Income inequality and alcohol related harm in Australia

Is there really a Gini in the bottle?

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Executive Summary

The overall aim of the research was to conduct an ecologic examination of the relationship between income disparity and the rates of key alcohol caused harms at a local-area level in Australia. This relationship has not been examined in previous research and an investigation of the nature of this relationship has the potential to significantly extend our understanding of geographic patterns in the extent and nature of alcohol caused harm and how this relates to social and contextual factors evident at a local area level in Australia. The findings from the research will have important public policy implications in relation to our attempts at intervention in the area of alcohol caused harms.

We chose to examine two measures of alcohol caused harm, hospitalisation and death. The specific objectives were for the period 1999-2001 to:

1. develop measures of income disparity (Gini coefficients) for Australian local areas
2. develop measures of the rate of key alcohol related harms (hospitalisation and death) for Australian local areas as well as rates for a number of control conditions (eg diverticulitis)
3. using multi-variate models, examine the relationship between income inequality and the rate of alcohol caused harms at a local area level
4. map the relationship between income inequality and the rate of alcohol caused harms in Australian local areas

These objectives were largely unaltered from those detailed in our original funding proposal. However, due to time and funding constraints we were unable to conduct the analysis of 1996 data for comparison.

Methods

Drawing on previous social epidemiological work on inequalities and health outcomes, we used an ecologic design in which the relationship between the distribution of income inequality and alcohol caused harm was examined across Australian Local Government Areas (LGAs). The key data sources were the hospitalisations obtained from the National Hospital Morbidity Database and deaths obtained from the ABS Mortality Datafile, as well as LGA-level income and other sociodemographic information obtained from the 2001 census. We had originally planned to analyse the data following the multi-level methods outlined by Galea et al. (2003). However, as we had few individual-level variables available, we chose to standardise the morbidity and mortality data at an LGA level (via indirect standardisation) in order to account for individual-level variation, and then use these standardised rates for LGAs in further area-level analyses. The resultant measures were a series of continuous outcomes specifying the extent of alcohol caused ill health or death within a given area relative to the national average, that were matched with the rate of occurrence of a series of control conditions. In general we only used measures of ill health or death that were wholly caused by drinking. We then fitted a series of multivariable linear regression models examining the relationship between income inequality and the alcohol measures after adjusting for the socio-economic disadvantage of the LGAs.

Results

We were able to develop a Gini coefficient to measure the extent of income inequality at an LGA level in Australia. Through our analyses we were able to show that this measure of income inequality was significantly and strongly associated with three measures of alcohol caused harm: hospitalisations wholly caused by acute drinking, hospitalisations wholly caused by chronic drinking, and deaths wholly caused by chronic drinking. The nature of this relationship was curvilinear across all three measures. An example of the relationship is given in Figure E1, which shows that with increasing inequality, the rate ratio of acute wholly-alcohol-caused hospitalisations first decreased but increased dramatically as the Gini coefficient approached 0.2. Maps of the model-predicted values are presented in the Appendix according to Australian LGAs.

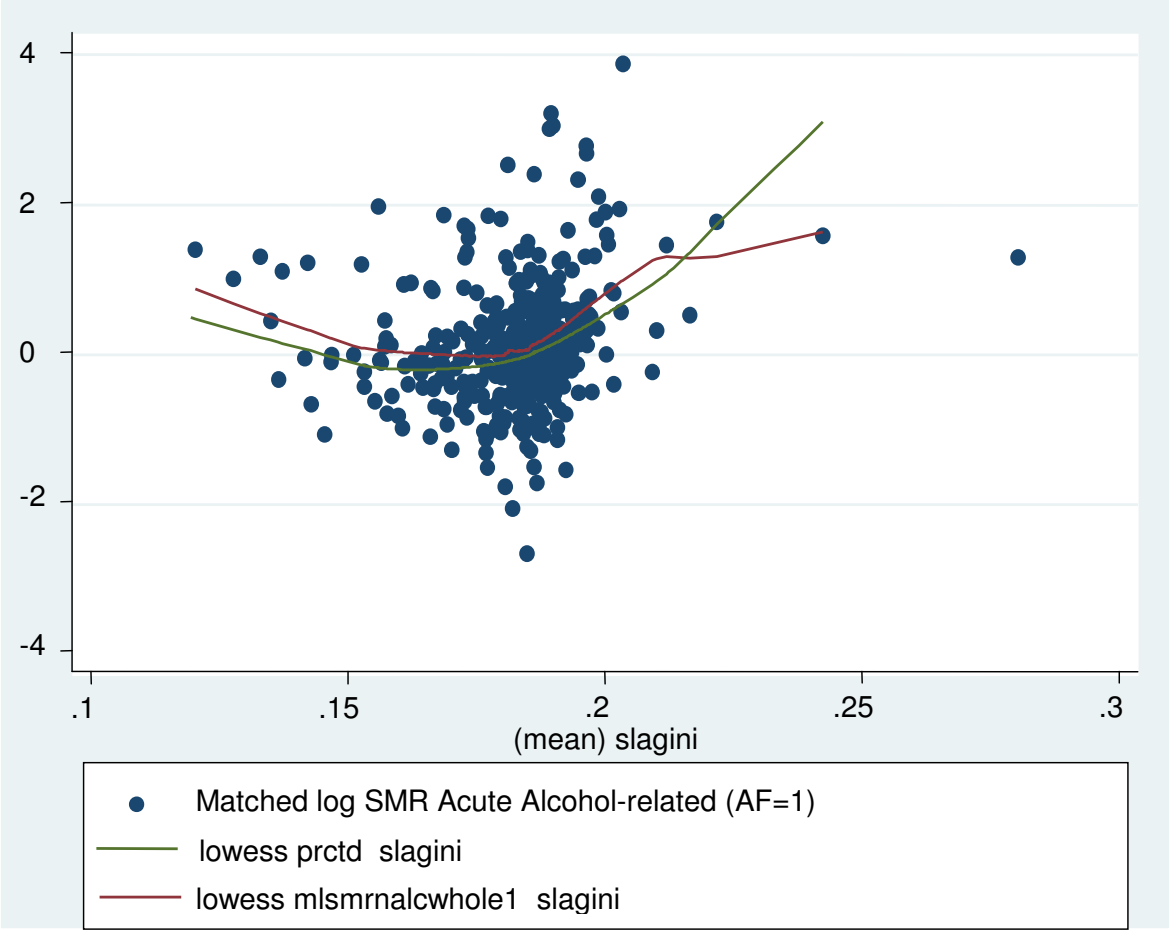

Figure E1: Gini coefficient by rate ratio for acute wholly-alcohol-caused hospitalisations for Australian LGAs in 99/00 fiscal year (trendlines show Loess curves of best fit for model predicted, (green) and raw scores (red))

There was no significant association between the GINI coefficient and either measure of acute alcohol caused (both wholly caused and partially caused) death we examined, probably due to the relatively small numbers of these deaths evident at an LGA level.

Conclusions

This study is unique in a number of ways. To our knowledge, it represents the first attempt at developing a Gini coefficient, based on income distribution alone, for use at a local area level in Australia. Further, it is the first to examine the ecological relationship between income inequality and alcohol related harms undertaken internationally. The study's findings build significantly upon previous study of these alcohol caused outcomes in Australia and point to further research both within and outside the alcohol field. We expect to publish the findings of the study soon, with an anticipated submission date to the Lancet of May 2007.

1 Introduction

Alcohol causes a significant amount of ill-health and death in Australia (Chikritzhs et al., 2003). These effects in the health domain are a significant component of the social costs attributed to drinking, conservatively estimated at $7.5 billion in 1998/99 (Collins & Lapsley, 2002) and alcohol is a major risk factor contributing to the burden of disease (Victorian Department of Human Services, 1999).

The extent and nature of alcohol caused harm in Australia is measured through a variety of mechanisms, notably the National Alcohol Indicators Project (NAIP) (Chikritzhs et al., 2003). This work has highlighted the incidence and prevalence of key alcohol caused harms such as death and hospitalisation across time and place in Australia. For example, it was estimated that there were 3290 alcohol caused deaths in the 1997 calendar year and 72302 alcohol caused hospitalisations in the 1996/97 financial year (Chikritzhs et al., 1999). Nevertheless, the work undertaken on the NAIP project has been largely descriptive in nature with little attention to the underlying causes of the findings that have emerged during the course of that project. The aim of the research undertaken in the current study has been to compliment NAIP work on alcohol caused harm in Australia through an ecologic examination of the relationship between measures of income disparity and the rates of key alcohol caused harms at a local area level in Australia.

Background

Previous research has shown that the harms caused by drinking are mediated by a variety of social and contextual factors operating at an individual as well as a community level. For example, social class (as represented by occupational categories) has been shown to independently account for the occurrence of alcohol caused harms (Hemmingsson, Lundberg, Romelsjo, & Alfredsson, 1997). Wider socio-economic status variables such as income and spending power have also been shown to be associated with acute, chronic and total alcohol caused death (Makela, 1999). This relationship is such that lowered socioeconomic status is associated with higher likelihood of alcohol caused death. Previous Australian work has demonstrated the extent to which these effects are evident in studies using data available at an ecologic level (Dietze, Rumbold et al., 2000; Jonas et al., 1999). This work has shown that the relationship between drinking and alcohol caused hospitalisation in local areas is mediated by factors such as income levels and unemployment (Jonas et al., 1999). Nevertheless, much of the variability in the data at a local area level remains unexplained by these social factors (Dietze, Rumbold et al., 2000), with evidence of a residual relationship between drinking and harms at a local area level even after adjustment for key sociodemographic variables included in analyses (Jonas et al., 1999). This residual relationship may be explained by variables related to drinking patterns (Bondy & Rehm, 1998; Bondy, 1996) or result from limitations in the measurement or use of socio-demographic indicators.

Social epidemiology and income inequality

Research in the field of social epidemiology has shown significant associations between social contextual factors (eg social capital, income disparities, socio-economic status) and a variety of health outcomes (Berkman & Kawachi, 2000). A considerable amount of work in the field has focused upon income inequality or disparity. Income inequality is typically measured through the Gini coefficient.

