Running Title: Burns and long-term infectious disease morbidity: A population-based study

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Background: There is a growing volume of data that indicates that serious injury suppresses immune function, predisposing individuals to infectious complications. With recent evidence showing long-term immune dysfunction after less severe burn injury, this study aimed to investigate post-burn infectious disease morbidity and assess if burn patients have increased long-term hospital use for infectious diseases.

Methods: A population-based longitudinal study using linked hospital morbidity and death data from Western Australia for all persons hospitalized for a first burn injury (n=30,997) in 1980 –2012. A frequency matched non-injury comparison cohort was randomly selected from Western Australia's birth registrations and electoral roll (n = 123,399). Direct standardisation was used to assess temporal trends in infectious disease admissions. Crude annual admission rates and length of stay for infectious diseases were calculated. Multivariate negative binomial and Cox proportional hazards regression modelling were used to generate adjusted incidence rate ratios (IRR) and hazard ratios (HR), respectively.

Results: After adjustment for demographic factors and pre-existing health status, the burn injury cohort had twice (IRR, 95% Confidence Interval (CI): 2.04,1.98-2.22) as many admissions and 3.5 times the number of days in hospital (IRR, 95%CI: 3.46, 3.05-3.92) than the uninjured cohort for infectious diseases. Higher rates of infectious disease admissions were found for severe (IRR, 95%CI: 2.37, 1.89-2.97) and minor burns (IRR, 95% CI: 2.22, 2.11-2.33). Burns were associated with significantly increased incident admissions: 0 to 30 days (HR, 95%CI: 5.18, 4.15-6.48); 30 days to 1 year (HR, 95%CI: 1.69, 1.53-1.87); 1 to 10 years (HR, 95%CI: 1.40:1.33-1.47); >10 years (HR, 95%CI: 1.16, 1.08-1.24). Respiratory, skin and soft tissue and gastrointestinal infections were the most common. The burn cohort had a 1.75 (95%CI: 1.37-2.25) times greater rate of mortality caused by infectious diseases during the 5-year period after discharge than the uninjured cohort.

Conclusions: These findings suggest that burn injury has long-lasting effects on the immune system and its function. The increase in infectious disease in three different epithelial tissues in the burn cohort suggests there may be common underlying pathophysiology. Further research to understand the underlying mechanisms are required to inform clinical interventions to mitigate infectious disease after burn and improve patient outcomes.

Keywords: burns; infections; long-term health; population-based; cohort.

1. Background

There is a growing volume of data that indicates that serious injuries suppress immune function, predisposing individuals who sustain such injuries to infectious complications.[1-4] The immune response after severe injury is complex and encompasses both innate and adaptive immunity [5, 6]. After an injury to the skin, a range of pathways are activated in an effort to restore tissue integrity and homeostasis, including the coagulation cascade, inflammation and the adaptive immune system [7, 8]. Post-burn there is an increased risk of blood stream infections and sepsis-related mortality [9, 10] and these outcomes are largely due to the disturbances in the skin, including changes in microflora [11]. Other problems related to burn injury include a persistent innate pro-inflammatory response or systemic inflammatory response syndrome [12], dendritic cell suppression and loss of adaptive immunity T-cell responses [3, 13]. The elevation in morbidity and mortality in the acute post-burn phase is further complicated by compensatory anti-inflammatory responses [3, 14].

While recent animal-based research indicates long-term immune dysfunction after non severe burn injury [15], to date the effect of severe and less severe burn injury on the long-term function of the immune system has not been comprehensively investigated. Initial investigations of long-term morbidity after burn injury using population-based data have identified increased post-burn circulatory morbidity, after both minor and severe burns [16], suggesting a substantial acute inflammatory response [15], as well as increased incidence of some types of cancers [17, 18], suggesting long-term effects of burns on the immune system.

Population-based linked administrative health data provide valuable opportunities to assess long-term health outcomes and morbidity in terms of diseases sufficiently serious to require admission [19]. This study used whole-of-population data from the Western Australian Population-based Burn Injury Project (WAPBIP) to assess post-burn infectious disease

admissions. To examine the potential for long-term immune dysfunction after burns, the data were analysed to assess if a cohort of burn patients had greater long-term infectious disease morbidity and mortality when compared to an uninjured cohort, after adjustment for socio-demographic and pre-existing health factors.

