# The projected impact of population and high-risk strategies for risk-factor control on coronary heart disease and stroke events

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## ABSTRACT

**Objective:** To model the impact of both population and high-risk strategies on cardiovascular disease (CVD) outcomes.

**Design, setting and participants:** A CVD risk-factor survey was carried out in rural south-eastern Australia from 2004 to 2006. Using a stratified random sample, data for 1116 participants aged 35–74 years were analysed. Applying the Framingham risk equations to risk-factor data, 5-year probabilities of a coronary heart disease event, stroke and cardiovascular event were calculated. The effect of different changes in risk factors were modelled to assess the extent to which cardiovascular diseases can be prevented by changing the risk factors at a population level (population strategy), among the high-risk individuals (high-risk strategy) or both.

**Results:** Among men, a population strategy could reduce cardiovascular events by 19.3% (193 per 1000 per 5 years), the high-risk strategy by 12.6% (126 per 1000) and a combined strategy by 24.1% (241 per 1000); and among women, by 21.9% (219 per 1000), 19.0% (190 per 1000) and 28.7% (287 per 1000), respectively.

**Conclusions:** For prevention of CVD in Australia, it is important both to treat high-risk individuals and to reduce the mean risk-factor levels in the population. We show how risk-factor survey data can be used to set targets for prevention and to monitor progress in line with the recommendations of the National Preventative Health Taskforce.


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Cardiovascular diseases account for 30% of deaths among men and 35% among women in Australia,[1] and are still the main contributors to mortality. However, the mortality from cardiovascular disease (CVD) has declined substantially during the past 30 years. Of disability-adjusted years of life indicating the burden of disease, 17% is due to CVD in Australia,[1] and the situation in rural areas is worse than the national average.[2]

The main CVD risk-factor levels in Australia are generally similar to those in many other Western countries. The mean cholesterol concentration among Australian adults is approximately 5.5 mmol/L;[3-4] mean systolic blood pressure (SBP) is approximately 120–130 mmHg;[1,4-5] and smoking rates vary between 15% and 20%,[4,6] depending on the area and age group. It is known that the majority of the disease burden and mortality occur in population groups with moderate risk-factor levels, despite their relatively lower risk, because the proportion of the population in these groups is the largest.[7] Strategies for reduction of risk in populations and in individuals at high risk are needed to effectively reduce the disease burden.

Estimation of the risks of chronic disease is important at the individual and population levels. Individual assessment of total risk is essential for control of risk factors and management of patients, and evaluation can be done using any of several risk tables, keeping in mind their validity and their limitations in different populations and population groups.[8] Risk assessment at the population level is needed for planning of policy and prevention and treatment strategies. Such analyses can indicate reasonable targets for population strategies and can also help in estimating the resources needed to carry out effective high-risk approaches in a population.[9-10]

The National Preventative Health Strategy has called for action to reduce obesity, alcohol consumption and smoking. It recommends setting up a national prevention agency that will adopt a cyclical approach of "do, measure, report; do, measure, report". Measurement will occur through national risk-factor surveys.[11] We report how survey data can be used to determine targets against which the success of both health promotion and clinical interventions can be assessed.

The aim of our study is to assess the effect of population or high-risk strategies, or both, on cardiovascular outcomes. The effect of changes in cholesterol, blood pressure and smoking are modelled using CVD risk-estimation functions[12] and recent population data from rural Australia.

## METHODS

Three cross-sectional surveys of CVD risk factors and related health behaviour were carried out in the Greater Green Triangle (GGT) region of south-eastern Australia between 2004 and 2006[4] to obtain rural data for comparison with existing urban data.

Each survey used a stratified random sample of the population drawn from the electoral roll. Stratification was done by sex and 10-year age groups. In these analyses, the age group 35–74 years is used, as the risk estimates are most appropriate in this age group. Cardiovascular events are very rare before age 35, and there is uncertainty about the role of risk factors among elderly people.