A Gini coefficient represents the area under a Lorenz curve of the proportion of the population in specific income categories given on the x-axis and the proportion of the total population's income on the y-axis (see Figure 3.1 below). It ranges from 0 (equitable income distribution) to 1 (maximum income inequality) and can be applied across time and place. Income inequality measured in this way has been shown to be associated with a variety of health and social outcomes including rates of all-cause mortality (Ross et al., 2000), violent crime (Hsieh & Pugh, 1993) and life expectancy (see Lynch et al., 2004, for a review).

However, the relationship between income inequality and health remains controversial, with some recent reviews and studies suggesting that the evidence of an association between income inequality and mortality is equivocal at best (Lynch et al., 2004). Nevertheless, some research by particular groups has continued to show an association between income inequality and measures of health (eg Subramanian & Kawachi, 2006).

In the field of alcohol and drug epidemiology there is a tradition of research that can be broadly construed as a type of social epidemiology, some findings from which have been detailed above (eg Makela, 1999). However, Sandro Galea and David Vlahov in particular have been instrumental in articulating a social epidemiologic framework for understanding patterns and trends in alcohol and drug use and related harms (Galea, Nandi, & Vlahov, 2004). One of the specific issues that Galea and others have focused on is the relationship between neighbourhood-level income inequality and a number of outcomes related to the use of alcohol and drugs. Indeed, there have been few studies of the association between income inequality and alcohol and drug related outcomes conducted outside of their group. Their work on alcohol and drugs, conducted largely in New York City, has demonstrated a positive association between neighbourhood income inequality and drug overdose mortality (Galea et al., 2003) as well as alcohol and cannabis use (Galea, Ahern, Tracy, & Vlahov, in press), that is independent of other neighbourhood characteristics and individual-level variables such as personal income.

As is the case with the general mortality studies reviewed by Lynch (2004), there have been some inconsistent results in terms of the association between alcohol and drug related harms and income inequality. For example, Blomgren et al. (2004) found no significant association between area-level income inequality and alcohol caused mortality in a Finnish study. Nevertheless, it should be noted that the Gini coefficient used in that study showed little variation (ranging between 0.20 and 0.24) which may explain why no significant association was found (the results, while non-significant, were in the expected direction). Indeed, the small variation in intra-country income inequalities in studied countries other than the USA has been proposed as an explanation for the mixed pattern of results found for income inequality in social epidemiology more broadly (Lynch et al., 2004).

While some of the previous work undertaken in relation to alcohol can be broadly understood as social epidemiology (eg Jonas et al., 1999), to our knowledge there has been no study of the relationship between income inequality and alcohol and drug related outcomes in Australia. This represents a missed opportunity for Australian alcohol and drug research as there is a variety of alcohol and drug related data amenable to such an analysis. Previous data collections in the alcohol field in particular require little manipulation to enable such a study, at least at an ecologic level.

1.1 The current study

The overall aim of the current study was to address the above gap in Australian alcohol epidemiology by conducting an ecologic examination of the relationship between income disparity and the rates of key alcohol caused harms at a local-area level in Australia. As indicated, this relationship has not been examined in previous research and an investigation of the nature of this relationship has the potential to significantly extend our understanding of geographic patterns in the extent and nature of alcohol caused harm and how this relates to social and contextual factors evident at a local area level in Australia. The findings from the research will have important public policy implications in relation to our attempts at intervention in the area of alcohol caused harms.

In epidemiological research ecologic designs are widely used but regarded as being weaker than traditional designs such as the case-control or prospective cohort (Morgenstern, 1995). Nevertheless, we chose an ecologic design as we assumed this would be the only mechanism by which sufficient numbers of cases could be generated and the methods have been widely used in examining related issues such as all-cause mortality (Ross et al., 2000), as well as the relationship between socio-demographic factors and alcohol caused morbidity (Jonas et al., 1999).

Galea et al.'s (2003) work is particularly pertinent to the current study, and our analytic strategy was initially designed on the basis of their work. As indicated, they found a significant association between income disparities or inequalities and drug overdose mortality at a local area level in New York City. This association was independent of other local area variables such as income levels and racial composition. The relationship was curvilinear for both measures of income inequality they used and was much stronger than that found for their 'control' deaths. Their use of controls, in this case deaths resulting from injury unrelated to drug use, is an important feature of their design in which they matched rates of drug overdose in New York neighbourhoods with rates of occurrence of these control deaths. This framework is similar to that used in previous work on alcohol caused harm in Australia (eg Chikritzhs et al., 1999; Jonas et al., 1999), where a number of control conditions unrelated to alcohol were devised in order to control for any biases inherent in some of the ecological data used. Our study has included these control conditions (unrelated to hazardous/high-risk drinking) in analysis as used by Jonas et al. (1999) and Chikritzhs et al. (2003).

We have chosen to examine two measures of alcohol caused harm, hospitalisation and death. In alcohol epidemiology there is considerable controversy around the purported benefits of drinking on these health outcomes at low risk levels, mainly thought to protect against cardiovascular disease (Chikritzhs et al., 2003). While recent research has suggested that these benefits may be overstated (Fillmore, Kerr, Stockwell, Chikritzhs, & Bostrom, 2006), we have chosen to focus on the effects of hazardous/high-risk drinking as ill health and death caused by drinking is best understood in relation to these patterns of drinking. Further, the logic of including a protective effect of drinking within our ecologic design is dubious (as detailed in the methods section below). We have, however, chosen to follow the methods of Chikritzhs et al. (2003) in determining the extent of alcohol caused hospitalisation and death at a local area level as these methods best take into account regional variation in the extent of exposure to hazardous/high-risk drinking.

1.1.1 Objectives

The specific objectives of the current study are to:

5. develop measures of income disparity (Gini coefficients) for Australian local areas
6. develop measures of the rate of key alcohol related harms (hospitalisation and death) for Australian local areas as well as rates for a number of control conditions (eg diverticulitis)
7. using multi-variate models, examine the relationship between income inequality and the rate of alcohol caused harms at a local area level
8. map the relationship between income inequality and the rate of alcohol caused harms in Australian local areas

2 Methods

2.1 Design Overview

Drawing on previous social epidemiological work on inequalities and health outcomes, (Berkman & Kawachi, 2000) the current study used an ecologic design in which the relationship between the distribution of income inequality and alcohol caused harm was examined across Australian Local Government Areas (LGAs). In order to explore this relationship the following information was required by LGA:

1. the number of persons dying or being hospitalised as a result of hazardous/high-risk alcohol consumption and their age and sex;
2. the number of persons dying or being hospitalised as a result of a series of non-alcohol-related conditions and their age and sex;
3. the age and sex distribution of the resident populations;
4. household income of the resident population; and
5. socio-economic characteristics of areas.

We had originally planned to analyse the data following the methods outlined by Galea et al. (2003). They used a multi-level analysis with individual-level variables such as age, sex, ethnicity included in their models along with area-level variables such as population composition and income inequality. However, in the data we obtained for the current study the only individual-level variables available and considered reliable across both morbidity and mortality datasets were the age and sex of the persons ill or deceased. Therefore, rather than adopt a multilevel analytic framework, we chose to standardise the morbidity and mortality data at an LGA level (via indirect standardisation, see below) in order to account for individual-level variation, and then use these standardised rates for LGAs in further area-level analyses. The resultant measures were a series of continuous outcomes specifying the extent of alcohol caused ill health or death within a given area relative to the national average. These alcohol ratios were then matched to the ratios observed for the control conditions within LGAs (to control for potential coding biases etc, see Jonas et al., 1999), with the resultant rate ratio used as the main outcome measure in analysis. We then fitted a series of multivariable linear regression models examining the relationship between income inequality and the alcohol measures after adjusting for the socio-economic disadvantage of the LGAs.

The remainder of this section of the report details the way in which the above information was obtained, manipulated and analysed for the purposes of the study.

2.2 Data Sources

2.2.1 Morbidity

Data on hospitalisations was obtained from the Australian Institute of Health and Welfare's (AIHW) National Hospital Morbidity Database (NHMD). This Database is a compilation of clinical information on hospital separations (equated here to hospitalisations) occurring within each Australian state/territory. It contains information on age at admission, sex, principal cause of hospitalisation and place of residence (according to Australian Standard Geographic Classification, ASGC, systems) for all cases, as well as

some additional demographic information of cases (eg country of birth). Unfortunately the NHMD does not include detailed data on place of residence for Queensland or South Australian hospitalisations, meaning that these two states were not included in the analysis of alcohol caused hospitalisations. Cause of hospitalisation (both primary diagnosis and any applicable external causes) is coded on the NHMD according to International Classification of Diseases *10th revision*, Australian Modification (ICD-10-AM). Hospitalisation data analysed in this report cover the 1999/2000 fiscal year. For the purposes of this report, the only patient-level demographic information included in the analytic strategy were age and sex as it was assumed that the remaining variables (eg ethnicity) were of limited reliability.

2.2.2 Mortality

Mortality data were sourced from the Australian Bureau of Statistics (ABS) Mortality Datafile, which is a compilation of details of all Australian deaths obtained from state and territory Death Registries. The ABS Mortality Datafile contains information on age at death, sex, date of registration of death, date of death, cause of death and place of residence for all cases (according to ASGC), as well as some additional demographic information of decedents (eg country of birth). Cause of death (both primary diagnosis and any applicable external causes of death, E-codes) is recorded on the Mortality Datafile according to ICD-10-AM. Mortality data were obtained for the 2000/01 fiscal year. For the purposes of this report, the only patient-level demographic information included in the analytic strategy were age and sex in order to maintain comparability to the analysis undertaken with respect to morbidity.

