



Australia's cholesterol crossroads: An analysis of 199,331 GP patient records

A report of cholesterol levels and management of dyslipidaemia in primary care from 2004 to mid 2009

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Abbreviations

AIHW	Australian Institute of Health and Welfare
CAD	Coronary artery disease
CSANZ	Cardiac Society of Australia and New Zealand
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CVD	Cardiovascular disease
DoHA	Department of Health and Ageing
GP	General Practitioner
GPRN	General Practice Research Network
HCN	Health Communication Network
HDL-C	High density lipoprotein cholesterol
HMG-CoA	3-hydroxy-3-methyl-glutaryl-co-enzyme A
LDL-C	Low density lipoprotein cholesterol
NHFA	National Heart Foundation of Australia
NVDPA	National Vascular and Disease Prevention Alliance
PBS	Pharmaceutical Benefits Scheme
TC	Total cholesterol
VLDL	Very low density lipoprotein

Executive Summary

Australia's Cholesterol Crossroads report outlines the results of Australia's largest ever study of cholesterol levels (otherwise known as lipid levels in this report to denote different forms of cholesterol). Data were derived from almost 200,000 patients managed in primary care by nearly 600 general practitioners from January 2004 to June 2009.

The importance of undertaking large-scale studies to monitor trends in lipid levels should not be underestimated. Dyslipidaemia (involving elevated total and low density lipoprotein levels and reduced high density lipoprotein levels) is a serious risk factor for coronary artery disease (CAD) which remains, in the midst of an overall reduction in death rates from cardiovascular disease (CVD), the nation's single largest killer. For example, CVD accounted for nearly 23,000 deaths in Australia (16.5% of all deaths) in 2007 (AIHW 2010). Despite some success in reducing the burden of CAD and the other components of atherosclerotic (i.e. cholesterol) - related CVD (namely cerebrovascular and peripheral arterial disease),

the reported number of Australians affected by dyslipidaemia (1 out of every 2 adults) has remained constant for the past few decades (AIHW 2010).

In view of the large burden of CVD in Australia and significant investment in lipid-modulating strategies, we sought to understand the contemporary pattern of lipid levels and management from a primary care perspective during a five and half year period. This report provides important insights into the optimal monitoring and management of dyslipidaemia in Australia and offers key clinical and public health messages for health professionals and the broader community.

Key findings

- Mean total cholesterol (TC) levels modestly declined in both men and women during 2004 to 2008.
- A small upward inflection in lipid levels was recorded in mid-2009, the significance of which is unclear at this point in time.
- Women had consistently higher lipid levels than men, even when adjusted for age.
- Overall, TC levels declined by 0.12 and 0.22 mmol/L in men and women, respectively.
- Despite modest improvements in lipid profiles overall, in mid-2009, 36% and 56% of men and 38% and 54% of women had sub-optimal TC and low density lipoprotein cholesterol (LDL-C) levels, respectively.
- There were limited differences in observed trends in lipid profiles according to region (urban vs. rural) and socio-economic status (low vs. high income areas).
- There were key differentials in lipid profiles according to location within Australia which were highest in the state of Tasmania (mean TC level of 5.41 mmol/L) and lowest in the state of Queensland (mean TC level of 5.05 mmol/L).
- The greatest improvements in lipid profiles were generally seen after five contiguous annual GP visits.
- Lipid-modulating treatment patterns over five or more contiguous GP visits were associated with significantly reduced TC levels (from 5.16 to 4.56 mmol/L) compared to those not prescribed such therapy (from 5.28 to 5.20 mmol/L).
- Despite the potential impact of lipid-modulating therapy combined with regular GP surveillance and management (particularly when active lifestyle modification is included) to address a high number of adults with dyslipidaemia, the "treatment gap" remained obvious relative to ideal targets.

Conclusions and recommendations

Overall, these data provide “food for thought” for continuing to improve lipid levels in Australia in order to subsequently reduce highly preventable (and often fatal) events caused by CAD and other forms of atherosclerotic CVD. Key recommendations to ultimately optimise lipid levels and attenuate the atherogenic process underlying many preventable events include:

For individuals

-  Adopt a healthy lifestyle approach for primary and secondary prevention of CVD in order to achieve desirable lipid target levels, including cessation of smoking, dietary modifications, physical exercise and weight control.
-  Know your lipid fraction targets and concentrate on reaching these (primary or secondary) levels or better.
-  Have regular lipid tests and attain a copy of the results for ongoing comparison and monitoring.
-  Understand what cholesterol is and the impact that elevated TC and LDL-C can have for your heart health.
-  Remember to take medications regularly (if prescribed) to help optimise your lipid profile.
-  Work with your GP to develop a long-term plan to treat and monitor your lipid levels in order to reach and maintain your individual, ideal targets.

For health professionals:

-  Utilise current guideline recommendations of an absolute risk approach to identify who may benefit most from intervention.
-  Initiate cost-effective risk factor management strategies.
-  Choose effective lipid-modulating medications and consider dose titrations and/or combination therapy in order to reach targets, particularly more stringent targets in higher risk individuals.
-  Acknowledge that there may be variations in the response and preference to therapy and a change to one of a number of lipid-modulating agents may be required to help optimise lipid levels and minimise side effects.
-  Emphasise the need for a long-term strategy to provide continuity of care in managing sub-optimal lipid profiles, including the development of an agreed action plan with your patient.

For health care policy and planning:

-  Develop educational campaigns for school children and the broader public that provide an understanding of the different types of lipids and the role these play in preventable forms of CVD.
-  Introduce wider policies that promote heart health in the community through healthy lifestyle approaches.
-  Consider the need to more closely monitor the overall impact of costly treatments designed to optimise lipid levels and associated risk factors.

Introduction

Cholesterol is an important risk factor for heart disease

Cholesterol is a lipid (fat) which is produced by the liver. It is essential for normal body functioning, performing a role in the formation of cell membranes (it exists in the outer layer of every cell in our body), the production of bile (to aid digestion) and certain hormones, and in converting light from the sun to vitamin D. Cholesterol forms part of a lipoprotein (containing both lipid and protein) and is carried in the blood via these transporter molecules. Triglycerides are another type of lipid found in the core of lipoprotein particles, as opposed to cholesterol which is a surface lipid.

Two important cholesterol-rich lipoproteins include low and high density lipoproteins (LDL-C and HDL-C, respectively). There are also other lipoproteins which contain more triglyceride than cholesterol such as very low density lipoproteins (VLDL) and a less common lipoprotein, chylomicrons.

Increasing total cholesterol (TC) levels are associated with a steady increase in the risk of developing coronary artery disease (CAD) and cerebrovascular disease. Unfortunately the most common manifestations of these diseases, acute myocardial infarction and stroke, are often sudden and fatal (LaRosa et al. 1990). There is no threshold below which cholesterol ceases to be a risk factor for CAD. Reduced HDL-C and elevated triglycerides are also accepted as major risk factors which contribute to CAD.

It follows that the lower the LDL-C level, the lower the risk of CAD and other forms of cardiovascular disease (CVD) caused by atherosclerosis (including stroke). Several randomised controlled trials have established that for every 1 mmol/L reduction in LDL-C, risk of major vascular events is decreased by 21% (Baigent et al. 2010). Furthermore, the relative risk reduction of having a primary or secondary CVD event is similarly reduced and the decrease in CVD risk is independent of the baseline level of LDL-C.

LDL-cholesterol

LDL-C is often referred to as “bad” cholesterol. LDL-C particles carry cholesterol from the liver to cells in the body; if too much is carried for the cells to use, there can be a harmful build-up of LDL-C particles which can increase the risk of CAD and stroke (**Figure 1**). CAD is the most common form of CVD in Australia and remains the most common cause of death and disability (AIHW 2010). LDL-C particles carry about 70% of blood cholesterol. Therefore, the amount of TC largely reflects the concentration of LDL-C. Small, dense LDL-C particles

are most atherogenic (likely to form atherosclerotic plaques) and qualify as the most dangerous.

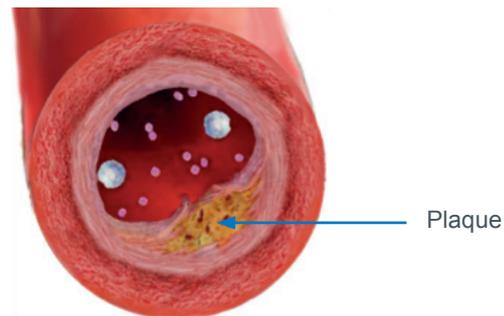


Figure 1. Excess cholesterol can cause fatty deposits (plaques) to form in arteries

HDL-cholesterol

HDL-C is often referred to as “good” cholesterol that helps to prevent atherosclerosis. In contrast to LDL-C particles, HDL-C particles take the excess cholesterol away from the tissues and deliver this back to the liver and to other lipoprotein particles (e.g. VLDL). Cholesterol is either broken down in the liver or expelled from the body as waste. Unlike LDL-C levels, therefore, it is desirable to have a higher HDL-C level.

Triglyceride

Triglyceride is a major source of energy that originates either from fats in our food or is made in the body by conversion of other energy sources such as carbohydrates. Calories we consume that are not used immediately by our tissues are converted into triglycerides and stored in fat cells to be released when our body needs energy. Lower levels of triglyceride are therefore more desirable from a “heart health” perspective.

Recommended lipid levels

Primary prevention targets

As shown in **Figure 2**, the Australian Government (AIHW 2010) and their national science agency, the Commonwealth Scientific and Industrial Research Organisation (CSIRO 2010) currently define a cut-off TC level equal to or below 5.5 mmol/L as normal. It has been disclosed however that this value is an “arbitrary definition” and not based on substantiated data (AIHW 2010). A TC level between 5.5 and 6.5 mmol/L is deemed to be associated with a “greatly increased risk” or a “slightly increased risk” of developing CAD, with the severity warning dependant on the advising authority. However, a level of 6.5 mmol/L or more is associated with extremely high risk of developing CAD requiring serious lifestyle changes and in turn, the consideration of drug therapy. There is less information available to define LDL-C primary prevention target levels, but it has been indicated that a level less than 3.0 mmol/L is recommended.

