

The Protein Leverage Hypothesis: What's Driving the Obesity Epidemic?

by Stephen Simpson



Periodically, in western North America, huge bands of Mormon crickets extending up to 10 km in length move en masse across the landscape – a striking example of mass animal movement. What makes them do it? Recently we showed that the reasons are nutritional: These creatures are on a forced march in search of protein and salt⁽¹⁾.

The tendency of Mormon crickets to prioritise protein over other macronutrients is not unique. It has been extensively studied in other insects⁽²⁾ and in other animals, including fish, birds, rodents, and free-ranging monkeys⁽³⁻⁵⁾. Recently, we hypothesised that humans too might have a dominating protein appetite which may partly explain the current epidemic of obesity and light the path to obesity management⁽³⁾.

PROTEIN LEVERAGE

Our hypothesis states that humans prioritise protein intake over the other macronutrients and will tend to eat until a protein target is reached. If true, there are very different implications for body weight depending on whether the available diet is relatively low or high in protein. If the diet is relatively high in protein, humans will reach their protein target early and then stop eating, under-eating kilojoules, carbohydrate and fat.

On the other hand, if the protein content is relatively low and the diet is dominated by carbohydrate and fat, humans will tend to over-eat these nutrients and kilojoules until the intake target for protein is finally met. Put simply, if protein is limiting, people will tend to overeat and get fat.

If this sounds implausible it is exactly what happens in the insect world. In one study, groups of locusts were offered 19 different foods varying in the protein:non-protein ratio. When confined to low protein foods locusts would continue to eat until their protein target was attained. They deposited excess lipid – the insect equivalent of obesity – and life expectancy diminished.

In modern western societies with abundant food supplies there is a surfeit of carbohydrate and fat. As humans have limited evolutionary experience of excess carbohydrates or fats it seems reasonable to infer that natural selection against their over-consumption would not have been strong.

IMPLICATIONS

If the Protein Leverage Hypothesis is correct, the implications are significant. Firstly, a relatively small fall in the proportion of dietary energy from protein would result in a disproportionately large increase in consumption of kilojoules from carbohydrate and fat – the protein leverage effect. We estimate that a fall in protein intake from 14 percent of energy to 12.5 percent, as has occurred in the United States since 1961, would require a 14 percent increase in kilojoules from carbohydrate and fat in order to maintain the amount of protein eaten. This is consistent with recent trends in diet and rates of obesity in the United States.

A second implication is that diets higher in protein should facilitate weight loss, which appears to be the case, at least in the short-term.

Thirdly, a fall in physical activity would need to be associated with an increase in the percentage energy from protein in order to maintain stable body weight.

If the same percentage of energy from protein is maintained while total energy requirements are lowered, the protein target will not be met and the likely consequence is over-consumption of carbohydrate and fat. This is the reverse of the first scenario.

The key assumption of our hypothesis is that when humans are forced to trade-off protein intake against that of carbohydrate or fat on nutritionally unbalanced diets, physiological regulatory mechanisms prioritise protein. If this is true then all other aspects of the hypothesis must follow – it is a mathematical inevitability. Further studies in humans using covertly manipulated foods under tightly controlled conditions will be required to provide the proof and are nearing completion.

References

1. Simpson SJ et al. *PNAS* 2006;103:4152-56.
2. Raubenheimer D, Simpson SJ. *Nutr Res Rev* 1997;10:151-79.
3. Simpson SJ et al. *Obesity reviews* 2005;6:133-142.
4. Sørensen, A et al. *Obesity* 2008;16:566-571.
5. Felton et al. *Behav. Ecol* 2009; in press.

Stephen Simpson is an ARC Federation Fellow in the School of Biological Sciences at the University of Sydney and a Visiting Professor at the University of Oxford.

