# Development of a Chronic Disease Risk Factor Index in the NSW Health Survey Program

**Yvette Miller**

**NSW Centre for Physical Activity and Health**
**University of New South Wales**

December 2003

Report No.: CPAH 03-0008

Suggested Citation:

Miller Y. Development of a chronic disease risk factor index in the NSW Health Survey Program. 2003. Report No.: CPAH 03-0008

# Table of Contents

# List of Tables



# List of Figures

The aim of this study was to explore the concept of multiple risk factors, and to identify population sub-groups at high risk for chronic disease based on clusters of risk behaviour as reported in NSW Health surveys. Initially, this involved summarising the evidence around multiple risk factors, their roles in health risk in populations, and current usage worldwide. Further, through secondary analyses of NSW health survey data, we explored methods of creating a risk factor index to be used to investigate the clustering of risk factors for chronic disease in the NSW population in order to identify population groups at very high risk. This work was completed with the goal of providing recommendations to inform the NSW Health Dashboard indicator project.

## Background

To date, there have been no systematic efforts to relate the concept of multiple risk factors to chronic diseases generally, although a number of studies have attempted to establish the relationship between multiple risk factors and individual chronic conditions. The majority of multiple risk factor assessment has been concerned with creating multiple risk factor profiles for cardiovascular disease risk, with concentration on clustering of risk factors such as smoking behaviour, overweight and/or obesity, hypertension, high blood cholesterol, low physical activity / sedentariness and diabetes or high blood glucose. Some studies have attempted to simply investigate the prevalence and clustering of established multiple risk factors for the identification of high-risk groups in a range of different healthy populations, including Chinese adults (Wu et al, 2001), older Kuwaiti men (Jackson et al, 2002), Adults (Schuit et al, 2002) and young adults (Twisk et al, 2001) in the Netherlands, Young adult African Americans (Murtagh et al, 2002) and New Zealand adults (New Zealand Ministry of Health, 1999). Similar risk factor clustering has been investigated in overweight and obese groups (Must et al, 1999) and among those with existing coronary artery disease (Mansur et al, 2001). Others have used prospective data to attempt to use multiple risk factors to predict mortality (Chang et al, 2001; Kaukua et al, 2001; Yusuf et al, 1998) or specific disease outcomes including stroke (Kaukua et al, 2001; Yusuf et al, 1998), coronary heart disease (Yusuf et al, 1998), myocardial infarction (Kaukua et al, 2001) and coronary artery disease (Mansur et al, 2001; Urbina et al, 2002).

The methods of data collection, clustering of the risk factors or creating the independent variable, and defining the 'existence' of the disease outcome being predicted, vary widely across both cross-sectional and prospective studies. Most multiple risk factor studies have simply summed the number of chronic disease risk behaviours or conditions present for each subject (Kaukua et al, 2001; Must et al, 1999; New Zealand Ministry of Health, 1999; Schuit et al, 2002; Twisk et al, 2001; Urbina et al, 2002; Wu et al, 2001; Yusuf et al, 1998), while others have looked at *a priori* defined combinations of specific risk factors (Chang et al, 2001; Jackson et al, 2002; Mansur et al, 2001) or which risk factors are more likely to co-exist or 'cluster' (Murtaugh, 2003; Schuit, 2002). In addition to applying each of the above approaches, Murtaugh and colleagues (2002) also calculated a numerical risk score for different levels of exposure (based on published guidelines for each risk factor) across four cardiovascular risk factors, and derived a total risk score for each subject from the sum of their scores from all risk factors. This was an attempt to account for the dose-response relationship that has been demonstrated between many risk factors and disease outcomes.

**What behavioural risk factor indicators are possible in NSW Health Surveys?**

The chronic disease outcomes or conditions included in the Chronic Disease Prevention Strategy 2003-2007 (NSW Department of Health, 2003) are cardiovascular diseases (including ischaemic heart disease, stroke and hypertension), cancers, asthma and chronic lung disease, non-insulin dependent (type II) diabetes, obesity, injuries from falls, and poor emotional and psychological well-being. The primary risk factors agreed upon as potentially contributing to the strategy include smoking, nutrition, hazardous alcohol use, physical inactivity, and psychosocial risk factors such as stress. This model formed the basis of the analysis plan presented here, with a number of modifications due to both theoretical and measurement issues.

The modifiable behavioural chronic disease risk factors described in the Chronic Disease Prevention strategy 2003-2007 that are currently measured through the NSW Health Survey program relate to smoking behaviour, physical activity behaviour, alcohol use, mental health (or psychological distress) and nutrition (serves of fruit and vegetables consumed). Psychological health is included as both a risk factor (called psychosocial risk factors such as stress) and an outcome in the above model. It was excluded from this analysis as a contributing risk factor [independent variable] to the index since it has not been applied as a risk factor in other studies, and because the only measure of psychological distress in the survey (the 10-item Kessler Psychological Distress Scale, or K10), already comprises part of the outcome of chronic disease.

Overweight and obesity has also been included as a risk factor (and therefore a contributor to the overall chronic disease risk factor index), rather than a disease outcome (as outlined in the Chronic Disease Prevention Strategy 2003-2007). This was considered more appropriate for this analysis given that increased risk of some of the other chronic disease outcomes with increasing overweight and obesity has been demonstrated. Self-reported height and weight is collected in the survey and body mass index (BMI) will be used to indicate overweight and obesity for inclusion in the risk factor index.

There are a number of possibilities for how these five factors can be defined for the purpose of constructing a chronic disease risk factor index.

## Methods

### Development of a Multiple Risk Factor Index using NSW Health Survey data

One possibility for constructing the chronic disease risk factor index was to use the dichotomous categories currently used for reporting by the NSW Continuous Health Survey program as a basis for scoring on each risk factor, and sum the scores across risk factors to calculate the final index for each respondent. The simplest method was allocating a score of 1 if the risk factor is present, and a score of 0 if absent, so the resulting index has a possible range from 0 to 5 (with higher scores indicative of higher risk of chronic disease). The resulting index is comparable to studies that have looked at 'numbers of risk factors'. The simplicity of such a scoring system makes it appealing, although it does assume the equal influence of each risk factor in developing chronic disease, as well as a dichotomous relationship between the risk factor and outcome/s (ie. it does not take into account the dose-response relationship between risk factor and outcome).

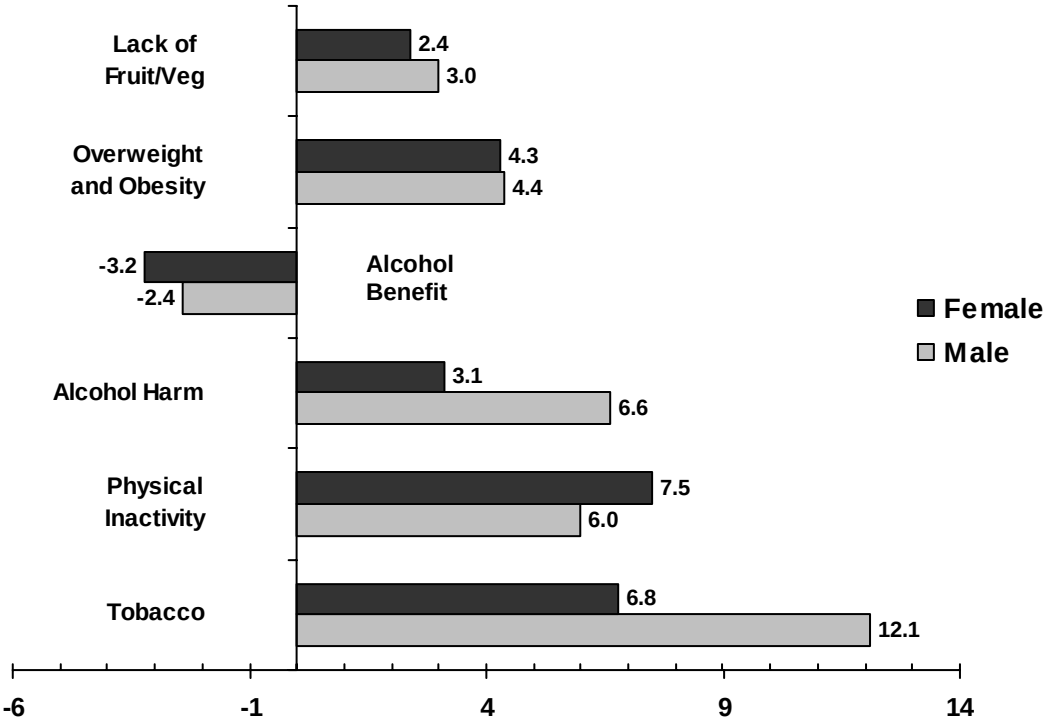


**Figure 1: Proportion of total burden attributed to selected risk factors, by sex, Australia, 1996 (Mathers, Vos & Stevenson, 1999).**

To account for the possibility that each risk factor contributes differentially to the burden of chronic disease, scoring for each risk factor was also weighted proportionate to it's contribution to the total burden of disease (measured in disability-adjusted life years, or DALYs; see Figure 1; Mathers, Vos & Stevenson, 1999). DALYs for each disease or condition are calculated as the sum of years of life lost (YLL) due to premature mortality in the population and the years lost due to disability (YLD) for incident cases of the health condition. As such, DALYS account for both potential years of life lost due to premature death and equivalent years of 'healthy' life lost by virtue of disability or being in poor states of health (the duration of the condition 'weighted' for it's impact on quality of life; Mathers, Vos & Stevenson, 1999). Weighting for contribution to the burden of disease was applied to the same dichotomous categories for each risk factor, but presence of the risk factor was scored relative to the contribution of other risk factors, and absence of the risk factor still resulted in a contribution of 0 to the final index.

In the example shown in Table 1 below, the score for presence of each risk factor has been developed relative to the contribution of smoking (set at a score of 1). Since the attributable burden of disease associated with each risk factor differs for men and women, the score for the presence of each risk factor and the resulting index are gender-specific (see column 2 in Table 1). Since scores for each risk factor are determined by the contribution relative to tobacco for men and women separately, analysis using the resulting index was within-sex (ie. not comparable by sex). Note that for alcohol, the total attributable risk is based on the sum of the contribution of alcohol harm and alcohol benefit (negative risk). Using this scoring method, the possible range of index scores for men was from 0 to 2.47 and from 0 to 3.08 for women. However, this method does not account for the possibility that for some risk factors, the association with chronic disease is not binomial, but linear.