As a comparison, the desirable TC level in the USA is less than 5.13 mmol/L (AHA 2010), and in the UK the optimum level is less than 5.0 mmol/L (DH March 2000). LDL-C targets in the USA are 3.3 and 4.0 mmol/L, depending on the level of risk whilst in the UK, a level of 3.0 mmol/L is considered ideal.

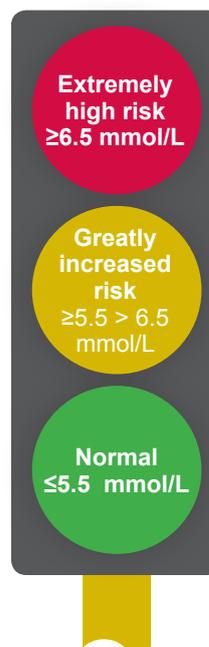


Figure 2. Classification of TC levels

Secondary prevention (treatment) targets

The targets for secondary prevention (i.e. for those individuals at increased risk or who have already developed CAD or experienced a stroke) are naturally lower than for primary prevention. The National Heart Foundation of Australia (NHFA) and the Cardiac Society of Australia and New Zealand (CSANZ) define targets for higher risk patients as a LDL-C below 2.0 mmol/L, a HDL-C above 1.0 mmol/L and triglyceride level below 1.5 mmol/L (Tonkin *et al.* 2005).

In the USA, the target for secondary prevention is a LDL-C less than 2.6 mmol/L with the option of reducing this to below 1.8 mmol/L for very high risk patients. In the UK, the equivalent target LDL-C is below 2.0 mmol/L. Lower LDL-C targets are particularly relevant for patients with diabetes.

Lipid levels in Australia

As indicated previously, dyslipidaemia (i.e. high TC and LDL-C levels and/or low HDL-C levels) is a serious risk factor for CAD that remains, in the midst of an overall reduction in death rates from CVD, the nation's single largest killer, accounting for nearly 23,000 deaths (16.5% of all deaths) in 2007 (AIHW 2010).

As with many fundamental parameters relating to Australia's risk profile, there is a lack of data to inform contemporary policies on the effectiveness of public health initiatives and specific health care strategies to optimise lipid profiles in Australia. Average lipid levels remained relatively static during the period 1980-2000 (AIHW 2010). As shown in **Figure 3**, there was little change in the percentage of males and females with elevated TC (above 5.5 mmol/L) during this time. The national prevalence of elevated TC was 51.2% in 2000; representing 6.5 million Australians, with males having slightly higher levels than females (Dunstan *et al.* 2001).

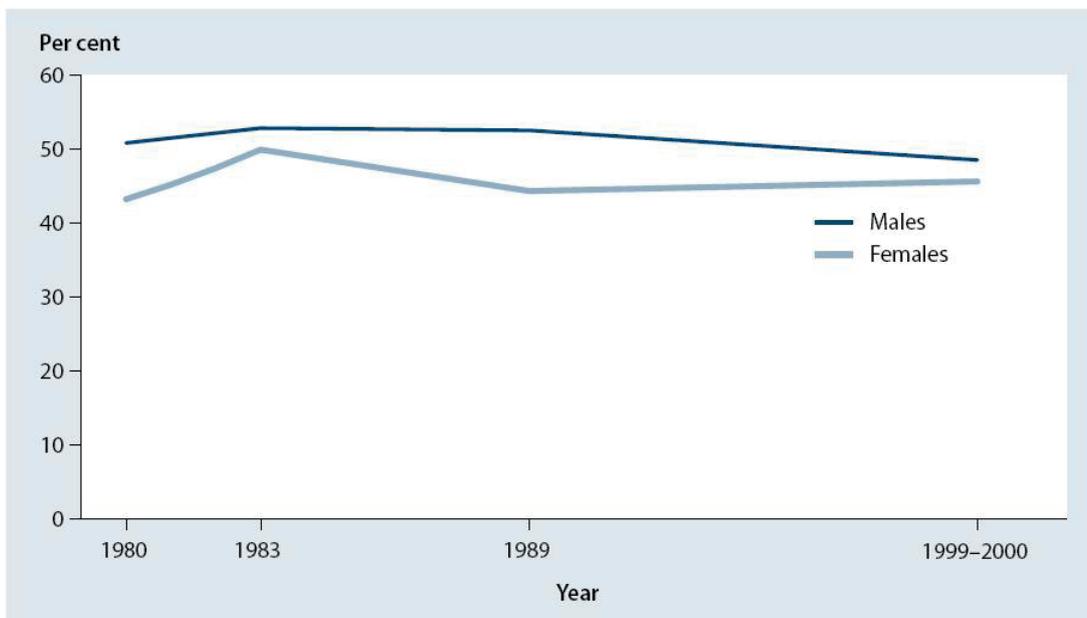


Figure 3. Proportion of Australian adults with high TC from 1980 to 2000

Source: Australian Institute of Health and Welfare 2010. Australia's health 2010 (AIHW 2010).

Figure 4 shows that there was an age-related increase in the prevalence of elevated TC during 1999 to 2000 which peaked at a younger age in males (55-64 years) than females (65-74 years). The prevalence of elevated TC was higher in males aged below 55 years. Above 55 years of age, more females than males had higher TC levels. With an ageing population overall and a greater number of women than men in older age categories, these age-related differences between men and women would certainly have an impact on TC levels in the early 21st Century.

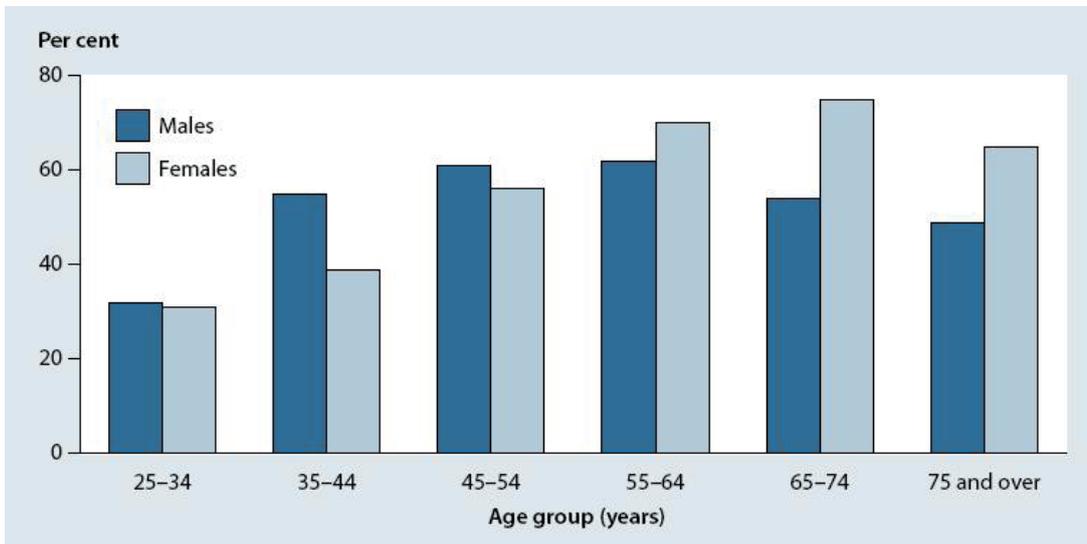


Figure 4. Age and gender prevalence of Australian adults with high TC levels in 1999 to 2000

Source: Australian Institute of Health and Welfare 2010. Australia's health 2010 (AIHW 2010).

As noted, whilst these data highlight the very high prevalence of elevated TC in Australia, the strength of evidence is overwhelmingly limited by out-dated, large-scale population studies (which were more customary in the past) (Bennett & Magnus 1994, Dunstan *et al.* 2001) and the lack of contemporary data. There is a paucity of detailed information on the long-term consequences of not only dyslipidaemia, but all major CVD risk factors, such as hospitalisations and deaths in Australia as well as a lack of data on the long-term trends and benefits of lifestyle factors of Australians on CVD outcomes. Measures of risk factors for CVD are sometimes self-reported, thereby reducing the accuracy and reliability of some surveys. Cross-sectional surveys that assess CVD risk factors and outcome at a single time point also reduce the quality of data that contribute to estimating CVD risk factor prevalence.

Treatment options for impaired lipid levels

There are two essential components for treatment to optimise an individual's lipid profile; non-pharmacological lifestyle advice and pharmacological lipid-modulating treatment.

Lifestyle

Making correct dietary choices can decrease TC, LDL-C and triglyceride levels whilst increasing HDL-C levels. This, in turn, can reduce the risk of developing CVD. Dietary treatment is part of the general lifestyle advice given to lower-risk patients for whom lipid-modulating drug therapy is not justified, but should also be employed as an adjunct in high-risk patients who would derive considerable benefit from lipid-modulating drug therapy (Tonkin *et al.* 2005). Dietary modification includes losing weight by eating less, particularly fat, and reducing energy intake. Exercise is also critical in keeping excess weight off. Decreasing saturated fat intake and replacing them with polyunsaturated or monounsaturated fats and carbohydrates (excluding sugars and syrup) lowers TC, LDL-C and triglyceride levels. Plant sterols and stanols as well as fruit and vegetables may also have a small effect in this regard.

When to treat?

The most common class of lipid-modulating medication that has transformed lipid management is the HMG-CoA reductase inhibitors (more commonly known as "statins"). Statins principally lower LDL-C (i.e. they are lipid-modulating) by increasing LDL-C removal and decreasing LDL-C production (as well as VLDL), but are not as effective in increasing HDL-C and are less effective in lowering triglycerides. Fibrates are another lipid-modulating class of drug which have proved less beneficial in reducing CVD events than statin therapy overall, but may play an important role in lipid management in certain individuals (particularly those intolerant to statin therapy). Fibrates primarily lower triglyceride by reducing its production and improving its clearance whilst also increasing HDL-C, but having nominal effects on LDL-C.