ADVISORY PANEL

Professor Jennie Brand-Miller
University of Sydney, Sydney

Professor Peter Clifton
CSIRO Health Sciences and Nutrition, Adelaide

Professor Caryl Nowson
Deakin University

Professor Linda Tapsell
University of Wollongong

Professor Jim Mann
University of Otago, Dunedin

EDITOR

Megan Cobcroft
*Nutrition Manager,
Unilever Australasia.*

ALL CORRESPONDENCE TO:

*Megan Cobcroft, Editor
Perspectives
20 Cambridge Street
Epping NSW 2121, Australia*

*The views expressed in
Perspectives are those of the
authors and do not necessarily
reflect those of the Advisory
Panel or Unilever's nutrition policy.*



Unilever

*Perspectives is a service
to health professionals by
Unilever Australasia.*

perspectives

NUTRITION NEWS AND VIEWS

CONTENTS

2

EDITORIAL
THE SILENT EPIDEMIC:
CARDIOVASCULAR DISEASE
IN WOMEN

Bill Shrapnel

3

SHOULD NUTRITIONISTS
RECOMMEND TEA OR COFFEE?

Colin Binns

4

TIME TO SOUP UP WEIGHT
MANAGEMENT!

Barbara Rolls

5

THE MENOPAUSE
AND CHOLESTEROL
(continued from page 1)

Heather Currie

6

HOLD THE BUTTER:
PREVENTING HEART DISEASE
IN NEW ZEALAND

Rod Jackson

8

THE PROTEIN LEVERAGE
HYPOTHESIS: WHAT'S DRIVING
THE OBESITY EPIDEMIC?

Stephen Simpson

RECEIVE PERSPECTIVES ELECTRONICALLY

Subscribe to the
Perspectives e-newsletter
by emailing your name to
[perspectives.nutrition@
unilever.com](mailto:perspectives.nutrition@unilever.com)

The Menopause and Cholesterol

by Heather Currie



Although there is widespread awareness of common early menopausal symptoms, there continues to be a poor appreciation of the important long-term effects of lack of ovarian estrogen on the cardiovascular system. Education and preventive action is needed to lower the risk of cardiovascular disease in menopausal women, which is by far the leading cause of death in this segment of society.

THE MENOPAUSAL TRANSITION

The time from the beginning of the ovarian decline until the cessation of menstruation lasts about five years. On average, women experience their last period at the age of about 52 years. During the menopause, hormone secretion is unpredictable and hormonal fluctuations can lead to a range of familiar physical and psychological symptoms including hot flushes, night sweats, urogenital problems and depressed mood. The gradual loss of metabolically active hormones produced by the ovaries also has significant but less obvious effects on women's whole body metabolism. It is widely accepted that the loss of ovarian estrogen affects skeletal health by reducing bone density, thus increasing the likelihood of developing osteoporosis. Though not widely recognised, it is also well documented that serum cholesterol rises and the risk of cardiovascular disease increases progressively throughout the menopausal transition. As women in developed countries can now expect to live up to a third of their lifetime after the menopause, long-term cardiovascular health is an increasingly important consideration.

More women die from cardiovascular disease than from the next five causes of death combined.

MENOPAUSE, CHOLESTEROL AND CARDIOVASCULAR DISEASE

During menopause declining estrogen levels progressively disrupt the serum lipid profile, increasing LDL-cholesterol by 10-20 percent⁽¹⁾. Most of the change in cholesterol concentration occurs early in the menopausal transition. Total cholesterol levels in women peak between 55 and 65 years of age, about a decade later than in men. There is also a shift in fat distribution from the lower body (pear shape) to the upper body, around the abdominal organs (apple shape). This central distribution of fat is characteristic of the metabolic syndrome and may be associated with lower HDL-cholesterol and higher triglyceride levels. Although this fat distribution resembles that of men, the adverse effects associated with it are actually greater in women. These combined changes in lipid levels, along with rising blood pressure and increased insulin resistance, significantly increase women's risk of cardiovascular disease, which was estimated in the Framingham study to increase four-fold over the 10 years after the menopause⁽²⁾.