The survey methodology has been described previously,[4] and comprised self-administered questionnaires, physical measurements and laboratory tests. The survey was approved by the Flinders Clinical Research Ethics Committee and all the participants gave their informed consent. A comparison of the socioeconomic background with population statistics available indicated that the participants closely resembled the true populations of the areas surveyed.

### Measures

The protocol in the risk-factor surveys closely followed the World Health Organization MONICA (Multinational Monitoring of Trends and Determinants in Cardiovascular Disease) Project protocol and expert group recommendations for risk-factor surveys.[4] Smoking status and history of heart disease, stroke and diabetes were assessed by a self-administered questionnaire. In the health check, systolic and diastolic blood pressure, fasting total cholesterol, high-density lipoprotein (HDL) cholesterol and blood glucose levels were ascertained. The venous blood samples were drawn after an overnight fast of at least 10 hours, and analysed at the then Flinders Medical Centre Clinical Trials Laboratory, which was internationally accredited for lipid measurement under the

**1 Projected 5-year cardiovascular event risks and current risk-factor profile of Greater Green Triangle risk-factor survey**

| | No. | Mean coronary event risk (%) (SE) | Mean stroke event risk (%) (SE) | Mean CV event risk (%) (SE) | > 10% CV event risk (%) | Current smoker (%) | Mean TC (mmol/L) (SE) | TC ⩾ 6.5 mmol/L (%) | Mean SBP (mmHg) (SE) | SBP ⩾160 mmHg (%) | Mean HDL-C (mmol/L) (SE) | T2DM (%) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Men | | | | | | | | | | | | |
| Age group (years) | | | | | | | | | | | | |
| 35–44 | 69 | 1.86 (0.19) | 0.19 (0.01) | 2.10 (0.22) | 0.0 | 23.2 | 5.57 (0.11) | 17.4 | 123.1 (1.7) | 2.9 | 1.31 (0.04) | 2.9 |
| 45–54 | 156 | 3.80 (0.19) | 0.45 (0.02) | 4.85 (0.23) | 5.8 | 17.9 | 5.69 (0.07) | 16.7 | 128.3 (1.2) | 5.1 | 1.33 (0.03) | 1.9 |
| 55–64 | 158 | 6.01 (0.30) | 1.10 (0.07) | 9.00 (0.44) | 31.6 | 13.9 | 5.43 (0.08) | 11.4 | 134.6 (1.5) | 10.8 | 1.37 (0.03) | 4.4 |
| 65–74 | 130 | 9.65 (0.37) | 2.42 (0.14) | 15.51 (0.59) | 79.2 | 13.1 | 5.21 (0.09) | 13.1 | 144.5 (1.8) | 21.5 | 1.31 (0.03) | 10.0 |
| Area | | | | | | | | | | | | |
| LC | 174 | 5.00 (0.19) | 0.96 (0.05) | 7.26 (0.27) | 23.7 | 14.7 | 5.52 (0.08) | 15.6 | 134.5 (1.4) | 14.2 | 1.29 (0.03) | 4.6 |
| CO | 133 | 4.78 (0.25) | 0.79 (0.05) | 6.64 (0.32) | 23.5 | 15.3 | 5.52 (0.09) | 17.9 | 126.6 (1.3) | 5.3 | 1.31 (0.04) | 1.3 |
| WI | 206 | 4.71 (0.18) | 0.92 (0.05) | 7.02 (0.26) | 23.7 | 16.9 | 5.45 (0.07) | 12.1 | 131.8 (1.1) | 6.4 | 1.37 (0.03) | 5.9 |
| *Total* | *513* | *4.79 (0.12)* | *0.88 (0.03)* | *6.91 (0.17)* | *23.1* | *15.9* | *5.51 (0.04)* | *15.0* | *131.2 (0.8)* | *8.7* | *1.33 (0.02)* | *4.1* |
| Women | | | | | | | | | | | | |
| Age group (years) | | | | | | | | | | | | |
| 35–44 | 91 | 0.52 (0.08) | 0.13 (0.01) | 1.00 (0.15) | 0.0 | 16.5 | 5.12 (0.09) | 6.6 | 117.3 (1.5) | 1.1 | 1.60 (0.04) | 2.2 |
| 45–54 | 188 | 1.60 (0.12) | 0.31 (0.02) | 2.34 (0.17) | 1.6 | 16.0 | 5.48 (0.08) | 16.5 | 123.5 (1.3) | 4.8 | 1.62 (0.03) | 3.2 |
| 55–64 | 181 | 2.80 (0.14) | 0.70 (0.03) | 4.46 (0.21) | 3.9 | 5.0 | 5.86 (0.08) | 26.0 | 132.0 (1.3) | 6.6 | 1.65 (0.03) | 3.9 |
| 65–74 | 143 | 3.77 (0.20) | 1.91 (0.12) | 8.47 (0.42) | 26.6 | 5.6 | 5.72 (0.09) | 25.2 | 144.6 (1.6) | 23.8 | 1.68 (0.03) | 9.8 |
| Area | | | | | | | | | | | | |
| LC | 204 | 2.17 (0.11) | 0.68 (0.04) | 3.93 (0.20) | 7.3 | 16.5 | 5.56 (0.08) | 19.6 | 129.9 (1.2) | 9.4 | 1.57 (0.03) | 5.3 |
| CO | 172 | 2.06 (0.11) | 0.62 (0.03) | 3.63 (0.18) | 6.2 | 7.1 | 5.53 (0.08) | 17.2 | 126.5 (1.2) | 5.6 | 1.58 (0.03) | 5.5 |
| WI | 227 | 1.79 (0.10) | 0.68 (0.05) | 3.42 (0.19) | 6.3 | 11.6 | 5.46 (0.08) | 16.2 | 126.0 (1.2) | 7.8 | 1.71 (0.03) | 3.3 |
| *Total* | *603* | *1.96 (0.06)* | *0.65 (0.02)* | *3.58 (0.11)* | *6.5* | *11.6* | *5.50 (0.04)* | *17.3* | *127.3 (0.7)* | *7.8* | *1.63 (0.02)* | *4.2* |