2.2.3 Population characteristics

Population statistics for each Australian Statistical Local Area (SLA) were obtained from the 2001 Census. We obtained estimates of Resident Population (ABS ERPs) for June 30 2001 by five year age cohort (0-4yrs, 5-9yrs…) and sex. Weekly income information for households within Australian LGAs was obtained directly from the ABS according to ABS income groupings ($1-39, $40-79, $80-119, $120-159, $160-199, $200-299, $300-399, $400-499, $500-599, $600-699, $700-799, $800-999, $1000-1199, $1200-1499, $1500-1999, $2000+) and household characteristics (number of persons and number of families per household). Socioeconomic characteristics of areas were indexed through the ABS *Socio-Economic Index for Areas, SEIFA* disadvantage score. This score summarises the socioeconomic disadvantage of areas focusing on the following area-level characteristics: low income earners, relatively lower educational attainment and high unemployment. Low scores show high levels of disadvantage while high scores show relatively less levels of disadvantage within areas.

2.3 Data extraction and manipulation

2.3.1 Unit of analysis

All of the data were obtained at an SLA level. We planned to undertake an initial exploration of the data in order to determine an appropriate event rate criterion for inclusion of areas in the analysis for each of the outcome variables under examination. Upon reflection such an exploration of the data was deemed unnecessary as SLA is an arbitrary geographic unit to which no governance structures are attached. We therefore chose Local Government Area (LGA) as the preferred geographic unit as these LGA

boundaries correspond to the administrative areas for which local governments are responsible. Local government is not only widely understood in defining community areas in Australia but also plays an important role in Australian alcohol policy; for example, in determining drinking by-laws, planning issues with respect to licensed premises, and safer city initiatives. This is also the level at which local community initiatives often operate (e.g. local liquor licensee accords, see Lang & Rumbold, 1997). For these reasons the LGA was selected as the preferred geographic unit. Further, as the geographic boundaries of LGAs are aggregations of SLAs, all of the data obtained were easily aggregated for LGA-level analysis.

2.3.2 Population characteristics

The manipulation of the mortality and morbidity data, as well the data analyses undertaken for this project more broadly, required detailed information on the characteristics of populations within areas. The number of people in LGAs, according to age and sex groupings was required in order to calculate age and sex standardised rates of alcohol related morbidity and mortality. The SEIFA index of disadvantage was required in order to provide a summary of the general socio-economic profile of LGAs. In order to describe inequalities in the income distribution within LGAs, a Gini coefficient was required.

The Gini coefficient

As indicated, the Gini coefficient represents the area under a Lorenz curve of the proportion of the population in specific income categories given on the x-axis and the proportion of the total population's income on the y-axis. In other jurisdictions such as the USA precise estimates of household-level income are asked of householders during the administration of the census. Unfortunately, in the Australian census the ABS merely asks householders to provide estimates of household income according to the categories listed above. This means that precise estimates of household income cannot be obtained from the Australian census.

In order to generate income distribution information for the purposes of this study we first determined the number of households within each LGA in each income category by aggregating across the number of persons resident and the type of household. We then took the midpoint of each income category in each LGA and multiplied this by the number of households in each income category in the LGA. However, the income data collected in the Australian census is right-censored because the largest response category available is $2000. In order to provide a parsimonious estimate of the midpoint of this income category we chose a value of $2250 which probably underestimates the true midpoint of this category. However, this means that the resultant income distribution will probably show less variation leading to more conservative estimates of the Gini coefficient. We ranked income categories within LGA, and then formed progressive cumulative totals of numbers of households and income, from lowest to highest. We then numerically integrated the Lorenz curve of cumulative income vs cumulative households, using a simple trapezoidal rule algorithm. For each LGA, a Gini coefficient was then calculated as the difference between 0.5 and the computed area under the LGA's Lorenz curve.

2.3.3 Alcohol related morbidity and mortality

The ABS mortality datafile and the AIHW's NHMD were extracted and manipulated in an almost identical fashion. Any differences in the analyses undertaken between the two datasets will be specified where appropriate.

Diagnostic categories

Five diagnostic categories for hospitalisations and deaths were derived on the basis of the extent of alcohol causation as well as the type of drinking with which the alcohol conditions have been associated. These five categories were:

1. Wholly attributable to acute hazardous/high-risk drinking
2. Wholly attributable to chronic hazardous/high-risk drinking
3. Partially attributable to acute hazardous/high-risk drinking
4. Partially attributable to chronic hazardous/high-risk drinking
5. Control (unrelated to hazardous/high-risk drinking)

Conditions caused by hazardous/high-risk drinking were derived from a meta-analysis of alcohol-caused morbidity and mortality originally published by English et al. (1995) and subsequently updated by Ridolfo and Stevenson (2001) and Chikritzhs et al. (2003). This body of work, described in detail below, involves the generation of alcohol aetiologic fractions which define the degree (either partially or wholly caused) to which alcohol is known to be a causal factor in any particular disease or injury. We used these fractions to derive rates of alcohol-caused morbidity and mortality. The distinction between 'acute' and 'chronic' alcohol-caused conditions relates to the pattern of drinking known to be associated with particular conditions. Chronic conditions are those that tend to develop over many years of alcohol misuse (e.g. oropharyngeal cancer, chronic gastritis) and reflect degenerative disease states. Acute conditions are generally those that result from episodes of drinking to intoxication (e.g. assault, road injury, drowning). Control conditions for the morbidity analyses were those thought to be unrelated to alcohol consumption as used in previous research (Jonas et al., 1999) and included: Acute appendicitis, Diverticulitis, Hyperplasia of prostate, Genital prolapse and Osteoarthritis. For the mortality analysis cases were included in the control groupings if the aetiologic fraction for hazardous/high-risk drinking was 0 (ie there was some association with low risk drinking, see below). Condition lists for all categories can be found in Appendix A.

English *et al*. (1995) identified 37 conditions for which there is sufficient evidence to conclude that hazardous/high-risk drinking is a causative factor in the development of disease or occurrence of injury[1] and calculated aetiologic fractions for each of these conditions. The aetiological fractions show the probability that a given diagnosis was caused by hazardous/high-risk drinking, on the basis of the patient's age and sex. Alternatively, aetiological fractions can also be seen to denote the proportion of cases that may be attributed to hazardous/high-risk drinking, for a given diagnosis. For example, a condition such as laryngeal cancer (partially attributable to alcohol consumption) has an aetiological fraction of 0.2 for males aged 30-34. Therefore, 20% of all laryngeal cancer cases may be attributed to hazardous/high-risk drinking. In contrast, alcoholic liver cirrhosis (wholly attributable to alcohol consumption) has an aetiological fraction of 1.0 for this age and sex specific group, and therefore 100% of cases may be attributed to hazardous/high-risk drinking.

[1] English *et al*. (1995) identified 38 conditions attributable to alcohol due to hazardous or harmful consumption. Thirty seven of these were shown to be caused by high-risk alcohol consumption while high-risk consumption was shown to have a protective effect for cholelithiasis. This protective effect was ignored in the current study. We have also followed Jonas et al. 1999 in assigning an aetiologic fraction of 0 to falls occurring among those aged >65.

The aetiologic fraction for conditions wholly attributable to alcohol consumption (ie 1.0) does not change over time or vary with the level of high-risk consumption in a community. However, in the case of conditions partially attributable to alcohol (eg assaultive injury, road injuries and stroke), the size of the aetiologic fraction is dependent on two main factors - a) the prevalence of high-risk drinking in the population and b) the magnitude of the association between exposure and disease – the relative risk (RR), or alternatively, an estimate of RR such as an Odds Ratio.

In order to better account for regional variations in drinking prevalence in populations across Australia, Chikritzhs et al. (2003) derived state-specific aetiological fractions on the basis of state-based differences in the prevalence of hazardous/high-risk drinking. Chikritzhs et al. (2003) used estimates of the state-wide prevalence of drinking obtained from the 2001 National Drug Strategy Household Survey (Australian institute of Health and Welfare, 2002, specifically from Graduated Frequency questions). The resultant updated state, age and sex –specific aetiological fractions were the ones we used in this research. A major complication was the introduction of the 10th Revision of the *International Classification of Diseases and Related Health Problems* (ICD-10) to Australian mortality and morbidity records in the late 1990's. The structure of the ICD-10 coding system is dramatically different to the ICD-9 version that had been in place for many years. However, Chikritzhs *et al*. (2002) have developed a map to translate conditions from ICD-9 to ICD-10 and we used this map in the current study.

Data extraction

To estimate the number of hospitalisations and deaths that resulted from hazardous/high-risk drinking in Australia, the following steps were undertaken with both the ABS mortality datafile and the AIHW NHMD:

- Cases containing alcohol caused or control diagnostic codes as a primary diagnosis or cause of death were extracted from the relevant dataset and grouped into age, sex and diagnosis categories and SLA of patient/decedent residence.
- Cases were assigned relevant aetiologic fractions on the basis of patient state, age, sex and diagnostic category.
- Data for each SLA were aggregated according to the diagnostic categories detailed above for each age and sex category, weighted by the relevant aetiological fractions. Data were then further aggregated into LGA of patient residence.

As indicated, there is some debate about the protective effect of drinking on health (Fillmore et al., 2006). In some analyses of morbidity and mortality data (eg Jonas et al., 1999), this protective effect is indexed by assigning a negative aetiologic fraction to cases involving the diagnosis code (eg some types of heart disease). The logic of this procedure is dubious as no protective effect could actually cause the ill health or death captured in a given case. Therefore, we did not included cases involving a negative aetiologic fraction (ie a protective effect of alcohol) in our analyses. Fortunately, most of the protective effect of drinking is conferred at low levels of consumption and we only considered conditions caused by hazardous/high-risk drinking. This means that the only protective effect of drinking missed in our analyses is that related to cholelithiasis, a condition for which drinking is only partially protective.

Standardisation

Age and sex are two variables that are known to be directly related to the prevalence of hazardous/high-risk drinking and the corresponding health sequelae (English et al., 1995; National Health and Medical Research Council, 2001). Therefore, the demographic distribution of the population within LGAs will have a profound impact on the rate of alcohol-related morbidity and mortality. In order to control for these effects of age and sex, indirect standardisation of the morbidity and mortality data from each LGA was undertaken (see Bland, 1987). In this procedure the number of hospitalisations or deaths observed in an LGA is compared against the number of hospitalisations or deaths expected in the LGA on the basis of the age and sex distribution of the population. The resultant statistic is known as a standardised morbidity or mortality ratio that allows for comparison across LGAs in relation to the whole of Australia, after adjustment for the age and sex structure of the population within the LGA.

