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Exploring disparities in acute myocardial infarction events between Aboriginal and non-Aboriginal Australians: Roles of age, gender, geography and area-level disadvantage

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A B S T R A C T

We investigated disparities in rates of acute myocardial infarction (AMI) between Aboriginal and non-Aboriginal people in the 199 Statistical Local Areas (SLAs) in New South Wales, Australia. Using routinely collected and linked hospital and mortality data from 2002 to 2007, we developed multilevel Poisson regression models to estimate the relative rates of first AMI events in the study period accounting for area of residence. Rates of AMI in Aboriginal people were more than two times that in non-Aboriginal people, with the disparity greatest in more disadvantaged and remote areas. AMI rates in Aboriginal people varied significantly by SLA, as did the Aboriginal to non-Aboriginal rate ratio. We identified almost 30 priority areas for universal and targeted preventive interventions that had both high rates of AMI for Aboriginal people and large disparities in rates.

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1. Main text

Aboriginal Australians are currently estimated to have a life expectancy 11.5 years lower for males and 9.7 years lower for females than other Australians (Australian Bureau of Statistics, 2009b), and a total burden of disease that is 2.5 times higher (Vos et al., 2009). Ischaemic heart disease (IHD) alone accounts for 14% of this gap in burden of disease (Vos et al., 2009) and IHD mortality has been estimated to be three times higher in Aboriginal compared with non-Aboriginal people (Gray and Thomson, 2011). Higher incidence of acute myocardial infarction (AMI) among Aboriginal people is likely to be a major contributor to higher mortality from IHD and its sequelae in this population.

Age-specific incidence of AMI has been found to be higher for Aboriginal people in the Northern Territory (NT) (You et al., 2009) and in Western Australia (WA), with the relative disparity particularly high in younger people and women (Katzenellenbogen et al., 2010), and the disparity persisting in urban, regional and remote areas (Katzenellenbogen et al., 2012). However, this research did not look at the influence of place of residence on AMI incidence or the disparity in incidence between Aboriginal and non-Aboriginal people.

While much of the research about how to reduce rates of IHD focuses on individual risk factors (Goldstein et al., 2004; Graham et al., 2007) the area in which someone lives can also have an important impact on IHD incidence (Chaix, 2009; Diez Roux, 2003). People living in the same area tend to be exposed to the same complex interplay of risk and protective factors including ease of access to services, exposure to public health campaigns, delivery of preventive interventions through primary care, walkability and public transport, and social cohesion and interactions. Studies of area of residence and IHD have shown that social disadvantage of areas has an impact on rates of AMI and IHD (Davies et al., 2009), even after adjustment for individual socioeconomic status (Diez Roux et al., 2001).

Identifying the relative contributions of individual factors and geography to disparities in AMI risk, and how disparities vary by area, can assist with making choices about which intervention strategies (universal or targeted) are likely to be most effective,
and where they should be targeted. However, inductive analyses at small area level require substantial population sizes. We took advantage of the availability of linked whole-of-population data for the state of New South Wales (NSW), which has the largest population of Aboriginal Australians of all the States and Territories, to explore in detail the roles of age, gender, geography and area-level disadvantage in disparities in AMI events between Aboriginal and non-Aboriginal people.

2. Methods

2.1. Study design

This was an observational study using routinely collected and linked hospital and mortality data for NSW, Australia between July 2000 and December 2007 and estimated resident population data for the same years.

2.2. Setting

NSW is the most populous state in Australia with an estimated 6.8 million residents (in 2006), 2.2% of whom identified as Aboriginal and/or Torres Strait Islander. NSW is home to approximately 30% of Australia’s Aboriginal peoples, the largest percentage of all the States and Territories in Australia. In 2006, 73% of the total NSW population lived in a major city (Population Health Division, 2006) compared with 42% of the NSW Aboriginal population (Australian Bureau of Statistics, 2006a).

