 PYs compared with 4.4 per 10 000 PYs for noninjured males, giving an unadjusted MRR of 2.1 (95% CI: 1.6–2.6). After adjustment for sociodemographic and previous health status factors, the adjusted MRR for males with burn injury was 1.3 (95% CI: 1.0–1.7, $P = .057$)

Adjusted Cox regression models for sub-cohorts of burn injury classified by TBSA% severity versus noninjury cohort resulted in adjusted MRRs for severe burns ($\geq 20\%$ TBSA) of 4.3 (95% CI: 1.8–10.7). Refer to Table 3 for summary of MRR analyses for TBSA% burn severity.

Although deaths were due to both disease and injuries, injuries were a predominant cause of death for both cohorts. No significant difference in injury-related mortality rates between the burn and uninjured cohorts (MRR, 95% CI: 1.0, 0.7–1.2) was found, adjusting for sociodemographic and preexisting health factors.

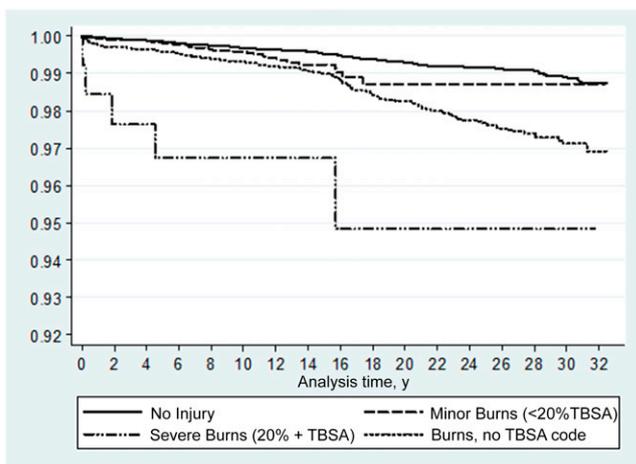


FIGURE 2 Kaplan-Meier estimates of the survivor function by burn injury classified by TBSA ($<20\%$ TBSA—minor burns; $\geq 20\%$ TBSA—severe burns; burns—TBSA not coded) and matched noninjury cohorts, follow-up from date of index discharge for period 1980 to 2012.

DISCUSSION

To our knowledge the WAPBIP is the first burn injury study using a large population-based sample of pediatric burn patients and a frequency

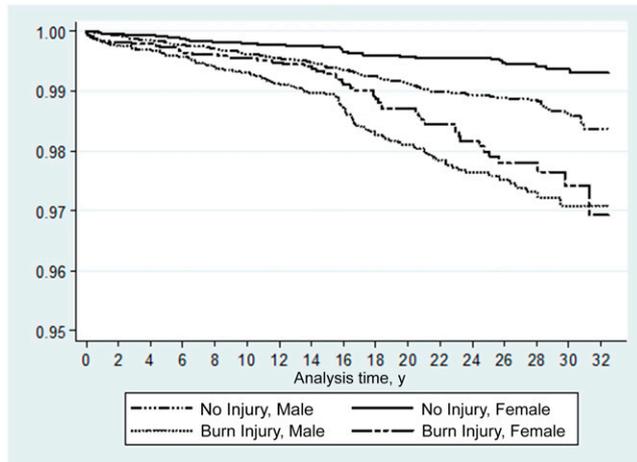


FIGURE 3

Kaplan-Meier estimates of the survivor function by burn injury and matched noninjury by gender, for children younger than 15 years index age with follow-up from date of index discharge for period 1980 to 2012.

matched noninjury comparison group to explore long-term mortality associated with burn injury. The use of a noninjured comparison cohort accounts for the normative risk of death regardless of injury. This study used data over a 33-year period from 1980 to 2012, in which 50% of the burn-injured cohort had >18 years of follow-up after the burn injury event. Excess mortality attributable to burn injury was observed after controlling for known potential confounders including age, gender, economic and social disadvantage, geographic location, and preexisting health status. The long-term increased risk of mortality after burn injury sustained during childhood has

significant consequences for the estimates of longer-term health burden, clinical care of burns, and burn prevention.