There is a common misconception that cardiovascular disease primarily affects middle-aged men. In fact, cardiovascular disease affects just as many women as men, though the onset is delayed by about 10 years due to the protection afforded by female hormone activity. When this fades

continued on page 5

EDITORIAL

The Silent Epidemic: Cardiovascular Disease in Women by Bill Shrapnel



SPECIAL NOTE FOR FOOD AUSTRALIA READERS:

Perspectives is going electronic! This will be the last printed edition of *Perspectives* to be included in Food Australia. The next edition will be released in October in a new e-newsletter format. To continue receiving articles on the latest nutrition science in *Perspectives*, email perspectives.nutrition@unilever.com with your NAME and 'PERSPECTIVES' in the subject line. You can then look forward to receiving the new-look e-newsletter in October! For information on Unilever's privacy policy, please go to www.unilever.com.au

Heather Currie's article in this issue of *Perspectives* is one of those articles that makes you sit up and think. Is it true that more women die from cardiovascular disease each year than men? Affirmative: In Australia the total number of female deaths from cardiovascular disease is about 23,000 annually, compared to just over 21,000 deaths in men ⁽¹⁾. Nearly 36 percent of all deaths in Australian females are due to cardiovascular disease. Certainly, in the younger age groups cardiovascular deaths in men exceed those of women but in older age groups, in which most cardiovascular events occur, the reverse is true.

The common belief that women are protected against coronary heart disease is misleading.

In New Zealand the differences in coronary rates between the sexes is less marked though the overall toll is higher with an astonishing 44 percent of all female deaths due to cardiovascular disease ⁽²⁾. That's 4300 deaths annually – the largest single cause of death among women, far exceeding the total number of deaths from all cancers combined. Rod Jackson's article on page 6 provides some insight into why cardiovascular disease, particularly coronary heart disease, exacts such a heavy toll in New Zealand.

The common belief that women are protected against coronary heart disease is misleading. The development of heart disease in women is not prevented, it is delayed. Although coronary rates lag behind those of men by about 10 years there is a substantial increase in risk in women from about 50 years of age. Adverse changes to serum lipids appear to be related to this increase in risk, the steady rise in serum cholesterol that occurs with age adding to the sharp increase associated with the menopause. This latter increase in cholesterol appears to start about three years before the onset of menopause and continues for about one year after the transition is complete ⁽³⁾.

Fortunately, most of the cardiovascular disease in women is preventable and the dietary recommendations for its prevention are the same as those for men ⁽⁴⁾. However, dietary intakes of key nutrients for cholesterol lowering still fall well outside the relevant dietary goals outlined in the Nutrient Reference Values – 10%E for saturated fat and 5-10% for polyunsaturated fat. In relation to the benefits of lowering serum cholesterol, no dietary trials have been conducted in women but the prevention trials using statins suggest

that women benefit as much as men. Even a modest reduction in cholesterol-related risk would have a large public health benefit. For example, it has been estimated that a reduction of just 0.1 mmol/L in the mean serum cholesterol of the New Zealand population would result in about 300 fewer deaths from cardiovascular disease each year ⁽⁵⁾.

Before further advances in prevention of heart disease in women are possible it will be essential to resolve the current paradox between actual risk and perceived risk. The results of European consumer surveys referred to by Dr Currie are consistent with consumer research conducted

by the American Heart Association that shows that only a small percentage of women believe heart disease and stroke constitute a significant threat to their long-term health ⁽⁶⁾. It is reasonable to expect that the same situation prevails on this side of the globe, especially given the media coverage given to other women's health issues.

Menopause is a time of change with implications for future health – a time when women are open to advice about diet and lifestyle. The onus is on dietitians and other health care professionals to take advantage of this opportunity to raise women's awareness of their increased risk of cardiovascular disease at this stage of life and the dietary changes needed to counter it. The average lifetime risk of cardiovascular disease in women is high so prevention is important for all.

On a final note, after 14 years as Editor of *Perspectives*, I have decided to move onto new opportunities outside Unilever and will hand over the baton to the new editor in time for our October issue. Thank you for supporting *Perspectives* over the past years. I trust you will continue to enjoy *Perspectives* into the future.

References

1. AIHW Australia's Health 2006.
2. Hay D. Cardiovascular disease in New Zealand, 2004. National Heart Foundation of New Zealand. Technical Report No. 82.
3. Akahoshi M et al 1996.
4. Mosca L et al. *Circulation* 2007;115:1481-1501.
5. Turley ML et al. *Aust NZ J Pub Health* 2006;30:252-257.
6. Mosca L et al. *Circulation* 2004;109:573-79.