In order to generate standardised rates the following procedure was undertaken:

- The expected number of cases for each diagnostic category in each LGA was computed by first calculating the overall national rates of hospitalisation or death for each diagnostic category in each age and sex category.
- These rates were then multiplied by the number of people in the same age and sex categories within each LGA.
- The raw number of cases for each of the five diagnostic categories detailed above was then divided by the expected number of cases for each Australian LGA as detailed in the following formula:

$$SMR = \frac{\begin{array}{c}Sum\ (\text{observed number of alcohol - related (or control condition) cases} \\ \text{for each age and sex category})\end{array}}{\begin{array}{c}Sum\ (\text{expected number of alcohol - related (or control condition) cases} \\ \text{for each age and sex category})\end{array}}.$$

2.4 Data analysis

All analyses were conducted using Stata/SE V9. The general principle we applied in our analyses was to first consider the relationships between wholly-alcohol-caused ill health or death and income inequality as these conditions are most clearly caused by hazardous/high-risk drinking. If no clear relationship was evident in a given diagnostic category for these wholly-caused conditions we then examined partially-alcohol-caused conditions, with the assumption that the increased number of cases would provide greater statistical power.

Prior to analysis the alcohol outcome data were log-transformed as they were highly skewed at an LGA level. Log-transformation also stabilises the variance of the outcome data. A matched rate ratio was then generated for each of the four categories of alcohol causation (acute wholly-caused, chronic wholly-caused, acute partially-caused, chronic partially-caused). This matched rate ratio was the log-transformed rate of the alcohol SMR divided by the control SMR for each LGA. For example, the matched rate ratio for wholly attributable acute alcohol related hospitalisation = logSMR(wholly attributable acute alcohol related hospitalisation) – logSMR(control hospitalisations).

The resultant matched rate ratios were then entered into a linear regression as outcome variables with LGA-level Gini coefficient and SEIFA disadvantage scores (broken into deciles) entered as predictor variables. There was a large variation evident in the number of cases occurring in LGAs, related in part to the size of the LGAs. In order to control for these variations in the models we weighted the models by the number of cases (both alcohol caused and control) using the analytic weights procedure available in Stata.

3 Results

3.1 LGA characteristics

Figure 3.1 shows the distribution of income across Australian households. Unsurprisingly, this Figure shows that the distribution of income is not wholly equitable (equitable represented by the diagonal). Table 3.1 shows that the mean value of the Gini coefficient (the area of the curve deviating from the diagonal) used in this study was around 0.18, ranging from .105 (most equitable) to 0.28 (most inequitable).

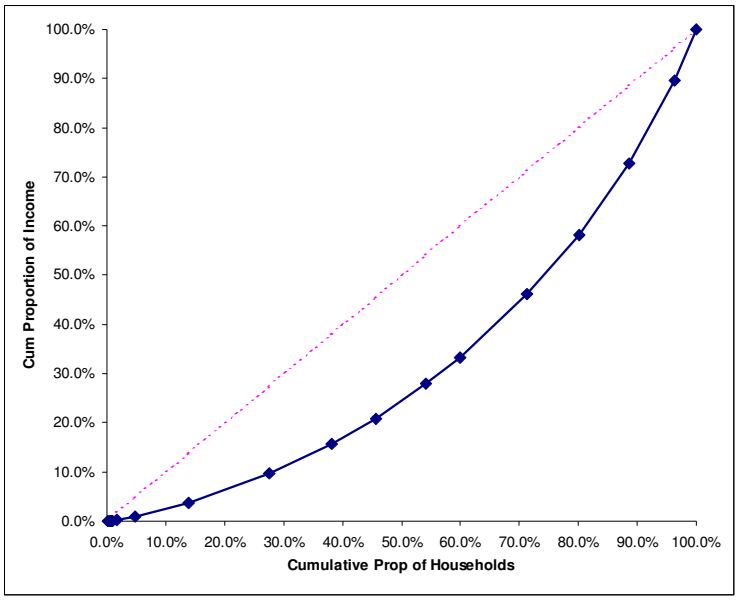

Figure 3.1: Lorenz curve of income distribution across all Australian LGAs

Across all LGAs there was a total of 885 wholly-alcohol-caused deaths and 19467 wholly-alcohol-caused hospitalisations. Table 3.1 shows the major descriptive characteristics of the LGA-level outcome data included in the analysis across all of the diagnostic categories, along with the Gini coefficients included in the different analyses undertaken. The Table highlights the differences in the number of LGAs available for analysis in the mortality data compared to the morbidity data, with around 140 more LGAs available for the mortality analyses because of the inclusion of Queensland and South Australia.

Table 3.1: Descriptive statistics for the key variables included in analyses

Variable	Total	LGA Mean	Min	Max	N LGAs
Population	18726897	29725	85	880519	630
SEIFA disadvantage	na	982.3	406.4	1151.5	630
Gini coefficient	na	.181	.105	.280	580
Morbidity					
Number acute wholly-alcohol-caused	9317	21.8	1	211	376
Number chronic wholly-alcohol-caused	10150	23.7	1	281	350
Number controls	64654	151.1	1	1304	423
(Gini coefficient used)	na	*.183*	*.119*	*.280*	*428*
Mortality					
Number acute wholly-alcohol-caused	170	0.3	1	5	128
Number acute partially-alcohol-caused	1159.2	2.1	.039	15.32	494
Number chronic wholly-alcohol-caused	715	1.3	1	16	265
Number controls (acute)	136	0.2	1	7	88
Number controls (chronic)	11161	19.9	1	191	491
(Gini coefficient used)	na	*.182*	*.105*	*.280*	*562*

3.2 Alcohol-caused morbidity

As shown in Table 3.1 there was a relatively large number of alcohol caused hospitalisations available for analysis. For the purposes of the current analysis we report only those LGA-level measures that were wholly caused by hazardous/high-risk alcohol (both acute and chronic). This is because such hospitalisations are, by definition, caused by hazardous/high-risk alcohol consumption.

3.2.1 Acute hospitalisations wholly caused by hazardous/high-risk alcohol consumption

After computing the rate ratios of acute wholly-alcohol-caused hospitalisations there were 373 LGAs with corresponding Gini coefficients available for analysis. Initial exploration suggested that the relationship was best described as quadratic so therefore we included a quadratic term for the Gini coefficient (Gini-squared) in the model. Table 3.2 shows that there was a highly significant association between the Gini coefficient (squared) and the rate ratio of acute wholly-alcohol-caused hospitalisations at an LGA level, after adjusting for SEIFA disadvantage scores. The model explained around 14% of the variance in the data with an R-squared of .143.

Table 3.2 Regression coefficients and 95% CIs for the predictor variables included in the model for acute wholly-alcohol-caused hospitalisations

Predictor	Coefficient	t	*p*	95%CI	
Gini	22.200	5.520	0.000	14.292	30.109
Gini-squared	566.261	4.580	0.000	323.204	809.319
SEIFA disad 1	N/A				
SEIFA disad 2	-0.172	-1.130	0.258	-0.469	0.126
SEIFA disad 3	-0.245	-1.540	0.124	-0.556	0.067
SEIFA disad 4	-0.185	-1.170	0.242	-0.495	0.125
SEIFA disad 5	-0.241	-1.630	0.103	-0.531	0.049
SEIFA disad 6	-0.401	-2.840	0.005	-0.678	-0.124
SEIFA disad 7	-0.268	-1.530	0.128	-0.614	0.078
SEIFA disad 8	-0.305	-2.040	0.042	-0.598	-0.012
SEIFA disad 9	-0.479	-3.510	0.000	-0.747	-0.211
SEIFA disad 10	-0.147	-1.040	0.299	-0.424	0.131
_cons	0.127	1.150	0.252	-0.091	0.345

The curvilinear quadratic relationship is detailed in Figure 3.2 which shows the raw scores as well as trend lines (plotted using Loess curves of best fit) of the model-predicted and raw scores. The relationship was such that with increasing inequality, the rate ratio of acute wholly-alcohol-caused hospitalisations first decreased but increased dramatically as the Gini coefficient approached 0.2.