The need for pharmacological treatment, and therefore who may benefit from medication, is determined by the assessment of absolute CVD risk (NVDPA 2009). Individuals with substantially high lipid levels and those deemed to be at high absolute CVD risk may not be offered dietary advice when lipid-modulating treatment can produce a substantially larger decrease in overall lipid levels.

For National Pharmaceutical Benefits Scheme (PBS) eligibility, patients identified as being at very high risk (refer to PBS guidelines for characterisation of high risk) may commence drug therapy with statin or fibrate therapy at any TC or LDL-C level (DoHA, 2009a). Otherwise, lipid profiles combined with patient history are taken into consideration for treatment initiation. Typically, if any of the following criteria are satisfied, treatment may be initiated depending on patient category:

-  TC > 6.5 mmol/L.
-  LDL-C > 5 mmol/L.
-  TC > 5.5 mmol/L and HDL-C < 1.0 mmol/L.
-  Triglyceride > 4 mmol/L.

The cost of treating impaired lipid levels

In the year ending 30 June 2009, lipid-modulating therapies were the top drug group in those listed as representing the highest cost to the Australian government and community collectively, totalling \$1.4 billion (DoHA 2009b). When ranked, three of the top five highest cost drugs were lipid-modulating agents.

Lipid-modulating agents were also the most frequently prescribed drug group totalling 22.4 million prescriptions and representing three out of the top ten highest volume PBS drugs prescribed. It is important to note that these figures do not include non PBS subsidised prescriptions nor is the cost of monitoring lipid levels accounted for.

The vast majority of lipid-modulating prescriptions occur in primary care and lipid management accounts for 3.9 per 100 GP encounters; representing 2.5% of total problems most often managed by GPs. Since 1999, the management of dyslipidaemia has consistently increased as a proportion of GP encounters. In respect to health initiatives, it could therefore be argued that a minimum of \$10 billion has been invested in the treatment of dyslipidaemia in Australia over the past decade without any substantial evidence that such an investment has been subsequently monitored to ensure that maximal gain has been achieved.

Objectives

Given the large burden of CVD in Australia, the enormous investment in lipid-modulating treatments and the paucity of data to determine their practical application and potential efficacy of management, we sought to understand the contemporary pattern of lipid levels and management of dyslipidaemia from a primary care perspective.

This report, therefore, is an analysis of the trends in lipid profiles in patients being managed in primary care in Australia over the period January 2004 to 30 June 2009. It provides important insights into the optimal monitoring and management of elevated cholesterol in Australia and provides underlying clinical and public health messages for health professionals and the broader community.

There are two key aspects which are addressed:

- Part I: Broad trends in lipid levels in patients seeking primary care across Australia.
- Part II: The effect of regular GP reviews and active management on lipid levels across contiguous GP visits (on an annual basis).

Part I

The initial set of analyses uses only patient records of the first lipid measurements within the five and a half years of available data. In instances where patients had multiple lipid measurements taken over this time period, only their first recorded results were used.

Part II

The second set of analyses uses patient records with repeat lipid measurements. For these analyses, records whereby patients had long periods between visits (over 1 year gap) were discarded and not included in this subset of data analyses. In instances where patients had more than one sequence of contiguous visits over the course of the five and a half years of available data, the largest set was taken.

All of the Figures in Part II of this report show average lipid levels over consecutive visits. Since a patient can have anywhere between two to six contiguous annual visits, they do not all provide data at every time point. Thus, a subsequent analysis for each sub-group of patients with 2,3,4,5 or 6 contiguous visits was undertaken to explore changes in lipid levels from baseline to their final visit.

This report summarises the following lipid levels:

-  Total cholesterol (TC)
-  Low-density lipoprotein cholesterol (LDL-C)
-  High-density lipoprotein cholesterol (HDL-C)

Each of these lipids are presented and compared according to a number of sub-groups of interest that include:

-  Sex (men vs. women)
-  Age (middle aged men vs. middle aged women)
-  Region (urban vs. rural dwelling individuals)
-  Treatment (lipid-modulating therapy vs. no lipid-modulating therapy)
-  Socio-economic status (low income vs. high income area)*
-  States and Territories

* Median household income was determined using postcode data of GP clinic locations in order to infer socio-economic status. This is a technique commonly applied to measure the relative prosperity of populations in different geographical locations.

Throughout this report, the percentage of patients not meeting national recommended targets is based on primary prevention ideal cut-offs except in analyses comparing lipid profiles amongst treatment groups (whereby secondary prevention targets are used). This represents a conservative estimate and may thus underestimate the true prevalence of dyslipidaemia.

Methods used to evaluate lipid levels and management of dyslipidaemia

Data source

This report was compiled using data from the research division of the Health Communications Network (HCN), the General Practice Research Network (GPRN).

HCN is the leading provider of clinical and practice management software for Australian General Practitioners (GPs) and Specialists. The most notable of these is the Medical Director software to record and manage patient consultations. Over 18,000 GPs and Specialists, representing 85% of GPs using computerised systems, use HCN's Medical Director (HCN 2010).

The GPRN is a national network of Australian GPs who supply de-identified data (after removal of patient's name, address, telephone number, Medicare number or any other information that could "reasonably" identify the person) on a weekly basis that is used to support research and development in general practice. Of the 14,500 Australian GP users of Medical Director, the GPRN is a random sample of GPs who form part of an ongoing prospective observational cohort of GPs and practices. The GPRN aims to maintain an active sample of 300 GPs with replacement of drop-outs.

From 1 January 1999, almost 1,000 GPs have contributed to prescribing data to create a longitudinal patient-based database containing over 24 million scripts in excess of 25 million encounters of more than 2.7 million patients

(HCN 2010). As such, the GPRN is a leading electronic provider on the use of medicines in general practice. Data are extracted and provided electronically to participating GPs taking part in quality assurance and research activities and are used by government, academic institutions and industry for educational and research initiatives. Available data includes:

-  GP demographics
-  Patient demographics
-  Prescriptions
-  Reason for visit
-  Reason for prescription
-  Pathology and radiology requests
-  Clinical measurements (e.g. height, weight, blood pressure)
-  Other data from Medical Director

Consent

The information provided is collected in accordance with privacy guidelines (refer to HCN's patient privacy policy) (HCN 2010). Upon giving informed consent to use the information for research and statistical purposes, de-identified data are provided to the HCN (hence the de-identification process begins with the GP/Specialist). However, HCN provides an information statement policy for display at the point of care outlining for the consumer the possible uses of the information and data and also the de-identification process. Both the GP/Specialist and consumer retain the right to refuse the use of any personal health information for research and statistical purposes.

The use of these data has been approved by the Alfred Hospital Human Research Ethics Committee.

Limitations

These data have a number of important limitations. They were collected as part of routine clinical practice and not in a systematic and prospective manner for the purpose of this report. For example, although a patient may undergo lipid profiling, the results may not have been entered into the data-set. Furthermore, due to the de-identified nature of data (both from a patient and GP perspective), we are unable to determine if individual patients consulted another GP and in turn were included twice in the data-set. In addition to having to apply a series of conservative assumptions to standardise between individual comparisons (for example we only accepted records where the age and gender of individuals were clearly identifiable), we have no way of verifying the veracity of individual data. Moreover, these data describe a specific primary care patient cohort and caution should be applied when making any extrapolations (i.e. beyond within cohort comparisons) to the wider patient population being managed within primary care in Australia and, indeed, the wider Australian population. Owing to the timing of preparing this report, the data for 2009 are not fully complete and records up to June 2009 are utilised, yet still yield 17,500 patient encounters for this period.

Nevertheless, there are many positive aspects to the utilisation of these data in this report. These include **a)** the standardised manner in which these data were collected, **b)** the reliance on pathology services for lipid levels, **c)** the broadly representative nature of contributing GPs and their patients, and **d)** the overall size of available records (close to 200,000 records over five and a half years). Thus, this report represents an important “barometer” of Australia’s lipid profile and our ability to cost-effectively improve lipid levels to reduce highly preventable hospital admissions and deaths due to CAD and other forms of atherosclerotic disease.

GP characteristics

Figure 5 shows the distribution of GPs and primary care clinics in Australia with the overall number of TC measurements recorded for each State and Territory. Information from a total of 199,331 patients was collected by 596 GPs from 237 primary care clinics across Australia. Overall, there was an average rate of 334 patient records per GP between January 2004 to June 2009.