CV = cardiovascular. TC = total cholesterol. SBP = systolic blood pressure. HDL-C = high-density lipoprotein cholesterol. T2DM = type 2 diabetes mellitus. LC = Limestone Coast. CO = Corangamite Shire. WI = Wimmer. ◆

Centers for Disease Control and Prevention Lipid Standardization Program.

Five-year coronary, stroke and cardiovascular event risks were calculated separately for men and women by using the Framingham risk function.[12-13] The function takes into account age, sex, smoking status, total cholesterol, HDL cholesterol, SBP and type 2 diabetes mellitus (T2DM).

Data from the GGT region were used to calculate the individual risk of a fatal or non-fatal cardiovascular event in the next 5 years. Those who had smoked regularly during the previous month were classified as smokers. T2DM status was assessed as positive if fasting plasma glucose was ⩾ 7.0 mmol/L or the person was undertaking treatment with insulin or oral hypoglycaemic agents.

Statistical analyses were undertaken using Stata, version 11 (StataCorp, College Station, Tex, USA). Means, standard errors and percentages are presented for current projected risks and risk factors (Box 1). Where age groups are combined, data are weighted to the age distribution of the local areas.

Change in risk resulting from a successful population strategy, high-risk strategy or both was modelled by reducing the risk-factor levels either by a percentage or by reducing the risk factors of those who had high levels (Box 2 and Box 3). Optimal treatment targets selected were SBP of 140 mmHg (using multiple antihypertensive drugs) and serum cholesterol concentration of 4.5 mmol/L (using diet and lipid-lowering drugs), based on the evidence that the risk of coronary heart disease (CHD) decreases progressively, at least down to total cholesterol concentrations of 4.5 mmol/L.[14] In the analyses combining both strategies, the population approach was applied first, and after that those remaining in the high-risk group were treated. The estimated numbers of events prevented per 1000 people per 5 years were calculated by multiplying 5-year relative reduction percentages by 10, and are presented in the boxes and in parentheses in the text.