Should Nutritionists Recommend Tea or Coffee? by Colin Binns



Tea and coffee are two of the most commonly consumed beverages worldwide and any impact they might have on chronic disease, positive or negative, has considerable implications for public health.

ASSESSING THE EVIDENCE

Evidence for formulating dietary guidelines is usually guided by evidence from epidemiological studies, with additional insight from animal studies, in vitro studies and short-term human intervention trials. Once evidence from these sources suggests a positive or negative effect on chronic disease the onus is on nutritionists to communicate the likely benefits or risks to the general public. In relation to the effects of tea and coffee, the chronic diseases that have been studied the most are cancer and cardiovascular disease – two of the largest causes of morbidity and mortality in the world. Our recent review of this evidence contains a full list of references ⁽¹⁾.

EFFECTS ON CANCER

Although coffee has been suspected of increasing the risk of several cancers this has not been confirmed by recent research. The latest evidence suggests that moderate consumption of coffee is not associated with cancer of the breast, pancreas, ovary, kidney, thyroid, prostate or bladder. There is some evidence from the epidemiology and animal studies that coffee consumption may be protective against colorectal cancer.

Quite strong evidence for a cancer protective effect of tea has emerged from animal and in vitro studies. The majority have shown a protective effect against cancers in the gut, oral cavity, lung, skin, liver, pancreas, bladder, mammary gland and prostate. The antioxidant property of tea polyphenols is the most researched preventive mechanism. Evidence from epidemiological studies is less clear cut and

Tea is a superior choice to coffee and nutritionists should advocate tea as part of a healthy diet.

complicated by sometimes differing findings for green and black tea. Overall, there is evidence of protection by green tea against cancer of the stomach, colon and rectum, breast and ovary, and protection by black tea against prostate cancer.

CARDIOVASCULAR DISEASE

Short-term human intervention studies indicate that coffee consumption can increase cholesterol levels and increase blood pressure slightly, which has raised the prospect that coffee consumption might increase the risk of cardiovascular disease. However, a recent review of epidemiological studies concluded that no clear association between coffee and the risk of hypertension, myocardial infarction or other cardiovascular disease can be demonstrated.

Evidence that both green and black tea is protective against cardiovascular disease is building. Prospective cohort studies have shown inverse associations between tea drinking and cardiovascular disease, myocardial infarction, important vascular

events and mortality. Flavonol intake, largely but not exclusively from tea, has also been associated with lower coronary risk. The epidemiology is supported by evidence from short-term human intervention studies, animal studies and in vitro studies.

In relation to possible mechanisms of action, tea has been shown to improve the vascular epithelium and affect homocysteine, cholesterol, LDL oxidation and atherogenesis.

Among other chronic conditions to have been investigated, type 2 diabetes is attracting some interest. There is possible evidence of protection by coffee but no likely effect of tea.

In considering guidelines for tea and coffee consumption a maximum intake of caffeine of 400 mg per day is recommended and not associated with any adverse effects. This is equivalent to approximately 4-5 cups of coffee or 13 cups of tea.

CONCLUSIONS

Although coffee has been suspected of having a variety of adverse effects, research has confirmed that moderate consumption (4-5 cups over a day) is safe. Tea is more than safe: it is a healthier option because of possible roles in the prevention of several cancers and cardiovascular disease. Despite the wide use of tea and coffee, nutritionists currently provide little dietary advice about their consumption. In light of the evidence, tea is a superior choice to coffee and nutritionists should advocate tea as part of a healthy diet.

References

1. Binns CW et al. *Public Health Nutrition* 2008;11:1132-41.
Colin Binns is Professor of Public Health, School of Public Health, Curtin University, Perth.

KEY POINTS

- Both tea and coffee are safe.
- Tea is the healthier option.
- Nutritionists should advocate tea as part of a healthy diet.

Time to Soup Up Weight Management!

by Barbara Rolls



Identifying dietary factors that influence energy intake is important for developing effective weight management strategies. Several studies suggest that routinely eating soup can reduce energy intake, enhance satiety and promote weight loss.