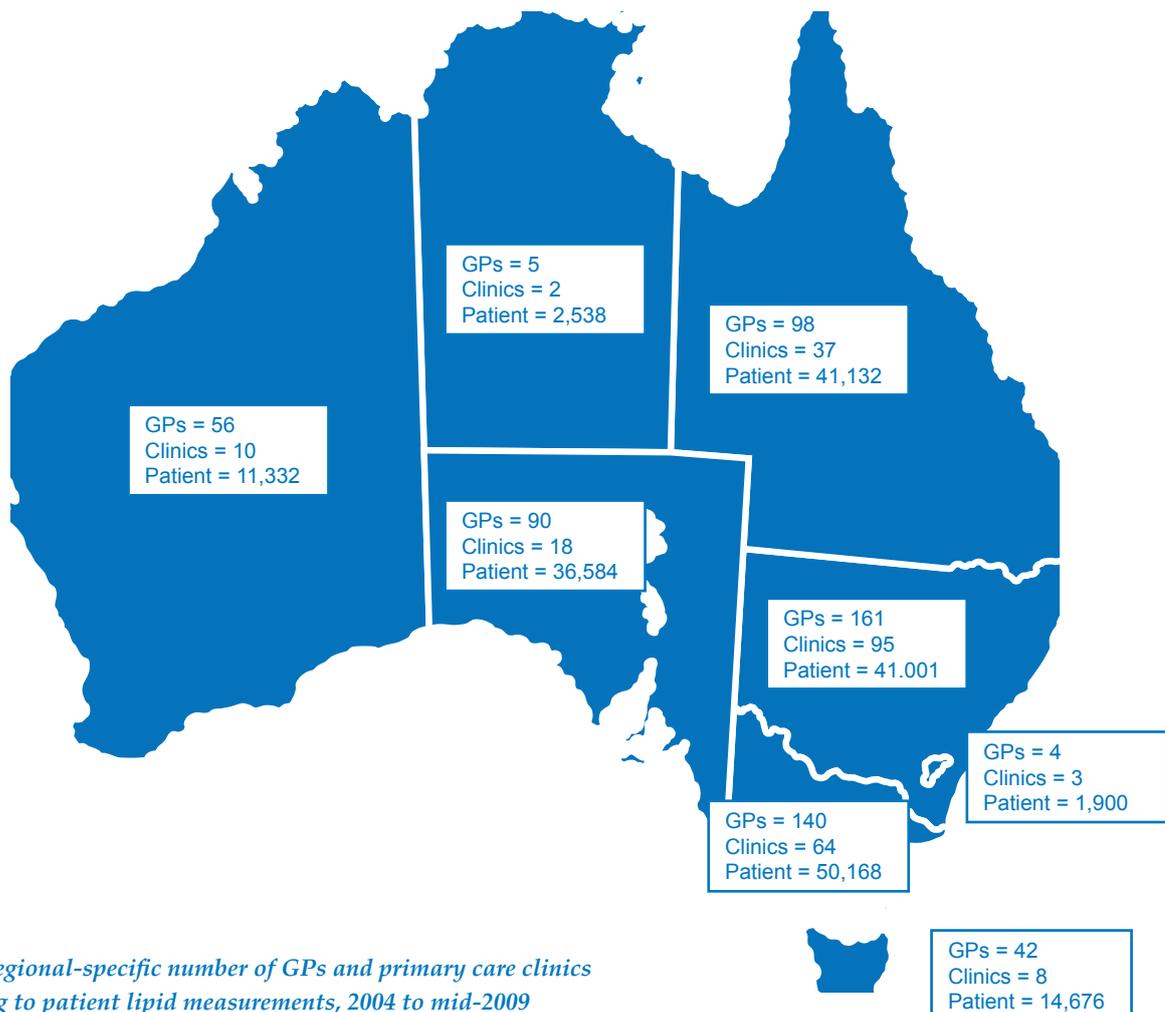


Figure 5. Regional-specific number of GPs and primary care clinics contributing to patient lipid measurements, 2004 to mid-2009

Patient characteristics

Only individuals aged over 18 years and with basic demographic data (age and sex) were included in data analyses for this report. **Table 1** shows the breakdown per year of males and females who had a TC measurement recorded. Overall, there were 47% men and 53% women who visited their GP between January 2004 to mid-2009 and had a TC measurement taken.

Table 1. Number of gender-specific TC records by year, 2004 to mid-2009

Year	Males (n=94,250)	Females (n=105,081)	Total (n=199,331)
2004	14,924	14,774	29,698
2005	12,603	13,503	26,106
2006	18,589	20,778	39,367
2007	20,409	23,867	44,276
2008	19,701	22,689	42,390
2009*	8,024	9,470	17,494

* Available data up until 30 June 2009.

Figure 6 shows the age distribution of the total number of men and women who contributed TC measurements. As can be seen, consistent with the demographic profile of the Australian population, there were more middle-aged adults (45 to 64 years of age) and a greater number of women than men in the majority of age groups. Overall, approximately 40% of men and women were middle-aged.

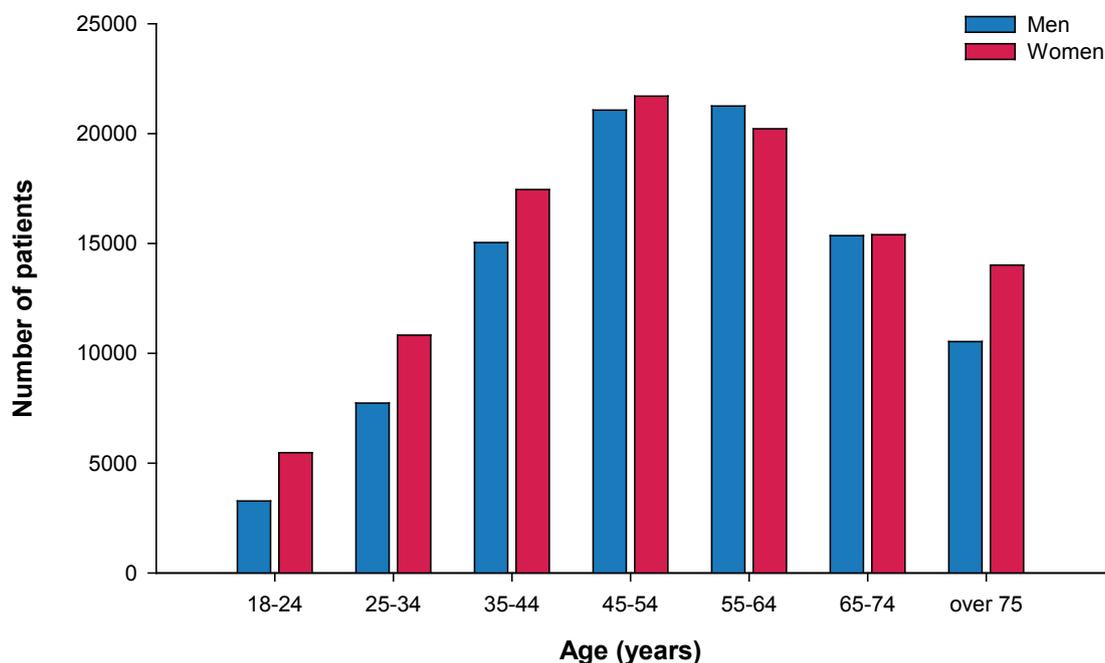


Figure 6. Age and gender-specific records of TC measurements, 2004 to mid-2009

Part I. Lipid profiles for first patient encounters

Overall trend in lipid levels

Based on nearly 200,000 first patient visits between 2004 and mid-2009, TC levels decreased from 5.3 mmol/L in 2004 to just below 5.15 mmol/L in mid-2009, during which time average levels remained below national recommendations of 5.5 mmol/L (**Figure 7**). There was a continuous reduction in TC to 2008 before increasing slightly in mid-2009 to levels similar to the 2007 average. The largest drop in average TC was from 2004 to 2005 (a fall of 0.11 mmol/L).

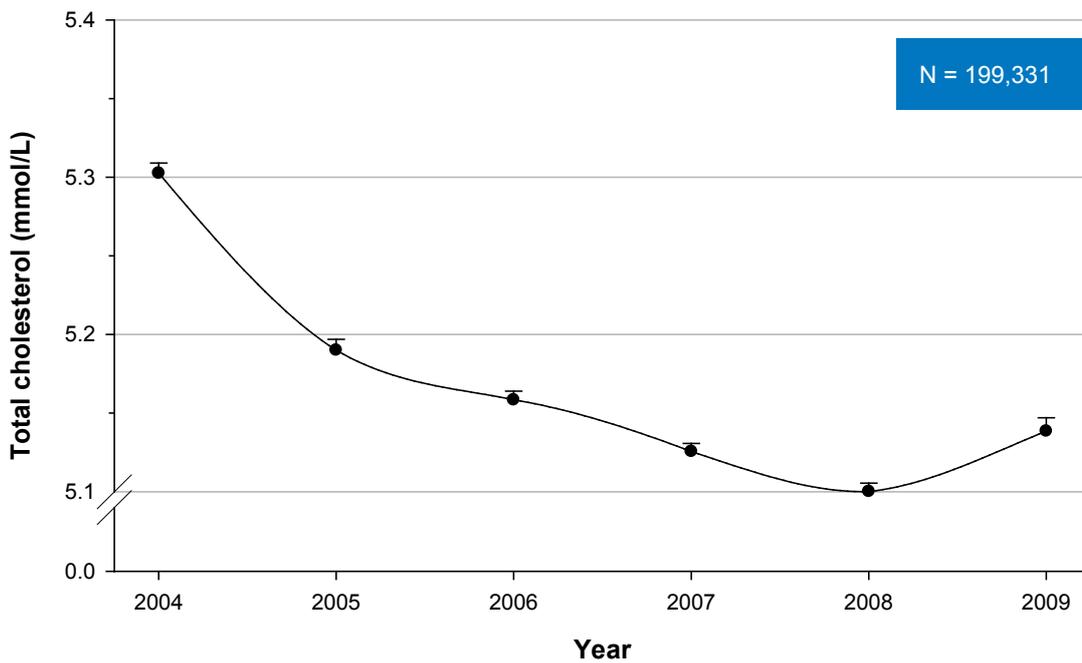


Figure 7. Average TC levels of first patient records, 2004 to mid-2009

Figure 8 shows the percentage of patients with elevated TC above 5.5 mmol/L, the level deemed to be desirable in Australia from a primary prevention perspective (AIHW 2010). In 2004, 43% of patients had elevated TC levels whilst in mid-2009, the percentage dropped to 37%.