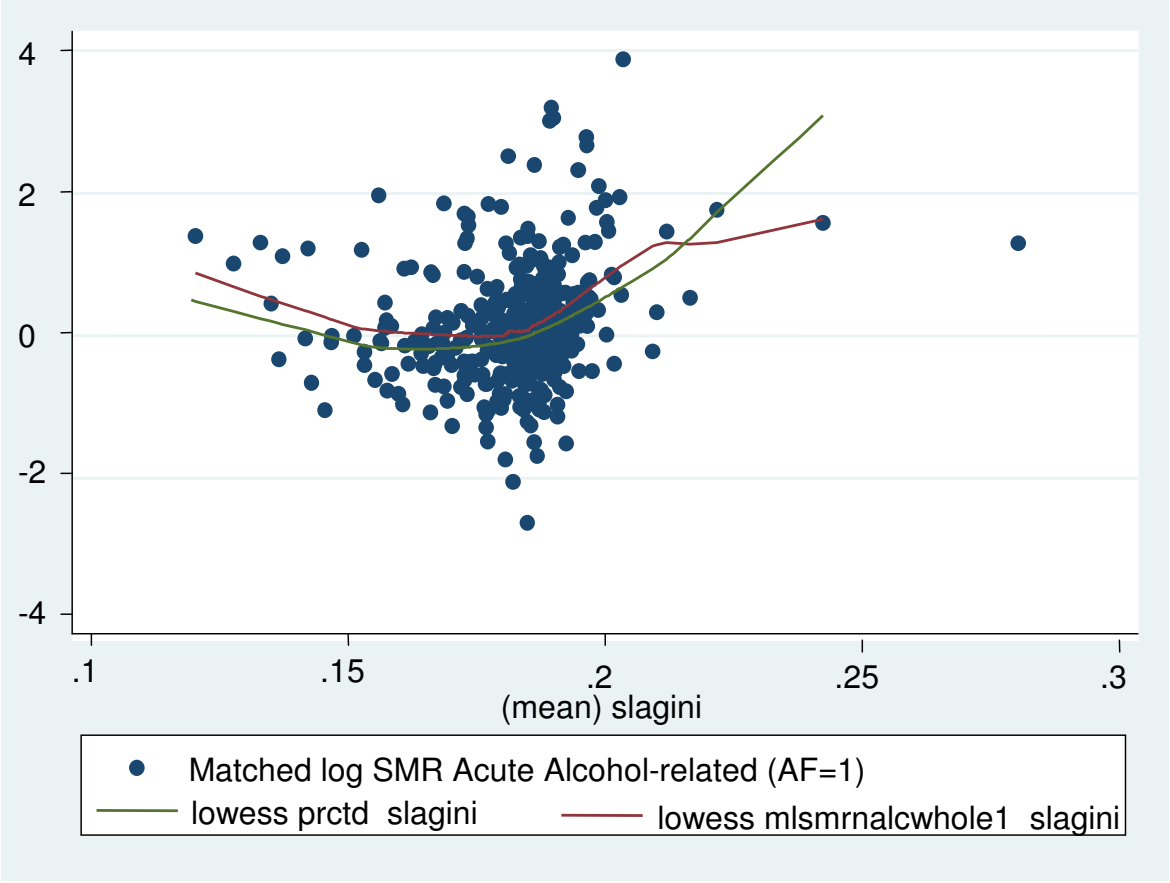

Figure 3.2: Gini coefficient by rate ratio for acute wholly-alcohol-caused hospitalisations for Australian LGAs in 99/00 fiscal year (trendlines show Loess curves of best fit for model predicted, (green) and raw scores (red))

3.2.2 Chronic hospitalisations wholly caused by hazardous/high-risk alcohol consumption

After computing the rate ratios of chronic wholly-alcohol-caused hospitalisations there were 349 LGAs with corresponding Gini coefficients available for analysis. As with the acute data described above, initial exploration suggested that the relationship was best described as quadratic so therefore we included a quadratic term for the Gini coefficient (Gini-squared) in the model. Table 3.3 shows that there was a highly significant association between the Gini coefficient (squared) and the rate ratio of chronic wholly-alcohol-caused hospitalisations at an LGA level, after adjusting for SEIFA disadvantage scores. The model explained around 11% of the variance in the data with an R-squared of .112.

Table 3.3 Regression coefficients and 95% CIs for the predictor variables included in the model for chronic wholly-alcohol-caused hospitalisations

Predictor	Coefficient	t	*p*	95%CI	
Gini	17.853	3.710	0.000	8.396	27.310
Gini-squared	404.838	2.740	0.006	114.461	695.215
SEIFA disad 1					
SEIFA disad 2	0.168	0.960	0.340	-0.178	0.513
SEIFA disad 3	0.027	0.150	0.881	-0.335	0.390
SEIFA disad 4	-0.124	-0.670	0.501	-0.484	0.237
SEIFA disad 5	-0.252	-1.460	0.146	-0.591	0.088
SEIFA disad 6	0.100	0.610	0.542	-0.222	0.422
SEIFA disad 7	0.312	1.540	0.124	-0.086	0.709
SEIFA disad 8	-0.020	-0.120	0.907	-0.362	0.321
SEIFA disad 9	0.198	1.260	0.210	-0.112	0.509
SEIFA disad 10	0.516	3.170	0.002	0.196	0.837
_cons	-0.273	-2.100	0.037	-0.529	-0.017

The curvilinear relationship is detailed in Figure 3.3 which shows the raw scores as well as trend lines (plotted using Loess curves of best fit) of the model-predicted and raw scores. The relationship was almost identical to the acute hospitalisations described above such that with increasing inequality, the rate ratio of chronic wholly-alcohol-caused caused hospitalisations first decreased but increased dramatically as the Gini coefficient approached 0.2.

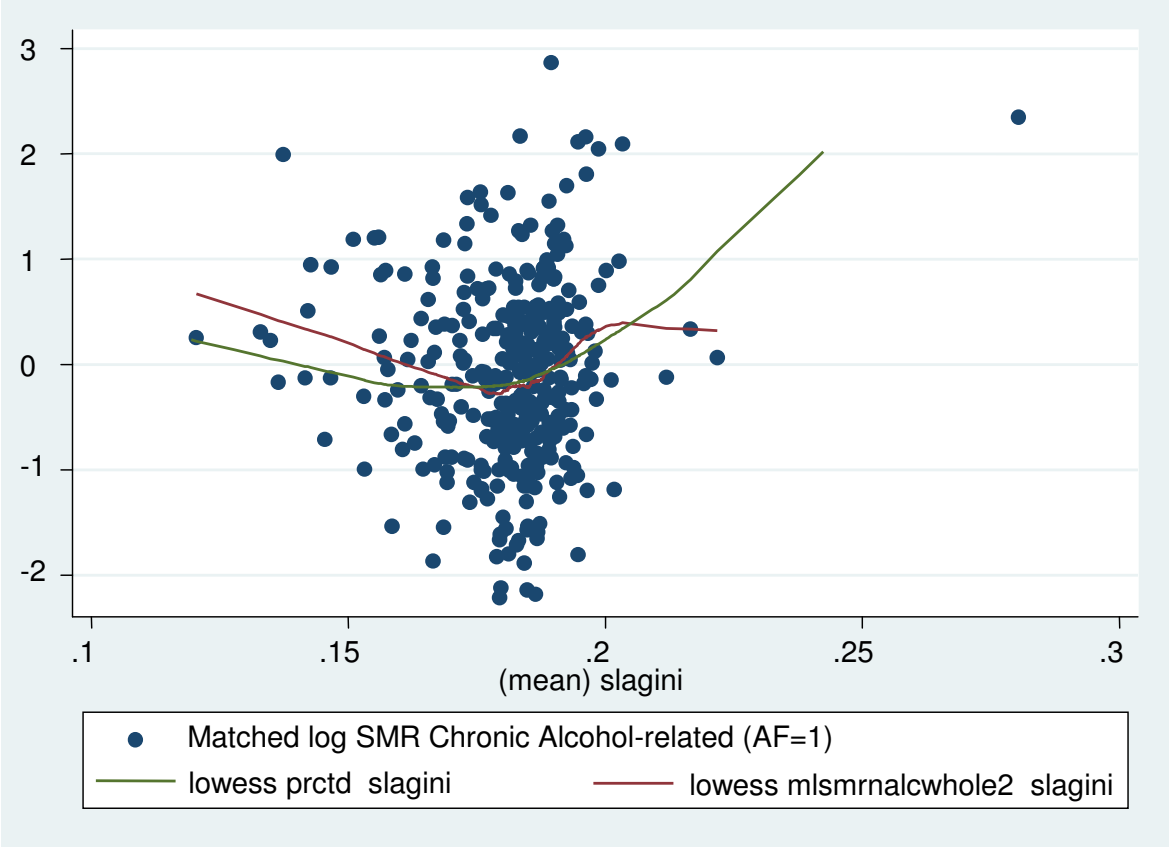

Figure 3.3: Gini coefficient by rate ratio for chronic wholly-alcohol-caused hospitalisations for Australian LGAs in 99/00 fiscal year (trendlines show Loess curves of best fit for model predicted, (green) and raw scores (red))

3.3 Alcohol related mortality

Table 3.1 shows that in comparison to the alcohol caused hospitalisations there was a much smaller number of alcohol related deaths available for analysis. As a consequence of these relatively small numbers we chose to examine first the relationship at an LGA level between the Gini coefficient and cases wholly attributable to hazardous/high-risk drinking (both acute and chronic), before turning to consider those with a positive alcohol aetiologic fraction for hazardous/high-risk drinking to increase the number of cases available for analysis where necessary.

3.2.1 Acute deaths wholly caused by hazardous/high-risk alcohol consumption

After computing the rate ratios of acute wholly-alcohol-caused deaths there were only 180 LGAs with corresponding Gini coefficients available for analysis. Table 3.4 shows that there was no significant association between the Gini coefficient (in either straight or quadratic form) and the rate ratio of acute wholly-alcohol-caused caused deaths at an LGA level. This was the case whether or not SEIFA disadvantage scores were included in the model. Interestingly, SEIFA disadvantage scores were associated with acute wholly-alcohol-caused deaths with the most disadvantaged decile having higher rate ratios than the remaining deciles, significantly so in comparison to deciles 2-4 (areas of relatively high disadvantage). The model explained around 18% of the variance in the data with an R-squared of .181.

Table 3.4 Regression coefficients and 95% CIs for the predictor variables included in the model for acute wholly-alcohol-caused deaths

Predictor	Coefficient	t	*p*	95%CI	
Gini	4.621645	0.55	0.585	-12.0438	21.28709
Gini-squared	319.3543	1	0.32	-313.056	951.7648
SEIFA disad 1	N/A				
SEIFA disad 2	-1.42046	-3.94	0.000	-2.13247	-0.70844
SEIFA disad 3	-1.02009	-2.96	0.004	-1.70039	-0.33978
SEIFA disad 4	-1.1155	-3.07	0.002	-1.83274	-0.39826
SEIFA disad 5	-0.3558	-0.92	0.357	-1.11583	0.404234
SEIFA disad 6	-0.96908	-2.89	0.004	-1.63181	-0.30634
SEIFA disad 7	-0.7406	-1.53	0.128	-1.69662	0.215418
SEIFA disad 8	-0.37517	-0.84	0.403	-1.25855	0.508212
SEIFA disad 9	-0.24156	-0.65	0.516	-0.97498	0.491866
SEIFA disad 10	-0.12109	-0.35	0.728	-0.80765	0.565461
_cons	0.492808	1.84	0.067	-0.03534	1.020953

The relationship between the Gini coefficient and the acute wholly-alcohol-caused deaths rate ratio is detailed in Figure 3.4. This figure highlights not only the fact that no clear relationship was evident but also just how sparse the data were in comparison to the hospitalisation figures detailed above (reflected in the low number of LGAs included in the analysis). For this reason we chose to examine the relationship between the Gini coefficient and the acute partially-alcohol-caused deaths at an LGA level.