Although little is known about the specific properties of soup that are involved in reducing food intake and increasing satiety, several characteristics have been suggested, including temperature, the physical form of the soup (pureed or chunky) and its viscosity. In a recent study we examined the effects of consuming different forms of a low energy-dense soup prior to a meal on the intake of that meal and the total energy consumed ⁽¹⁾. We hypothesised that consuming soup at the start of a meal would decrease subsequent intake and total meal energy intake, compared to when no soup was consumed, and that a chunky soup would be the most satiating form.

when no soup was consumed, subjects ate 20 percent less energy from the test meal when a soup was consumed, confirming the results of our earlier study ⁽²⁾. The lower energy intake was not accompanied by increased ratings for hunger or decreased fullness at the end of the meal. Women and men responded similarly. Importantly, the reduction in energy intake was observed when any of the soups was consumed, so our hypothesis that the chunky soup would have the most satiating was not confirmed.

What accounts for these findings? The most likely explanation is that eating a large volume of a food containing few calories at the start of a meal

Eating a large volume of a food containing few calories at the start of a meal displaces intake from more energy-dense foods that follow.

SOUP STUDY

Subjects with a range of ages and body mass indices were recruited so that the findings would be relevant to the general population. They came to our laboratory once a week for five weeks for breakfast and lunch. Lunch was served three hours after a standard breakfast. At the beginning of each lunch meal, subjects were served one of four vegetable soups or no soup at all. The four soups which were all prepared from identical ingredients were broth and vegetables served separately, chunky vegetable soup, chunky-pureed vegetable soup and pureed vegetable soup. All soups had the same energy density (1.4 kJ/g). Men and women consumed 475 ml and 350 ml of soup, respectively. Fifteen minutes after the soup was served a test meal consisting of pasta was served and subjects could eat or drink as much or as little as they wanted. Subjects rated their hunger and fullness before and after breakfast, before and after the soup and after lunch. Sixty subjects, 30 women and 30 men, completed the study.

EFFECTS ON HUNGER, SATIETY AND ENERGY INTAKE

Our subjects' ratings of hunger and fullness immediately before the lunch test meal was served were significantly lower following all four types of soup than when no soup was eaten. Compared to

displaces intake from more energy-dense foods that follow. Other low energy density foods, such as salad, have a similar effect when consumed as a first course ⁽³⁾. While the mechanisms underlying the effect of soup have not been established, it is likely that the enhanced satiety results from a combination of sensory, cognitive and physiological responses. Soup has also been shown to help promote weight loss ⁽⁴⁾. We found that adding two servings of soup a day to an energy-controlled diet resulted in 50 percent greater weight loss after one year than adding two servings of energy-dense snacks.

In conclusion, the results from our study offer additional support for recommending the inclusion of various forms of low energy-dense soups in the diet as a strategy for controlling energy intake for weight management, while allowing individuals to consume satisfying amounts of food.

References

1. Flood JE, Rolls BJ. *Appetite*. 2007;49:626-34.
2. Rolls BJ et al. *Am J Clin Nutr* 1999;70:448-455.
3. Rolls BJ et al. *J Am Diet Assoc* 2004;104:1570-76.
4. Rolls BJ et al. *Obes Res* 2005;13:1052-60.

Barbara J. Rolls is Professor, Department of Nutritional Sciences at The Pennsylvania State University, USA.

KEY POINTS

- Soup has a satiating effect.
- Consuming soup prior to a meal can lower energy intake at the meal by 20 percent.
- Soup consumption can enhance weight loss.

at menopause women’s risk of cardiovascular disease increases and rates of the disease begin to rise.

At about the age of 50 the prevalence of cardiovascular disease in women is roughly equal to that of men and at older ages it exceeds that of men. Although the age-standardised rates in women remain below that of men, the toll is considerable. In Europe, cardiovascular disease accounts for over half of all female deaths and in the United States it accounts for one in every 2.5 female deaths.