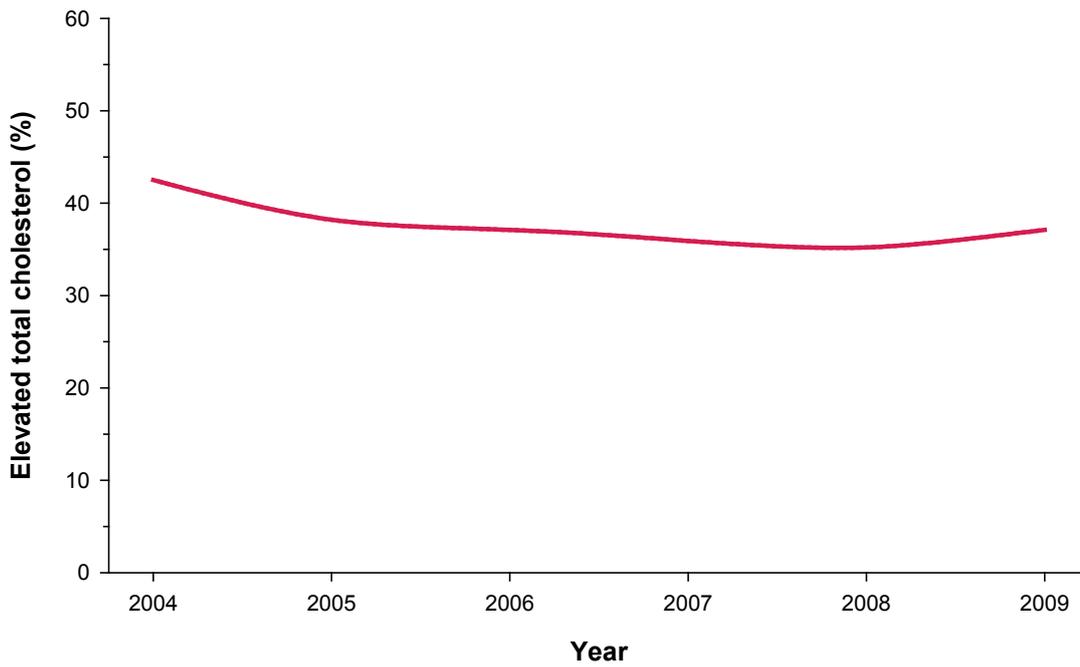


Figure 8. Percentage of patients with elevated TC levels, 2004 to mid-2009

LDL-C was simultaneously recorded in over 112,000 measurements and showed an overall decrease from 2004 to mid-2009 but remained above the ideal target of 3.0 mmol/L during this time (**Figure 9**). On average, LDL-C decreased steadily from 2004 to 2006 before rising slightly in 2007 to 3.12 mmol/L, a level around which it remained to mid-2009.

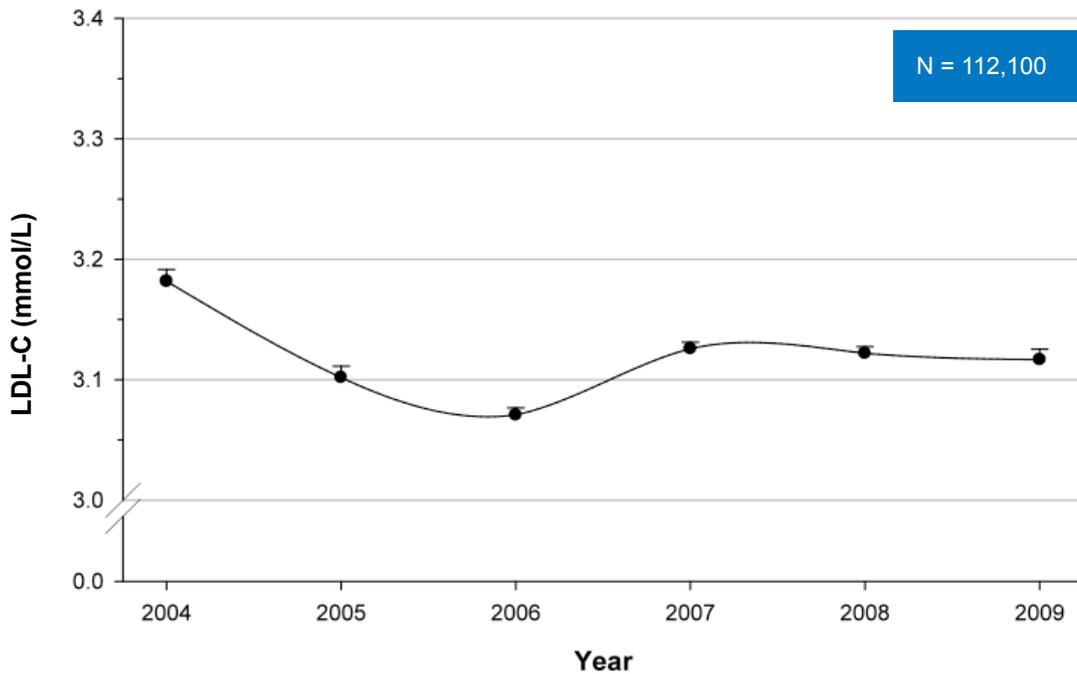


Figure 9. Average LDL-C levels of first patient records, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Figure 10 shows the percentage of patients with elevated LDL-C above 3.0 mmol/L. Between 2004 and mid-2009, the proportion of those with elevated LDL-C remained static at around 55%.

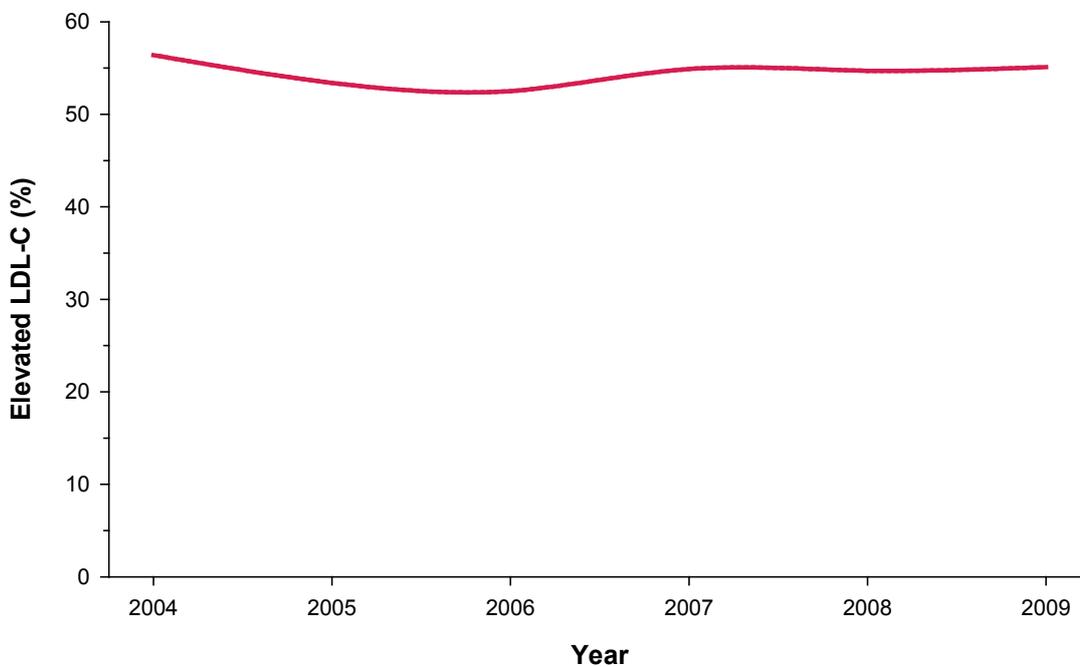


Figure 10. Percentage of patients with elevated LDL-C levels, 2004 to mid-2009

Approximately 145,000 patient measurements indicated that HDL-C levels decreased almost progressively from 2004 to mid-2009 by an average of 0.8 mmol/L, from 1.13 mmol/L to 1.05 mmol/L (**Figure 11**). The average level of HDL-C remained above 1.0 mmol/L, the recommended target for optimal cardio-protection.

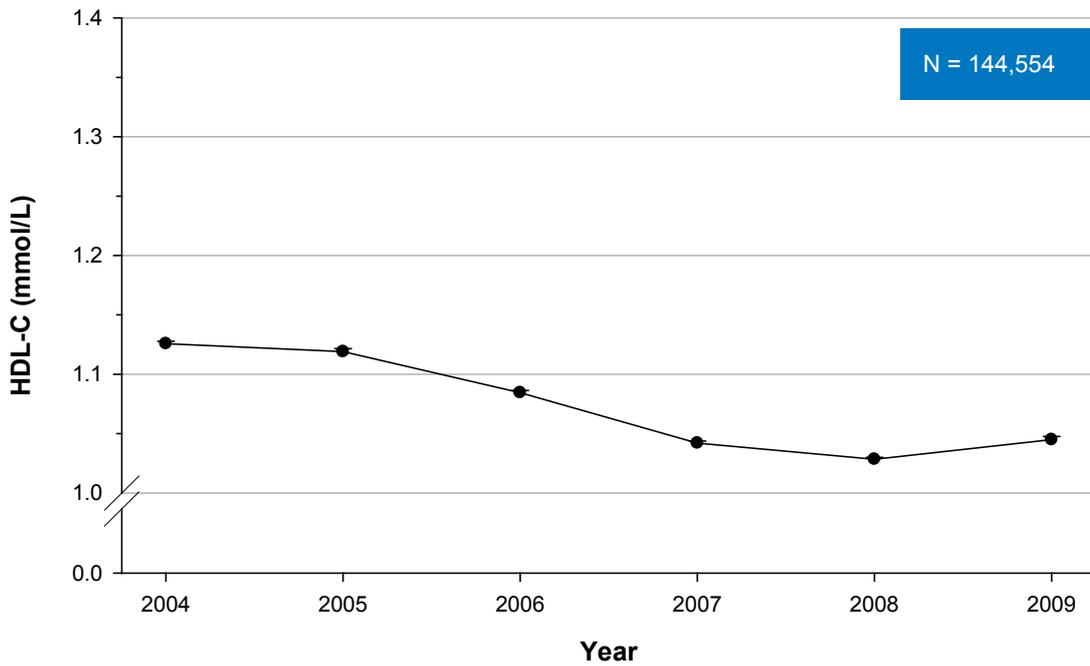


Figure 11. Average HDL-C levels of first patient records, 2004 to mid-2009

Note: Standard error bars are contained within the data points due to the very small variability.