2.   Methods

The WAPBIP is a retrospective longitudinal project that supports multiple investigations of long-term health, trends and costs of burns. Detailed information on the Western Australian Data Linkage System [20, 21], cohort selection, data extraction and standard methods applied, has been previously published [16, 22-24]. This study used data of 30,997 individuals hospitalised for an incident burn (burn cohort) in Western Australia during the period 1 January 1980 to 30 June 2012 and an (~4:1) age and gender frequency matched cohort of 123,399 individuals with no record of injury admission (uninjured cohort). This study had human research ethics committee approvals from the Western Australian Department of Health and the University of Western Australia.

The linked hospital and death data provided information on the following variables included in the analyses: International Classification of Diseases (ICD9-CM and ICD10AM) coded diagnosis, causes of injury, age and gender, indigenous status, admission and discharge dates, burn features (total burns surface area percent (TBSA%), depth, site), residential postcode and date and cause of death. TBSA % was classified as minor (TBSA<20%), severe burns (TBSA≥20%) or burns of unspecified TBSA %. The Charlson comorbidity index (CCI) [25] was used to classify pre-existing comorbidity (0 CCI=0; 1 CCI>0) using hospital records [26]. Quintiles of social disadvantage (Socio-economic Indices for Areas (SEIFA)[28]) and geographic remoteness and access to services (Accessibility Remoteness Index of Australia

(ARIA+)[29]), derived from the national census data, were assigned to each member of both cohorts. ICD9-CM codes were mapped to ICD10-AM [27].

An infectious disease admission was defined as an episode of care with a principal diagnosis belonging to the ICD code set of major infectious diseases developed by Baker et al [30]. Refer to Appendix I for the ICD-based infectious disease code set used in this study. The Baker et al code set builds on a coding system established by the United States of America Centers for Disease Control and Prevention [31, 32] and incorporates ICD9-CM and ICD10-AM codes.

Univariate analyses were conducted using chi square and Kruskal–Wallis tests with 5% level of significance. The total number of years of person risk (person-years) was estimated from the final discharge date for the index or first burn admission; this date was used for the corresponding frequency matched uninjured controls. The number of annual admissions (total) and summed length of hospital stay (LOS) for infectious diseases identified during the study period were used as outcome measures.

To assess trends over time, rates of hospitalizations for infectious diseases (total) for the burn and uninjured cohorts were standardised to the age structure of the Australian population at the 2001 national census [33]. Admission rates, total and by decade of study entry (1980-89; 1990-1999; and 2000-2012), were age-standardised and classified by major infectious disease category (e.g. lower respiratory, skin and soft tissue, enteric etc.).

Multivariate negative binomial regression analyses were used to assess the link between burn injury and infectious disease admissions. Multivariate Cox proportional hazards regression was used to assess the effects of burns on first time (incident) admissions and long-term mortality for infectious diseases. Zero truncated negative binomial regression was used to examine the effect of burn injury on LOS for infectious disease. All models were adjusted for the following

confounders: socio-demographic (gender, indigenous status, 5-year age group, social disadvantage, remoteness of residence and health factors (pre-existing comorbidity, prior infectious disease admission) and year of study entry (to allow adjustment for treatment and referral patterns). Additional adjusted analyses were conducted for sub-cohorts defined by i) age at study start (<15 years; 15 to 45 years; ≥45 years); ii) burn TBSA severity; iii) and, iii) decade of study entry with adjustment for follow-up time (1980-1989; 1990-1999; 2000-2012).

To address potential differences in post-burn infectious disease rates related to prior record of infectious disease admissions (yes/no), additional subcohort analyses were undertaken. Analyses were conducted: firstly, comparing those in the burn and uninujured cohorts that had a record of prior infectious disease admission (using a 5-year look-back period); and secondly, comparing those in the burn and uninjured cohorts with no record of prior infectious disease admission. These models were adjusted for the effects of comorbidity, year of study entry and socio-demographic variables that included geographic remoteness, indigenous status and social disadvantage. Additional analysis using start of follow up from 30 days after discharge was undertaken to assess the effect of potentially excluding admissions for infections related to the index burn.