## RESULTS

After excluding participants who had ever had a heart attack, stroke, cerebral haemorrhage, coronary bypass surgery, or coronary angioplasty, 1116 out of 1319 eligible participants had complete information for calculating coronary and stroke event risk. CVD risk-factor levels and the estimated risk of a coronary event or stroke event in the next 5 years are shown in Box 1. The risk of a cardiovascular event in the next 5 years was 6.9% in men and 3.6% in women. A greater than 10% risk of a cardiovascular event was present in 23.1% of men and 6.5% of women.

Estimated effects of population and high-risk strategies on CHD events are shown in Box 2 and Box 3. In the population strategy, a modest 5% (0.28 mmol/L) reduction in

**2 Potential effects of population and high-risk strategies on 5-year risk of cardiovascular events in men, using Framingham functions**

| | Coronary events | | | | Stroke events | | | | Cardiovascular events | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Mean risk (%) (SE) | Mean absolute reduction (%) (SE) | Mean relative reduction (%) (SE) | ER (per 1000) | Mean risk (%) (SE) | Mean absolute reduction (%) (SE) | Mean relative reduction (%) (SE) | ER (per 1000) | Mean risk (%) (SE) | Mean absolute reduction (%) (SE) | Mean relative reduction (%) (SE) | ER (per 1000) |
| No change | 4.79 (0.12) | | | | 0.88 (0.03) | | | | 6.91 (0.17) | | | |
| **Total cholesterol** | | | | | | | | | | | | |
| Population strategy | | | | | | | | | | | | |
| 5% TC reduction | 4.48 (0.11) | 0.32 (0.01) | 6.6 (0.1) | 66 | 0.88 (0.03) | 0.00 (0.00) | 0.2 (0.0) | 2 | 6.61 (0.16) | 0.31 (0.01) | 4.4 (0.0) | 44 |
| 10% TC reduction | 4.16 (0.11) | 0.64 (0.01) | 13.3 (0.1) | 133 | 0.87 (0.03) | 0.00 (0.00) | 0.4 (0.0) | 4 | 6.30 (0.16) | 0.62 (0.01) | 8.9 (0.1) | 89 |
| High-risk strategy | | | | | | | | | | | | |
| TC reduced to 4.5 mmol/L if TC ≥ 6.5 mmol/L | 4.34 (0.10) | 0.45 (0.06) | 9.4 (1.0) | 94 | 0.88 (0.03) | 0.00 (0.00) | 0.2 (0.0) | 2 | 6.50 (0.15) | 0.42 (0.05) | 6.1 (0.7) | 61 |
| Population and high-risk strategy | | | | | | | | | | | | |
| 10% TC reduction, to 4.5 mmol/L if TC still ≥ 6.5 mmol/L | 4.03 (0.10) | 0.77 (0.04) | 16.0 (0.6) | 160 | 0.87 (0.03) | 0.00 (0.00) | 0.4 (0.0) | 4 | 6.18 (0.15) | 0.73 (0.03) | 10.6 (0.4) | 106 |
| **SBP** | | | | | | | | | | | | |
| Population strategy | | | | | | | | | | | | |
| 2.5% SBP reduction | 4.59 (0.12) | 0.20 (0.00) | 4.2 (0.0) | 42 | 0.80 (0.03) | 0.08 (0.00) | 9.1 (0.0) | 91 | 6.52 (0.16) | 0.39 (0.01) | 5.6 (0.1) | 56 |
| 5% SBP reduction | 4.39 (0.11) | 0.40 (0.01) | 8.4 (0.1) | 84 | 0.72 (0.02) | 0.15 (0.00) | 17.5 (0.0) | 175 | 6.14 (0.15) | 0.77 (0.01) | 11.2 (0.1) | 112 |
| High-risk strategy | | | | | | | | | | | | |
| SBP reduced to 140 mmHg if ≥ 160 mmHg | 4.56 (0.11) | 0.23 (0.03) | 4.9 (0.6) | 49 | 0.74 (0.02) | 0.14 (0.02) | 16.1 (2.0) | 161 | 6.43 (0.14) | 0.48 (0.07) | 7.0 (0.9) | 70 |
| Population and high-risk strategy | | | | | | | | | | | | |
| 5% SBP reduction, to 140 mmHg if still ≥ 160 mmHg | 4.28 (0.10) | 0.52 (0.03) | 10.8 (0.4) | 108 | 0.65 (0.02) | 0.22 (0.02) | 25.6 (1.5) | 256 | 5.90 (0.14) | 1.01 (0.05) | 14.6 (0.6) | 146 |
| **Smoking** | | | | | | | | | | | | |
| No smoking | 4.44 (0.10) | 0.36 (0.04) | 7.5 (0.8) | 75 | 0.82 (0.03) | 0.06 (0.01) | 7.0 (1.1) | 70 | 6.30 (0.14) | 0.62 (0.08) | 8.9 (1.0) | 89 |
| **All risk factors** | | | | | | | | | | | | |
| Reduction of 10% in TC, 5% in SBP | 3.80 (0.10) | 1.00 (0.02) | 20.8 (0.2) | 208 | 0.72 (0.02) | 0.16 (0.01) | 17.8 (0.0) | 178 | 5.58 (0.14) | 1.34 (0.02) | 19.3 (0.2) | 193 |
| TC reduced to 4.5 mmol/L if ≥ 6.5 mmol/L and SBP to 140 mmHg if ≥ 160 mmHg | 4.13 (0.09) | 0.66 (0.07) | 13.8 (1.2) | 138 | 0.74 (0.02) | 0.14 (0.02) | 16.3 (2.0) | 163 | 6.04 (0.13) | 0.87 (0.09) | 12.6 (1.1) | 126 |
| Combined strategy for TC and SBP | 3.57 (0.09) | 1.22 (0.05) | 25.6 (0.7) | 256 | 0.65 (0.02) | 0.23 (0.02) | 25.9 (1.5) | 259 | 5.24 (0.12) | 1.67 (0.07) | 24.1 (0.6) | 241 |
| Combined strategy for TC and SBP + no smoking | 3.29 (0.07) | 1.51 (0.07) | 31.5 (0.9) | 315 | 0.60 (0.01) | 0.27 (0.02) | 31.2 (1.6) | 312 | 4.74 (0.10) | 2.17 (0.10) | 31.4 (1.0) | 314 |