To account for this linear association, a third scoring option was developed, whereby the total risk associated with each risk factor was divided across levels of exposure to the risk factor. Within each risk factor, potential categories of exposure for differential weighting in the index were limited to those that are able to be established from responses to items in the 2002 NSW Health Survey (ie. the pre-determined response categories for each item). Using this method, risk was distributed across levels of exposure according to the estimated relative risk of chronic disease for each level of exposure to the risk factor. The sum of scores for each individual across risk factors was used to derive the index (as for the first scoring option), resulting in a possible index range from 0 to 3.5. The definition of categories and justification for the relative weighting of categories for each risk factor are included in Appendix A, and were based on relative risk of different disease outcomes in the epidemiological literature. The risk factor indexes were only calculated for those participants who had values for all of the risk factor variables from which the index was derived.

**Table 1: Risk factor score assignment**

| | DEFINITIONS OF RISK AND ATTRIBUTABLE WEIGHT | | | |
|---|---|---|---|---|
| | **RISK FACTOR INDEX 1** | **RISK FACTOR INDEX 2** | | **RISK FACTOR INDEX 3** |
| | **Dichotomous categories currently used for NSW Health reports (unweighted) (Range = 0-5)** | **Dichotomous Categories weighted for different contributions to the score proportionate to their contribution to total DALYs (Range for Males = 0 – 2.47) (Range for females = 0 – 3.08)** | | **Unweighted multiple categories developed according to linear risk associated with differing levels of the risk factor (Range = 0- 3.8)** |
| | | MALES | FEMALES | |
| **SMOKING** | Smoker = 1<br>Non–smoker = 0 | Smoker = 1<br>Non-smoker = 0 | Smoker = 1<br>Non-smoker = 0 | Smoke daily = 1<br>Smoke occasionally = 0.8<br>Ex-smoker = 0.5<br>Never smoked = 0 |
| **NUTRITION Fruit & Vegetable Intake** | Inadequate = 1<br>Adequate = 0 | Inadequate = 0.25<br>Adequate = 0 | Inadequate = 0.35<br>Adequate = 0 | Tertiles for total serves per day<br>Low = 0.4<br>Moderate = 0.2<br>High = 0 |
| **ALCOHOL** | Any risk drinking = 1<br>No risk drinking = 0 | Any risk = 0.35<br>No risk = 0 | Any risk = 0<br>No risk = 0 | Non-drinker/Low risk = 0<br>Hazardous = 0.3<br>Harmful = 0.4 |
| **PHYSICAL ACTIVITY** | Inadequate PA = 1<br>Adequate PA = 0 | Inadequate = 0.5<br>Adequate = 0 | Inadequate = 1.1<br>Adequate = 0 | Sedentary = 1<br>Inadequate = 0.4<br>Adequate = 0.1<br>High = 0 |
| **OVERWEIGHT AND OBESITY** | Not O'weight/ Obese = 0<br>O'weight or Obese = 1 | Not O'weight/ Obese = 0<br>O'weight/ Obese = 0.37 | Not O'weight/ Obese = 0<br>O'weight/ Obese = 0.63 | Underweight/ Healthy weight = 0<br>Overweight = 0.3<br>Obese = 1 |

### Data Analysis

This analysis was conducted using data from all adult (aged 16 years or over) respondents from the 2002 NSW Health Survey. Cases were excluded where the participant had not responded to all survey items used in constructing all three of the risk factor indices.

In an attempt to identify populations at special risk of multiple sets of behavioural risk factors, differences in mean index resulting from all three scoring options above were examined across categories of sex, age group, area health service, indicators of socioeconomic status (highest level of education achieved and quintile of socioeconomic disadvantage) and ethnicity (country of birth and language spoken at home). Independent samples two-tailed t-tests [for sex (male, female), country of birth (Australia, Other) and Non-English speaking at home (yes, no)] and One-way ANOVAs were used to examine differences in mean index across demographic groups.

Risk factor indices were then categorised as 'high' (vs. 'other') based on whether they were in the highest quartile of scores (ie. upper 25%). For risk factor 1 and risk factor 3, the cut-point was based on the distribution of scores for the whole sample; for risk factor 2, it was based on the distribution of scores for men and women separately. Logistic regression analysis was performed to assess the risk of having a 'high' score based on sociodemographic variables. The models for each risk factor index were gender specific, and were calculated both with and without adjustment for other sociodemographic variables.

## Results

Of the total sample aged 16 years or over, 92.8% (N = 11 710) responded to all items necessary for calculation of the indices and were included in the analysis. The distribution of each of the indices among men and women is described in Table 2.

### Gender

The mean risk factor index was significantly higher among men than women for both risk factor index 1 ($t(11708) = 17.357$, $p<0.001$), and risk factor index 3 ($t(11708) = 10.704$, $p<0.001$). Risk factor index 2 was not compared between men and women since construction of the score was gender-specific.

**Table 2: Distribution of each risk factor index, by sex**

| Sex | | | Dichotomous risk factor index | Weighted Dichotomous risk factor index | Unweighted multiple category risk factor index |
|---|---|---|---|---|---|
| **Male** | **N** | | 5016 | 5016 | 5016 |
| | **Mean** | | 2.63 | 1.06 | 1.21 |
| | **Std. deviation** | | 1.04 | .58 | .71 |
| | **Minimum** | | 0.0 | 0.0 | 0.0 |
| | **Maximum** | | 5.0 | 2.47 | 3.80 |
| | **Percentiles** | | | | |
| | | **25** | 2.00 | .62 | .70 |
| | | **50** | 3.0 | .97 | 1.10 |
| | | **75** | 3.0 | 1.47 | 1.70 |
| **Female** | **N** | | 6694 | 6694 | 6694 |
| | **Mean** | | 2.30 | 1.38 | 1.07 |
| | **Std. deviation** | | 1.04 | .77 | .71 |
| | **Minimum** | | 0.00 | 0.00 | 0.00 |
| | **Maximum** | | 5.00 | 3.08 | 3.80 |
| | **Percentiles** | | | | |
| | | **25** | 2.00 | .9800 | .5000 |
| | | **50** | 2.00 | 1.45 | 1.00 |
| | | **75** | 3.00 | 2.08 | 1.50 |

**Socioeconomic Disadvantage**

About one fifth of the sample fell in the two least disadvantaged quintiles of socioeconomic status, a further 21.1% were in the mid quintile of disadvantage, and 30.9% and 27.1% of respondents were in the second most or most disadvantaged quintile respectively. Mean risk factor index for the total sample increased significantly with quintile of socioeconomic disadvantage for both risk factor index 1 [$F(4, 11705) = 12.212$, $p<0.001$] and risk factor index 3 [$F(4, 11705) = 31.326$, $p<0.001$]. The relationship between socioeconomic disadvantage and the mean for each risk factor index for males and females is shown in Figure 2. It is important to note that Risk Factor Index 2 (RFI2) should not be compared between genders since calculation of the index is gender-specific. Mean risk factor index across all three indices increased significantly with increasing socioeconomic disadvantage for both men [RF1 – $F(4, 5011) = 7.659$, $p<0.001$; RF2 – $F(4, 5011) = 9.612$, $p<0.001$; RF3 – $F(4, 5011) = 16.476$, $p<0.001$] and women [RF1 – $F(4, 6689) = 5.794$, $p<0.001$; RF2 – $F(4, 6689) = 19.354$, $p<0.001$; RFI3 – $F(4, 6689) = 16.062$, $p<0.001$).

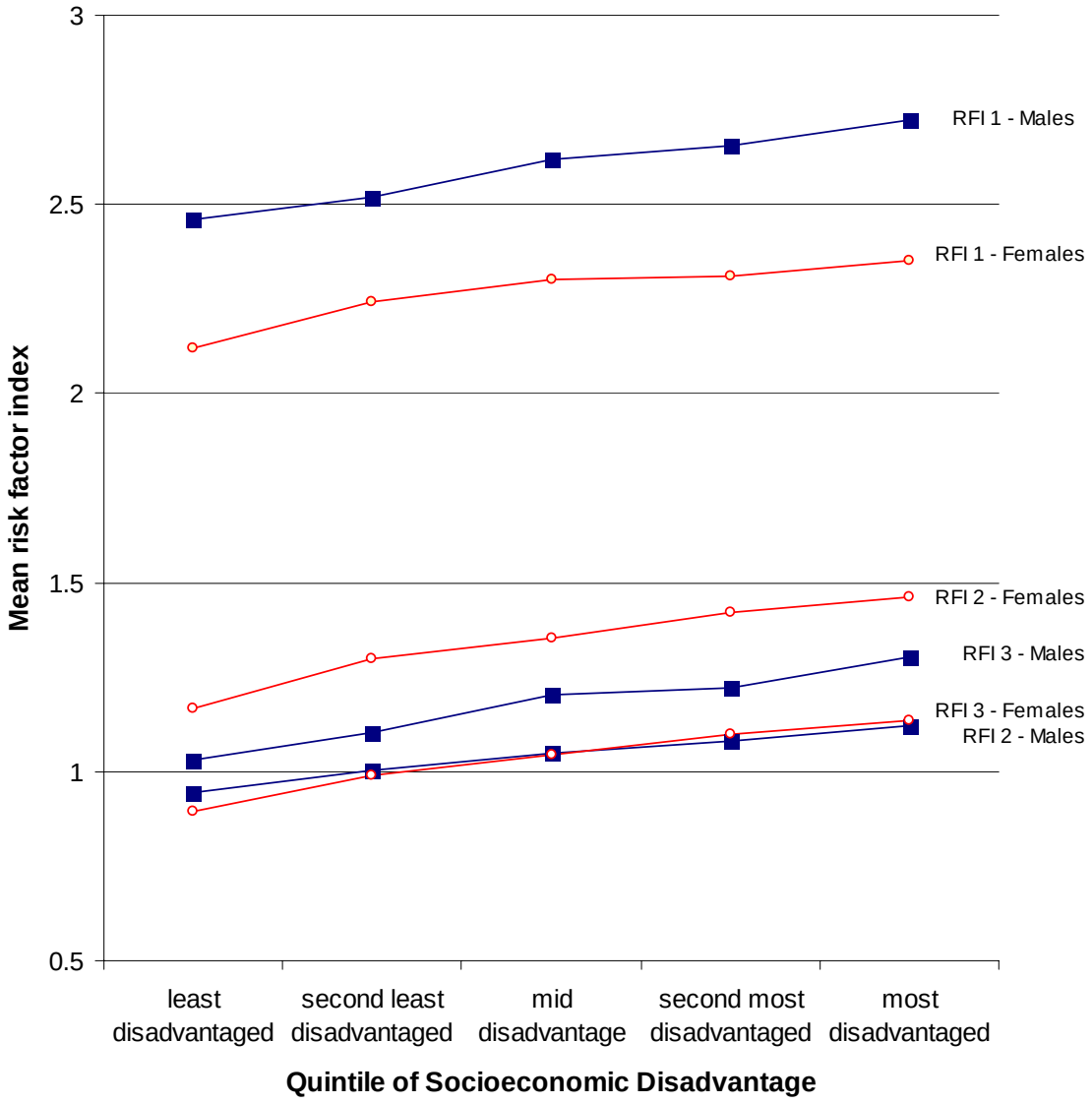


**Figure 2: Mean risk factor indices by quintile of socioeconomic disadvantage**

## Age

The mean and 95% confidence interval for the mean is shown by age group for risk factor index 1 and risk factor index 3 in Figure 3. Risk factor index 2 is not shown for the whole sample since calculation of the index is gender specific and combining males and females would not be appropriate. There was a significant difference in mean risk factor index across age categories for both risk factor index 1 [$F(13, 11696) = 19.414$, $p<0.001$] and risk factor index 3 [$F(13, 11696) = 17.373$, $p<0.001$]. The pattern of difference across age groups was similar for both indices, with those aged 16-19 years significantly lower ($p<0.05$) than all others aged up to 60-69 years for risk factor 1, and significantly lower than all other ages for risk factor 3. For risk factor 1, highest mean index was evident for those aged between 20-24 and 45-49, with a steady decline across older age groups such that those aged 65-69 and older had significantly lower mean index scores compared with all those aged between 20-24