Incident analyses were conducted on cohort data that excluded any person (burn or uninjured) with a prior admission for infectious disease, and also any person in the burn cohort with an additional record of non-burn injury admission. The latter exclusion was applied to reduce the potential for confounding associated with systemic effects caused by non-burn injury [15]. Standard tests of the proportional hazards assumption were applied [36]. If non-proportionality was identified, Cox regression analyses were undertaken in conjunction with Aalen's linear hazard models [37]. Attributable risk percentage (AR%) [34] was generated from the adjusted hazard ratio and used to estimate the proportion of incident admissions attributed to the burn

injury [16, 35]. Statistical analyses were performed using Stata version 12 (StataCorp. LP, College Station, United States of America).

3. Results

3.1 Cohort characteristics

The median age of the burn and uninjured cohorts was 23 years (interquartile range (IQR) 7-39); males represented 68% of each cohort. Refer to Table 1 for a summary of characteristics for the burn and uninjured cohorts.

Burn cohort characteristics: Full thickness burns were sustained by 14% (n=4,390) of the burn cohort, 40% (n=12,307) had partial thickness burns, 17% had erythema (first degree) burns (n=5,335) and for 31% the burn depth was unspecified (multiple anatomical sites and burn depths were recorded in individual files). Burn sites included: 21% (n=6,384) head and neck; 23% (n=7,262) trunk; 42% upper limbs/hands (n=13,144); 34% (n=10,537) lower limbs/feet; 7% (n=2303) eyes; 2% (n=624) respiratory tract or other internal organs; 3% (n=904) unspecified site; and, 4% (n=1,237) were coded as having burns to multiple regions. A third of the burn cohort (n=10,351) had a record of non-burn injury.

Using ICD codes for TBSA: 48% (n=14,854) had minor burns (<20% TBSA), 3% (n=911) had severe burns (≥20% TBSA); and for 49% (n=15,232) TBSA% was unspecified. LOS (median days; IQR) for the index burn admission by TBSA category: unspecified TBSA burns (3 days; IQR 1-10); severe burns ≥ 20% TBSA (25 days; IQR 12-48); and, minor burns <20% TBSA (4 days; IQR 1-10). LOS comparisons suggest that members of the unspecified TBSA sub cohort predominantly had minor burns [38].

There were 273 (0.9%) deaths during the index burn admission (114 ≥ 20% TBSA; 96 <20% TBSA; 70 unspecified TBSA) and a total of 3,647 (11.8%) members of the burn cohort died

after discharge during the study follow-up (87 ≥ 20% TBSA; 1383 <20% TBSA; 2177 unspecified TBSA). In the uninjured cohort there were 8,566 deaths during study follow-up.

Over the study period the burn and uninjured cohorts contributed 485,707 and 2,009,370 person-years of observation, respectively. The median years (IQR) of follow up by age group for burn versus non-injured: <15 years: 18.1 (10.3-25.6) vs 17.9 (10.2-25.5); 15 to 45 years: 16.7 (8.4-25.0) vs 16.7 (8.8-25.0); ≥45 years: 9.0 (3.0-16.0) vs 13.0 (6.0-23.0).

3.2   Admission rates and summed length of stay

There were 25,463 infectious disease post-burn admissions (principal diagnosis) and 31,994 admissions in the uninjured cohort during the study follow-up (refer to Table 2 for classification of infectious diseases). The burn and uninjured cohorts experienced total hospital LOS for an infectious disease of 159,944 and 130,369 days, respectively. The median (IQR) LOS for an infectious disease admission for the burn and uninjured cohorts were 3 (1-6) days and 2 (0-4) days, respectively (0 days LOS corresponds to same day admission and discharge).

The burn cohort had higher age-standardised admission rates when compared to the uninjured cohort over the study period (Figure 1). However, from 1980 to 2012, the age-standardised rate of infectious disease admissions in the burn cohort decreased by an average annual rate of 1.8% (95%CI: -1.6% to -2.1%) while for the uninjured cohort, the average annual rate increased by 0.8% (95%CI: 0.4% to 1.3%). Age standardised hospitalisation rates for major categories of infectious diseases, classified by decade of burn admission (study entry) are presented in Figure 2. For the burn cohort, the infectious disease categories with the most admissions included the lower respiratory tract, digestive tract and the skin and soft tissue, which collectively accounted for 42% of infectious disease admissions during the study period (Table 2).

