Author's response to reviews

Title: Income inequality and alcohol related harm in Australia

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Author's response to reviews: see over
We thank the reviewers for their careful consideration of the manuscript. Our response to their queries is presented as indented italicised text directly after each of their comments.

Referee 1
Title: Income inequality and alcohol related harm in Australia
Version: 1 Date: 21 July 2008
Reviewer: Gerhard Gmel
Reviewer’s report:
The present study looks at the interplay between income inequalities and alcohol-related harm in an ecologic analysis. The authors claim to have found an uncommon relationship (U-shaped). Unfortunately, I can not follow the authors’ conclusion about a U-shaped curve, and I'm afraid that some of their findings may be related to methodological artifacts, which have to be addressed in a future version of the MS.

1) All coefficients for the Gini and Gini-squared in tables 2 and 3 are strictly positive. Therefore, there is no way that the quadratic function can take a U-shaped form (given that the independent variable is strictly positive too); the resulting function must be monotonically increasing. Thus, most of the arguments made in the discussion are hard to follow. At least the authors should explain the differences between regression models and the Loess fits in figures. May it be that Loess fits did not adjust for the same variables as the regression models? The authors need to explain more the link between the U-shape and the coefficients from regression models that do not show a U-shape.

Our description of input data to the quadratic models was unclear, and we thank the reviewer for identifying this problem. In fact, the linear and quadratic terms used in all our models was the centred Gini score, ie. $X = \text{Gini} - \text{mean( Gini)}$ and $X^2$. Thus the reviewer’s assumption that the independent variable is strictly positive is untrue, but this was poorly documented. The use of a centred Gini coefficient is now described in the data analysis section of the manuscript. We used the centred Gini score in order to minimise the correlation between coefficients. Thus, we submit, the U-shape from loess fits and the quadratic coefficients are consistent.

2) I am very much concerned about the ecologic validity of the study. The risk of running into an ecologic bias is well known. The risk of an ecologic bias increases with the heterogeneity within the ecologic units. An ecologic analysis would be best if there was large heterogeneity across units but a strong homogeneity within units. The large variation of unit sizes (LGA) in representing Australian inhabitants is striking. It ranges from 85 inhabitants in the smallest LGA to more than 880’000 (see table 1) in the largest. Hence it ranges between a street block and a metropolitan city.

We accept the reviewer’s point and share his concerns about ecologic bias. In order to address this issue, and hopefully put his mind at rest,
we have performed a sensitivity analysis (see suggestion in relation to 4(b)) in which we restricted our analysis to the LGAs in the middle 50% of population distribution (restricted analysis). There was little change in our results, best demonstrated through the predicted values of the various models. The predicted values of the model derived from these analyses are shown in the following scatterplots against the relevant original model (according to each of the three dependent measures of interest).

Figure r1: predicted values from original model plotted against the predicted values derived from the restricted analysis for the chronic alcohol attributable mortality.
Figure r2: predicted values from original model plotted against the predicted values derived from the restricted analysis for the acute alcohol attributable morbidity.

Figure r3: predicted values from original model plotted against the predicted values derived from the restricted analysis for the chronic alcohol attributable morbidity.
3) Related to 2) is the problem that one aim of the study is partly in contradiction with the need for homogenous units. Either a unit is homogeneous, than there should be no or little income inequality, or vice versa. The authors should discuss this.

*We thank the reviewer for making this point. The key here is that the Gini coefficient is measuring heterogeneity within units. This means that our exposure at least should not be interpreted as being subject to a classical ecological fallacy because it is the characteristics of the population that are being measured – the Gini coefficient is a measure of the heterogeneity of the unit under examination. We have added a sentence to this effect into the discussion.*

4) I’m wondering, how much of the variation in Gini coefficients is actually related to the size of the LGA. The larger the LGA the more likely are income inequalities within the LGA. Thus, much of the effects found in the paper may just be related to size of LGAs. This problem is even aggravated by the fact that the authors weighted the analysis with the size of the LGA (namely the number of deaths or hospitalizations, see page 6 “data analysis”). The rationale for this should be given. Personally I’m rather opposed to weighting by size, because this only favors large units. I suggest as a sensitivity analysis a) an unweighted analysis and b) an analysis that controls for the size of LGA’s.