ER = event reduction. TC = total cholesterol. SBP = systolic blood pressure. ◆

serum cholesterol in men would reduce coronary events by 6.6% (66 per 1000 per 5 years), and a 10% decline (0.55 mmol/L) would reduce coronary events by 13.3 (133 per 1000). Using a high-risk strategy (reducing serum cholesterol to 4.5 mmol/L among those who have cholesterol of 6.5 mmol/L or higher) would reduce coronary events by 9.4% (94 per 1000) in men and 17.2% (172 per 1000) in women.

Similar analyses were done for blood pressure. Using a population strategy, a 2.5% (3.3 mmHg) decline in SBP in men would reduce coronary events by 4.2% (42 per 1000), and a 5% (6.6 mmHg) decline by 8.4% (84 per 1000). Decline in blood pressure would also reduce stroke events. The same 2.5% and 5% declines in men's blood pressure would reduce stroke events by 9.1% (91 per 1000) and 17.5% (175 per 1000). In women, the estimated effects of

**3 Potential effects of population and high-risk strategies on 5-year risk of cardiovascular events in women, using Framingham functions**

| | Coronary events | | | | Stroke events | | | | Cardiovascular events | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Mean risk (%) (SE) | Mean absolute reduction (%) (SE) | Mean relative reduction (%) (SE) | ER (per 1000) | Mean risk (%) (SE) | Mean absolute reduction (%) (SE) | Mean relative reduction (%) (SE) | ER (per 1000) | Mean risk (%) (SE) | Mean absolute reduction (%) (SE) | Mean relative reduction (%) (SE) | ER (per 1000) |
| No change | 1.96 (0.06) | | | | 0.65 (0.02) | | | | 3.58 (0.11) | | | |
| **Total cholesterol** | | | | | | | | | | | | |
| Population strategy | | | | | | | | | | | | |
| 5% TC reduction | 1.81 (0.06) | 0.15 (0.00) | 7.9 (0.1) | 79 | 0.65 (0.02) | 0.00 (0.00) | 0.2 (0.0) | 2 | 3.40 (0.10) | 0.18 (0.00) | 5.1 (0.1) | 51 |
| 10% TC reduction | 1.66 (0.06) | 0.31 (0.01) | 15.6 (0.1) | 156 | 0.65 (0.02) | 0.00 (0.00) | 0.4 (0.0) | 4 | 3.22 (0.10) | 0.37 (0.01) | 10.2 (0.1) | 102 |
| High-risk strategy | | | | | | | | | | | | |
| TC reduced to 4.5 mmol/L if TC ⩾ 6.5 mmol/L | 1.62 (0.05) | 0.34 (0.03) | 17.2 (1.4) | 172 | 0.65 (0.02) | 0.00 (0.00) | 0.4 (0.0) | 4 | 3.19 (0.10) | 0.40 (0.04) | 11.1 (1.0) | 111 |