Breast cancer is often wrongly reported in the media as the leading cause of death in women but in fact, more women die from cardiovascular disease than from the next five causes of death combined. Globally, women are nine times more likely to die from cardiovascular disease than breast cancer. In Europe, cardiovascular disease accounts for 57 percent of all female deaths compared to just three percent from breast cancer.

The symptoms of heart disease may manifest themselves differently in women compared to men. For example, women are more likely than men to present with unstable angina and once they have a myocardial infarction their prognosis is worse. Almost two-thirds of women who die suddenly from heart disease have no prior symptoms (3). In the immediate period following an acute myocardial infarction, more women die than men. Also, there are distinct gender differences in responses to some treatments, for example aspirin, and in rates of intervention carried out.

LOW PUBLIC AWARENESS

Expert bodies including the European Society of Cardiology (4) and the American Heart Association (3) have highlighted the growing problem of cardiovascular disease in women and the need to improve prevention and treatment. For this to happen, health professionals and women themselves need to be aware of the problem and sufficiently well-informed to take preventive action. Recent surveys conducted in five European countries revealed that menopausal women seriously underestimate their risk of cardiovascular disease, being more concerned about osteoporosis, breast cancer and physical menopausal symptoms (5). Fewer than one in four women listed heart disease as a concern and only one in four women associated menopause with high blood cholesterol. On the general issue of dealing

with menopausal symptoms, women indicated a strong preference for adopting the right diet and lifestyle, rather than taking medication.

The problem of awareness extends to health care professionals. According to the American Heart Association, “Women’s heart risk (is) underestimated by doctors, resulting in less preventive care than men”. The situation is similar in Europe. According to a recent survey of health care professionals, more than two-thirds were unaware that women of menopausal age are at equal or greater risk of cardiovascular disease than middle-aged men. Advice to women to have their blood cholesterol checked rated seventh in doctors’ recommendations for preventive treatment after taking more exercise, losing weight, stopping smoking, a self-check for breast cancer, a smear test and increasing calcium intake.

DIET AND LIFESTYLE MODIFICATION FOR LDL-LOWERING

The World Health Organisation estimates that 80 percent of cardiovascular disease could be prevented by positive diet and lifestyle changes (6). Elevated LDL-cholesterol is widely accepted as a key modifiable risk factor for coronary heart disease and is a primary target for risk reduction. Studies estimate that lowering LDL-cholesterol by 10 percent will reduce the risk of coronary disease by about 20 percent. Dietary changes shown to lower LDL-cholesterol are summarised in Table 1. Replacing saturated fat with polyunsaturated fat and increasing plant sterol intake are the most potent strategies.

References

1. Hall G et al. *Maturitas* 2002;41:177-85.
2. Kannal WB, Levy D. *Arch Int Med* 2004;164:479-81.
3. American Heart Association. *Women and cardiovascular disease facts*. AHA, 2007.
4. *Eur Soc Cardiol. Eur Heart J* 2006;27:994-1005.
5. Taylor Nelson Sofres (for Unilever). *Survey of attitudes to the menopause and cholesterol*. August 2007.
6. WHO. *Diet, Nutrition and prevention of chronic disease*. WHO Technical Report Series 916, 2003.

Dr Heather Currie is Associate Specialist Gynaecologist and Obstetrician, Dumfries and Galloway Royal Infirmary, Dumfries, United Kingdom. She is the founder and Managing Director of Menopause Matters which runs the award winning website (www.menopausematters.co.uk) and publishes the quarterly magazine Menopause Matters.

KEY POINTS

- During menopause declining estrogen levels progressively disrupt the serum lipid profile, increasing LDL-cholesterol.
- Fat storage is redistributed from the lower to the upper body, changing the body shape from ‘pear’ to ‘apple’. This shift in fat storage may be associated with lower HDL-cholesterol and higher triglyceride levels.
- In the 10 years following the onset of menopause cardiovascular risk in women increases four-fold.
- More must be done to inform women of the problem so that preventive action can take place.