*We followed the usual statistical practice to use weights which are inversely proportional to the known variance of each datapoint. Since \( \text{var}(\log \text{SMR}) = 1/D_1 + 1/D_2 \), then the appropriate weights are the numbers of events included in each datum. We feel strongly that the best analysis is that which weights each datum according to its variance. Further, we would argue that omission of the weights would produce biased results that we would be unable to interpret. In relation to comment (b) we have conducted a sensitivity analysis that controls for the population size of LGAs as detailed above.*

5) How reliable is an analysis with 885 deaths and more than 500 LGA, i.e. less than 2 deaths per LGA on average. This will probably mean that in many small LGAs there were no deaths at all! This leads to a serious problem: How did the authors deal with those zeros? As an SMR is the ratio of observed deaths divided by expected deaths, the SMR will remain zero for LGAs without deaths. The log of zeros (see also my point 8), however, is undefined. From the visual inspection of e.g. Figure 3 with only 28 datapoints (a datapoint should represent a LGA), I’m afraid that I have to assume that all LGAs with zero deaths were just left out of the analysis, and thus the mortality analysis probably only refers to large LGAs.

*The reviewer is right in pointing out that LGAs with zero events will have zero weight. Therefore, we agree with the reviewer that LGAs with zero deaths were omitted from the mortality regressions.*

I do have a couple of minor comments.

   *We thank the reviewer for pointing out this error and it has been changed from ‘short term’ to ‘long term’*

7. Abstract: I can not see that there is a “strong” association given that the overall R-squared (including control variables) are around 0.15; authors should avoid labeling their results.

   *Corrected as requested, ‘strongly’ has been deleted from abstract*

Generally, the conclusions could be more concrete. What was the direction of the association; what is "complex" in the relationship; with which findings are the current findings inconsistent?

   *The conclusions section has been changed to be more concrete. It now reads: “We found a curvilinear relationship between income inequality and the rates of some types of alcohol-attributable hospitalisation and death at a local area level in Australia. While alcohol-attributable harms generally increased with increasing income inequality, alcohol-attributable hospitalisations actually showed the reverse relationship at low levels of income inequality. The curvilinear patterns we observed are inconsistent with monotonic trends found in previous research making our findings incompatible with previous explanations of the relationship between income inequality and health related harms.”*

8. Why were SMRs logged? The distribution of the SMRs is not important for the assumptions of linear regression, because the dependent variable was not the SMR directly, but a ratio of SMR (alcohol-related versus other). Was this ratio also skewed? (I don't want to be a nitpicker, but in fact, not the distribution of the dependent variable is important, but the distribution of errors). Not logging may solve some of the problems (see above).

   *Generally, SMRs are measures on a multiplicative scale, and log(SMR) has a distribution which is more suitable for linear modelling. This is particularly the case when there a small numbers of events involved in the calculation of SMR. Furthermore, the sampling variability of log(SMR) is much simpler than that of SMR, leading to simpler weighting variables. Hence, our reason is not based only on skewness of distribution, but on homogeneity of variance.*

9. Why did the author adjust for the Socio-Economic Index for Areas (SEIFA). Wouldn’t this be in the causal path between Gini and alcohol-related harm. Variables in the causal path should not be adjusted for (see Rothman, Introduction to Epidemiology). Similarly the remoteness; again I think this variable just reflects larger (major cities) versus smaller LGAs and thus eliminates some of the variance of the Gini (which is in turn related to size of LGAs). At least, the authors should explain why they think both variables are confounders. Again a sensitivity analysis (leaving out the confounders) may help here.