The graph of annual rates of observed admissions (unadjusted) for infectious diseases for the burn and uninjured cohorts are shown in Figure 3. After adjustment for covariates, the burn

cohort experienced increased hospitalisations for infectious diseases at twice the rate of that experienced by the uninjured cohort (incidence rate ratio (IRR), 95%CI: 2.04, 1.98-2.22). This increase was found for those with a record of infectious disease admission (IRR, 95%CI: 1.94, 1.87-2.01) and for those without prior infectious disease admission (IRR, 95%CI: 2.32, 2.16-2.50). The burn cohort spent 3.46 times as long in hospital with infectious diseases (95%CI: 3.05-3.92) than the uninjured cohort. Adjusted analyses using follow-up from 30 days after discharge also found the burn cohort had increased infectious disease admissions over the study period (IRR, 95%CI: 1.91, 1.85-1.97).

Adjusted sub cohort analyses by decade of study entry identified significantly higher infectious disease admissions rates for the burn cohort for each time period examined, when compared to the uninjured cohort. The lowest adjusted rate ratio was identified for the earliest time period (1980-1989: IRR, 95%CI: 1.82, 173-1.91) with higher rates from 1990 (1990-1999: IRR, 95%CI: 2.16, 2.05-2.28; 2000-2012: IRR, 95%CI: 2.25, 2.10-2.41).

Increased hospital admissions for infectious diseases were found for those who sustained a burn when younger than 15 years of age (IRR, 95%CI: 1.66, 1.58-1.75), 15 to 45 years (IRR, 95%CI: 2.35, 2.23-2.47) and those 45 years and older (IRR, 95%CI: 2.23, 2.07-2.40). Higher rates of admissions for infectious diseases were found for severe burns (IRR, 95%CI: 2.37, 1.89-2.97), minor burns (IRR, 95% CI: 2.22, 2.11-2.33) and burns of unspecified TBSA (IRR, 95%CI: 1.97, 1.89-2.06).

3.3   Incident – first time admissions

Incident analyses were conducted on data of 16,758 individuals in the burn cohort and 112,021 uninjured individuals that excluded those in both cohorts that had a prior admission for infectious disease. A total of 3,741 (burn cohort) and 18,011 (uninjured cohort) first time

admissions for infectious diseases were identified. A significant increase in first time infectious disease admissions in the burn cohort was found when compared to the uninjured cohort. The difference in adjusted admission rates was highest during the first 30 days after the burn discharge (HR, 95%CI: 5.18, 4.15-6.48) decreasing in magnitude with increasing time after burn hospital discharge; 30 days to 1 year after burn (HR, 95%CI: 1.69, 1.53-1.87); 1 year to 10 years after burn (HR, 95%CI: 1.40, 1.33-1.47), and 10 years to 32.5 years after burn (HR, 95%CI: 1.16, 1.08-1.24). In total, 1,058 (28%) of the first time post-burn infectious disease admissions were attributable to burn injury.

3.4   Mortality

The burn cohort had significantly higher adjusted mortality caused by infectious diseases in the first 5-year period after discharge (HR, 95%CI: 1.75, 1.37-2.25), after which time there was no significant effect (HR, 95%CI: 0.94, 0.76-1.15).

4.   Discussion

This study identified increased infectious diseases in pediatric and adult burn patients for a prolonged period after discharge from hospital for the initial burn. Rates of post-burn infectious disease admissions were found to be significantly elevated for those with a burn injury. A marginal dose response effect was observed for burn TBSA severity whereby those with severe burns experienced a 20% higher rate of infectious disease admissions than those with minor burns, when compared with the uninjured cohort. When compared with the uninjured cohort, the burn cohort had a 1.8-fold greater adjusted rate of mortality caused by infectious diseases in the first five years after discharge; thereafter, no difference was found.