TABLE 1: DIETARY STRATEGIES FOR LOWERING LDL-CHOLESTEROL

DIETARY STRATEGY	RECOMMENDATION	LDL-C REDUCTION
Lower saturated fat	<7 percent of energy	5-10 percent
Increase polyunsaturated fat	Up to 10 percent of energy	3 percent
Increase plant sterols	2.0-2.5g/day	10 percent
Increase soluble fibre	3g/day	2-5 percent
Increase soy protein	25g/day	3-5 percent

Hold the Butter: Preventing Heart Disease in New Zealand

by Rod Jackson



We now have a good understanding of the causes of coronary heart disease. The big three risk factors are high blood cholesterol, high blood pressure and smoking. In a country like New Zealand these factors alone explain about 80 percent of all coronary heart disease. Despite good progress, there is still a long way to go with heart disease prevention and one simple dietary change would help.

High blood cholesterol appears fundamental to coronary risk. The Japanese smoke much more and have higher blood pressure than New Zealanders yet, historically, Japanese rates of coronary disease have been amongst the lowest in the world while those in New Zealand have been among the highest. The Japanese are not protected by their genes. The famous Ni-Hon-San study, which followed Japanese men who migrated to the USA via Honolulu, found that the migrating Japanese took on the heart disease rates of the country they lived in. Those living in the USA had the highest rates and those living in Honolulu had intermediate rates. The main determinant of the different coronary rates between these populations was their environment, especially their diet. The Japanese experience tells us that the underlying risk for coronary disease has a lot to do with blood lipids. When cholesterol is raised, smoking and high blood pressure make it much worse. But if blood cholesterol is low, smoking and raised blood pressure do not seem to increase the risk for heart disease very much.

THE 'HIGH CHOLESTEROL' MYTH

Figure 1 shows the relationship between blood cholesterol and the risk of coronary heart disease. This figure is based on the biggest cohort study in the world involving over one million people, followed up for 10 years, and shows the relation clearly – as blood cholesterol increases, risk of coronary disease increases⁽¹⁾. The relationship is a straight line – there is no indication of any threshold,

The relationship between cholesterol and heart disease is evident in all age groups.

so the lower the cholesterol level the better. The ideal total blood cholesterol is below about 4.0-4.5 mmol/L and every cholesterol level above this is associated with an increased risk of coronary heart disease. So the most meaningful definition of high blood cholesterol is any level over 4.0-4.5 mmol/L, which means most of us have high blood cholesterol.

The relationship between cholesterol and heart disease is evident in all age groups. Note that the scale in Figure 1 is a log scale, which means that the slope for older people at the top is actually much steeper for than that for younger people at the bottom.

It doesn't matter where you live – Asia, New Zealand or Australia, or who you are – European, Asian, Maori or Pacific Islander, as your cholesterol goes up your risk of coronary heart disease goes up⁽²⁾.

If cholesterol is lowered, everybody benefits. The statin trials have shown that it doesn't matter if people have very high or modestly raised cholesterol before they start treatment; in relative terms they all get a similar benefit.

THE GOOD NEWS

The story of prevention of coronary heart disease in New Zealand is overwhelmingly positive. The death rate has been plummeting since the late 1960s, declining at a rate of two to three percent per year. It is happening across all age groups

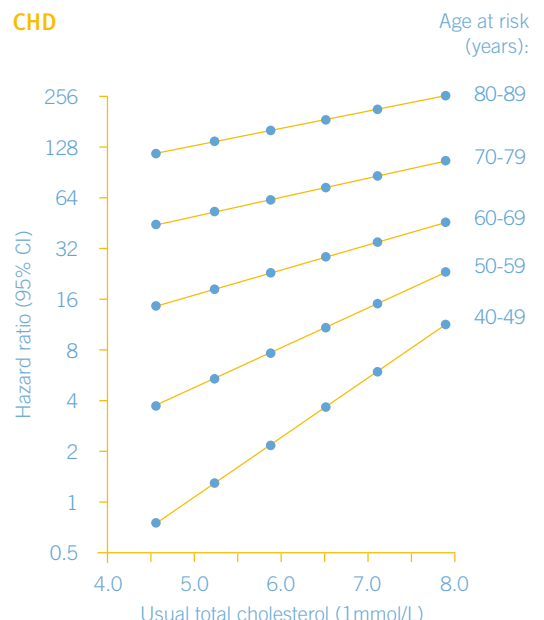
and now hospitalisations for heart attacks are also declining. Most of this occurred before the advent of statins, so it is not a statin effect. This is seriously good news but why is it happening?