and 55-59 years (see Figure 3a). For risk factor index 3, there was less comparative decline in older age groups, although those aged 70-74 maintained significantly lower mean index($P<0.05$) compared with those in age groups from 25-29 to 60-64 years (see Figure 3b). This difference in pattern for older age groups between risk factor index 1 and risk factor index 3 may suggest that those in older age groups may still be exposed to many risk factors but at lower levels of exposure, which is accounted for in risk factor 3 but not in the calculation of risk factor 1.

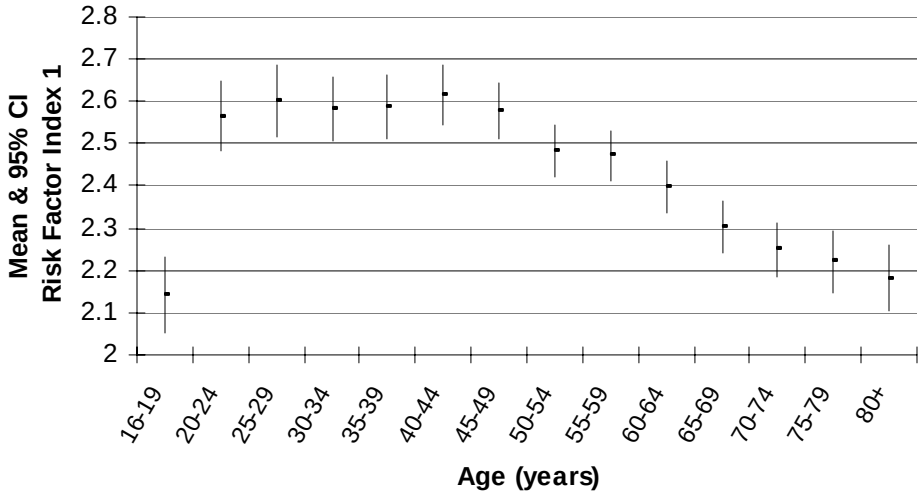


**Figure 3a: Mean and 95% confidence interval for risk factor index 1, by age group**

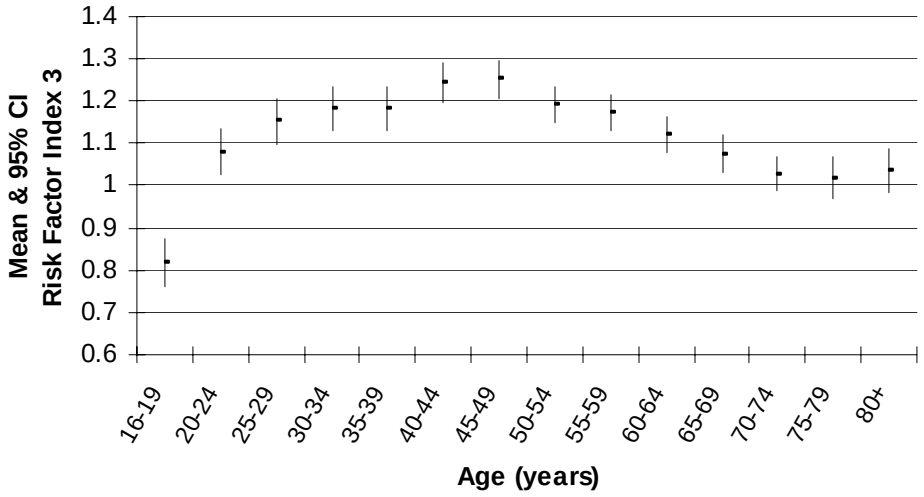


**Figure 3b: Mean and 95% confidence interval for risk factor index 3, by age group**

The relationship between age and the mean for each risk factor index for males and females is shown in Figure 4. Mean risk factor index across all three indices was significantly different between age groups for both men [RF1 – $F(13, 5002) = 16.269$, $p<0.001$; RF2 – $F(13, 5002) = 6.409$, $p<0.001$; RF3 – $F(13, 5002) = 11.312$, $p<0.001$] and women [RF1 – $F(13, 6680) = 6.701$, $p<0.001$; RF2 – $F(13, 6680) = 3.935$, $p<0.001$; RFI3 – $F(13, 6680) = 7.916$, $p<0.001$). Differences between males and females in the pattern of mean risk factor index 3 in older age groups suggests that the lack of decline for risk factor 3 compared with risk factor 1 shown above in Figure 3 is due to a similar pattern among men, but a steady decline with age does occur for women (see Figure 4).

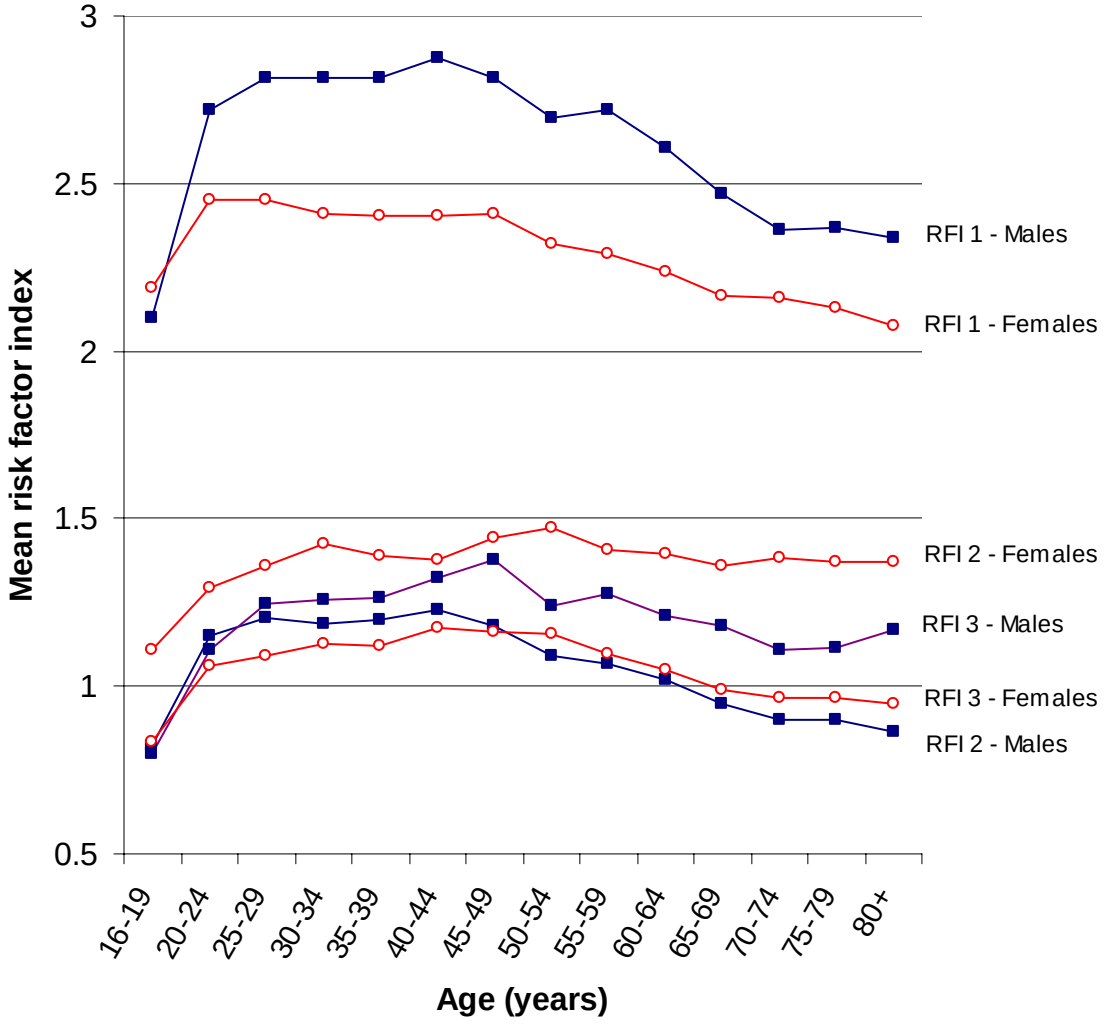


**Figure 4: Mean risk factor indices by age**

### Education

Those participants who responded to the question about highest level of education achieved with 'Don't Know' [N (males) = 28; N (females) = 37], refused to respond [N (males) = 5; N (females) = 3] or were not asked this item [N (males) = 12; N (females) = 8] were

excluded from the comparison of mean indices based on educational status. The mean and 95% confidence interval for the mean (risk factor index 1 and risk factor index 3) by education category are shown in Figure 5. There was a significant difference in mean index between categories of educational attainment for risk factor index 1 [F (3, 11613) = 35.52, P<0.001] and risk factor index 3 [F (3, 11613) = 78.831, p<0.001]. For both indices, those with a tertiary degree had a significantly lower mean score than those in all other categories. For risk factor 3, those who completed secondary school also had a significantly lower mean risk score than those who had not completed secondary school.