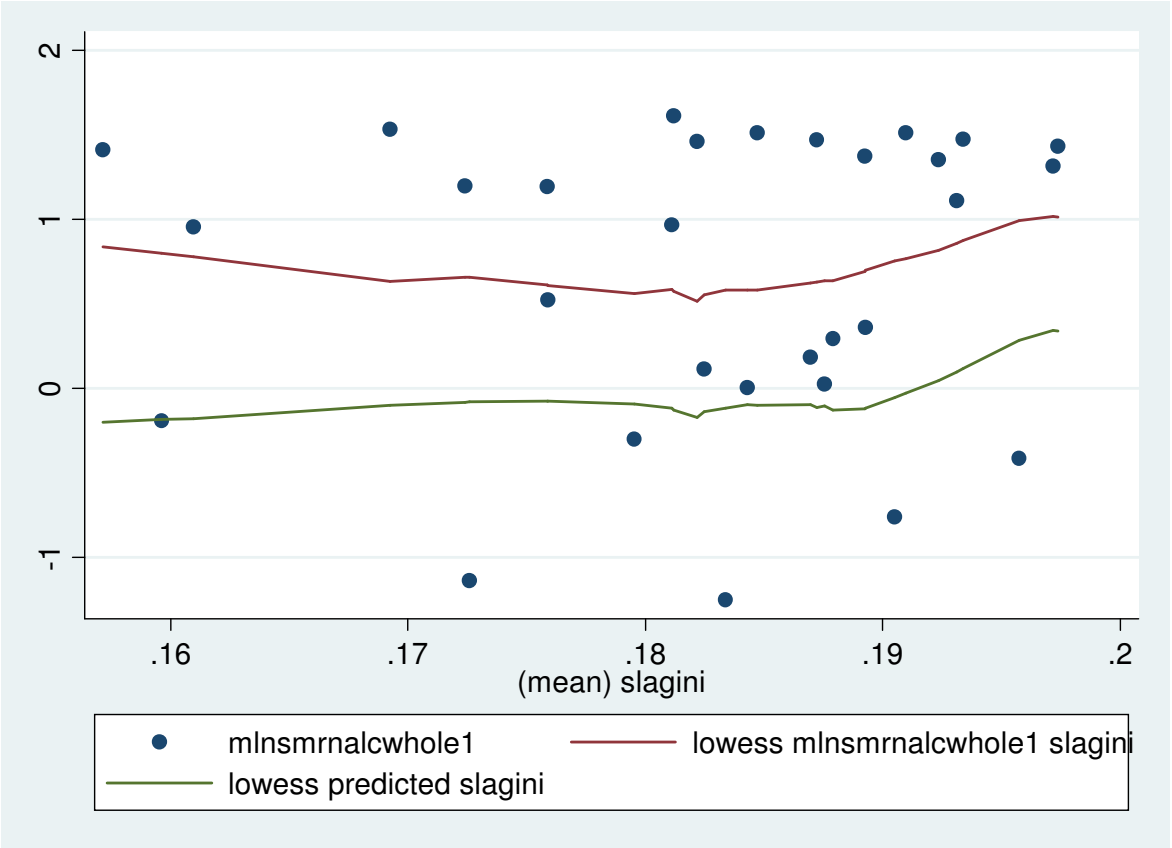

Figure 3.4: Gini coefficient by rate ratio for acute wholly-alcohol-caused deaths for Australian LGAs in 0001 fiscal year (trendlines show Loess curves of best fit for model predicted, (green) and raw scores (red))

3.2.2 Acute deaths partially caused by hazardous/high-risk alcohol consumption

After computing the rate ratios of acute partially-alcohol-caused deaths there were 498 LGAs with corresponding Gini coefficients available for analysis. Table 3.5 shows that there was no significant association between the Gini coefficient (in either straight or quadratic form) and the rate ratio of acute partially-alcohol-caused deaths at an LGA level. Again, this was the case whether or not SEIFA disadvantage scores were included in the model. Further, SEIFA disadvantage were associated with the rate ratio of partially-alcohol-caused deaths with the most disadvantaged decile having higher rate ratios than the remaining deciles, significantly so for almost all of the remaining deciles (which did not vary markedly). The model explained around 13% of the variance in the data with an R-squared of .127.

Table 3.5 Regression coefficients and 95% CIs for the predictor variables included in the model for acute partially-alcohol-caused deaths

Predictor	Coefficient	t	*p*	95%CI	
Gini	-0.509	-0.160	0.874	-6.811	5.793
Gini-squared	90.953	0.960	0.339	-95.727	277.633
SEIFA disad 1					
SEIFA disad 2	-0.870	-5.740	0.000	-1.167	-0.572
SEIFA disad 3	-0.793	-5.680	0.000	-1.067	-0.519
SEIFA disad 4	-0.701	-4.660	0.000	-0.997	-0.406
SEIFA disad 5	-0.275	-1.840	0.067	-0.569	0.019
SEIFA disad 6	-0.744	-5.530	0.000	-1.009	-0.479
SEIFA disad 7	-0.488	-2.820	0.005	-0.827	-0.148
SEIFA disad 8	-0.291	-1.880	0.061	-0.596	0.013
SEIFA disad 9	-0.284	-2.000	0.046	-0.564	-0.005
SEIFA disad 10	-0.433	-3.090	0.002	-0.708	-0.158
_cons	0.676	6.610	0.000	0.475	0.877

The relationship between the Gini coefficient and the rate ratio for the acute partially-alcohol-caused deaths is detailed in Figure 3.5. This figure shows that while there was no clear significant relationship evident between the Gini coefficient and the acute partially-alcohol-caused rate ratio, the values followed the general form of the curves detailed above in relation to the hospitalisation data. Nevertheless, even with the extra LGAs included in this analysis using partial causation there was no significant relationship. This probably reflects the relatively small numbers of acute deaths even partially attributable to hazardous/high-risk alcohol consumption at an LGA-level.

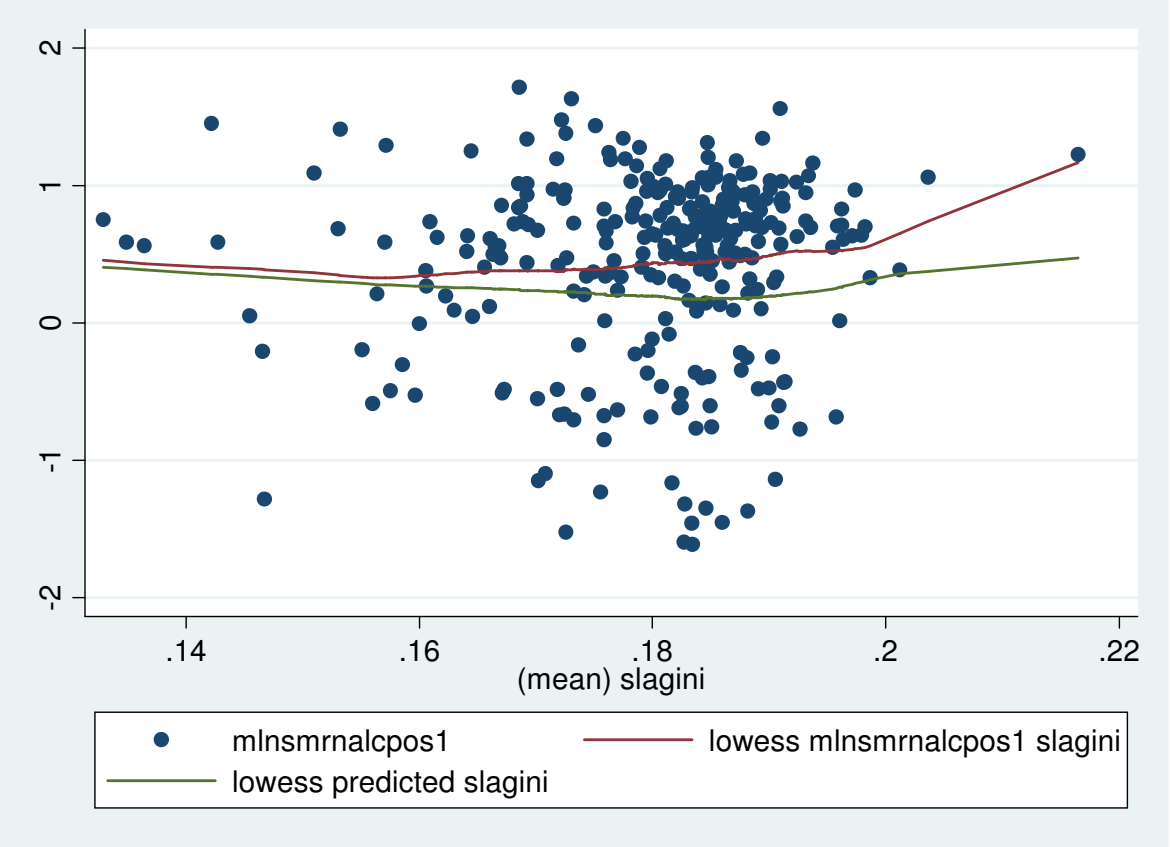

Figure 3.5: Gini coefficient by rate ratio for acute partially-alcohol-caused deaths for Australian LGAs in 00/01 fiscal year (trendlines show Loess curves of best fit for model predicted, (green) and raw scores (red))

3.3.3 Chronic deaths wholly caused by hazardous/high-risk alcohol consumption

After computing the rate ratios of chronic wholly-alcohol-caused deaths there were 499 LGAs with corresponding Gini coefficients available for analysis. Table 3.6 shows that there was a highly significant association between the Gini coefficient (squared) and the rate ratio of chronic wholly-alcohol-caused deaths at an LGA level, after adjusting for SEIFA disadvantage scores. The model explained around 15% of the variance in the data with an R-squared of .153.