2.3. Data

The Admitted Patients Data Collection (APDC) is a routinely collected administrative dataset containing records for all NSW public and private hospital separations (hospital admissions ending in a discharge, transfer, type-change or death). Patient demographics and multiple diagnoses and procedures are recorded for each separation and coded according to the Australian modification of the International Statistical Classification of Diseases and Related Problems (diagnoses) (National Centre for Classification in Health, 2006). The NSW Register of Births, Deaths and Marriages (RBDM) captures all deaths registered in NSW. The Australian Bureau of Statistics (ABS) codes the underlying cause of death and RSBDM data, as well as including demographic information such as date of birth, sex and Aboriginal status. APDC data (July 2000 to December 2007), RBDM (July 2000 to December 2007) and ABS Mortality Data (July 2000 to December 2007) were linked using probabilistic methods by the Centre for Health Record Linkage (Centre for Health Record Linkage, 2012). We were supplied with de-identified APDC, RBDM and ABS data and merged these using a project-specific unique person number.

Estimated resident populations for each of 199 Statistical Local Areas (SLAs) in NSW were obtained by age, sex and Aboriginal status using the 2001 and 2006 Australian Census data (unpublished data, Australian Bureau of Statistics) and combined with year-specific population projections (Australian Bureau of Statistics, 2009a) to obtain synthetic estimates of the mid-year populations of Aboriginal and non-Aboriginal people by SLA, year, age group and sex (Office of Economic and Statistical Research (OESR), 2010).

2.4. Subjects

We used the linked data to identify NSW residents aged 25 to 84 who were admitted to a public or private hospital with a primary diagnosis of AMI (ICD-10-AM: I21) or a diagnosis of AMI in the second or third diagnosis field, accompanied by a primary diagnosis of IHD (ICD-10-AM: I20-I25) (Randall et al., 2012), or who died with an underlying or contributing cause of death coded as AMI (ICD-10-AM: I21). The death record cases were restricted to those with a coded cause of death of AMI rather than IHD after an internal validation study was run examining the concordance between the coded cause of death and diagnoses recorded in linked hospital records. In this study, the broader IHD definition resulted in a poor positive predictive value (PPV), and the AMI as underlying or contributing COD had a higher PPV, sensitivity and specificity than IHD as underlying COD. We chose the first such event for each person in the period January 2002 to December 2007 as the index event for analysis, with at least an 18 month clearance period for previous AMI events. These first-ever events in the study period thus consisted of first-ever AMI events as well as events for those people who may have had a previous AMI before July 2000. This variable clearance period (where not all patients had exactly the same clearance period) is suitable for the current study because time trends were not the main focus and outcomes were not investigated. Additionally, this minimised the number of prevalent cases included in the analysis. We performed a sensitivity analysis using the last three years of data (2005 to 2007) to investigate the impact on the relative Aboriginal to non-Aboriginal ratio of AMI index events of using various clearance periods of up to four years to remove prevalent AMI cases. For each individual with an index event, we had information on age, sex, Aboriginal status, SLA of residence, and date of event (either date of admission or date of death). We excluded non-NSW residents and those whose address was not able to be assigned to an SLA from the analysis.

2.5. Variables

We used the following individual-level variables: (i) age (in 10-year age groups); (ii) sex (male and female); (iii) year of index event (from 2002 to 2007); and (iv) Aboriginal status.

An audit of NSW public hospitals in 2007 found that Aboriginal people were correctly recorded as Aboriginal on 88% of admissions (Australian Institute of Health and Welfare, 2010). We classified subjects whose index AMI event was a hospital record as Aboriginal if they were recorded as such in their most recent public hospital record (for any diagnosis type). This method for enhancing reporting of Aboriginal status is not biased by the number of hospital records available for individuals (which is related to their level of morbidity) and takes advantage of improved recording of Aboriginality in more recent years (Randall et al., 2013). We classified subjects whose index AMI event was a death record as Aboriginal if this was recorded in the death record or their most recent public hospital record (if there was one). This increased the number of AMI index events reported as Aboriginal by 7% compared to using Aboriginal status as recorded in the index event record alone (either hospital admission or death record). We also conducted a sensitivity analysis using two other definitions; (i) status as recorded on the index event record; and (ii) ever having been recorded as Aboriginal on any record (hospital or death) in the dataset (‘ever identified’). Due to the small proportion of admissions recorded as Torres Strait Islander but not Aboriginal in the NSW hospital data (0.1%), and the fact that Torres Strait Islanders were not coded separately in the mortality data, we considered Aboriginal and Torres Strait Islander peoples as one group for the analysis (referred to as ‘Aboriginal’).