Figure 12 shows the percentage of patients with sub-optimal HDL-C below 1.0 mmol/L. In 2004, 28% of patients had sub-optimal HDL-C levels whilst in mid-2009, the percentage increased to 38%.

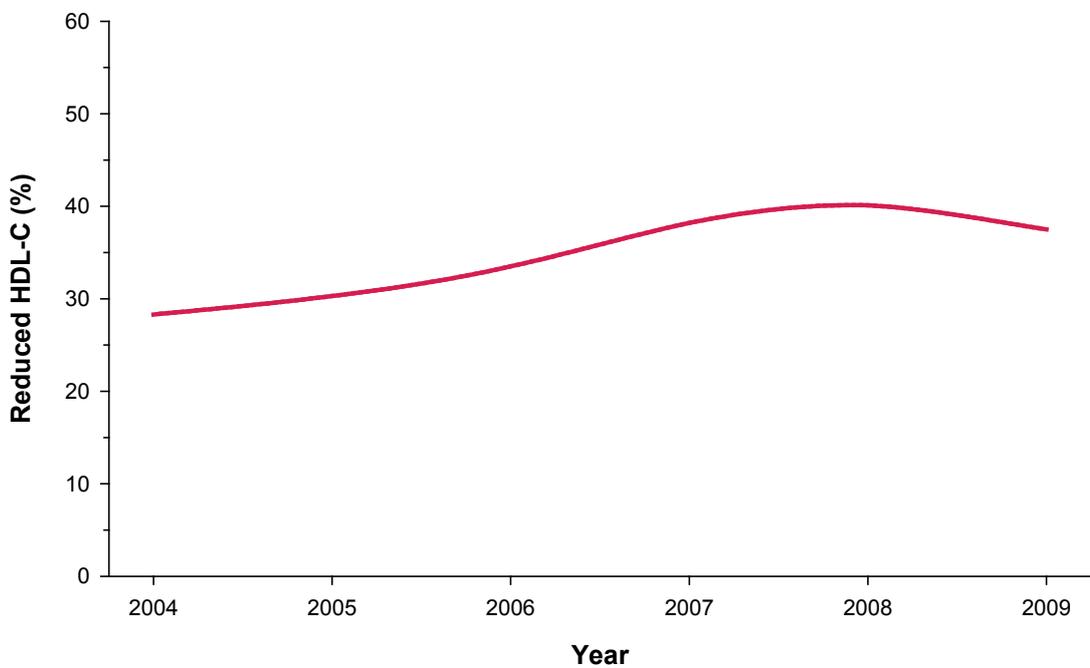


Figure 12. Percentage of patients with reduced HDL-C levels, 2004 to mid-2009

Gender differences in lipid levels

Overall and as shown in **Figure 13**, average TC levels were below the national recommendation of 5.5 mmol/L between 2004 and mid-2009. Women had higher TC levels than men by a relatively consistent margin of at least 0.10 mmol/L. TC decreased from 2004 to mid-2009 in both sexes but the change over this period was nearly double for women (0.22 mmol/L) than men (0.12 mmol/L) considering the higher starting point. Consistent with overall trends, there was a slight increase in TC levels in mid-2009 to 5.19 mmol/L for women and 5.07 mmol/L for men.

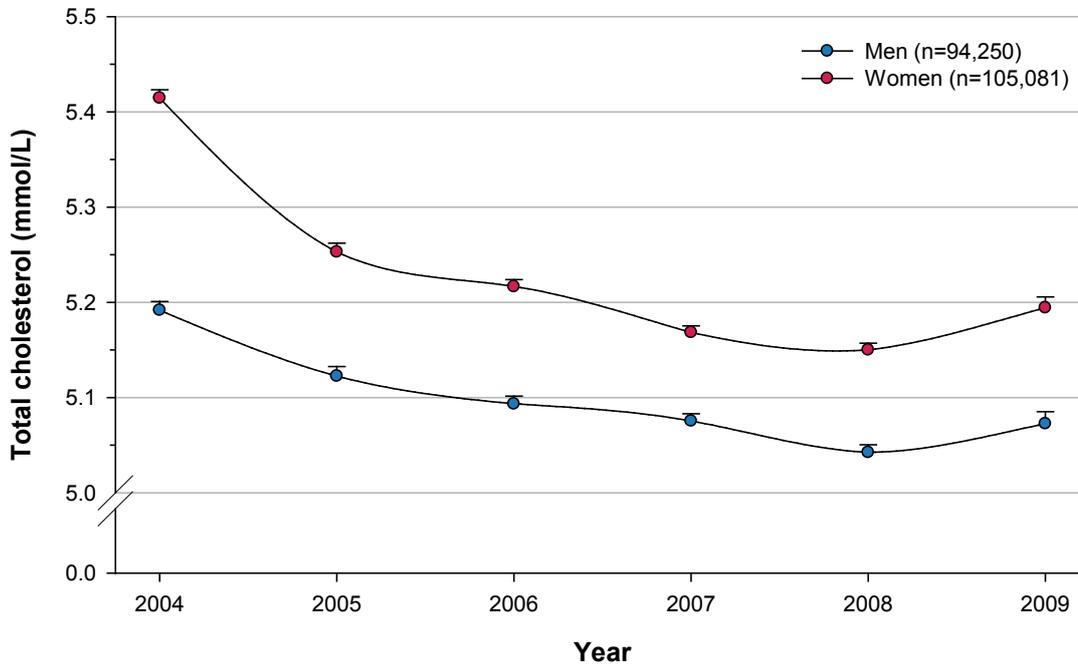


Figure 13. Average TC levels in men and women, 2004 to mid-2009

Note: Standard error bars are contained within the data points due to the very small variability.

As shown in **Figure 14**, the proportion of men and women with elevated TC levels above 5.5 mmol/L decreased from 2004 to mid-2009 and a higher proportion of women than men had increased TC levels. The proportion of women with elevated TC greater than 5.5 mmol/L was 46% in 2004 compared to 39% in mid-2009, whereas for men, the rates were 39% and 36%, respectively.

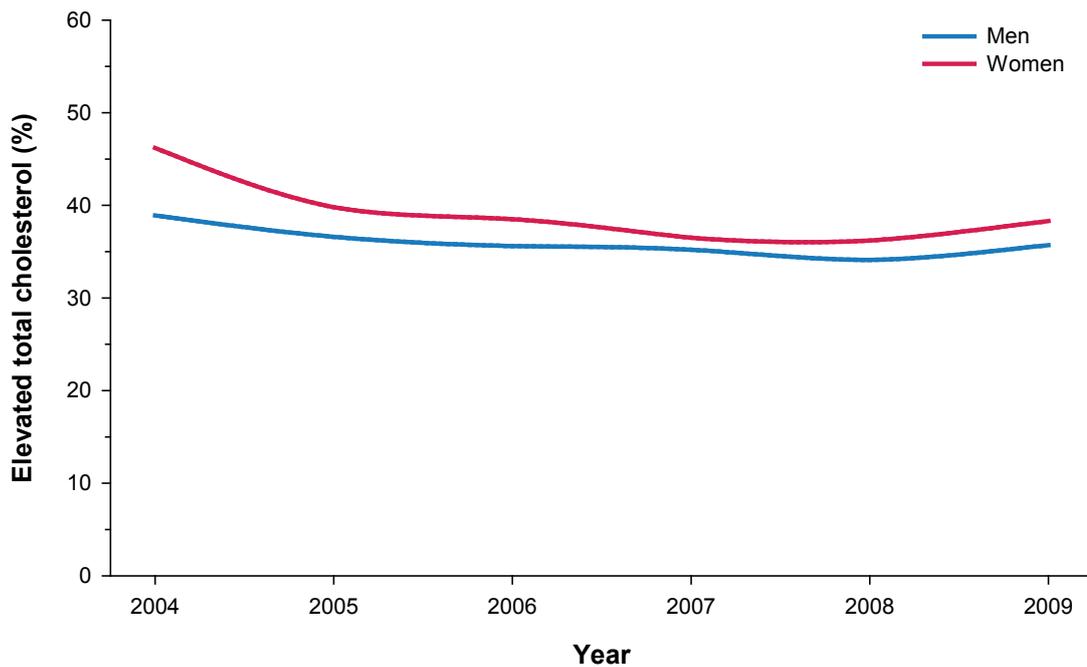


Figure 14. Gender-specific proportion of patients with elevated TC levels, 2004 to mid-2009

Generally, LDL-C levels between 2004 and mid-2009 were broadly the same in men and women, although women had higher average levels in 2004 (**Figure 15**). Aside from a dip in 2006, LDL-C was relatively unchanged from 2004 to mid-2009 in both sexes, remaining close to 3.1 mmol/L and consistently above the ideal target of 3.0 mmol/L.