Mark Tobias and colleagues from the Ministry of Health have looked at the contributions of changes in the major risk factors to falling heart disease rates⁽³⁾:

- Blood cholesterol is declining. In men, the mean has fallen from the high figure of about 6.2 mmol/L in the mid-1980s to nearly 5.8 mmol/L in 2000. The fall in women may be even greater.
- Blood pressure is declining quite rapidly, in both sexes. This does not appear to be directly related to medication and is probably largely related to changes in diet.
- There were major declines in smoking rates in the 1970s and less impressive falls in the 1980s and 1990s. But smoking rates are still falling.

FIGURE 1: IHD MORTALITY (33744 DEATHS) VERSUS USUAL TOTAL CHOLESTEROL

PSC. Lancet 2007; 370: 1829-39



So all the major risk factors are declining and between 70-90 percent of the observed decline in coronary heart disease rates can be accounted for by changes in these major risk factors. The one thing that is going in the wrong direction is body weight.

The fall in cholesterol accounts for 20-40 percent of the fall in heart disease. However, although blood cholesterol levels are coming down across all age groups, the bad news is that even the best group,

New Zealanders need to replace high saturated fat foods with low saturated fat foods to further drive down cholesterol levels and coronary heart disease rates.

women aged 35-44 years, has a mean total cholesterol of about 5.0 mmol/L. So two-thirds of these women have blood cholesterol levels above the ideal range of 4.0-4.5 mmol/L. Of the rest of the population, about three-quarters have cholesterol levels above the ideal range. This is why New Zealand's heart disease rate still remains near the top of the OECD comparative tables of coronary disease – five times the rate that prevailed in Japan in the 1950s and 1960s. So New Zealanders still have a long way to go with lowering cholesterol and preventing coronary heart disease. Even the Australians are doing better than we are and we hate that.

HIGH BUTTER INTAKE

Why does Australia have a lower mean blood cholesterol level and lower coronary heart

disease rates than New Zealand? Saturated fat intake, and in particular butter intake, appears to be the key. Figure 2 shows the sources of saturated fat in the New Zealand diet, the major single source being butter. In 2003, the average New Zealander ate 11 kg of butter per head per year whereas the average Australian ate about 3.2 kg. This difference in diet alone probably accounts for about half of the difference in coronary rates between New Zealand and Australia.

Other major sources of saturated fat include beef, lamb and milk, but you can take the fat out of these products. The beef, lamb and dairy industries have done fantastic things with lean meats and low fat dairy products. There is now a wealth of low saturated fat products available – we just need to eat them instead of the high fat products we still eat too much of.

CONCLUSION

My message is simple: New Zealanders need to replace high saturated fat foods with low saturated fat foods to further drive down cholesterol levels and coronary heart disease rates. In particular, hold the butter, which alone accounts for about one fifth of our total saturated fat intake!

References

1. *Prospective Studies Collaboration. Lancet 2007; 370: 1829-39.*
2. *Asia Pacific Cohort Studies Collaboration. Int J Epidemiol 2003;32:563-572.*
3. *Tobias et al. ANZJPH 2008;32: 117-25.*

Rod Jackson is Professor of Epidemiology and Head of Epidemiology & Biostatistics at the School of Population Health, University of Auckland.

KEY POINTS

- The causes of coronary heart disease are now well understood – raised blood cholesterol is fundamental.
- The relationship between blood cholesterol and heart disease is linear, present in all age groups, and across all races. Lowering blood cholesterol lowers coronary risk.
- Despite good progress in prevention, cholesterol levels and rates of coronary disease remain high in New Zealand.
- Butter is the major single source of saturated fat in the New Zealand diet, intake per capita being three times that in Australia.
- The difference in butter intake probably accounts for about half the difference in coronary rates between New Zealand and Australia.

FIGURE 2: SOURCES OF SATURATED FAT IN NEW ZEALAND DIET