We used two area-level variables, assigned on the basis SLA of residence at the time of the index event: (i) remoteness, classified according to the Accessibility/Remoteness Index of Australia (ARIA+) and grouped into four categories (major city, inner
3. Statistical analysis

We calculated age-standardised AMI event rates using direct standardisation to the 2001 Australian Population. We used single-level and multilevel Poisson models to estimate rate ratios (RRs) for the disparity in AMI events between Aboriginal and non-Aboriginal people, using first AMI events grouped by year as the numerator and estimated mid-year populations by SLA as the denominator. In the multilevel model, we investigated trends in events by year (also stratified by Aboriginal and non-Aboriginal people) and interactions between Aboriginal status and all other variables (age, sex, year, remoteness, and SES) to determine whether the influence of the other variables on AMI rates were the same for Aboriginal and non-Aboriginal people. In order to aid interpretability of the interaction terms, they were presented as new composite variables, such as ‘non-Aboriginal males’, ‘non-Aboriginal females’, ‘Aboriginal males’, and ‘Aboriginal females’ with one reference category, in this case, ‘non-Aboriginal males’. The multilevel analysis included a random intercept and allowed the overall rate of AMI events to vary by SLA. Variation at the SLA level ($\tau^2$) was expressed as a median rate ratio, which was the median of the rate ratios of pair-wise comparisons of people with identical characteristics taken from randomly chosen SLAs. This was an extension of the technique described by Merlo and colleagues (Merlo et al., 2006) for calculating median odds ratios for multilevel logistic regression models and was calculated using the formula:

$$\text{median rate ratio} = e^{0.95\sqrt{\tau^2}}.$$

To investigate the variation in AMI events for Aboriginal and non-Aboriginal people across SLAs, the model was reparameterised to include a random slope for both Aboriginal and non-Aboriginal rates. The relative differences in the rates of AMI in each SLA compared to the average (the area residuals) were calculated for Aboriginal and non-Aboriginal people, and the linear relationship between these residuals was assessed using Pearson’s correlation coefficient. Finally, to investigate whether the magnitude of the relative Aboriginal to non-Aboriginal disparity differed between SLAs, a random slope for Aboriginal status was added to the random intercept model. Estimated Aboriginal to non-Aboriginal RRs by SLA were generated from this model by adding the fixed effect for Aboriginal status to the slope residuals for each SLA (the degree to which the disparity in a SLA differed from the disparity in the ‘average’ SLA) and exponentiating. The estimated RRs by geographic area used ‘shrunken’ residuals from the multilevel models that borrow information from the average to stabilise area-level estimates (Merlo et al., 2005). We tested specific spatial models including a spatial multiple membership model and a conditional autoregressive (CAR) model, and these did not improve model fit over the Poisson multilevel model or change the parameter estimates and as such were not preferred over the multilevel model. Confidence intervals were calculated at the 95% level. Data analyses were carried out using SAS 9.3 (SAS Institute, 2010) and in the multilevel modelling software MLwiN 2.25 (Rasbash et al., 2012) using iterated generalised least squares (IGLS) estimation.

4. Results

We identified a total of 65 548 index AMI events between 2002 and 2007 for NSW residents aged 25 to 84 years (1168 Aboriginal and 64 380 non-Aboriginal). Of these index events, 78% were for people admitted to hospital (80% and 78% for Aboriginal and non-Aboriginal people, respectively) and 22% were for those who died of AMI with no linked AMI hospital admission. The age-standardised rate of first AMI events in the study period was 464 per 100 000 for Aboriginal people and 234 per 100 000 for non-Aboriginal people (Table 1). Among the 25 to 84 year olds included in the study, the average age at first AMI was 56 for Aboriginal people and 68 for non-Aboriginal people. Rates of AMI increased markedly by age group for both Aboriginal and non-Aboriginal people and males had higher rates than females. There was no clear trend by year for AMI rates in Aboriginal people, but AMI rates decreased steadily for non-Aboriginal people from 2002 to 2007. AMI rates were higher in inner regional, outer regional and remote areas than in major cities, and increased with increasing socio-economic disadvantage for both Aboriginal and non-Aboriginal people.

