#### Part 2 - Evaluation

This section will be released to the candidate prior to the Oral Examination and to those candidates required to revise and resubmit

Candidate's name: Elizabeth Lillian Bolton

Thesis Title: Longitudinal predictors of mortality inequalities in Aotearoa New Zealand

The examiner is requested to provide a report on the quality of the work, according to the following criteria. Please note that in order to accommodate your detailed and comprehensive comments, the boxes below are expandable.

## Is an original contribution to knowledge or understanding in its field.

(Please comment)

This PhD submission is of a breadth and standard that it fulfils the requirements for a PhD. There are some areas where arguments could have been better developed but overall the PhD has been well done. The candidate has traversed the use of varying advanced epidemiological methods to investigate a series of hypotheses related to SEP, mortality and the role of religion in SEP-mortality related risk. The candidate has illustrated a competent use and understanding of a range of epidemiological methods, while also attempting to address previous criticisms of similar work in this area (e.g. well done on attempting to move on from p-values as the 'easy' way out to decide what is meaningful). The candidate has done a particularly nice job of explaining the mediation analysis and the 'cross-world'.

I also appreciate the honest discussion of Indigenous Data Sovereignty and that this has advanced considerably since the conceptualisation of the studies at hand.

## Meets internationally recognised standards for such work.

(Please comment)

Yes, this work meets international standards for a body of work investigating the predictors of mortality and extends this with additional investigations into weighting the NZLC.

Demonstrates knowledge of the literature relevant to the subject and the field or fields to which the subject belongs, and the ability to exercise critical and analytical judgement of it. (Please comment)

Some of the discussion of the previous literature could be strengthened – there are some places where the thesis could have more fully demonstrated a deep view of where this literature has been. I was surprised there was not more in-depth discussion of the clustering that occurs with SEP and how this interacts with contagion of health behaviours, and/or access to health services. Perhaps this is less of a problem in NZ? Otherwise, the candidate has illustrated a substantial working knowledge of the area and communicated the strengths and limitations of the PhD thesis in an appropriate context.

Is satisfactory in its methodology, in the quality and coherence of its expression, and in its scholarly presentation and format.

(Please comment)

The varied use of methods was a strength of the PhD – particularly the use of mediation analyses. The thesis for the most part was a pleasure to read, and the candidate should be congratulated for in places – explaining complicated concepts quite simply.

#### Additional comments:

The results of the cross-sectional and longitudinal analysis are interesting – although in some ways it makes sense the results don't differ dramatically (if we believe one measure contains information on other time points). Of course, the really interesting but difficult question this raises is – do we stop investing in longitudinal studies? This particular implication could have been explored more. Does this mean having more data doesn't do anything? Or that census data is not enough to truly capture trajectories in SEP? Or is this simply a reflection of the low-mortality count of some SEP patterns?

I was surprised not to see a more in-depth discussion of the role of risk prediction in investigating life-course hypotheses. It is true that formal risk prediction (which in its full form involves estimating sensitivity, specificity, positive predictive values, as well as generating measures of discrimination such as the AUC and calibration such as the ratio of observed/predicted within deciles of predicted risk) can potentially be used to understand the characteristics and number of individuals that could be considered in a 'high-risk' population. However, I find it interesting that only the AUC was used as the measure of performance, when in real life we would of course set a risk threshold and explore other metrics like PPV, sensitivity etc. It would be possible for this to be extended to understand the implications of targeting say the population with the highest 10% of risk – and (assuming an effective intervention) what prevention benefit this might offer individuals and society as a whole.

There might have been interesting considerations on what is preventable when considering targets for support/intervention to reduce mortality. As you note, it is difficult here to isolate the mechanism that results in religion increasing odds of mortality. Religion itself would seem to be a difficult thing to 'intervene' on- whereas SEP is much more clearly modifiable.

The exploration of choice of weighting scheme and the observations around the issues of missing Census and mortality linkages and the resultant possible selection bias introduced some interesting ideas.

It would have been helpful to have had a dedicated section exploring missing data (with a focus on related to non-response from participants), particularly as some analyses seemed to be based on response samples that varied according to measure of SEP, and not complete case samples. If it is correct that this has been run on a response sample where missing on one SEP indicator is recoded to some default (e.g. high SEP), and this was not altered to a complete case sample for the remaining analysis than this is problematic. If the n's under each age cohort across the three SEP indicators represent the different response samples – this possibly means a good chunk of the sample had varying opportunities to be exposed to all trajectories. This can introduce bias into the classification of individuals into different SEP trajectories through what I am assuming is differential misclassification – where some individuals have a greater probability of being misclassified into higher SEP trajectories (assuming this was the default recoding). Of course, this simply may not be clear and could be dealt with through additional explanation.

### Questions for the oral examination:

How does this body of work help inform what governments should invest in to reduce inequalities in mortality?

How do we consider the results of the study on religion in the context of targets that are modifiable and may impact inequalities in morality?

# Part 2 continued – Corrections and/or Revisions

Recommended minor corrections, if any: (lists of typographical errors, while welcome, are not expected)
Methods: consider the merits of adding some detail on missing data to fully explain how this was dealt with across the studies and what proportion of missing data is across all key variables. For example - Table 3.2 appears as if it is based on a "Response sample" i.e. the sample included is the maximum available based on who responded to the questions underlying each SEP indicator. Although based on information in chapter 2 it seems there was some imputation of household equivalised income? Was this mean imputation? MICE? It appears as if the other SEP measures were not imputed. What proportion of each variable had missing data and how this was treated, could be clearer.
Chapter 3- If mortality data from 2006-2010 was used, but the data included people linked between 1981 and 2006 – does this means deaths prior to 2006 are ignored?