| Population and high-risk strategy | | | | | | | | | | | | |
| 10% TC reduction, to 4.5 mmol/L if TC still ⩾ 6.5 mmol/L | 1.55 (0.05) | 0.41 (0.02) | 21.1 (0.9) | 211 | 0.65 (0.02) | 0.00 (0.00) | 0.5 (0.0) | 5 | 3.09 (0.10) | 0.49 (0.03) | 13.7 (0.6) | 137 |
| **SBP** | | | | | | | | | | | | |
| Population strategy | | | | | | | | | | | | |
| 2.5% SBP reduction | 1.86 (0.06) | 0.10 (0.00) | 5.0 (0.0) | 50 | 0.59 (0.02) | 0.06 (0.00) | 9.1 (0.0) | 91 | 3.35 (0.10) | 0.23 (0.01) | 6.5 (0.1) | 65 |
| 5% SBP reduction | 1.77 (0.06) | 0.19 (0.00) | 9.9 (0.1) | 99 | 0.54 (0.02) | 0.11 (0.00) | 17.5 (0.0) | 175 | 3.12 (0.10) | 0.46 (0.01) | 12.8 (0.1) | 128 |
| High-risk strategy | | | | | | | | | | | | |
| SBP reduced to 140 mmHg if ⩾ 160 mmHg | 1.84 (0.06) | 0.12 (0.02) | 6.2 (0.7) | 62 | 0.54 (0.01) | 0.11 (0.02) | 16.9 (1.9) | 169 | 3.27 (0.09) | 0.32 (0.04) | 8.9 (1.0) | 89 |
| Population and high-risk strategy | | | | | | | | | | | | |
| 5% SBP reduction, to 140 mmHg if still ⩾ 160 mmHg | 1.71 (0.06) | 0.25 (0.01) | 12.8 (0.5) | 128 | 0.49 (0.02) | 0.16 (0.01) | 25.0 (1.3) | 250 | 2.98 (0.09) | 0.61 (0.03) | 16.9 (0.7) | 169 |
| **Smoking** | | | | | | | | | | | | |
| No smoking | 1.82 (0.06) | 0.14 (0.02) | 7.2 (0.9) | 72 | 0.61 (0.02) | 0.04 (0.01) | 5.6 (1.1) | 56 | 3.28 (0.09) | 0.30 (0.04) | 8.4 (1.1) | 84 |
| **All risk factors** | | | | | | | | | | | | |
| Reduction of 10% in TC, 5% in SBP | 1.49 (0.05) | 0.48 (0.01) | 24.2 (0.2) | 242 | 0.54 (0.02) | 0.12 (0.00) | 17.8 (0.0) | 178 | 2.80 (0.09) | 0.79 (0.02) | 21.9 (0.2) | 219 |
| TC reduced to 4.5 mmol/L if ⩾ 6.5 mmol/L and SBP to 140 mmHg if ⩾ 160 mmHg | 1.52 (0.05) | 0.44 (0.04) | 22.4 (1.5) | 224 | 0.54 (0.01) | 0.11 (0.02) | 17.2 (1.9) | 172 | 2.90 (0.08) | 0.68 (0.06) | 19.0 (1.3) | 190 |
| Combined strategy for TC and SBP | 1.34 (0.05) | 0.62 (0.03) | 31.7 (0.9) | 317 | 0.49 (0.02) | 0.16 (0.01) | 25.3 (1.3) | 253 | 2.55 (0.08) | 1.03 (0.05) | 28.7 (0.8) | 287 |
| Combined strategy for TC and SBP + no smoking | 1.24 (0.04) | 0.72 (0.04) | 36.6 (1.1) | 366 | 0.46 (0.01) | 0.19 (0.01) | 29.3 (1.5) | 293 | 2.33 (0.07) | 1.25 (0.06) | 35.0 (1.2) | 350 |