Using a single-level Poisson regression model adjusting for age group, sex and year of event, we estimated that the rate of AMI events in Aboriginal people was 2.30 (95% CI 2.17–2.44) times higher than in non-Aboriginal people. When a random intercept for area was added, allowing the rate of AMI events to vary between SLAs and comparing Aboriginal and non-Aboriginal people within SLAs, the Aboriginal RR decreased slightly to 2.10 (95% CI 1.98–2.23). Table 2 shows the RRs for the adjusted multilevel model including all individual-level variables. Consistent with the age-standardised rates, this model demonstrated that the rate of AMI events increased dramatically with age, and was higher in males than females. Overall, there was a downward trend in total AMI events by year ($p$ for linear trend $< 0.01$). A stratified analysis showed a significant decreasing trend in non-Aboriginal AMI events ($p$ for linear trend $< 0.01$), while the trend in Aboriginal events was of a similar magnitude but did not reach significance ($p$ for linear trend $= 0.08$).

We identified a significant interaction between Aboriginal status and age group ($p < 0.01$) and Aboriginal status and sex ($p < 0.01$), but there was no significant interaction between Aboriginal status and year ($p = 0.94$). Fig. 1 plots these interactions. For age, the significant interaction was evidenced by the large disparity in the younger age groups that decreased steadily with age. Within age strata, the Aboriginal to non-Aboriginal RR was 4.75 (95% CI 3.48–6.49) among 25–34 year-olds decreasing to 0.95 (95% CI 0.77–1.18) for 75–84 year-olds. The significant interaction effect by sex was due to the higher disparity for females than males, with a stratified RR of 2.33 (95% CI 2.12–2.57) for females and 1.98 (95% CI 1.84–2.13) for males.

After adjusting for the individual-level variables, we identified significant variation in the overall AMI event rate according to SLA of residence ($\tau^2 = 0.82$, $p < 0.01$). This variation equated to a median rate ratio of 1.31; in other words, for any population group defined by age, sex, year and Aboriginal status from two randomly chosen areas, the rate of AMI events in one area was on average 31% higher than in the other area. Area-level variation was even greater for AMI rates in Aboriginal people, with a median rate ratio of 1.77, suggesting that area of residence had a greater impact on AMI rates in Aboriginal people than in non-Aboriginal people. The correlation between the Aboriginal and non-Aboriginal area-level residuals was 0.72, indicating that there was reasonable agreement in those areas with high and low rates for both Aboriginal and non-Aboriginal people. However, there was significant variation in the Aboriginal to non-Aboriginal disparity by area ($p < 0.01$). The Aboriginal to non-Aboriginal RRs by SLA from
the random slope multilevel model are shown in Fig. 2, grouped by remoteness. Aboriginal people had higher rates of AMI events than non-Aboriginal people in almost all SLAs in NSW, and in many SLAs, this disparity was significant at the 95% confidence level (those areas where the confidence limits do not cross 1). This is a relative measure and could be influenced by areas with particularly low rates of AMI in non-Aboriginal people, so areas of particular note were those where the relative rate was higher for Aboriginal people and the rate of AMI events for Aboriginal people was higher than average. These “high rate, high disparity” SLAs were spread throughout NSW, and are highlighted on the map in Fig. 3.

We added the area-level variables of remoteness and SES into the fully-adjusted individual-level model one at a time to see how much of the variation in overall AMI rates by SLA they explained (Table 3). Adding remoteness of residence demonstrated that rates of AMI events were significantly higher outside the major city areas, but remoteness only explained 6% of the area-level variation. In contrast, SES explained 37% of the variation by area, and there was a clear gradient of higher AMI rates with increasing area disadvantage. Interactions between remoteness (p < 0.01) and SES (p < 0.01) and Aboriginal status are shown in Fig. 4. These show increasing disparities in AMI rates between Aboriginal and non-Aboriginal people with increasing remoteness and increasing socioeconomic disadvantage.

### 4.1. Sensitivity analysis 1: Clearance of prevalent cases

To identify the influence of the duration of the clearance period, we estimated the Aboriginal to non-Aboriginal AMI RR for the years 2005 to 2007 in a multilevel model accounting for age group and sex. This yielded RRs of 2.26 (95% CI 2.09–2.45) with no clearance period, 2.20 (95% CI 2.03–2.39) with a one-year clearance, 2.17 (95% CI 2.00–2.35) with a two-year clearance, 2.15 (95% CI 1.98–2.33) with a three-year clearance, and 2.11 (95% CI 1.94–2.29) with a four-year clearance.
4.2. Sensitivity analysis 2: Reporting of Aboriginal status