To address the possibility that individuals with a prior history of infectious disease admission may be systematically different and have unresolved infectious disease prior to the study start that may influence admissions captured in the study, additional analyses were conducted. These

analyses, compared firstly those with a history of infectious disease admission in the burn and uninjured cohorts, and secondly, those in the burn and uninjured cohorts with no prior admission for infectious disease. While the admission rates compared were higher in the burn and uninjured sub cohorts that had a history of infectious disease admission compared with those for the burn and uninjured sub cohorts with no record of infectious disease, results identified consistently elevated post-burn infectious disease admissions (Figure 3) and adjusted rate ratios of similar magnitude for both scenarios.

Assessment of the time to a first (incident) post-burn admission for infectious disease, excluding those in the burn and uninjured cohorts with prior infectious disease admission, found increased risk of admission. Burn patients were at the greatest risk or vulnerability during the first year after discharge with incident admission rates 5.2-fold higher during the first 30 days, 1.7-fold higher for the remainder of the first year, with the risk reducing to 1.4-fold higher over the subsequent nine years, compared with the uninjured cohort. These incident results support the temporality of the association between burns and infectious disease admissions; however, it appeared that the effect of burn injury on infectious disease admissions was modified by increasing time from the initial injury. The AR% indicated that 28% of all first time (incident) post-burn infectious disease admissions could be attributed to the burn injury.

The age standardised rates revealed that the burn cohort had substantially higher infectious diseases (combined) admissions rates from 1980 to 2012 when compared with the uninjured cohort. However, admissions declined in the burn cohort by an average rate of 1.8% per year and it is most likely that this decline was related to the medical and surgical advances in acute burn treatment over the past few decades [39, 40]. We identified specific categories of infectious diseases, namely, respiratory, skin and soft tissue and gastrointestinal, that contributed disproportionately to the higher admission rate experienced by the burn cohort.

For the burn cohort, comparison of age standardised admission rates by infectious disease category for each decade of study entry, showed declines in respiratory tract (lower and upper) and skin and soft tissue infections with advancing decade of study entry; however, this trend was not observed for gastrointestinal infections. The uninjured cohort experienced an increase in age-standardised admission rates for all categories of infectious diseases except respiratory infections, with advancing decade of study entry. The overall trend of increased infectious disease hospitalisations in the uninjured cohort selected from the general community is consistent with other reports in Australia [41], New Zealand [30] and the United States [32].

The increase in infectious disease in three different epithelial tissues in the burn cohort suggests there may be common pathophysiological mechanisms underlying the increased infection rates after burn injury. Epithelial integrity is critical to reduce infection risk both as an intact structure and through the innate immune functions of epithelia through anti-microbial peptide production. Burn injury has been demonstrated to impact epithelial integrity and epithelial function, with reduced anti-microbial peptide production [42, 43]. This is likely to increase infection risk, whilst disruption to epithelial barriers can also lead to changes to the microbiome which can contribute to increased infection and potentially severe polymicrobial infections [44, 45]. In addition to epithelial changes, suppression of the immune response is likely to contribute to increased infection. In particular, this may contribute to increased severity of infection and therefore increase the likelihood of presentation to hospital for infectious disease. Burn injury is known to induce acute immune suppression [9, 46] and recent studies suggest this suppression may extend long after the injury is healed [15, 47]. Both these systems may ultimately recover post-injury, which is supported by the decreased risk as time post-injury increased, observed in this study.

Strengths and limitations

Linked health data allowed us to investigate long-term hospital patterns based on ICD coding with a comparison uninjured cohort. The Baker infectious disease ICD code set used in this study has been reported to be conservative as it accounted only for admissions that were considered to be entirely or predominantly infectious diseases, and excluded those caused in-part by infections [30]. Our study analyses were based on principal diagnosis data only and the results may underestimate admissions due to infectious diseases that were recorded as additional diagnoses in the hospital records.