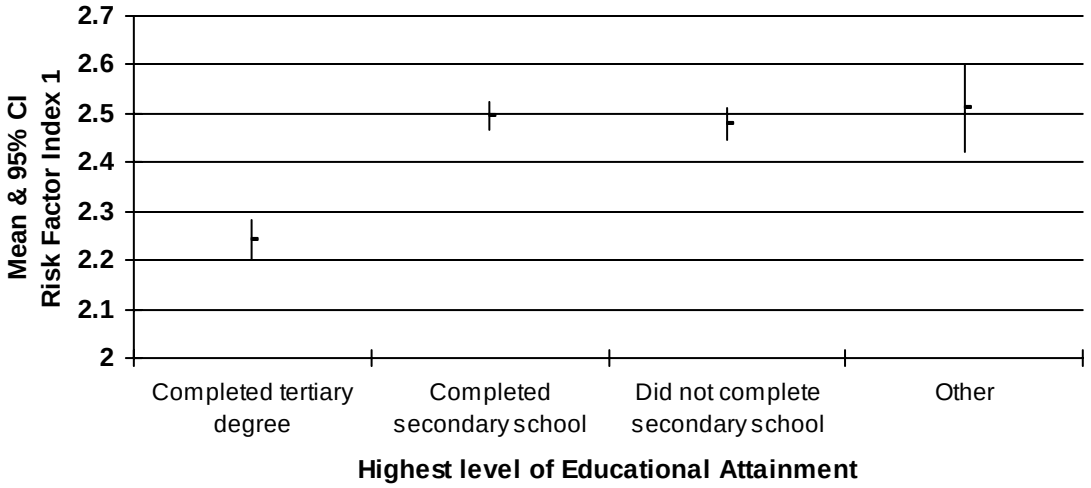


**Figure 5a: Mean and 95% confidence interval for risk factor index 1, by education**

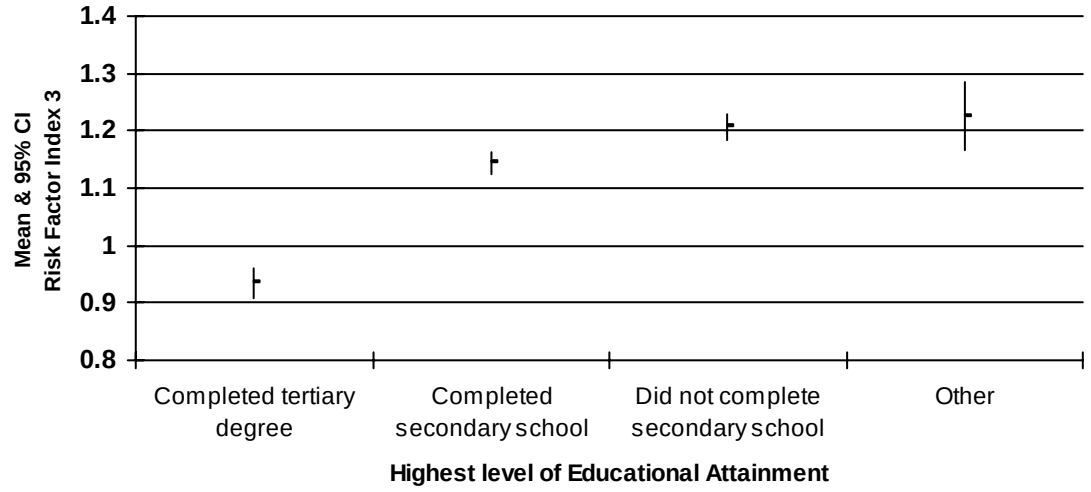


**Figure 5b: Mean and 95% confidence interval for risk factor index 1, by education**

The mean risk factor indices for men and women across highest level of completed education are shown in Figure 6. Mean risk factor index across all three indices was significantly different between education levels for both men [RF1 – $F(3, 4967) = 21.371$, $p<0.001$; RF2 – $F(3, 4967) = 23.635$, $p<0.001$; RF3 – $F(3, 4967) = 52.357$, $p<0.001$] and women [RF1 – $F(3, 6642) = 16.87$, $p<0.001$; RF2 – $F(3, 6642) = 40.575$, $p<0.001$; RFI3 – $F(3, 6642) = 34.498$, $p<0.001$). Similar patterns across levels of education were found for both men and women using each risk factor index, and those who had completed a tertiary degree having lowest risk factor scores across all indices for both men and women. However, an increase in risk factor score for those who had not completed secondary school compared with those who had (shown above in Figure 5b) was greater among men than women for risk factor 3. For risk factor index 2, there seemed to be a greater increase among women than men for those who had not completed secondary school compare to those who had.

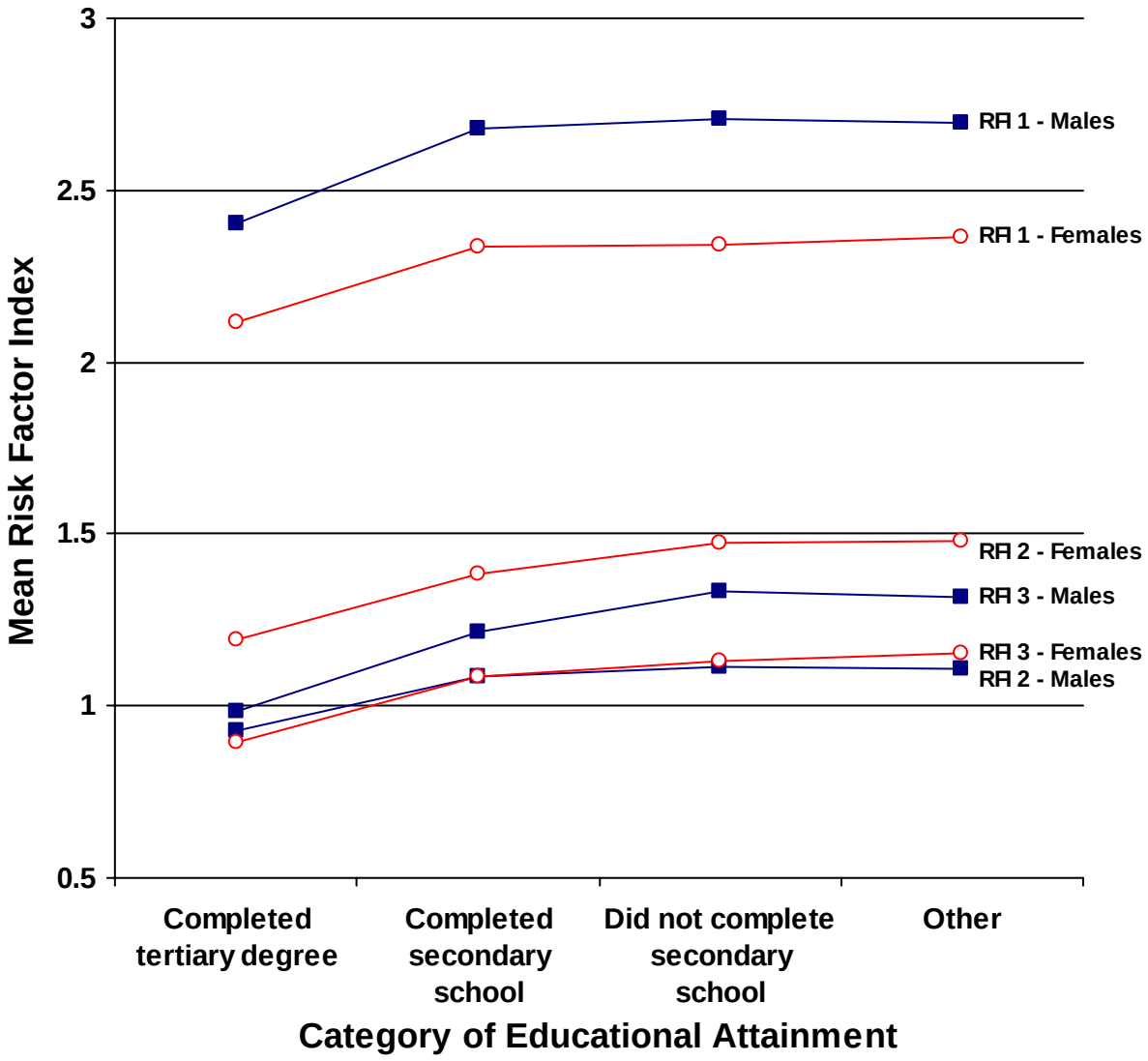


**Figure 6: Mean risk factor indices by education level**

**Country of Birth**

Those participants who refused to respond to the survey item about country of birth [N (males) = 3; N (females) = 2] or said they didn't know their country of birth [N (males) = 1] were excluded from the comparison of mean indices based on country of birth. The majority of the sample were born in Australia (80.2%), and had a marginally but significantly higher mean risk factor index 1 (2.47; t (11702) = 6.795, p<0.001) and mean risk factor 3 (1.14; t (11702) = 4.829, P<0.001) compared with those born elsewhere (Mean (RF1) = 2.31 and Mean (RF3) = 1.06). When separated by gender, the mean of all indices was significantly higher among those born in Australia than those born elsewhere (p<0.001).

**Language**

Those participants who were not asked what language they spoke at home [N (males) = 55; N (females) = 82), refused to respond [N (females) = 1] or said they didn't know [N (males) = 1] were excluded from the comparison of mean indices for English and non-English speaking participants. The majority of the remaining sample spoke English at home (92.8%), and had a significantly higher mean risk factor index 1 (t (11569) = -5.306, p<0.001) and risk factor 3 (t (11569) = -4.994, p<0.05) compared to those who spoke a language other than English. Among men, higher mean scores were evident among English-speaking respondents for all risk factor indices [RF1: t (4958) = -4.559, p<0.001; RF2: t (4958) = -2.727, p<0.01; RF3: t (4958) = -3.477, p<0.01]. English-speaking women had a significantly higher mean risk factor index 1 [t (6609) = -3.213, p<0.01) and risk factor index 3 [t (6609) = -3.661, p<0.001] compared to those who spoke a language other than English at home, but there was no significant difference in mean risk factor 2 between English-speaking and non-English speaking women.

**Area Health Service**

The means and 95% confidence intervals for the mean (risk factor index 1 and risk factor index 3) by Area Health Service are shown in Figure 7. There was a significant difference between Area Health Services in mean risk factor index 1 [F (16, 11693) = 7.890, p<0.001] and mean risk factor index 3 [F (16, 11693) = 12.771, p<0.001]. Those in the Far West Area Health Service had significantly higher mean scores than all other Ashes for both risk factor scores. Those in Northern Sydney and South Eastern Sydney Area Health Services had a similar and significantly lower mean risk factor index 3 (based on risk associated with differential levels of exposure to each factor) than all other Ashes. However, for risk factor index 1 (dichotomous unweighted exposure), South Eastern Sydney was not significantly lower than Central Sydney or Northern Rivers, and Northern Sydney was not significantly lower than Central Sydney, South Western Sydney, South Eastern Sydney, Northern Rivers or Mid North Coast Ashes.