We explored two alternative ways of classifying Aboriginal status: Aboriginal status as recorded on the AMI index event (either hospital admission or death record); and ‘ever identified’ as Aboriginal in any record in the entire linked dataset. Index event records classified 1091 subjects as Aboriginal and the ‘ever identified’ algorithm classified 1953 as Aboriginal, compared with 1168 for the method used in the main analysis. When entered into a multilevel model adjusted for age group, sex, year and SLA the Aboriginal to non-Aboriginal RR estimated using Aboriginal status from index event records was 1.95 (95% CI 1.83–2.08); while that estimated using the ‘ever identified’ algorithm was 2.58 (95% CI 2.44–2.72).

5. Discussion

We found that the age-standardised rate of first AMI events in NSW between 2002 and 2007 was 464 per 100 000 for Aboriginal
people and 234 per 100,000 for non-Aboriginal people. After adjusting for age, sex and year of event, the rate of AMI events in Aboriginal people was 2.3 times higher than in non-Aboriginal people (95% CI 2.17–2.44). This disparity persisted, only slightly reduced (RR 2.10, 95% CI 1.98–2.23), when we accounted for SLA of residence using a multilevel model. The disparity in AMI rates was particularly high in the younger age groups, and was larger in females than in males. AMI rates in NSW for all people decreased by 12% over the study period, and while there was also a decrease over time for Aboriginal people, this trend was not significant. It must be noted, however, that the decreasing trend found in this study may have been due to the short clearance period and the possibility that there were prevalent cases of AMI in the earlier years of the study. Conversely, the number of confirmed AMI events may have increased over time due to the roll-out of the troponin test, a more sensitive and specific biochemical marker for the diagnosis of AMI (Alpert et al., 2000). The change in definition of an AMI to include the troponin test in 2000 resulted in events previously defined as angina being defined as small AMIs and has been shown to increase the number of NSTEMI events identified (Roger et al., 2010). However, a validation study in WA found that the hospital administrative data underestimated the number of AMIs defined with the new troponin biomarker in 2003 (Sanfilippo et al., 2011). There is no equivalent validation study in NSW, so it is difficult to estimate how the new definition impacted on trends in our study.

We identified significant variation in AMI rates by area of residence both overall and for Aboriginal people. The SES of an area accounted for a greater proportion of this variation than its remoteness, and the rate of AMI events was highest in the most disadvantaged areas. AMI rates were higher in Aboriginal than non-Aboriginal people in almost all SLAs in NSW, and the size of this disparity varied significantly by area. Combining information from the variation in AMI rates by area for Aboriginal people and variation in the Aboriginal to non-Aboriginal disparity by area highlighted almost 30 “high rate, high disparity” areas for Aboriginal people. These were predominantly SLAs classified as inner and outer regional areas, comprising mainly medium to large sized

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**Fig. 3.** Map of New South Wales, Australia, marking the “high rate, high disparity” Statistical Local Areas, where the rate of AMIs for Aboriginal people is higher than for non-Aboriginal people as well as being higher than the average rate for Aboriginal people.

**Table 3**

<table>
<thead>
<tr>
<th>Remoteness of residence</th>
<th>RR</th>
<th>95% CI</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major city</td>
<td>1.00</td>
<td></td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Inner regional</td>
<td>1.16</td>
<td>1.04–1.28</td>
<td></td>
</tr>
<tr>
<td>Outer regional</td>
<td>1.11</td>
<td>1.01–1.23</td>
<td></td>
</tr>
<tr>
<td>Remote/very remote</td>
<td>1.22</td>
<td>1.02–1.45</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SES quintile</th>
<th>RR</th>
<th>95% CI</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 least disadvantaged</td>
<td>1.00</td>
<td></td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>2</td>
<td>1.26</td>
<td>1.11–1.43</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>1.40</td>
<td>1.24–1.58</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1.46</td>
<td>1.30–1.64</td>
<td></td>
</tr>
<tr>
<td>5 most disadvantaged</td>
<td>1.70</td>
<td>1.52–1.91</td>
<td></td>
</tr>
</tbody>
</table>

*Rate ratio.  
*Confidence interval.

Area-level factors added one at a time to the fully adjusted individual-level model (adjusted for Aboriginal status, age, sex and year) due to being highly associated.
rural towns and their hinterlands, and were more likely to be situated in Northern NSW.