Inclusion of indigenous status, social disadvantage, that is also an indicator of lifestyle risk factors (alcohol use, smoking, nutrition, physical activity) and pre-existing comorbidity in the analyses allowed for adjustment of factors that had the potential to increase an individual's risk of admission for infections [48-51]. Inclusion of the national census derived indicator of geographic remoteness allowed adjustment for access to services, including health services, as well as diversity in lifestyle and environmental factors relative to urban, rural and remote areas of Western Australia that may affect admission rates [52]. The majority of unspecified TBSA burn data occurred before 1999; however, subcohort analyses identified significant increases in infectious disease admissions for all TBSA categories. The use of hospital data to measure post-burn morbidity has limitations; episodes of infectious diseases experienced by burn patients managed by primary care in the community are excluded from these data. In addition, hospital administrative data are typically not collected for research purposes. Our analyses are limited by the variables available in the datasets and it is possible that a level of residual confounding may exist. This is an observational study using health administrative data and as such this study does not have the capacity to elucidate specific underlying pathophysiology that may be involved. We expect these results will be applicable to other countries of similar models of burn care.

5.   Conclusions

Increased infectious disease morbidity was found for an extended period after the initial burn, suggesting prolonged effects of burns on immune function. The increase in infectious diseases in three different epithelial tissues (skin, gastrointestinal, respiratory tract) in the burn cohort suggests there may be common pathophysiology underlying the increased infection rates after burns. However, future research is required to understand how burns activate specific mechanisms within the body that would increase an individual's longer term susceptibility to infectious diseases.

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Table 1   **Baseline socio-demographic and pre-existing health status factors**

Characteristics	No Injury N (%)	Burn injury N (%)	p-value
Total	123,399	30,997	
Demographic			
Aboriginality			
Yes	2,993 (2.4)	4,481 (14.5)	<0.001
Social disadvantage quintiles*			
Quintile 1. (Most disadvantaged)	14,597 (12.0)	6,579 (21.6)	<0.001
Quintile 2.	28,339 (23.4)	9,878 (32.4)	
Quintile 3.	22,142 (18.2)	6,354 (20.8)	
Quintile 4.	21,671 (17.9)	3,833 (12.6)	
Quintile 5. (Least disadvantaged)	34,609 (28.5)	3,857 (12.6)	
Remoteness**			
Major city	88,278 (72.8)	15,810 (51.7)	<0.001
Inner regional	11,725 (9.7)	3,360 (11.0)	
Outer regional	11,653 (9.6)	4,958 (16.2)	
Remote	5,897 (4.9)	3,434 (11.2)	
Very remote	3,697 (3.0)	3,011 (9.8)	
Health status			
Any comorbidity (CCI>=1)†	4,691 (3.8)	3,131 (10.1)	<0.001
Prior admission for infectious disease ‡	3,497 (2.8)	3,145 (10.1)	<0.001

* SEIFA socio-economic disadvantage quintiles; missing values 1.3% burn, 1.7% no injury

** ARIA+ remoteness classification; missing values 1.4% burn, 1.8% no injury

† Comorbidity based on derived Charlson Comorbidity Index (CCI) using 5-year look-back

‡ Principal diagnosis record of hospitalisation for infectious disease using 5-year look-back period