Advances in the management of patients with burns over the past decades have resulted in improved in-hospital survival after burn injury,^{8,22-24} and there were only 8 (0.1%) in-hospital fatalities in this pediatric burn cohort. After adjusting for known potential confounders, the pediatric burn cohort was found to have a 1.6 times greater risk of long-term all-cause mortality compared with a frequency matched population-based noninjured cohort. Although injuries were a predominant cause in both cohorts,

no significant difference in the injury-related mortality rates was found between the burn and uninjured cohorts. The excess mortality risk attributable to burn injury after adjustment for known confounders was 38%, accounting for 59 of the 156 deaths in this pediatric burn cohort during the study period. This finding has implications for long-term clinical care and management of burn injury patients and also challenges the definition of fatal outcomes used in burn injury assessments.

Compared with male burn patients, female burn patients have poorer outcomes with respect to in-hospital mortality²⁵⁻²⁸ and cancer incidence²⁹ and yet improved prognoses for sepsis³⁰ and multiple organ dysfunction syndrome³¹ after burn injury. It is possible that a gender dimorphism in the immune response to burn³²⁻³⁵ may have contributed to the greater risk of long-term death observed for female burn patients compared with their male counterparts.

Although there was no significant difference in the adjusted MRRs and the 95% CIs of the burn severity subcohorts, a relationship between burn severity and increased mortality was demonstrated. Children with minor (<20% TBSA) burns had a mortality rate of 1.4 to 1.5 times greater than the noninjured cohort, whereas for those with severe burns, the mortality rate was 4.3 times greater. The similarity of the adjusted MRR for burns with no coded TBSA% and minor burns suggests that the majority of the records with no coded TBSA% were minor burns.

Burn injury causes significant depression of humoral and cell-mediated immunity,^{36,37} sustained high levels of oxidative stress,³⁸ and prolonged elevation stress hormones.^{39,40} Research of pediatric patients with severe burns has shown that metabolic and inflammatory changes persist for up to at least

TABLE 2 Log-Rank Tests to Assess Equality of Survivorship Between Children Younger Than 15 Years of Age at First Burn Injury Hospitalization Compared With Noninjured Frequency Matched Cohort

	Observed Events	Expected Events	P
Burn (total) vs no injury			
No injury	248	321.32	<.001
Burns	156	82.68	
Burn (by TBSA) vs no injury			
No injury	248	321.32	<.001
Minor burns (<20% TBSA)	26	19.35	
Severe burns (20%+ TBSA)	5	0.73	
Burns—TBSA not coded	125	62.6	
Burn and no injury by gender			
No injury—male	192	198.29	<.001
No injury—female	56	123.10	
Burns—male	106	51.73	
Burns—female	50	30.98	

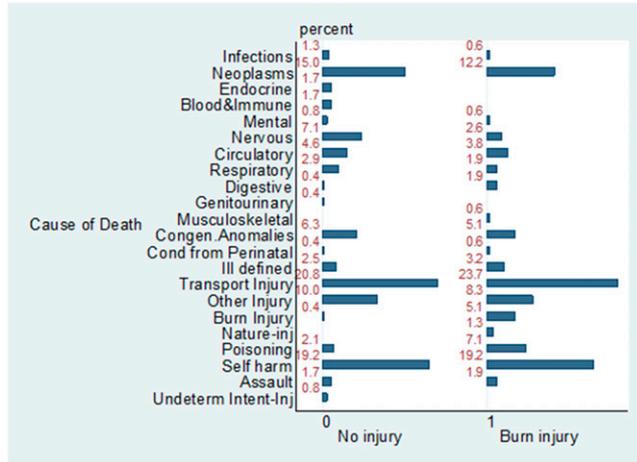


FIGURE 4 Cause of death for children younger than 15 years of age at first burn injury hospitalization and noninjured frequency matched cohort, Western Australia, 1980–2012.

3 years after the injury.⁶ The significance of these effects is that they induce a spectrum of health and medical issues including insulin resistance,⁴¹ increase in fracture risk,⁴² increased risk of sepsis and infections,^{40,43} enlargement of the liver,^{44,45} cardiac stress and dysfunction,^{40,46} and hormonal abnormalities.^{41,44} Recent research has shown that systemic responses may also occur after minor and moderate burns,^{47,48} and as such, these systemic responses may have contributed to the increased mortality risk for minor burns observed in this study.