The mean risk factor indices for men and women in each Area Health Service are shown in Figure 8. The mean for each risk factor index varied significantly across Area Health Services for both men [RF 1: F(16, 4999) = 5.266, p<0.001; RF 2: F (16, 4999) = 4.267, p<0.001; RF 3: F (16, 4999) = 7.812, p<0.001] and women [RF 1: F (16, 6677) = 3.98, p<0.001; RF 2: F (16, 6677) = 7.104, p<0.001; RF3: F(16, 6677) = 6.932, p<0.001]. Although patterns of mean index across different Area Health services were similar for men and women and for

each index, separate examination by gender revealed a slightly higher mean risk for all indices among women in the Hunter and Wentworth AHSs relative to other AHSs that was not seen among men. Similarly, for risk factor index 2, a higher mean risk was evident among women in South Western Sydney relative to mean index for other AHSs, but this was not apparent among men in South Western Sydney.

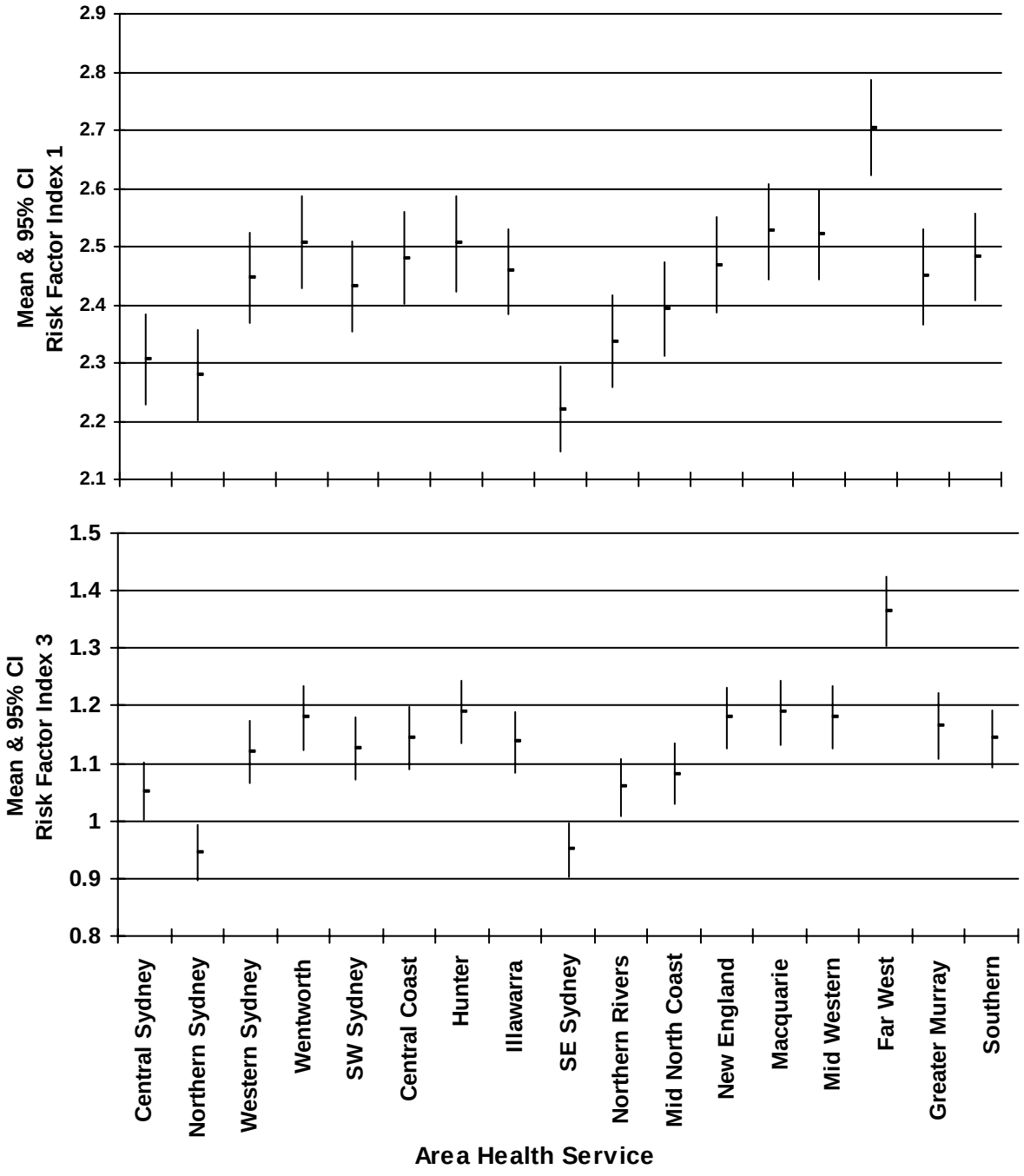


**Figure 7: Mean and 95% confidence interval for risk factor indices 1 and 3, by Area Health Service**

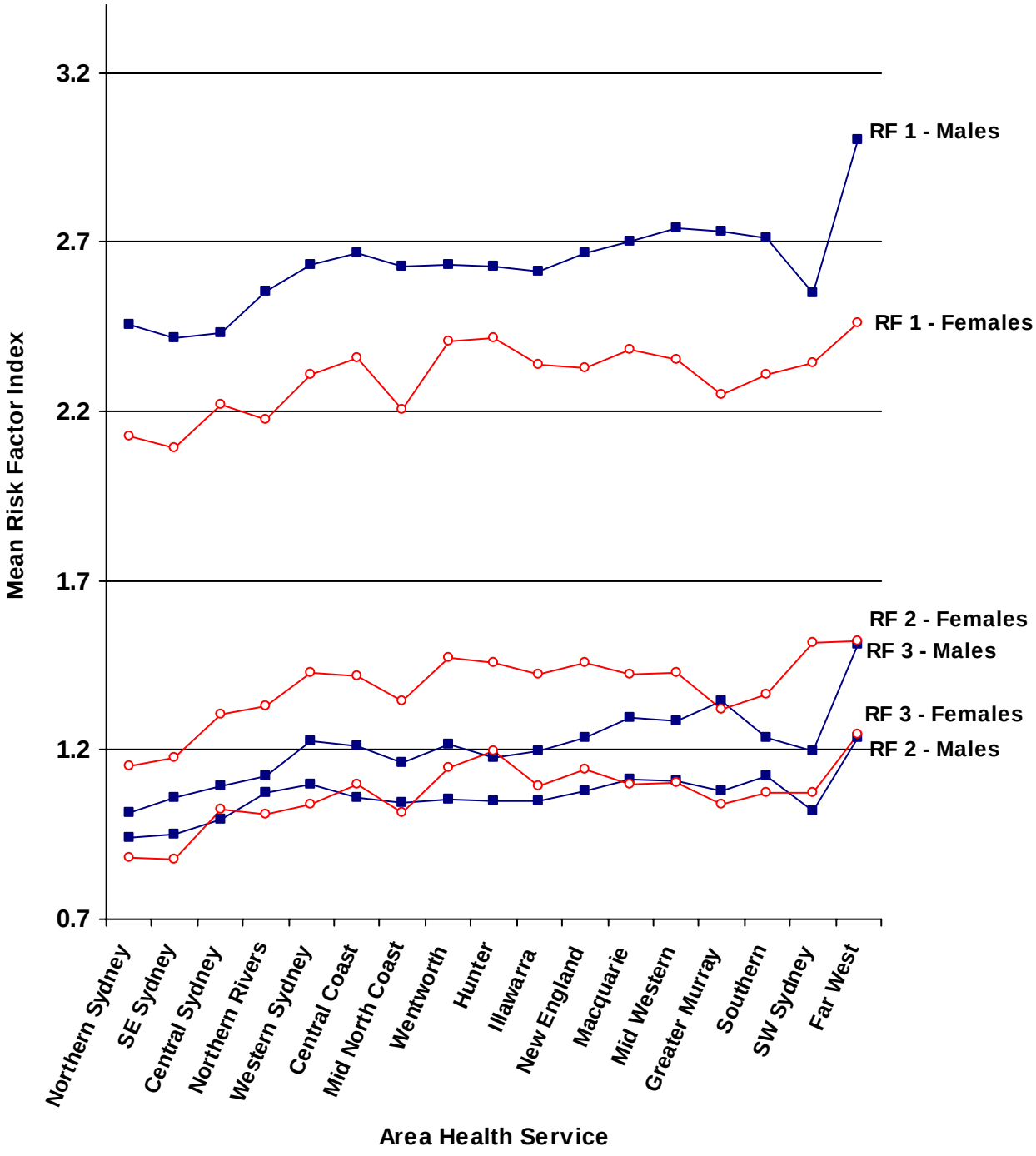


**Figure 8: Mean risk factor indices by Area Health Service**

## Likelihood of being at 'high risk'

Categorisation of those at 'high risk' was based on having a risk factor score in the highest quartile of the distribution. In risk factor 1, 'high' number of risk factors was based on a score of more than 3 (i.e. presence of either 4 or 5 risk factors). Since a high proportion of the sample had a score of 3, this resulted in only 15.2% of the sample (11.5% of women and 20.2% of men) being classified as at 'high' risk. For risk factor index 2, men were scored as 'high' if they had a score of greater than or equal to 1.47 (28%), and women if they scored 2.08 or higher (29.3%). For risk factor 3, people were classified as 'high' if their risk factor index was greater than or equal to 1.6. This was 26.8% of the whole sample. Among men, 30.2% were categorised as 'high risk' based on risk factor 2, compared to 24.3% of women.