Table 2   **Number of admissions (%) for infectious diseases classified by sub-conditions in the burn and uninjured cohorts, 1980-2012**
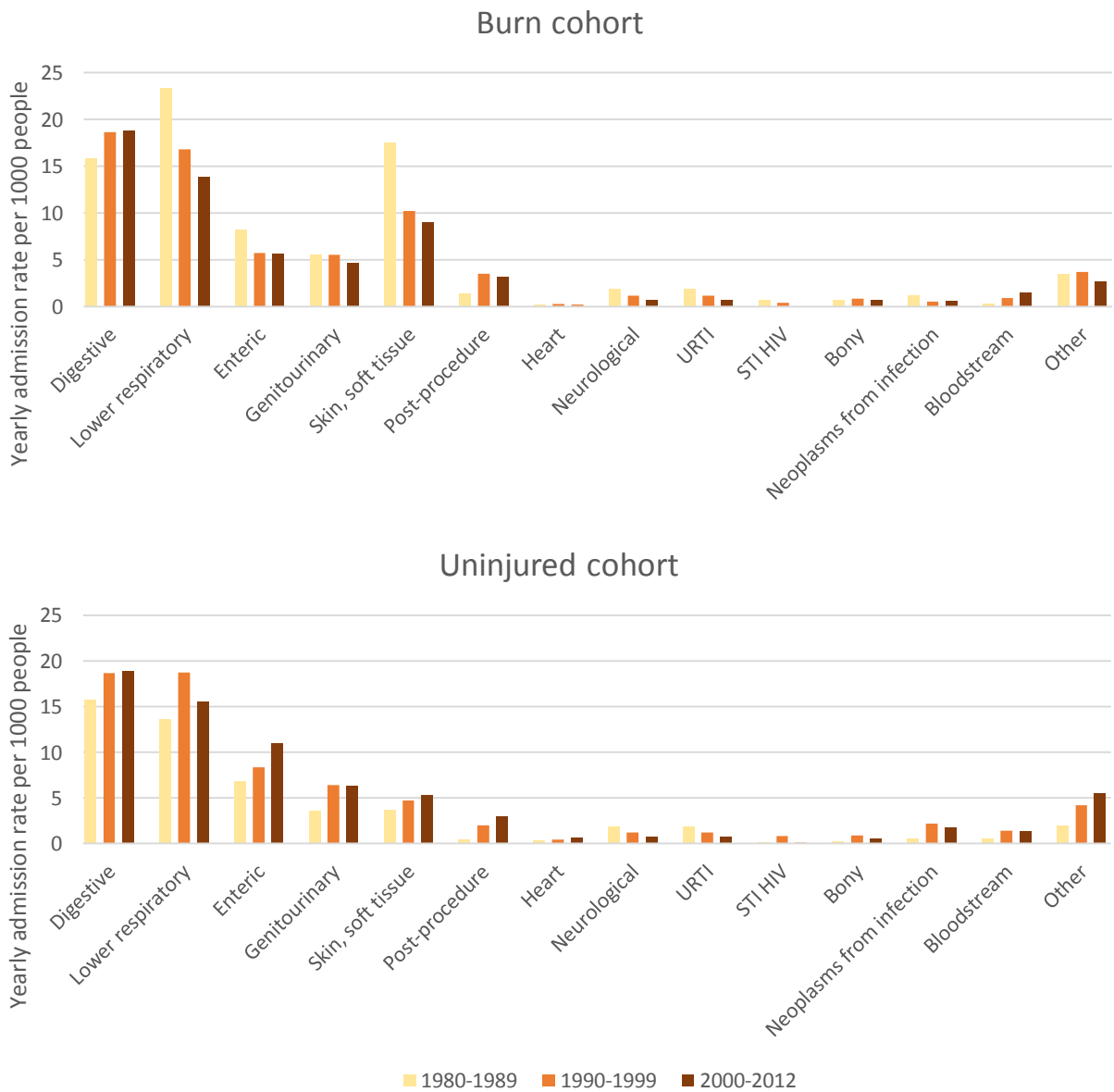
	No Injury N (%)	Burn injury N (%)
Enteric infections	723 (2.3)	412 (1.6)
Enteric symptoms	3417 (10.7)	2170 (8.5)
Septicemia	423 (1.3)	476 (1.9)
Sexually transmitted infections	106 (0.3)	108 (0.4)
HIV/AIDS	65 (0.2)	6 (0.0)
Meningococcal disease	27 (0.1)	14 (0.1)
CNS viral infections	102 (0.3)	57 (0.2)
CNS general infections	137 (0.4)	157 (0.6)
Eye infections	149 (0.5)	241 (0.9)
Ear infections	2269 (7.1)	987 (3.9)
Upper respiratory tract infections (RTI)	2794 (8.7)	1717 (6.7)
Tuberculosis	28 (0.1)	16 (0.1)
Acute Lower RTI	3451 (10.8)	3963 (15.6)
Chronic Lower RTI	1884 (5.9)	2021 (7.9)
Heart & circulatory infections	209 (0.7)	114 (0.4)
Oral infections	2331 (7.3)	800 (3.1)
Gastrointestinal tract infections	5056 (15.8)	1956 (7.7)
Hepatic infections	104 (0.3)	38 (0.1)
Viral Hepatitis	135 (0.4)	105 (0.4)
Kidney infections	219 (0.7)	233 (0.9)
Urinary tract infections	1088 (3.4)	1225 (4.8)
Reproductive system infections, male	350 (1.1)	220 (0.9)
Reproductive system infections, female	790 (2.5)	408 (1.6)
Skin infections, typical	1853 (5.8)	3699 (14.5)
Skin infections, other	259 (0.8)	940 (3.7)
Breast infections	59 (0.2)	71 (0.3)
Osteomyelitis	48 (0.2)	40 (0.2)
Joint infections	117 (0.4)	229 (0.9)
Connective tissue	98 (0.3)	77 (0.3)
Neoplasms from infection	672 (2.1)	229 (0.9)
Postoperative infections	904 (2.8)	1305 (5.1)
Adverse effect of ID treatment	73 (0.2)	54 (0.2)
Other Bacterial infections	97 (0.3)	126 (0.5)
Other Viral infections	1164 (3.6)	564 (2.2)
Other Mycoses	135 (0.4)	77 (0.3)
Other Protozoan infections	31 (0.1)	14 (0.1)
Other infectious diseases	72 (0.2)	50 (0.2)
Infections of pregnancy puerperium	546 (1.7)	544 (2.1)
Perinatal infections	9 (0.0)	0
Total	**31,994**	**25,463**