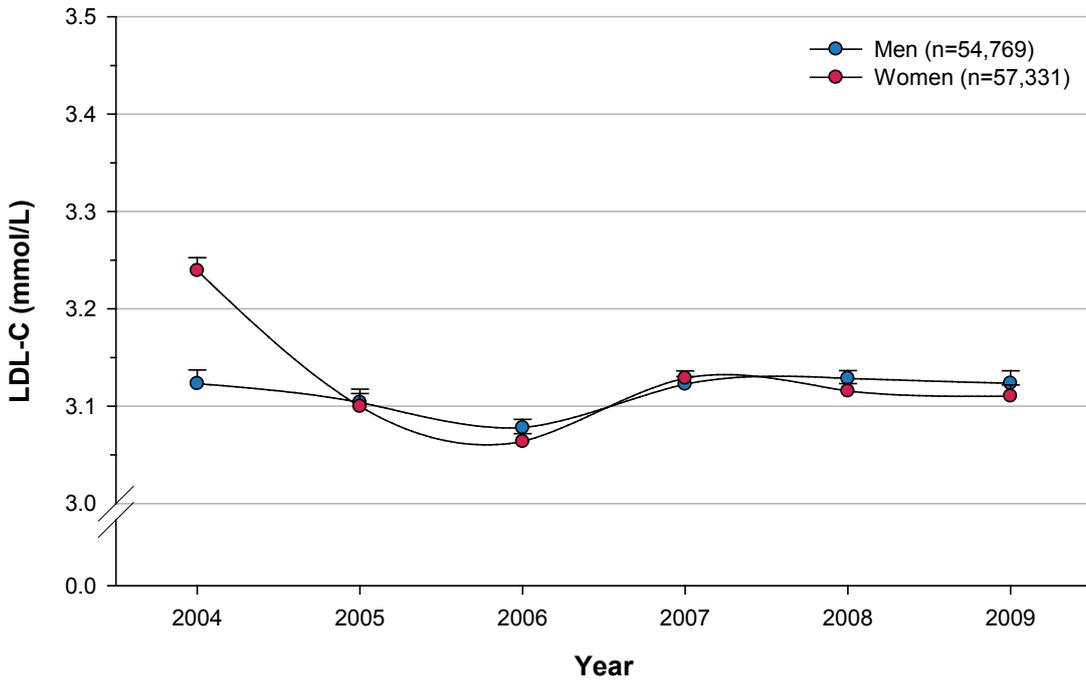


Figure 15. Average LDL-C levels in men and women, 2004 to mid-2009

Note: Standard error bars are contained within the data points due to the very small variability.

Figure 16 shows that the proportion of men and women with elevated LDL-C levels above 3.0 mmol/L was essentially static between 2004 and mid-2009. A higher percentage of women than men had elevated LDL-C in 2004 (58% vs. 55%, respectively) but this was reversed in mid-2009 and a higher percentage of men had elevated LDL-C (56% vs. 54%, respectively). From these data, the proportion of women with elevated LDL-C decreased by 4% in mid-2009 compared to a 1% increase for men.

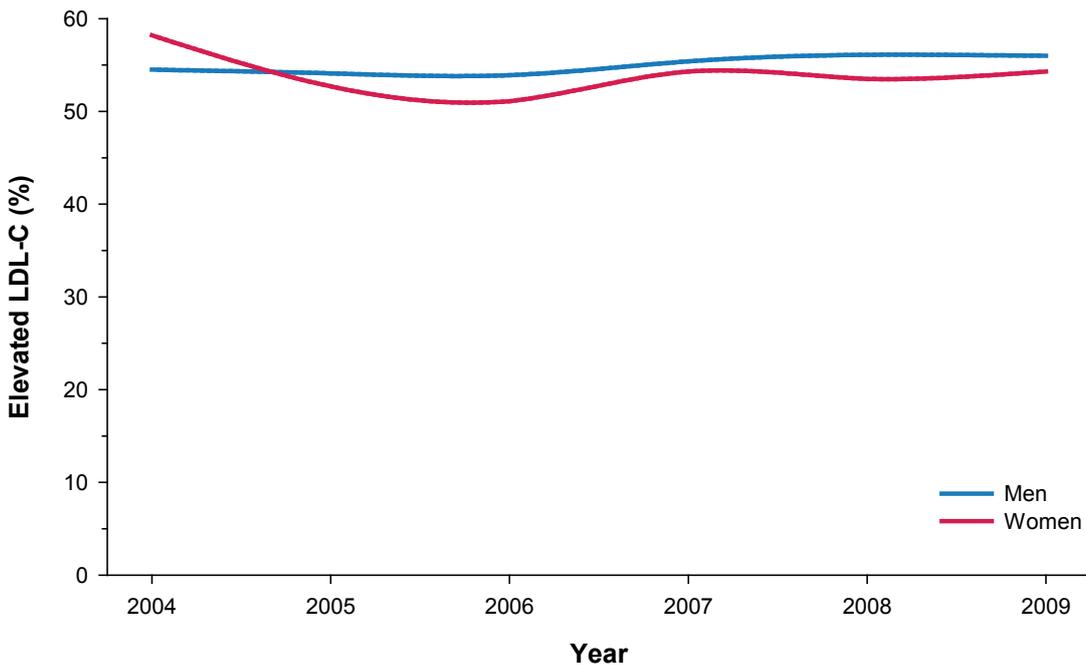


Figure 16. Gender-specific proportion of patients with elevated LDL-C levels, 2004 to mid-2009

As shown in **Figure 17**, women had consistently higher HDL-C levels than men. Initially, this difference was close to 0.09 mmol/L (in 2004/05) but fell to 0.03 mmol/L from 2007 onwards. There was an overall decline in average HDL-C of 0.11 mmol/L for women and 0.05 mmol/L for men from 2004 to mid-2009, yet levels remained above recommended targets of 1.0 mmol/L over this time period. In mid-2009, HDL-C levels slightly increased to a level of 1.06 mmol/L for women and 1.03 mmol/L for men.

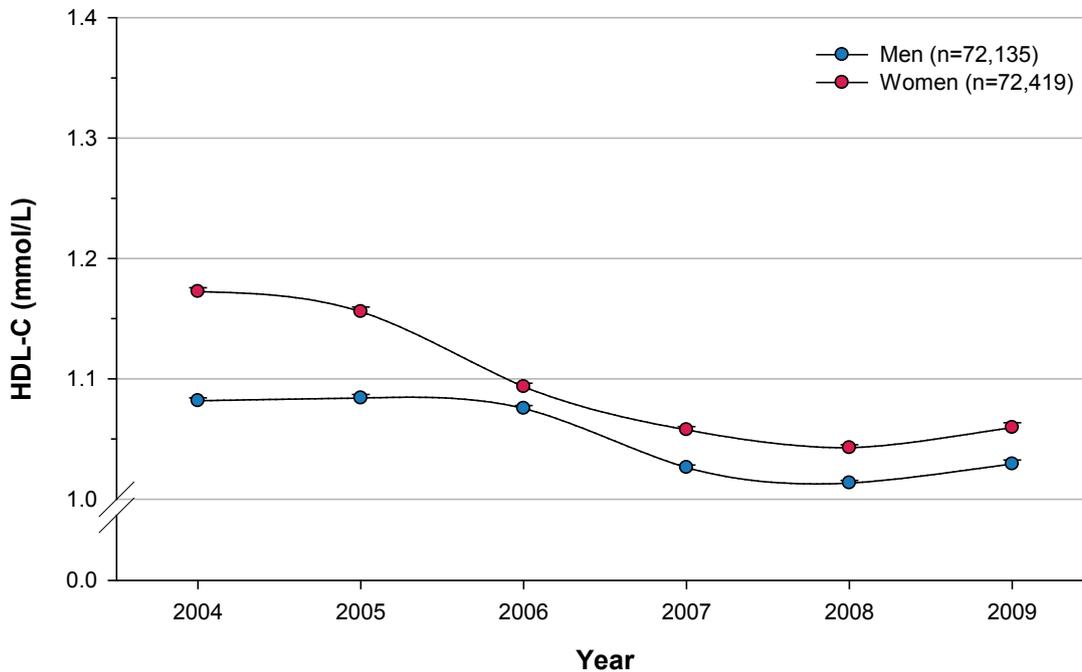


Figure 17. Average HDL-C levels in men and women, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

The proportion of patients with sub-optimal HDL-C below 1.0 mmol/L increased from 2004 to mid-2009 in both men and women (**Figure 18**). Earlier, in 2004, more men (31%) than women (25%) had sub-optimal levels of HDL-C whilst in mid-2009, an equal proportion of men and women (37%) had sub-optimal HDL-C levels.

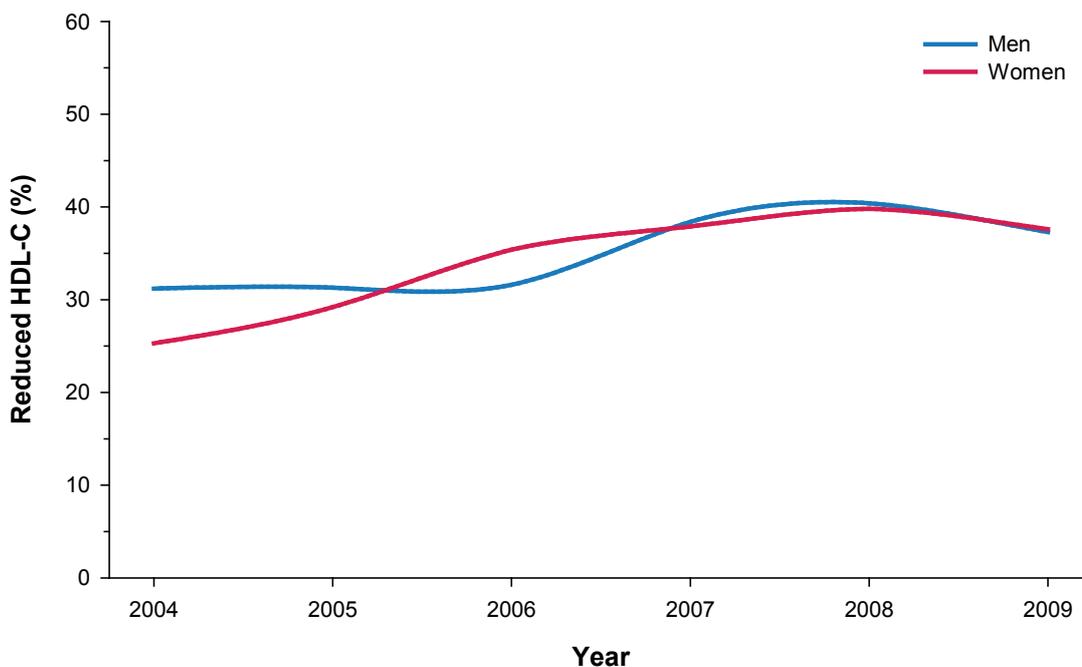


Figure 18. Gender-specific proportion of patients with reduced HDL-C levels, 2004 to mid-2009

Gender differences in lipid levels of middle-aged (45 to 64 years) adults

Consistent with the overall difference between men and women of all ages, **Figure 19** shows middle-aged women had consistently higher TC levels than middle-aged men. In middle-aged women, TC decreased most rapidly from 2004 to 2005 where it dropped to within recommended levels and then remained static at the same level of around 5.5 mmol/L until mid-2009. In middle-aged men, TC levels decreased progressively from 2004 to 2008 before returning to similar levels seen in 2005 by mid-2009, although these levels always remained below national recommendations across this time. Whilst the average TC levels recorded for middle-aged men and women were typically below 5.5 mmol/L, 49% and 42% of middle-aged women and men, respectively, recorded a TC level above 5.5 mmol/L.