Although the incidence of burn injury hospitalizations in high-income

countries (HICs) has declined over the past decades, children remain at high risk of burns.^{8,49} Of importance is that the majority of pediatric burn hospitalizations in HICs are for minor burns, and the results of this study demonstrated that an excess risk of long-term mortality exists for both minor and severe burns. Deaths were caused by both disease and injury, and these results highlight potential opportunities for continuity of care and regular primary health care surveillance after completion of initial burn injury treatment, as well as injury prevention interventions during well-child visits with health practitioners (eg, immunization), injury-related admissions and during

TABLE 3 MMR and AR% for Children Younger Than 15 Years of Age at First Burn Injury Hospitalization Compared With Noninjured Frequency Matched Cohort, Western Australia, 1980–2012

	Deaths, n	Unadjusted MRR (95% CI)	Adjusted MRR (95% CI) ^a	Adjusted AR% ^a
Burn vs no injury				
Burns	156	2.5 (2.0–3.0)	1.6 (1.3–2.0)	38
Burn vs no injury by gender				
Males	106	2.1 (1.6–2.6)	1.3 (1.0–1.7)	23 ^b
Females	50	3.6 (2.4–5.2)	2.3 (1.5–3.5)	57 ^b
Burn by TBSA vs no injury				
Minor burns (<20% TBSA)	26	1.7 (1.1–2.6)	1.4 (0.9–2.2)	29
Severe burns (≥20% TBSA)	5	8.9 (3.5–20.9)	4.3 (1.7–10.6)	77
TBSA not coded	125	2.6 (2.1–3.2)	1.5 (1.2–1.9)	33

^a Adjusted for sociodemographic (age at index, gender, Aboriginal status, social disadvantage, remoteness, index year) and health status variables (any comorbidity, record of congenital anomaly).

^b Adjusted for sociodemographic (age at index, Aboriginal status, social disadvantage, remoteness, index year) and health status variables (any comorbidity, record of congenital anomaly).

the life course. Of those who died in the burn cohort, 75% were younger than 25 years, with the oldest aged 41 years. Given that 75 years of age is often used as the limit for premature death in Australia,⁵⁰ the years of potential life lost attributed to burn injury is significant among this pediatric burn cohort.

Study Strengths and Limitations

Although data of single-center studies of pediatric severe burns have been reported with follow-up of 14 years to evaluate quality of life (no control)⁵¹ and 3 years to investigate pathophysiology (with control),⁶ the strengths of this research are driven by the use of linked administrative data and the ability to perform a “whole-of-population” study of all children hospitalized for burn injury with a long follow-up time and a comparison noninjured cohort. The use of an unexposed-noninjured cohort provided a measure of the background risk of death that exists in the absence of burn injury and the means for attributing effects that have occurred after the initial exposure (burn injury).²¹

The use of linked health data allowed identification for preexisting comorbidity and included an index of socioeconomic status that is correlated with lifestyle factors such as alcohol use and smoking, which are associated with poor health outcomes.^{52,53} Such data have been omitted from other population-based injury studies.⁵⁴ Although infants and young children (0–4 years) explore their physical environment before they understand and have the skills to respond to hazards, older children (5–14 years) have increasing capacity to make decisions about safety issues.^{55,56} For this age group, injuries sustained are increasingly influenced by behavior in addition to their physical and social environment.⁵⁶ Variables measuring appraisal of injury risk and risk-taking behaviors, were not included in the health administrative data.

However, MRR analysis showed no significant difference in injury-related mortality rates between the burn and uninjured cohorts.

The WAPBIP was developed to analyze multiple aspects of burns including spatial analyses and inequities in health care. Although burns tend to cluster in disadvantaged populations, rural groups, and ethnic minorities, matching of the control cohort on all of these factors was not feasible and is a study limitation. It is possible that burn injury is a marker for subsequent injurious behavior associated with long-term deaths.

The majority of the missing TBSA% ICD codes were for records before 1996; however, results of survival analyses for burns classified by TBSA% suggest that the majority of burns with missing TBSA% were most likely minor burns. The Western Australia hospital morbidity data set is assessed for both accuracy and quality.⁵⁷ It is expected that the finding of increased risk of all-cause mortality after burn injury is generalizable to populations with similar demographic characteristics and comparable health care systems to those in Australia.⁵⁸

CONCLUSIONS

This research confirms that after adjusting for measurable sociodemographic and preexisting health conditions, children who sustained burn injury, both minor and severe, had a higher long-term all-cause mortality rate than the noninjured. Eight in-hospital deaths occurred after the first burn admission; however, burn injury accounted for 38% ($n = 59$) of the total all-cause deaths that occurred during the long-term follow-up after discharge. Thus, we estimate that the long-term deaths in those who sustained burn injury as a child may presumably have been reduced by 38% if the burn injury had not occurred. An estimate of the mortality

burden from burn injury based on in-hospital deaths alone underestimates the total mortality burden. These findings have implications for clinical management and burn prevention.

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