Figure 1: Age standardised rates of infectious disease admissions, burn injury vs. no injury
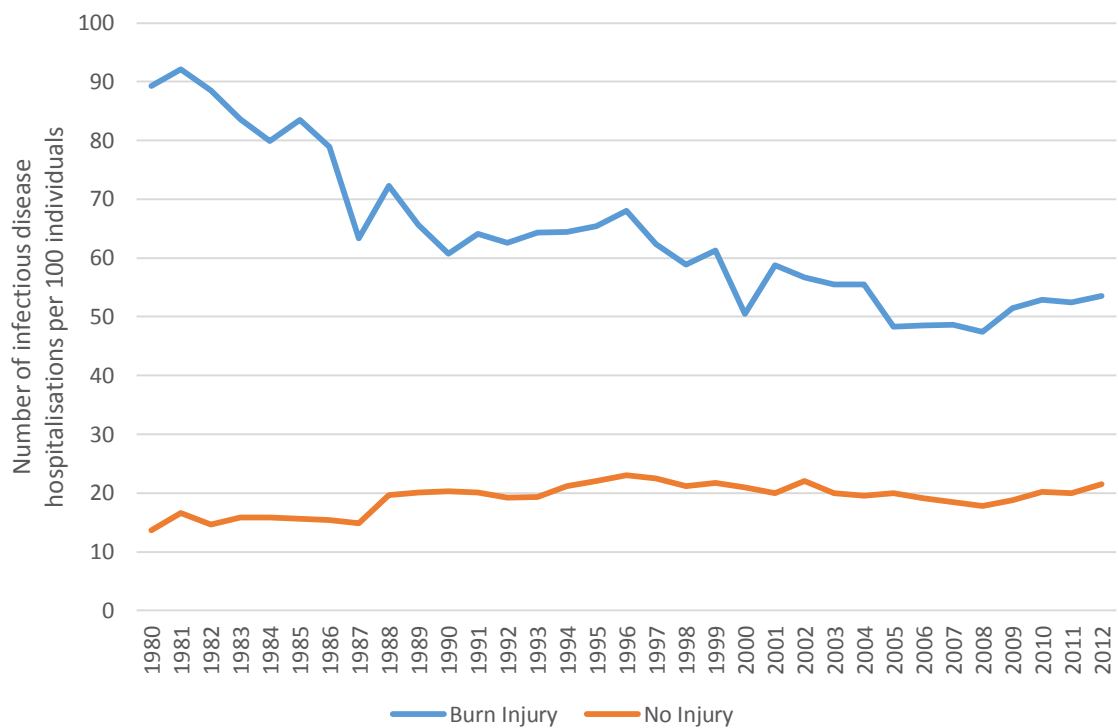
Figure 2: Age standardised annual rates of hospital admissions for major categories of infectious diseases by time period, burn injury vs. no injury

Figure 3: **Unadjusted annual rates of hospital admissions (per 100 person years (PYs)) for infectious disease admissions, burn injury vs. no injury**