   *Previous research in Australia has shown that a range of health-related harms vary according to socioeconomic characteristics and remoteness of areas across the country. Therefore, we have included*
measures of these in order to control for any possible confounding of the relationship between income inequality and alcohol-attributable harm. The key point in previous work on income inequality and health related harms is that inequality has been shown to predict health-related harms independently of the distribution of other socio-economic variables such as income. In this way SEIFA should not be seen as part of the causal path, instead it is a representation of absolute disadvantage (eg low income) within an LGA whereas the Gini coefficient represents the spread of income within an LGA. We have now included a more detailed discussion of the confounders in the introduction in which we articulate the rationale for their inclusion more clearly.

As suggested we have conducted a sensitivity analysis without including the confounders (no-confounder analysis). Again, there was little change in our results.

10. I had sometimes difficulties to follow the different numbers of LGAs in different analyses. For example, what does it mean that “There were around 140 more LGAs available for the mortality analyses” (page 6)? More than what? More than the 373 LGAs for the analysis of acute alcohol-attributable hospitalizations or more than the 349 LGAs for chronic hospitalizations (same page)? How do these numbers match with the 580 Gini coefficients in table 1, respectively with the 428 and 562 Gini “coefficients used” in table 1? Authors could be a bit more explicit about the n’s of LGAs for different analyses; could a flowchart help here?

The number of LGAs does vary between analyses. We thank the reviewer for the suggestion of a flowchart but feel that this would be unnecessarily complicated. Instead, we have taken the LGA numbers out of the Table (as these were confusing) and inserted the number of LGAs included in each analysis at the top of the relevant tables. We have also explained the smaller number of LGAs for the hospitalisations analysis more clearly at the end of the data analysis section.
The introduction has been restructured and re-written emphasising previous research on the relationship between income inequality and health related harms and specifying more clearly the rationale for the study along with what we expected to find at the outset.

2. I tried to read the “Methods” very carefully but I am not convinced that the income level is controlle for. Therefore, one explanation for the result may be that the same level of income inequality includes some LGAs with very high and some LGAs with very low income levels, and the income level may have a greater effect on alcohol-related problems than the income inequality. Why should the above mentioned LGAs be more similar and differ from a LGA with on average medium high income level but higher income inequality.

The SEIFA index was included in order to control for variations in socio-demographic characteristics of areas. As indicated in the methods section, this index is a summary score of a variety of census-derived variables derived from a factor analysis of a suite of census variables undertaken by the Australian Bureau of Statistics. The proportion of low income earners in an LGA is represented in the SEIFA index. The use of one variable to summarise a suite of socioeconomic measures is the most efficient means of controlling for relevant variation in socioeconomic disadvantage. It is important to remember here that our main interest in modelling the relationship between income inequality and alcohol attributable harms.

Minor essential revisions
1. Abstract. ...“alcohol caused harm: acute (primary related to the short term consequences of drinking”). Both death in alcohol poisonings and alcohol related liver cirrhosis are short term consequences when the person passes away but alcohol poisoning is more an outcome of one drinking episode (with no need for previous long term alcohol consumption) whereas dying in liver cirrhosis mortality presupposes long term heavy alcohol consumption. In the next sentence the word “short” is definitely wrong.

We thank the reviewer for pointing out this error and it has been changed from ‘short’ to ‘long’

2. Introduction, line 6: “typically” may be too strong word here.

“typically” has been deleted

3. According to my mind there are some inconsistencies with
a. the wording in “Conclusion” of the abstract (“Rates of alcohol-attributable hospitalisation and death are strongly associated with measures of income inequality at a local level in Australia”,

b. presenting the results on pages 6 and 7, (“Figure 3 highlights not only the fact that no clear relationship was evident ...; However, unlike hospitalisation data shown in Figures 1 and 2, there was no evidence of the concave decrease, ...”, and

c. the first paragraph in “Discussion” (“The nature of the relationship was consistent across acute and chronic alcohol-attributable hospitalisations and was similar for chronic alcohol-attributable deaths,”; “... , there was no evidence of a relationship between income inequalities and acute alcohol-attributable deaths,...”
We thank the reviewer for pointing out this inconsistency. The conclusion to the abstract has been changed to be consistent with the remainder of the paper. The revisions undertaken in relation to referee 1’s comments (see above) have hopefully eliminated the inconsistency.