ER = event reduction. TC = total cholesterol. SBP = systolic blood pressure. ◆

blood pressure reduction on coronary events and stroke were very similar to that of men.

SBP reduction to 140 mmHg among men whose blood pressure is at least 160 mmHg would reduce coronary events by 4.9% (49 per 1000) and stroke events by 16.1% (161 per 1000). The blood pressure treatment effects were very similar in men and women.

The combination of high-risk and population strategies provide the highest estimated benefit. After benefits from population strategies, 4.3% of men and 5.2% of women would still have cholesterol of 6.5 mmol/L or higher and 4.2% of men and 4.1% of women would still have SBP of 160 mmHg or higher. In men, the combined strategies for cholesterol would reduce coronary events by 16.0% (160 per 1000) and combination strategies for blood pressure would reduce coronary events by 10.8% (108 per 1000). The estimated effects of combined strategies on stroke events would be 0.4% (4 per 1000) for cholesterol and 25.6% (256 per 1000) for blood pressure. Eliminating

smoking would reduce coronary event risk by 7.5% (75 per 1000) in men and 7.2% (72 per 1000) in women.

Among men, combining population and high-risk strategies for both cholesterol and blood pressure would reduce coronary events by 25.6% (256 per 1000) and total cardiovascular events by 24.1% (241 per 1000). Stopping smoking would give an additional 6%–7% reduction. Among women, the results were similar.

In the ideal situation (in which no one smokes, cholesterol is reduced to 4.0 mmol/L, SBP to 110 mmHg, and no one has T2DM), the average man would reduce his 5-year coronary event risk by 64.9% (649 per 1000) and the average woman would reduce her 5-year coronary event risk by 68.4% (684 per 1000). Five-year total cardiovascular event risk for men would reduce by 65.8% (658 per 1000), and for women by 67.3% (673 per 1000).

## DISCUSSION

In this analysis, the population approach produced a greater overall impact on risk reduction than the high-risk strategy. However, in practice, this will depend on the extent to which the goals are achieved. Most importantly, the results of the analyses support the need for combining high-risk and population strategies. These combined strategies apply to Australia and other countries overall, although our data were from a particular rural Australian population. In the population we studied, the high-risk strategy for both SBP and cholesterol would reduce cardiovascular events in men by 12.6% and the population strategy would reduce the events by 19.3%. Applying both strategies would reduce cardiovascular events by 24.1%. Among women, the pooled effect would be 28.7%. In the combined analyses, the risk-factor distribution was first reduced for the whole population, and then those still remaining in the high-risk group were considered eligible for the high-risk strategy. Thus, the combined effect is not simply additive. For coronary event prevention, the cholesterol changes seem to be more important than SBP reduction, but for total cardiovascular effect, SBP would be more important. This is because cholesterol is not a strong risk factor for stroke, but blood pressure affects both coronary events and stroke.