Table 3.6 Regression coefficients and 95% CIs for the predictor variables included in the model for chronic wholly-alcohol-caused deaths

Predictor	Coefficient	t	*p*	95%CI	
Gini	32.245	7.380	0.000	23.657	40.833
Gini-squared	513.775	3.310	0.001	209.116	818.435
SEIFA disad 1					
SEIFA disad 2	-0.460	-2.600	0.010	-0.808	-0.113
SEIFA disad 3	-0.214	-1.320	0.189	-0.533	0.106
SEIFA disad 4	-0.264	-1.550	0.122	-0.599	0.071
SEIFA disad 5	-0.078	-0.450	0.654	-0.419	0.263
SEIFA disad 6	-0.315	-2.020	0.044	-0.622	-0.009
SEIFA disad 7	0.023	0.120	0.904	-0.350	0.395
SEIFA disad 8	0.102	0.580	0.562	-0.243	0.446
SEIFA disad 9	-0.103	-0.620	0.533	-0.428	0.221
SEIFA disad 10	-0.076	-0.480	0.634	-0.390	0.238
_cons	-0.063	-0.480	0.633	-0.320	0.195

The curvilinear relationship is detailed in Figure 3.6 which shows the raw scores as well as trend lines (plotted using Loess curves of best fit) of the model-predicted and raw scores. The relationship was different to the patterns described above for the hospitalisation data. While the relationship was curvilinear, there was no evidence of the concave decrease observed in the hospitalisation data, with a flat relationship evident until values of the Gini coefficient of around .17, above which the increase appears to follow a similar pattern to the hospitalisation data described above.

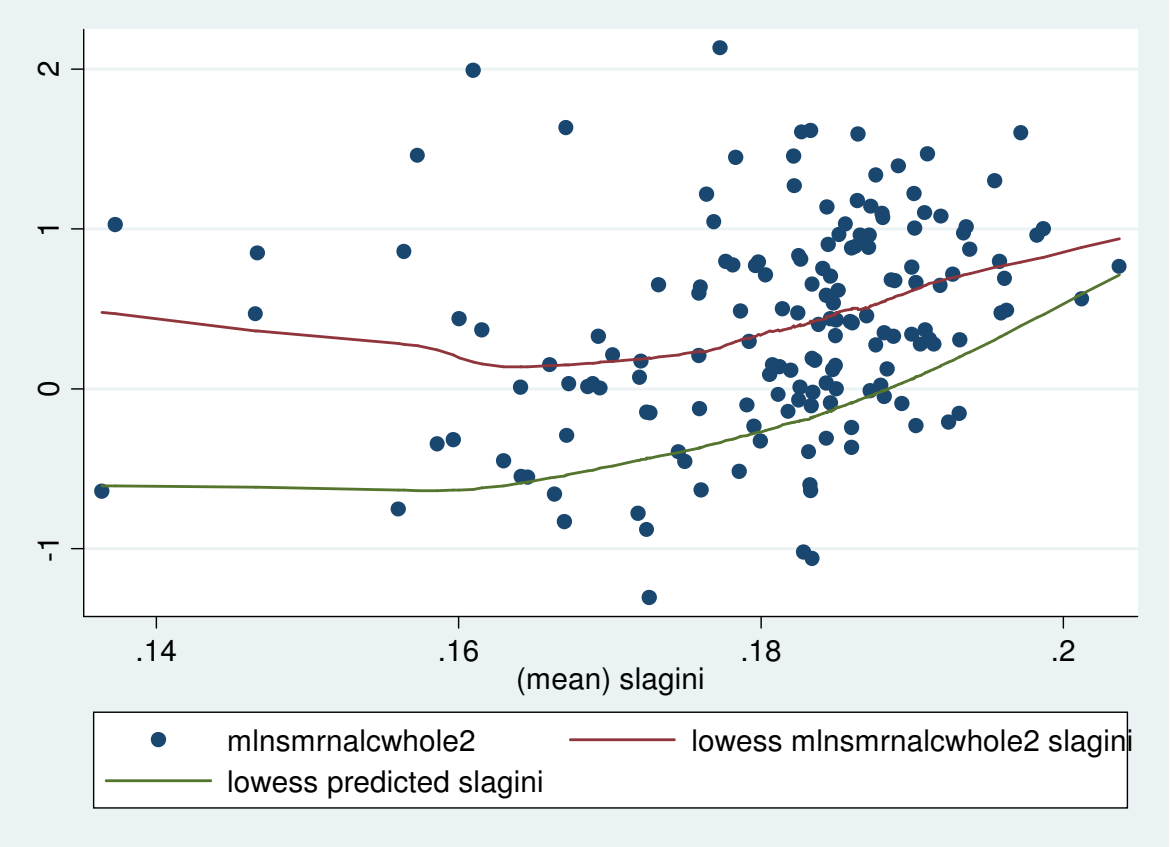

Figure 3.6: Gini coefficient by rate ratio for chronic wholly-alcohol-caused deaths for Australian LGAs in 00/01 fiscal year (trendlines show Loess curves of best fit for model predicted, (green) and raw scores (red))

4 Discussion

We have argued that there is a clear need for studies of the relationships between socio-demographic variables and alcohol caused harms in Australia. The existence of sophisticated systems for monitoring and surveillance of alcohol caused harms provides an opportunity for the relatively straightforward implementation of such study. Research in the field of social epidemiology has demonstrated the relationship between social contextual factors (eg social capital, income disparities) and a variety of health outcomes (Berkman & Kawachi, 2000). Borrowing from the tradition of this work in social epidemiology, our study represents the first attempt at understanding the relationship between income inequality and alcohol caused harms at a local area level.

To reiterate, the objectives of this research were to:

1. develop measures of income disparity (Gini coefficients) for Australian local areas
2. develop measures of the rate of key alcohol related harms (hospitalisation and death) for Australian local areas as well as rates for a number of control conditions
3. using multi-variate models, examine the relationship between income inequality and the rate of alcohol caused harms at a local area level
4. map the relationship between income inequality and the rate of alcohol caused harms in Australian local areas

The findings of the study will be considered in relation to each of the first three objectives. Maps of the model-predicted values for key outcomes measures are presented in Appendix 2.

4.1 Income disparity at a local area level in Australia

To our knowledge, the results presented in this study represent the first attempt at developing a Gini coefficient, based on income distribution alone, for use at a local area level in Australia. Turrell and Mathers (2001) examined local area variation in mortality as a function of the ABS Index of Relative Socio Economic Disadvantage (IRSED) where one of the key measures was a Gini coefficient they developed for mortality inequality, rather than income inequality. Our income-derived Gini coefficient is consistent with that used in the majority of studies in social epidemiology, (Berkman & Kawachi, 2000; Kawachi & Kennedy, 1997) that will be able to be used in studies of other health outcomes in Australia.

The Gini coefficient we developed was limited by the income categories used by the ABS in the collection of census information. These categories necessitate the use of frequency-weighted means in calculating income within areas, which assumes equal distribution of values within categories. Further, the upper category is unbounded which undoubtedly resulted in an underestimate of the mid-point of this category, making our Gini coefficient conservative. Nevertheless, even with these caveats the use of this measure provided new information about local area socio-demographics and alcohol caused harms. Importantly the relationship we observed was different to that evident for other measures of socio-economic disadvantage available for local areas such as the ABS SEIFA index.

4.2 Local area level alcohol caused harms

This study has provided the first LGA level examination of key alcohol related health outcomes undertaken for the whole country. Drawing on the tradition established by the MAPP undertaken in Western Australia (Midford et al., 1998), and Turning Point Alcohol and Drug Centre's Alcohol Epidemiology Project (Laslett, Dietze, & Rumbold, 1996), we have demonstrated that the data available in Australia on alcohol caused harms are amenable to area-based ecological analysis for the whole country. Nevertheless, this analysis is limited in the case of hospitalisation because area of residence information was not available for Queensland or South Australia. Further, it should be noted that the use of hospitalisation data alone will dramatically underestimate the true extent of alcohol caused morbidity in Australia, as the majority of morbidity will be expressed in settings outside of inpatient hospitals, such as emergency departments, general practitioners and specialist treatment agencies (Dietze, Laslett, & McElwee, 2000).

4.3 The ecological relationship between income inequality and alcohol caused harm

This study is the first to provide evidence of a relationship between income disparity and alcohol caused harm in Australia. The nature of the relationship was consistent across the two measures of morbidity (acute and chronic wholly-alcohol-caused hospitalisations) and was similar for chronic wholly-alcohol-caused deaths. In general the results showed that increasing LGA-level income inequality was associated with increasing rates of alcohol caused harm, after adjusting for general socio-economic disadvantage for LGAs. While these relationships appeared strong and robust, there was no evidence of a relationship

between income inequality and either wholly- or partially-alcohol-caused acute deaths, possibly due to the relatively small number of these deaths (even after the application of aetiological fractions).

As indicated in the introduction, there remains considerable controversy about the relationship between income inequality and health outcomes such as all-cause mortality (Lynch et al., 2004; Subramanian & Kawachi, 2004). However, where relationships between income inequality and various health outcomes have been observed, they have typically been shown to be a monotonically increasing function (a straight line); that is, as income inequality increases so too do rates of ill-health or death (Berkman & Kawachi, 2000). In this context our findings of a curvilinear function were unexpected; especially the apparent decline in rates of alcohol caused hospitalisation with initial increases in income inequality. In contrast, the significant relationship between income inequality and chronic wholly-alcohol-caused death appeared to follow a pattern similar to that found by Galea et al (2003) in relation to rates of drug overdose. However, Galea et al.'s matched analysis (where they included other injury death as controls) showed a clear monotonic trend with no statistically significant differences in odds between different percentiles of their Gini coefficient. In this way even our mortality findings differ from those found in previous research.