**Table 3: Proportion with a high risk factor index across levels of socioeconomic disadvantage, and estimated odds ratios with and without adjustment for all other sociodemographic variables**

| | MEN | | | WOMEN | | |
|---|---|---|---|---|---|---|
| | % (high risk) | OR (95% CI) | Adjusted OR[1] (95% CI) | % (high risk) | OR (95% CI) | Adjusted OR [1] (95% CI) |
| **RFI 1** | | | | | | |
| **Least disadvantaged** | 15.3 | 1 | 1 | 9.0 | 1 | 1 |
| **Second least disadvantaged** | 17.3 | 1.2 (0.8-1.6) | 1.1 (0.7-1.5) | 11.6 | 1.3 (0.9-1.9) | 1.3 (0.9-1.9) |
| **Mid disadvantage** | 19.3 | 1.3 (1.0-1.8) | 1.2 (0.9-1.7) | 12.3 | 1.4 (1.0-2.0) | 1.4 (1.0-1.9) |
| **Second most disadvantaged** | 20.6 | 1.4 (1.1-1.9)* | 1.2 (0.9-1.7) | 11.4 | 1.3 (0.9-1.8) | 1.3 (0.9-1.8) |
| **Most disadvantaged** | 23.4 | 1.7 (1.3-2.3)* | 1.5 (1.1-2.0)* | 11.5 | 1.3 (1.0-1.9) | 1.3 (0.9-1.9) |
| **RFI2** | | | | | | |
| **Least disadvantaged** | 19.9 | 1 | 1 | 19.3 | 1 | 1 |
| **Second least disadvantaged** | 23.8 | 1.3 (0.9-1.7) | 1.2 (0.9-1.6) | 23.7 | 1.3 (1.0-1.7) | 1.2 (0.9-1.6) |
| **Mid disadvantage** | 26.6 | 1.5 (1.1-1.9)* | 1.4 (1.0-1.8) | 27.8 | 1.6 (1.3-2.1)* | 1.5 (1.2-1.9)* |
| **Second most disadvantaged** | 28.9 | 1.6 (1.3-2.1)* | 1.5 (1.3-2.0)* | 31.6 | 1.9 (1.5-2.5)* | 1.8 (1.4-2.2)* |
| **Most disadvantaged** | 32.5 | 1.9 (1.5-2.5)* | 1.8 (1.4-2.4)* | 33.3 | 2.1 (1.6-2.6)* | 1.8 (1.4-2.3)* |
| **RFI3** | | | | | | |
| **Least disadvantaged** | 21.8 | 1 | 1 | 16.0 | 1 | 1 |
| **Second least disadvantaged** | 23.1 | 1.1 (0.8-1.4) | 1.0 (0.7-1.3) | 21.1 | 1.4 (1.1-1.9)* | 1.3 (1.0-1.8) |
| **Mid disadvantage** | 30.7 | 1.6 (1.2-2.1)* | 1.4 (1.1-1.8)* | 23.7 | 1.6 (1.3-2.1)* | 1.5 (1.1-2.0)* |
| **Second most disadvantaged** | 30.0 | 1.5 (1.2-2.0)* | 1.3 (1.0-1.7) | 25.6 | 1.8 (1.4-2.3)* | 1.7 (1.3-2.2)* |
| **Most disadvantaged** | 35.9 | 2.0 (1.6-2.6)* | 1.6 (1.2-2.1)* | 27.2 | 2.0 (1.5-2.5)* | 1.8 (1.4-2.4)* |

[1] Adjusted for age, language spoken at home and highest level of education.
* Significantly different from those in the least disadvantaged quintile.

The odds ratio estimates for being at 'high risk' (based on each index) across levels of socioeconomic disadvantage for both men and women are shown in Table 3. After controlling for age, language spoken at home and highest level of educational attainment, socioeconomic disadvantage did not significantly predict having 4 or more risk factors (high

risk based on RFI1) among women, but men in the most disadvantaged quintile were 1.5 times significantly more likely to be at high risk than those in the least disadvantaged quintile. Both men and women in the three highest quintiles of socioeconomic disadvantage were significantly more likely to be at high risk than those in the least disadvantaged quintile based on RFI2, although men in the third most disadvantaged quintile were no longer at significantly greater risk after adjusting for other sociodemographic variables. Among men, those in the third and fifth quintiles of disadvantage were 1.4 and 1.6 times respectively more likely to be at high risk than those who were least disadvantaged based on RFI3. Women in the three most disadvantaged quintiles were at least 1.5 times significantly more likely than those in the least disadvantaged group to be at high risk based of RFI3.

The odds ratio estimates for being at 'high risk' (based on each index) based on language spoken at home for both men and women are shown in Table 4. For high risk classification using RFI1 (number of risk factors), both men and women who spoke a language other than English at home were half as likely as those who spoke English to be at high risk after adjustment for other sociodemographics. There was no significant difference between English and non-English speaking groups in likeliehood of being at high risk based on RFI2 among either men or women. However, women who spoke a language other than English at home were significantly less likely to be at high risk based on RFI3, after adjusting for other sociodemographic variables.

**Table 4: Proportion with a high risk factor index according to language spoken at home, and estimated odds ratios before and after adjusting for other sociodemographic variables**

| | MEN | | | WOMEN | | |
|---|---|---|---|---|---|---|
| | % (high risk) | OR (95% CI) | Adjusted OR[1] (95% CI) | % (high risk) | OR (95% CI) | Adjusted OR[1] (95% CI) |
| **RFI1** | | | | | | |
| **English speaking** | 20.9 | 1 | 1 | 12.0 | 1 | 1 |
| **Non-English speaking** | 12.8 | 0.6 (0.4-0.8)* | 0.5 (0.4-0.8)* | 6.6 | 0.5 (0.4-0.8)* | 0.5 (0.3-0.7)* |
| **RFI2** | | | | | | |
| **English speaking** | 28.4 | 1 | 1 | 29.6 | 1 | 1 |
| **Non-English speaking** | 24.0 | 0.8 (0.6-1.0) | 0.8 (0.6-1.0) | 27.1 | 0.9 (0.7-1.1) | 1.0 (0.8-1.3) |
| **RFI3** | | | | | | |
| **English speaking** | 30.4 | 1 | 1 | 25.0 | 1 | 1 |
| **Non-English speaking** | 25.6 | 0.8 (0.6-1.0) | 0.9 (0.7-1.2) | 17.9 | 0.7 (0.5-0.8)* | 0.7 (0.5-0.9)* |

[1] Adjusted for age, socioeconomic disadvantage and highest level of education.
* Significantly different from those who speak English at home.

The odds ratio estimates for being at 'high risk' (based on each index) for those who do not have a tertiary degree compared to those who do are shown in Table 5, by gender. Not having a tertiary degree significantly increased the likelihood of being at high risk for both

men and women across all indices, after adjusting for age, language spoken at home, and socioeconomic disadvantage.

**Table 5: Proportion with a high risk factor index according to educational attainment, and estimated odds ratios before and after adjusting for other sociodemographic variables**

| | MEN | | | WOMEN | | |
|---|---|---|---|---|---|---|
| | % (high risk) | OR (95% CI) | Adjusted OR[1] (95% CI) | % (high risk) | OR (95% CI) | Adjusted OR[1] (95% CI) |
| **RFI1** | | | | | | |
| **Tertiary educated** | 13.0 | 1 | 1 | 8.4 | 1 | 1 |
| **No tertiary education** | 22.1 | 1.9 (1.6-2.3)* | 1.8 (1.5-2.2)* | 12.3 | 1.5 (1.2-1.9)* | 1.7 (1.3-2.1)* |
| **RFI2** | | | | | | |
| **Tertiary educated** | 19.3 | 1 | 1 | 19.9 | 1 | 1 |
| **No tertiary education** | 30.3 | 1.8 (1.5-2.2)* | 1.7 (1.5-2.1)* | 31.5 | 1.9 (1.6-2.1)* | 1.6 (1.4-1.9)* |
| **RFI3** | | | | | | |
| **Tertiary educated** | 18.8 | 1 | 1 | 16.2 | 1 | 1 |
| **No tertiary education** | 33.0 | 2.1 (1.8-2.5)* | 2.0 (1.7-2.4)* | 26.2 | 1.9 (1.6-2.2)* | 1.8 (1.5-2.1)* |

[1] Adjusted for age, socioeconomic disadvantage and language spoken at home.
* Significantly different from those with a tertiary degree.

The odds ratio estimates for being at 'high risk' (based on each index) by Area Health Service (relative to Central Sydney Area Health Service) for both men and women are shown in Table 6. Compared to men in Central Sydney Area Health Service, men in Far West Area Health Service were 2-3 times significantly more likely to be at high risk when categorised on the basis of all three indices. Men in Macquarie, Mid Western, and Greater Murray AHS were also more likely than those in Central Sydney AHS to be at high risk based on both RFI1 and RFI3. Men in Northern Sydney were 30% less likely than those in Central Sydney to be at high risk based on RFI2 and women in Northern Sydney were significantly less likely than those in Central Sydney to be at high risk based on RFI2 and RFI3. Women in South Eastern Sydney Area Health Service were also significantly less likely than women in Central Sydney to be at high risk based on RFI1 and RFI3, but not when categorised as high risk based on RFI2. However, women in Far West and South Western Sydney Area Health Services were 1.8 times significantly more likely than women in Central Sydney Area Health Service to be at high risk based on RFI2.

Table 6: Proportion with a high risk factor index in each Area Health Service, and estimated odds ratios (unadjusted), separated by gender.