The CVD risk-factor levels observed in the GGT region were slightly higher than those observed in studies carried out in urban areas in Australia,[4] but similar to other industrialised countries. Smoking rates were comparable with Canada and some states in the United States.[15] Mean SBP levels were higher in our study than, for example, in the US and Canada (where the adult population mean SBP is about 120 mmHg[16]), but slightly lower than in Finland, which, compared with other countries, has high SBP levels (where the adult population mean SBP is 136 mmHg among men and 129 mmHg among women[17]). Mean cholesterol concentrations in the GGT region are high (5.50 mmol/L in men and women), exceeding those in the US (5.26 mmol/L[15]) and in Finland (5.29 mmol/L in men and 5.19 mmol/L in women[17]).

The Framingham risk-estimation function was used in this study to assess the absolute CHD, stroke and total CVD risk in the rural GGT population. The 3.3–6.6 mmHg reduction in SBP at the population level (2.5%–5% reduction in the GGT population) is a realistic target for a population strategy. With long-term reduction of salt intake to 6 g/day, a 7 mmHg reduction in SBP among hypertensive patients can be achieved.[18] Moderate-intensity physical activity reduces SBP by an average of 5 mmHg.[19] An approximate 4% reduction in weight reduces SBP by up to 6 mmHg,[20] and a reduction in heavy alcohol consumption by four drinks per day among hypertensive patients reduces SBP by about 5 mmHg.[21]

A reduction in total cholesterol in the GGT population by 5%–10% corresponds to a 0.28–0.55 mmol/L reduction. At the population level, such a reduction could be achieved with dietary interventions. By reducing the intake of saturated fat, increasing the intake of polyunsaturated fat, increasing the intake of fibre (especially soluble fibre) and by reducing dietary cholesterol intake, a 3%–10% reduction in total cholesterol can be obtained.[22]

In many countries, such reductions in risk-factor levels have been seen, followed by a major reduction in CHD mortality. In Finland, for example, the CHD mortality has declined since the early 1970s by more than 80%. This is a result of effective policies and improvements in treatment, especially in primary and secondary prevention, including a combination of population-based screening programs in community health centres and workplaces and opportunistic screening in general practice.[17] Behind this Finnish CHD mortality reduction there were reductions of over 20% in the total cholesterol level, about 7% in SBP and 40% in smoking rates. With such major nationwide social and cultural changes as occurred in Finland, population approaches outperform clinical trials of modifying multiple risk factors by lifestyle changes.[23]

Strategies similar to those used in Finland could be effectively implemented in Australia. In the United Kingdom, an approximate 55% reduction in CHD mortality was seen between 1980 and 2000 following reductions of 4% in total cholesterol, 8% in SBP and 35% in smoking.[24] A remarkable decline in CVD mortality has been seen in Australia since the late 1960s (around 70%).[6] Smoking has reduced from nearly 30% to 16% among people aged over 14 years from 1985 to 2007, and SBP decreased from 134 mmHg to 128 mmHg among men, and from 127 mmHg to 121 mmHg among women aged 25–64 years between 1980 and 2000.[1] No notable change has been seen in cholesterol concentrations.[1] As the smoking rates are already low, the possibilities for further reducing smoking are challenging, but through effective population and high-risk strategies, major reduction in both blood pressure levels and total cholesterol levels should be achievable.

For effective strategies to reduce cardiovascular risk, the population-level data on risk factors and their predictive value for morbidity and mortality are needed, preferably from regular population surveys, to estimate the possible effects of different interventions. Data are also needed to estimate the proportion of the population at high risk and the resources required to intervene. These estimates are crucial for planning national preventive and management programs. In most Western countries, there has been a major reduction in CVD risk factors, resulting in a considerable decline in mortality. These analyses indicate that still more can be done to reduce the CVD burden.

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## COMPETING INTERESTS

None identified.

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