Various mechanisms have been postulated through which income inequality may manifest an effect on health outcomes (Lynch et al., 2004; Subramanian & Kawachi, 2004). Typically the mechanisms are indirect in that income inequality is thought to be associated with social-contextual processes that may result in biased policy producing 'social capital' favouring the wealthy in an area. Such social capital may be expressed in policy terms such as better resource allocation, but may also reflect better social connectedness (eg having trusted others) for those at higher income levels. Direct effects have also been postulated, whereby living near rich neighbours produces a kind of 'economic envy' among poorer people that results in greater stress and therefore poorer health (and possibly more drinking) (Lynch et al., 2004). These types of processes may explain the area-level increases in adjusted rates of alcohol caused harms observed in our study at the upper end of the Gini coefficient we devised.

However, neither of the above pathways can explain the observed decline in the rate of alcohol-caused morbidity at the lower values of the Gini coefficient. It is unlikely that 'economic envy' of neighbours would be worse for those areas of lesser inequality and it is also unlikely that other forms of social capital would be lesser in these areas, unless there are some confounding factors for which we were unable to control. One candidate explanation here may be the rapid development of the urban fringe around Australia's cities which are typically homogenous with respect to a variety of socio-economic characteristics. Further analysis is required in order to examine the impact of these other area-level characteristics on the relationship we observed. In this light it is not possible to formulate direct policy recommendations (eg interventions designed to reduce income inequalities) as direct intervention to affect income inequality may indeed increase the rate of alcohol related harms in our study.

4.4 Limitations

The proposed study was cross-sectional in nature. Ready interpretation of cross-sectional ecological studies requires the exposure (eg drinking, income inequality) and outcome (eg hospitalisation, death) to occur within a similar timeframe. In this framework it is

reasonable to infer that hazardous/high-risk drinking produced the acute alcohol-caused outcome for which a relationship to income inequality was observed (for hospitalisation at least). The same is not true of the chronic alcohol caused conditions that result from sustained patterns of hazardous/high-risk drinking over time. In this case we need to assume relative stability in persons residence over time, and relatively static levels of inequality. We had originally proposed to test this issue by replicating the analysis for 1996. However, time and resource constraints did not allow us to undertake this analysis in time for this report. We hope to have the opportunity to undertake this analysis prior to further publication of the results of this study.

4.5 Future dissemination

As indicated, the findings of the study are unique in the Australian and international context. We now plan to publish the findings in suitable peer-reviewed journals. We expect to submit first to the Lancet (a prestigious journal that has published some of the findings regarding sociodemographics and health in the past) and then to a range of other journals if not accepted (the rejection rate at the Lancet is high).

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Appendices

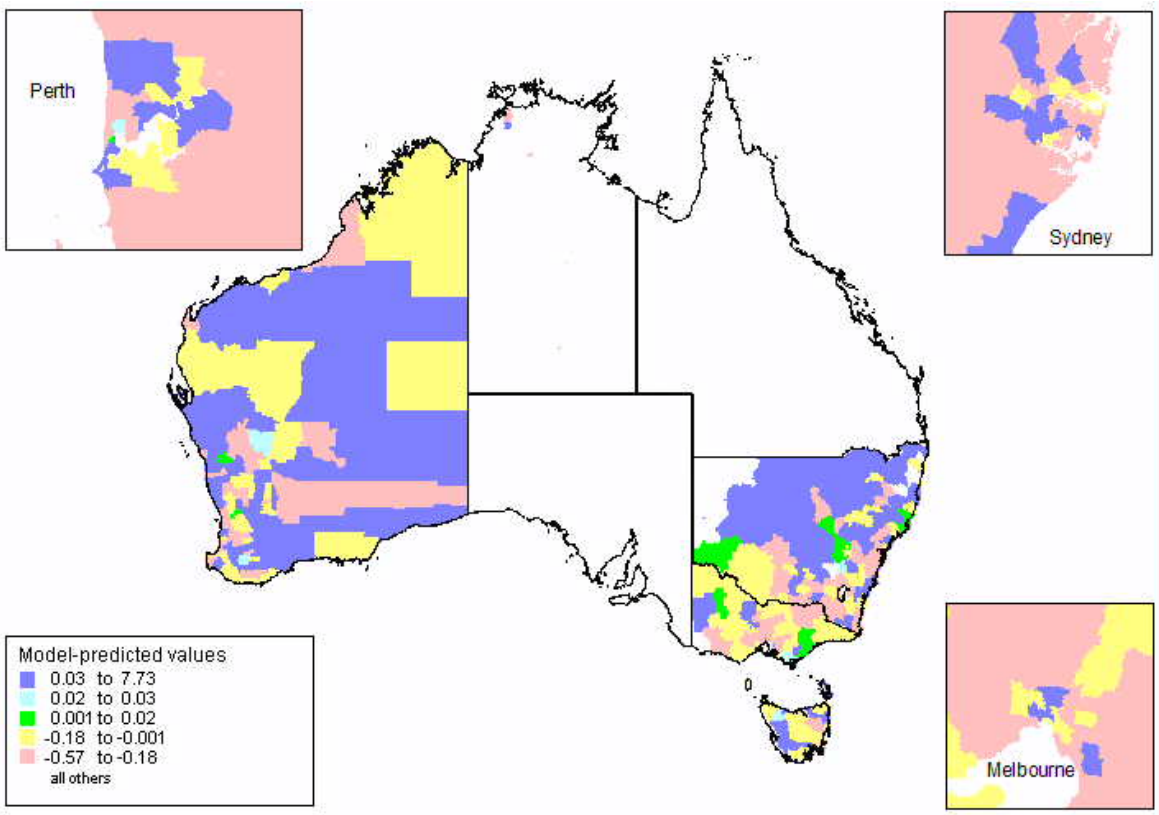
Appendix A: Alcohol-caused conditions and their classifications as used in this study

Acute	Chronic	Mixed
Acute pancreatitis	Alcoholic cardiomyopathy[1]	Stroke
Alcohol abuse[1]	Alcoholic dependence[1]	Suicide
Alcoholic beverage poisoning[1]	Alcoholic liver cirrhosis[1]	
Alcoholic gastritis[1]	Alcoholic poly neuropathy[1]	
Alcoholic psychosis[1]	Chronic pancreatitis	
Aspiration[1]	Epilepsy	
Assault	Female breast cancer	
Child abuse	Hypertension	
Drowning	Laryngeal cancer	
Ethanol toxicity[1]	Liver cancer	
Fall injuries	Oesophageal cancer	
Fire injuries	Oesophageal varices	
Gastro-oesophageal haemorrhage	Oropharyngeal cancer	
Low birthweight	Psoriasis	
Methanol toxicity[1]	Unspecified liver cirrhosis	
Occupational and machine injuries		
Other ethanol and ethanol poisoning[1]		
Road injuries		
Spontaneous abortion		
Supraventricular cardiac dysrhythmias		

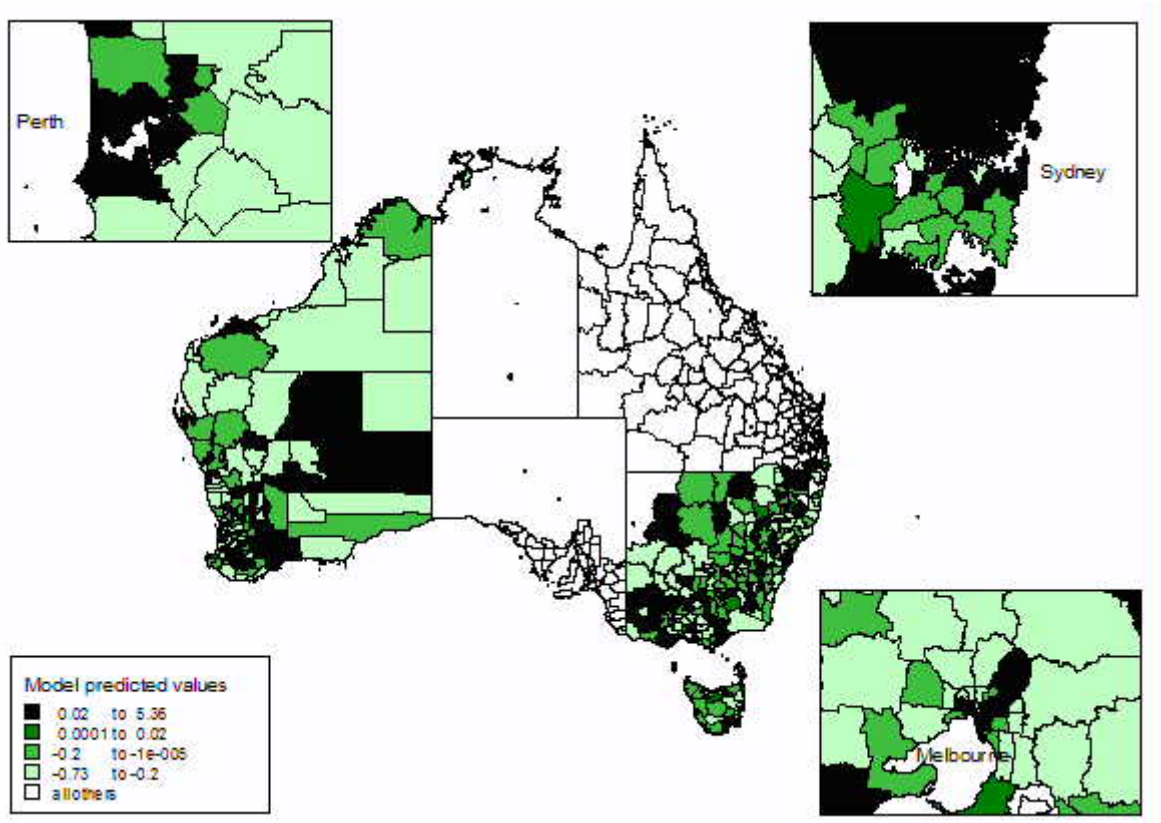
[1]conditions wholly attributable to alcohol consumption

Appendix B: Maps of model-predicted outcomes

B.1 model-predicted values for acute wholly-alcohol-caused hospitalisation in Australia, 99/00



B.2 model-predicted values for chronic wholly-alcohol-caused hospitalisation in Australia, 99/00



B.3 model-predicted values for chronic wholly-alcohol-caused death in Australia, 2000/01