The main strengths of the study include the large Aboriginal population size of NSW and the complete population coverage that was available using linked routinely collected data. This allowed us to look at small-area variation in AMI rates and also to compare across years. Additionally, our application of multilevel modelling techniques allowed us to account for clustering by area of residence and produce “shrunken” small-area estimates, which are not as prone to random fluctuations as crude or standardised rates. However, the linked data brought with them some limitations. In particular, data were only available from July 2000 onwards, so it was not possible to remove all prevalent cases of AMI using a substantial clearance period. We used a minimum of an 18-month clearance period to maximise the amount of data available for analysis, and conducted a sensitivity analysis to investigate the impact of longer clearance periods on the Aboriginal to non-Aboriginal AMI RR. The RR attenuated slightly with longer clearance periods, suggesting that using an 18-month clearance may have overestimated the disparity. However, this effect was likely to be minimal in comparison with the potential for underestimation associated with the known under-recording of Aboriginal status in hospital (Australian Institute of Health and Welfare, 2010) and death (Australian Bureau of Statistics, 2008; Taylor et al., 2012) data, particularly in more urban areas. We used a ‘most recent’ algorithm to enhance reporting of Aboriginal status, which increased the numbers of AMI events reported as Aboriginal by 7% (15% in major cities), but our sensitivity analysis using an ‘ever identified’ algorithm produced a far higher Aboriginal to non-Aboriginal AMI rate ratio, suggesting that our estimates of the disparity were indeed conservative. Differences in recording of Aboriginal status by area may have increased the geographic variability in Aboriginal AMI event rates; however, our enhancement algorithm differentially increased numbers of events reported as Aboriginal in major cities, redressing at least in part the differential under-recording in urban areas. The probabilistic linkage may have resulted in some false positive links as well as missed links but quality assurance measures at the Centre for Health Record Linkage ensure that these are kept to a minimum. At the time of extraction of the current study data, the false positive rate was estimated to be 4/1000 records (0.4%) and the false negative rate was estimated to be < 5/1000 records (<0.5%).

The standardised rates found in our study were not as high as those found in a study in the NT from 1992 to 2004, also using an 18-month clearance period (647 per 100 000 for Aboriginal residents and 381 per 100 000 for non-Aboriginal residents) (You et al., 2009). The NT study also found that while the non-Aboriginal AMI rates for people aged over 40 years decreased over the study time period, rates increased in non-Aboriginal 20–39 year olds and Aboriginal people aged 20 years and over. The differences in results are not unexpected due to the earlier time period in their study and the higher proportion of people in the NT who live in regional and remote areas, but the definition used to identify incident AMI events was also broader than that used in the current study.

The incidence of AMI in 25–74 year old Aboriginal and non-Aboriginal people (Katzenellenbogen et al., 2010) and the relationship with remoteness (Katzenellenbogen et al., 2012) was examined in WA between 2000 and 2004. The first study found similar results to ours: a greater relative disparity in rates of AMI for younger Aboriginal people and for females, but the magnitude of the disparities was larger. The study had a much longer clearance period (15 years) than ours, but used an ‘ever identified’ method for reporting Aboriginal status. The different analysis method (stratified standardised rates vs multilevel modelling) makes it difficult to directly compare the results by remoteness with our
study. Consistent with our results, the WA study found that AMI incidence for Aboriginal people was higher than for non-Aboriginal people in all remoteness strata. However, in contrast, there was not a clear pattern of increasing disparities for Aboriginal people with increasing remoteness. Comparisons between our findings and those of other Australian studies must be made with caution, not only because of the methodological differences, but also potential differences in culture, geographic distribution of Aboriginal people, and access to and provision of services in different States and Territories.

The higher rate of AMI events in Aboriginal people compared with non-Aboriginal people, particularly in younger age groups, points to the importance of primary care and interventions to prevent the early development of heart disease and contributing conditions such as diabetes. In an audit of randomly selected primary care clients in various locations in Australia, Peiris et al. (2009) identified gaps in the preventive care of Aboriginal Australians: 53% of the sample were not adequately screened for cardiovascular disease (CVD) risk and under-screening was higher in younger age groups. While these health care gaps were similar to those found in non-Aboriginal care settings, improvements are more urgently needed for Aboriginal Australians.