| | RFI1 | | RFI2 | | RFI3 | |
|---|---|---|---|---|---|---|
| **Area Health Service** | % (high risk) | OR (95% CI) | % (high risk) | OR (95% CI) | % (high risk) | OR (95% CI) |
| **MEN** | | | | | | |
| **Central Sydney** | 15.3 | 1 | 25.6 | 1 | 25.9 | 1 |
| **Northern Sydney** | 14.1 | 0.9 (0.6-1.4) | 18.6 | **0.7 (0.5-1.0)*** | 21.9 | 0.8 (0.6-1.2) |
| **Western Sydney** | 17.7 | 1.2 (0.8-1.9) | 27.9 | 1.1 (0.8-1.6) | 31.7 | 1.3 (0.9-1.9) |
| **Wentworth** | 20.2 | 1.4 (0.9-2.2) | 25.9 | 1.0 (0.7-1.5) | 29.1 | 1.2 (0.8-1.7) |
| **South Western Sydney** | 15.1 | 1.0 (0.6-1.6) | 25.9 | 1.0 (0.7-1.5) | 29.5 | 1.2 (0.8-1.7) |
| **Central Coast** | 19.1 | 1.3 (0.8-2.0) | 25.3 | 1.0 (0.7-1.4) | 30.7 | 1.3 (0.9-1.8) |
| **Hunter** | 21.1 | 1.5 (1.0-2.3) | 28.9 | 1.2 (0.8-1.7) | 27.5 | 1.1 (0.8-1.6) |
| **Illawarra** | 16.9 | 1.1 (0.7-1.7) | 25.0 | 1.0 (0.7-1.4) | 28.9 | 1.2 (0.8-1.7) |
| **South Eastern Sydney** | 14.9 | 1.0 (0.6-1.5) | 21.1 | 0.8 (0.5-1.1) | 22.8 | 0.9 (0.6-1.2) |
| **Northern Rivers** | 19.4 | 1.3 (0.9-2.0) | 31.7 | 1.4 (1.0-1.9) | 25.1 | 1.0 (0.7-1.4) |
| **Mid North Coast** | 20.3 | 1.4 (0.9-2.2) | 24.6 | 1.0 (0.7-1.4) | 26.9 | 1.1 (0.7-1.5) |
| **New England** | 22.0 | **1.6 (1.0-2.4)*** | 28.2 | 1.1 (0.8-1.7) | 30.7 | 1.3 (0.9-1.8) |
| **Macquarie** | 23.1 | **1.7 (1.1-2.5)*** | 32.3 | 1.4 (1.0-2.0) | 33.7 | **1.5 (1.0-2.1)*** |
| **Mid Western** | 24.8 | **1.8 (1.2-2.7)*** | 31.4 | 1.3 (0.9-1.9) | 34.5 | **1.5 (1.1-2.1)*** |
| **Far West** | 32.8 | **2.7 (1.8-4.0)*** | 40.7 | **2.0 (1.4-2.8)*** | 49.3 | **2.8 (2.0-3.9)*** |
| **Greater Murray** | 23.4 | **1.7 (1.1-2.6)*** | 28.4 | 1.2 (0.8-1.7) | 36.8 | **1.7 (1.2-2.4)*** |
| **Southern** | 22.9 | **1.6 (1.1-2.5)*** | 33.0 | **1.4 (1.0-2.0)*** | 29.9 | 1.2 (0.9-1.7) |
| **WOMEN** | | | | | | |
| **Central Sydney** | 11.2 | 1 | 23.7 | 1 | 21.9 | 1 |
| **Northern Sydney** | 8.5 | 0.7 (0.5-1.2) | 17.4 | **0.7 (0.5-1.0)*** | 14.0 | **0.6 (0.4-0.8)*** |
| **Western Sydney** | 10.7 | 1.0 (0.6-1.5) | 28.1 | 1.3 (0.9-1.7) | 21.1 | 1.0 (0.7-1.3) |
| **Wentworth** | 13.2 | 1.2 (0.8-1.9) | 33.5 | **1.6 (1.2-2.2)*** | 29.5 | 1.5 (1.1-2.1)* |
| **South Western Sydney** | 10.2 | 0.9 (0.6-1.4) | 36.4 | **1.8 (1.4-2.5)*** | 26.7 | 1.3 (0.9-1.8) |
| **Central Coast** | 12.6 | 1.2 (0.8-1.8) | 32.4 | **1.5 (1.1-2.1)*** | 26.8 | 1.3 (1.0-1.8) |
| **Hunter** | 13.8 | 1.3 (0.9-1.9) | 33.8 | **1.6 (1.2-2.2)*** | 30.4 | **1.6 (1.2-2.1)*** |
| **Illawarra** | 11.1 | 1.0 (0.7-1.5) | 30.7 | **1.4 (1.1-2.0)*** | 26.8 | 1.3 (1.0-1.8) |
| **South Eastern Sydney** | 7.2 | **0.6 (0.4-1.0)*** | 19.9 | 0.8 (0.6-1.1) | 15.5 | **0.7 (0.5-0.9)*** |
| **Northern Rivers** | 10.8 | 1.0 (0.6-1.5) | 27.6 | 1.2 (0.9-1.7) | 21.5 | 1.0 (0.7-1.4) |
| **Mid North Coast** | 9.2 | 0.8 (0.5-1.3) | 28.0 | 1.3 (0.9-1.7) | 23.0 | 1.1 (0.8-1.5) |
| **New England** | 12.7 | 1.2 (0.8-1.7) | 29.9 | **1.4 (1.0-1.9)*** | 25.3 | 1.2 (0.9-1.7) |
| **Macquarie** | 13.0 | 1.2 (0.8-1.8) | 34.3 | **1.7 (1.2-2.3)*** | 25.4 | 1.2 (0.9-1.7) |
| **Mid Western** | 13.7 | 1.3 (0.9-1.9) | 33.0 | **1.6 (1.2-2.1)*** | 24.2 | 1.1 (0.8-1.6) |
| **Far West** | 14.3 | 1.3 (0.9-2.0) | 36.2 | **1.8 (1.4-2.5)*** | 31.5 | **1.6 (1.2-2.3)*** |
| **Greater Murray** | 11.0 | 1.0 (0.6-1.5) | 26.1 | 1.1 (0.8-1.6) | 24.5 | 1.2 (0.8-1.6) |
| **Southern** | 12.2 | 1.1 (0.7-1.7) | 28.6 | 1.3 (1.0-1.7) | 26.1 | 1.3 (0.9-1.7) |

* Significantly different from Central Sydney Area Health Service. Note: Where '1' is included in the 95% Confidence Interval shown and * denotes significance, the CI did not include 1 before rounding to one decimal place.

## Discussion

The findings presented here highlight that different methods of calculating a chronic disease risk factor index can result in different conclusions when attempting to identify most at-risk groups. Profiling those most at risk based on the crude number of risk factors, as most previous studies have done, may be useful for targeting initiatives that address multiple health risk behaviours. However, the findings presented here suggest that it may be inappropriate to define these groups as most 'at risk' of chronic disease outcomes based on number of risk factors alone, without accounting for the differential contribution of behavioural risk factors to chronic disease outcomes and the potential risk associated with lower levels of exposure that may misclassify people as 'not at risk' based on dichotomous definitions.

However, there were particular sociodemographic identifiers that showed consistent patterns of risk across different methods of calculating a behavioural risk factor index. Risk increased with increasing quintile of socioeconomic disadvantage and the crude rate ratio of increased mean index for each subsequent level of disadvantage was similar for all indices among both men and women. Patterns of risk according to highest education level were also similar for both men and women across all indices, with a marked decrease in risk for those with tertiary qualifications and little difference between other educational categories. These findings suggest that risk behaviours decrease with increasing socioeconomic advantage and education among both men and women, regardless of whether methods used to calculate the risk index account for the differential contribution of risk factors to the total burden of disease or the risk associated with graded levels of exposure to the risk factor. Inconsistencies between the different indices in their association with other sociodemographic characteristics may be due to variations in patterns of risk across different sub-groups for different individual risk factors. This suggests an ongoing need to monitor specific health behaviours (smoking, physical activity, etc) among particular at-risk groups, rather than focusing only on a 'dashboard' approach to assess risk relative to other groups or change in risk over time.

Comparison of risk across age groups suggests that those aged 16-19 years have significantly less risk behaviours compared with those in all other age groups. However, this may be partly attributable to misreporting of certain risk behaviours that are legislatively discouraged for those at the lower end of this age group (such as tobacco and alcohol use). Risk behaviours appear to steadily decrease with age among those 50 years and older, although this may be due to a selective survival bias. There is a steep decrease in number of risk factors among men from age 60 which is not evident among women and may be attributable to the onset of cardiovascular disease in men around this age who have had high levels of risk behaviour. Patterns of risk according to age group also revealed some interesting differences between an index based on crude number of risk factors compared to that which accounts for differing levels of exposure to each risk factor. Most notably, the decline with increasing age was steeper for risk factor index 1 than for risk factor index 3. Since risk factor index 3 accounts for lower levels of exposure to risk behaviours, this suggests that older groups may not be engaging in substantially less risk behaviours than younger groups, but may be doing so at lower levels of exposure that are not accounted for when risk is categorised dichotomously based on minimum exposure levels.

Speaking a language other than English at home and being born outside Australia were significantly associated with lower mean risk, with the exception of risk factor index 2 among women (for which there was no significant difference based on language). Since risk factor index 2 is more heavily weighted for physical inactivity because of its substantial contribution to ill-health among women, lack of difference based on language may be explained by a high prevalence of physical inactivity among non-English speaking women. When the contribution of physical inactivity is not weighted heavily relative to other risk factors (as in the calculation of the other two indices), low rates of tobacco and alcohol use among non-English speaking women may account for their lowered risk. High levels of physical inactivity among non-English speaking women may also account for higher mean risk factor index 2 evident among women in South Western Sydney Area Health Service but not apparent for the other two indices, since the population in this area has greater representation from non-English speaking groups.

A chronic disease risk factor index can potentially be used to identify high-risk groups to be targeted for primary prevention of chronic disease outcomes. Prevention of chronic diseases through health promotion has a favourable cost-benefit ratio compared with treatment of these diseases. This is, in part, due to evidence that treatment or control of risk factors may not reduce the risk of disease to the equivalent of never having developed the risk factor. Since individuals with multiple major risk factors seem to experience a greater increased risk of a range of chronic diseases than would be expected from the summation of the independent risks, identification and health promotion targeting of subgroups with elevated multiple risk is likely to result in substantial improvement in chronic disease incidence.

**Potential Limitations**

Development of the chronic disease risk factor indexes described here was only able to utilise the questions and categorical response categories that were predetermined or pre categorized by the NSW Health survey program. For some of the variables, these categories do not allow sensitivity analyses using alternative categories across each risk factor. Other studies that have continuous measures available have determined categories of exposure based on the epidemiological evidence for the dose-response association with the specific disease being investigated. For smokers, there is evidence of a dose-response relationship for the number of cigarettes per day, total years of cigarette smoking, and degree of inhalation. In addition, the risk of outcomes for ex-smokers seems to vary according to the number of years since cessation (US Department of Health and Human Services, 1989). More sensitive measures of these risk behaviours and appropriate weighting of each level of exposure are likely to result in less misclassification for risk of the outcome. Nonetheless, the work here, based on the Australian BOD study, does allow comparisons with nationally defined norms of risk levels.

Construction of the index was also limited to inclusion of individual behaviours measured in the 2002 NSW Health survey. For example, inclusion of fruit and vegetable consumption to account for the contribution of nutritional behaviour to chronic disease may underestimate the total contribution of nutritional behaviour to the disease outcomes examined here. In addition, there is evidence exposure to environmental tobacco smoke may increase risk of some chronic diseases, and this was not accounted for in the index either.

This analysis is based on self-report measures of risk factors. There is evidence that self-report may be have some limitations, although self reported PA, tobacco use, and even relative body weight indices are usual in population surveys.

## References


Australian Bureau of Statistics (2001) Information Paper – Use of the Kessler Psychological Distress Scale in ABS Health Surveys. Cat No 4817.0.55.001 2001

Carstenson JM, Pershagen G & Eklund G (1987) Mortality in relation to cigarette and pipe smoking: 16 years' observation of 25,000 Swedish Men. *Journal of Epidemiology and Community Health, 41:* 166-172.

Chang M, Hahn RA, tetchy SM & Hutwagner LC (2001) Multiple risk factors and population attributable risk for ischemic heart disease mortality in the United States, 1971-1992. *Journal of Clinical Epidemiology, 54:* 634-644.

Jackson RT, Al-Mousa Z, Al-Raqua M, Prakash P & Muhanna AN (2002) Multiple coronary risk factors in healthy older Kuwaiti males. *European Journal of Clinical Nutrition, 56:* 709-714.

Kaukua J, Turpeinen A, Uusitupa M & Niskanen L (2001) Clustering of cardiovascular risk factors in type 2 diabetes mellitus: prognostic significance and tracking. *Diabetes, Obesity and Metabolism, 3:* 17-23.

Kenchaiah S, Evans JC, Levy D, Wilson PWF, Benjamin EJ, Larson MG, Kannel WB, Vasan RS. (2002) Obesity and the risk of heart failure. *The New England Journal of Medicine, 347 (5):* 305-313.

Mansur AP, Gomes EPSG, Avakian SD, Favarato D, Cesar LAM, Aldrighi JM, Ramires JAF (2001) Clustering of traditional risk factors and precocity of coronary disease in women. *International Journal of Cardiology, 81:* 205-209.

Mathers C, Vos T & Stevenson C (1999) The burden of disease and injury in Australia. AIHW cat. No. PHE 17. Canberra: AIHW

Murtagh KH, Borde-Perry WC, Campbell KL, Gidding SS & Falkner B (2002) Obesity, smoking, and multiple cardiovascular risk factors in young adult African Americans. *Ethnicity and Disease, 12:* 331-335.

Must A, Spadano J, Coakley EH, Field AE, Colditz G & Dietz WH (1999) The disease burden associated with overweight and obesity. *JAMA, 282(16):* 1523.

National Expert Advisory Committee on Alcohol (NEACA) (2001) Alcohol in Australia: Issues and strategies. Canberra: Commonwealth Department of Health and Ages Care.

National Health and Medical Research Council (2003) Dietary Guidelines for Australian Adults. ISBN: 1864961171

New South Wales Department of Health (2003) NSW Chronic Disease Prevention Strategy 2003-2007. ISBN: 0 7347 3592 8

New Zealand Ministry of Health (1999) Taking the pulse: the 1996/97 New Zealand Health Survey. Wellington, New Zealand: Ministry of Health. ISBN: 0 478 23511 9

Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Mamun AA, Bonneux L (2003) Obesity in adulthood and its consequences for life expectancy: a life-table analysis. Ann Intern Med., 138(1): 24-32.

Twisk JWR, Kemper HCG, van Mechelen W & Post GB (2001) Clustering of risk factors for coronary heart disease: the longitudinal relationship with lifestyle. Ann Epidemiol, 11: 157-165.

Schuit AJ, van Loon AJM, Tijhuis M & Ocke MC (2002) Clustering of lifestyle risk factors in a general adult population. Preventive Medicine, 35: 219-224.

Urbina EM, Srinivasan SR, Tang R, Bond MG, Kieltyka L & Berenson GS (2002) Impact of multiple coronary risk factors on the intima-media thickness of different segments of carotid artery in healthy young adults (The Bogalusa Heart Study). Am J Cardiol, 90: 953-958.

US Department of Health and Human Services (1983) The Health consequences of smoking: Cardiovascular disease. A report of the Surgeon General. Washington, DC: US Govt. Printing Office.

US Department of Health and Human Services (1989) Reducing the Health Consequences of Smoking: 25 years of progress. A report of the Surgeon General. Rockville, Maryland: Office on Smoking and Health.

van't Veer P, Jansen MCJF, Klerk M & Kok FJ (2000) fruits and vegetables in the prevention of cancer and cardiovascular disease. Public Health Nutrition, 3 (1): 103-107.

Wu DM, Pai L, Chu NF, Sung PK, Lee MS, Tsai JT, Hsu LL, Lee MC & Sun CA (2001) Prevalence and clustering of cardiovascular risk factors among healthy adults in a Chinese population: the MJ Health Screening Center Study in Taiwan. International Journal of Obesity, 25: 1189-1195.

Yusuf HR, Giles WH, Croft JB, Anda RF & Casper ML (1998) Impact of multiple risk factor profiles on determining cardiovascular disease risk. Preventive Medicine, 27: 1-9.

## Classification of Risk Factors Appendix A

**Dichotomous category definitions**

**Smoking:**

Indicator includes respondents who reported that they smoked daily or occasionally.

**Nutrition:**

NSW Health currently uses the following definitions for adequate fruit intake and adequate vegetable intake separately:

*Fruit Intake*: Adequate intake determined by meeting the recommendations for daily consumption according to the Australian Healthy Eating Guidelines (3 serves for people aged 16-18 years/ 2 serves for people aged 19 and over). One serve = one medium or 2 small pieces of fruit.

*Vegetable Intake*: Adequate intake determined by those who meet the recommended daily consumption of vegetables (4 serves per day if female or male aged 16-18 or more than 60 years/ 5 serves per day for males aged 19 to 60 years). One serve = ½ cup cooked vegetables or 1 cup salad vegetables.

For the purposes of this analysis, a combined variable for fruit and vegetable intake was defined to account for risk attributable to 'Nutrition' by the following:

*Inadequate Fruit and Vegetable Intake* (derived for this analysis): Less than 2 serves of fruit or less than 5 serves of vegetables per day (one serve as per above definitions), based on above categories for the majority of the population and on the recommendations for both men and women aged 19 to 60 years from the current Dietary Guidelines for Australians (NHMRC, 2003).

**Alcohol:**

Risk drinking defined as per guideline 1 of the Australian Alcohol Guidelines (National Expert Advisory Committee on Alcohol, 2001). One or more of the following:

- consuming alcohol every day
- consuming on average more than (4 if male/2 if female) standard drinks
- consuming more than (6 if male/4 if female) standard drinks on any one occasion or day

**Physical Activity:**

Adequate PA defined as 150 minutes per week (weighted x 2 for minutes in vigorous PA) on at least 5 separate occasions.

**Overweight & Obesity:**

Indicator includes respondents with a BMI greater than or equal to 25.

**Category definitions for multiple categories:**

**Smoking:**

Smoke daily (weighted as 1), Smoke occasionally (weighted as 0.8), ex-smoker (weighted as 0.5), non-smoker (those who reported that they had never regularly smoked; weighted as 0).

Overall, smokers have a 70% greater CHD mortality than non-smokers (US Department of Health and Human Services, 1983). Data from the Multiple Risk Factor Intervention Trial (MRFIT) have shown that ex-smokers reduced their risk of death from Coronary Heart Disease (CHD) by almost half, and risk of all-cause mortality by almost 30%, compared to current smokers (US Department of Health and Human Services, 1983). A 16 year prospective study of Swedish men has demonstrated that male smokers are between 2.9 (for cancer of the oral cavity/larynx) and 7.4 (lung cancer) times more likely to die from cancers compared with men who had never smoked (Carstenson, Pershagen & Eklund, 1987).

**Nutrition:**

Based on tertiles for total serves of fruit and vegetables per day. First tertile (lowest number of serves per day weighted as 0.4; Second tertile weighted as 0.2; Third tertile (highest number of serves per day) weighted as 0. The weightings for these categories are difficult to construct since the categorization is based on tertiles in the distribution (the cut-offs which are yet unknown). These weightings may change once the tertiles are calculated depending on the cut-offs for number of serves per day for each tertile and how these relate to demonstrated risk reduction for chronic diseases. One limitation to developing weightings for each category is that most of the research investigating risk of chronic diseases does not compare levels of total fruit and vegetable intake, but instead compare intake of specific subgroups of fruits and/or vegetables or associated vitamin levels (including fibre intake, consumption of legumes, levels of vitamin C, etc). There is much variation in definition of nutritional risk factors and levels of risk within them, and these inconsistencies makes it difficult to consolidate the evidence for the nature of a dose-response relationship between fruit and vegetable intake and chronic diseases. However, a review of observational studies that investigated the risk of cancer and cardiovascular disease attributable to 'low' (about 2.5 serves per day) versus 'high' intake (at least 4 serves per day) of fruits and vegetables suggests that the risk of upper respiratory tract and gastrointestinal cancers is reduced by about half, and the risk of cardiovascular disease (coronary heart disease and stroke) reduced by up to 40%, for those in the high compared to low consumption groups (van't Veer et al, 2000).

**Alcohol:**

Hazardous use when usually consume more than (2 if female/4 if male) standard drinks per occasion (weighted as 0.3); Harmful alcohol use when usually consumes more than (4 if female/6 if male) standard drinks per occasion (weighted as 0.4). Defined as per the International Guide for monitoring Alcohol Consumption and Related Harm.

**Physical Activity:**

Sedentary (nil reported time in activity; weighted as 1.0); categories of inadequate (but more than 0 minutes in PA; weighted as 0.4) and adequate (but less than 300 minutes in activity; weighted as 0.1) as per NSW Health dichotomous categories described above. 'High' was defined as 300 or more minutes (minutes in vigorous activity weighted by 2) and at least 5 sessions per week in activity (weighted as 0). The weighting applied to each category was informed by evidence from prospective studies that demonstrate the greatest reductions in risk for a range of chronic disease outcomes (diabetes, cardiovascular disease, cancer, etc) are for those engaging in low levels of physical activity compared with those that are sedentary.

**Overweight & Obesity:**

**'**Underweight' indicated by BMI <20 (weighted as 0); 'Healthy weight' by BMI from 20 to <25 (weighted as 0), 'Overweight' by BMI from 25 to <30 (weighted as 0.3), and 'Obese' by BMI greater than or equal to 30(weighted as 1.0).

Although the risk of all-cause mortality is slightly higher among underweight compared with normal weight persons, much of this is likely to be due to preexisting illness and higher rates of smoking among those with very low weight (Peeters et al, 2003). The association between risk of underweight and specific chronic disease outcomes is unclear, since most studies either exclude this group or compare the risk of being overweight and obese with those who 'are not overweight or obese'. Therefore, underweight was weighted as 0 (the same as healthy weight) in the index. Results from the Framingham Heart Study demonstrate that the risk of mortality from overweight is only significantly higher than normal weight participants for female non-smokers, although the risk among obese participants significantly increased by about 100% for obese participants compared to those of normal weight for all gender-specific groups of smokers and non-smokers (Peeters et al, 2003). A similar two-fold increase in risk for obese compared with healthy weight subjects, and 30% greater risk among overweight subjects, has been demonstrated for heart failure among participants in the Framingham Heart Study (Kenchaiah et al, 2002).