While we found that Aboriginal women had a lower rates of AMI than Aboriginal men, the relative disparity between Aboriginal and non-Aboriginal women was greater than in men. This greater disparity for women was also found in WA for AMI incidence (Katzenellenbogen et al., 2010) and in national data for coronary heart disease prevalence (Penm, 2008). One possible reason for this is the relatively higher rate of smoking and diabetes mellitus in Aboriginal women when compared with non-Aboriginal women (Penm, 2008). However, in research on predicting risk of coronary heart disease using the Framingham Index (which includes both smoking status and whether diabetes has been diagnosed) Wang and Hoy found that risk was particularly under-estimated for Aboriginal women (Wang and Hoy, 2005).

The reasons for the greater risk of coronary heart disease for Aboriginal women (relative to non-Aboriginal women) after accounting for known risk factors needs to be further investigated.

Our study found that the SES of the area was a more influential area-level factor than remoteness in explaining variation in rates of first AMI events. Davies et al. (2009) found that area-level SES was influential in explaining differences in AMI rates across areas in Scotland. The possible reasons for the impact of area-level SES on rates include clustering of individuals with certain SES characteristics within areas and the association between individual SES and AMI risk factor prevalence, however, studies that have been able to adjust for individual SES as well as area-level SES have found a unique contribution of the SES of an area to rates of chronic heart disease (Diez Roux et al., 2001; Sundquist et al., 2004).

The higher proportion of Aboriginal people living in more disadvantaged areas where there is a higher rate of AMI did not explain the higher rates of AMI for Aboriginal people overall; rather we found an increase in the disparity with increasing disadvantage. This may point to a higher sensitivity among Aboriginal people to the factors associated with lower SES, such as poverty and lower education levels, which increase the risk of AMI. Another possibility is that there were unmeasured factors specific to Aboriginal people and correlating with SES, such as stress (Brown and Blashki, 2005), experience of racism (Brown and Blashki, 2005), early life predisposition to cardiovascular disease (McNamara et al., 2012) that increased the risk of AMI for Aboriginal people living in the more disadvantaged areas.

Our small-area analysis identified almost 30 “high rate, high disparity” SLAs, mainly in inner regional and outer regional areas of Northern NSW. These present priority areas for the introduction of both universal and targeted preventive intervention opportunities and will therefore be disseminated by the study team to the NSW Ministry of Health, the Aboriginal Health and Medical Research Council and other relevant organisations. Further research to characterise these areas may highlight possible reasons for the burden in these areas, such as access to primary prevention services, physical environment, social cohesion and social norms, and point to the types of interventions that are needed. In a study on smoking during pregnancy in WA, Aboriginal women mentioned smoking as a ‘normal’ and accepted behavior (Wood et al., 2008), suggesting that targeting whole communities, and not just individuals, is important in order to reduce individual behaviours such as smoking.

Unfortunately, there is only limited evidence about what interventions are likely to be effective. While longer term outcomes are yet to be assessed, the Audit and Best Practice for Chronic Disease (ABCD) Extension project has shown improvements in delivery of best practice services for prevention, detection and management of chronic diseases within Aboriginal primary health care settings (Squierhou et al., 2010). The Kanyini Vascular Collaboration has a number of research projects underway to evaluate primary and secondary prevention to improve Aboriginal health, including a trial of a polypill (containing low dose aspirin, a statin and two blood pressure lowering medicines) for those at a high risk of cardiovascular disease (Kanyini Vascular Collaboration, 2013). The results of these trials will provide direction for interventions to improve management of cardiovascular risk among Aboriginal people. Implementation research, conducted in partnership with Aboriginal communities, to support the wide-scale adoption of findings from these and other current research projects, is a pressing priority.

6. Conclusion

Rates of first AMI events occurring in the 7.5 year study period were higher in Aboriginal compared with non-Aboriginal people. The disparity was greatest in the younger age groups and in females. There was significant variation in overall AMI rates by area that was partly explained by area-level disadvantage. There was also significant geographic variation in Aboriginal AMI rates and the disparity in rates between Aboriginal and non-Aboriginal people, pointing to potential priority areas for implementing universal and targeted preventive interventions.

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References


Randh, J., Browne, W.J., Healy, M., Cameron, R., Charlton, C., 2012. MLwN Version 2.2.5 [software]. Centre for Multilevel Modelling, University of Bristol, Bristol.