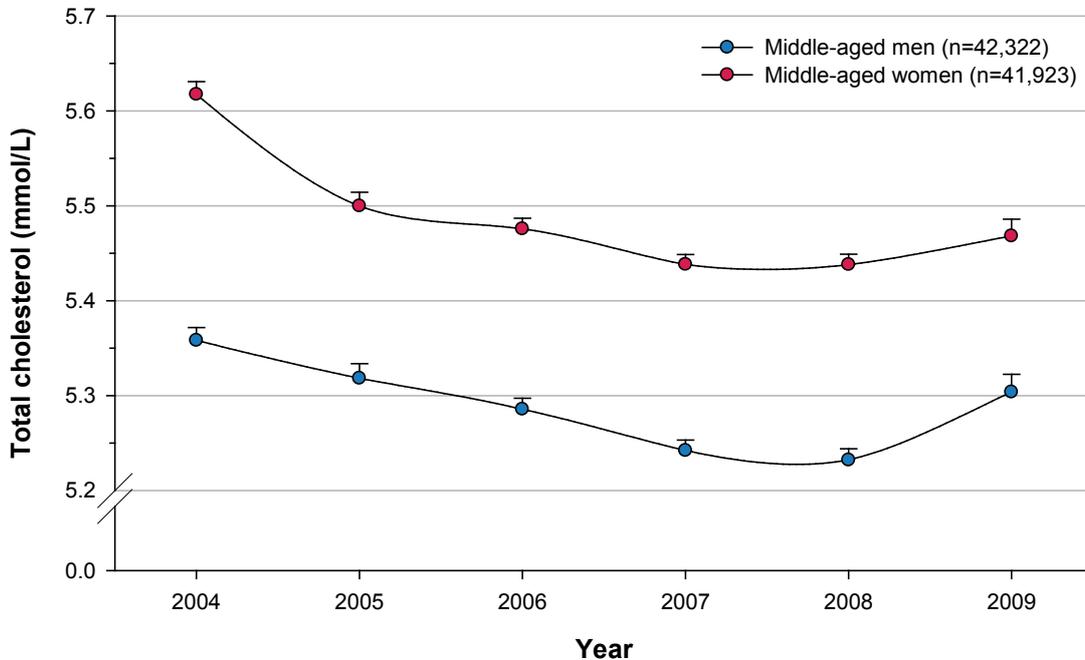


Figure 19. Average TC levels in middle-aged men and women, 2004 to mid-2009

Middle-aged women had consistently higher LDL-C levels than middle-aged men (**Figure 20**). For both sexes, LDL-C levels dipped in 2006 before increasing to approximately 3.3 mmol/L in mid-2009. The difference in LDL-C between middle-aged men and women typically ranged from 0.01 to 0.07 mmol/L over this period. Overall, average LDL-C levels were above primary prevention targets of 3.0 mmol/L for both middle-aged men and women and two out of five had a recorded LDL-C level that exceeded 3.0 mmol/L.

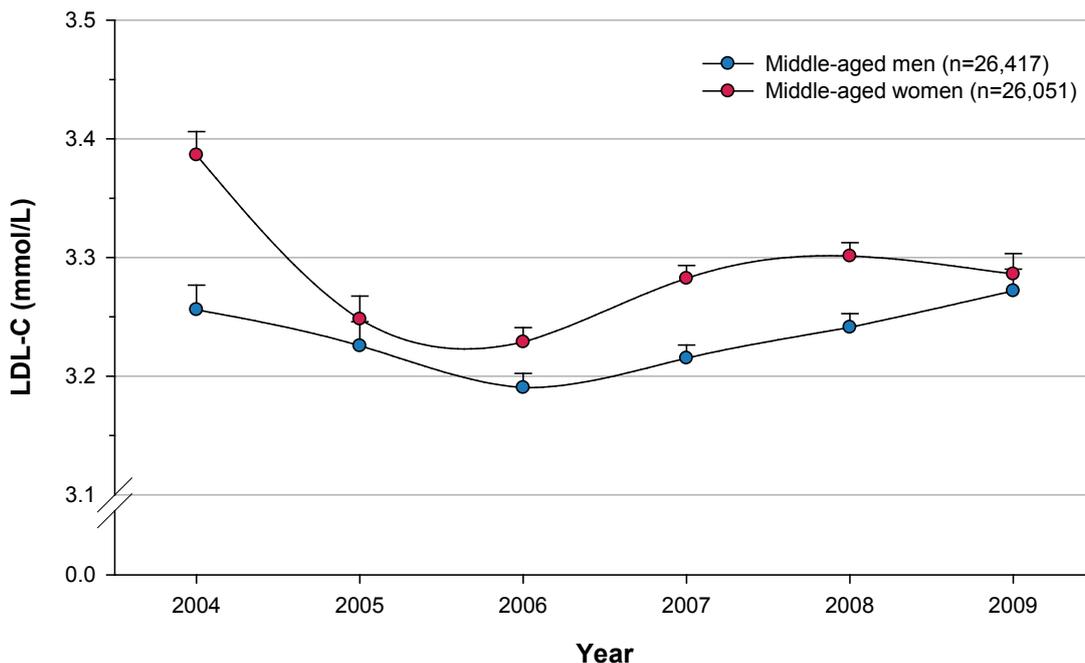


Figure 20. Average LDL-C levels in middle-aged men and women, 2004 to mid-2009

Consistent with observed trends for HDL-C levels in men and women of all ages, **Figure 21** shows that middle-aged women had higher HDL-C levels than middle-aged men, except in 2006 when they were equal (1.12 mmol/L) for both sexes. From 2004 to mid-2009, there was an overall decrease in HDL-C of 0.11 mmol/L for middle-aged women and 0.05 mmol/L for middle-aged men whereupon levels increased to 1.07 mmol/L and 1.05 mmol/L, respectively. Whilst the average HDL-C levels recorded for middle-aged men and women remained above 1 mmol/L, one third had a HDL-C level below 1.0 mmol/L.

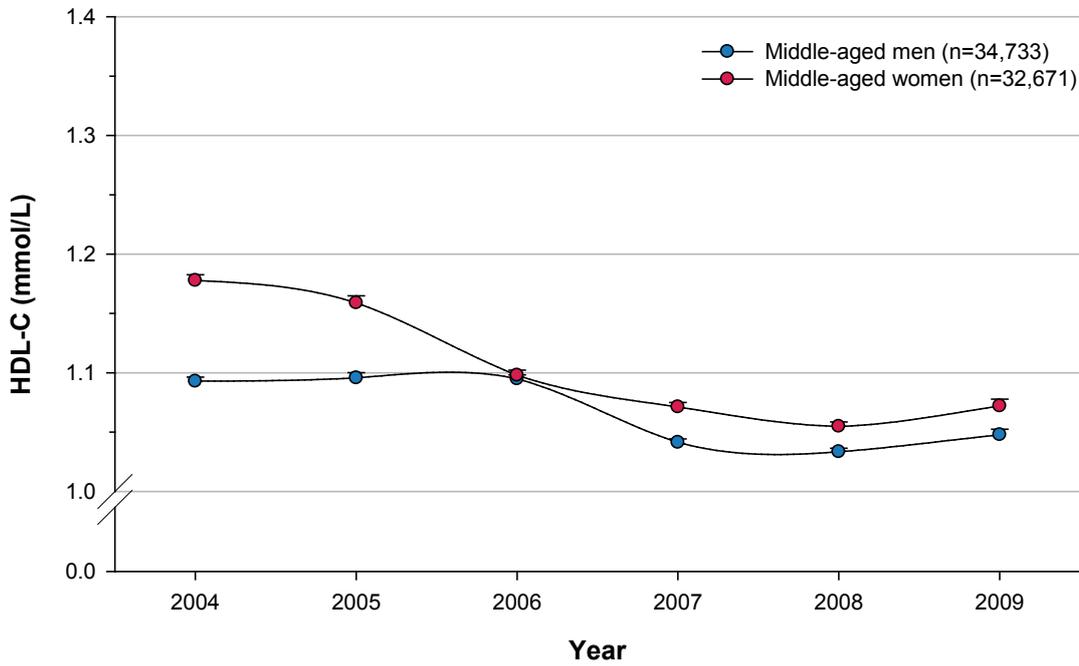


Figure 21. Average HDL-C levels in middle-aged men and women, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Regional differences in lipid profiles

Consistent with the distribution of the Australian population, there were more than twice as many people with a lipid level recorded who were located in an urban region (n=135,289) compared to a rural/remote region (n=63,905). **Figure 22** indicates that TC levels from 2004 to mid-2009 were similar in residents from both locations and, on average, below national recommendations. Further, there were a similar proportion of adults from urban and rural/remote areas who had a TC level above 5.5 mmol/L (37% vs. 39% respectively).

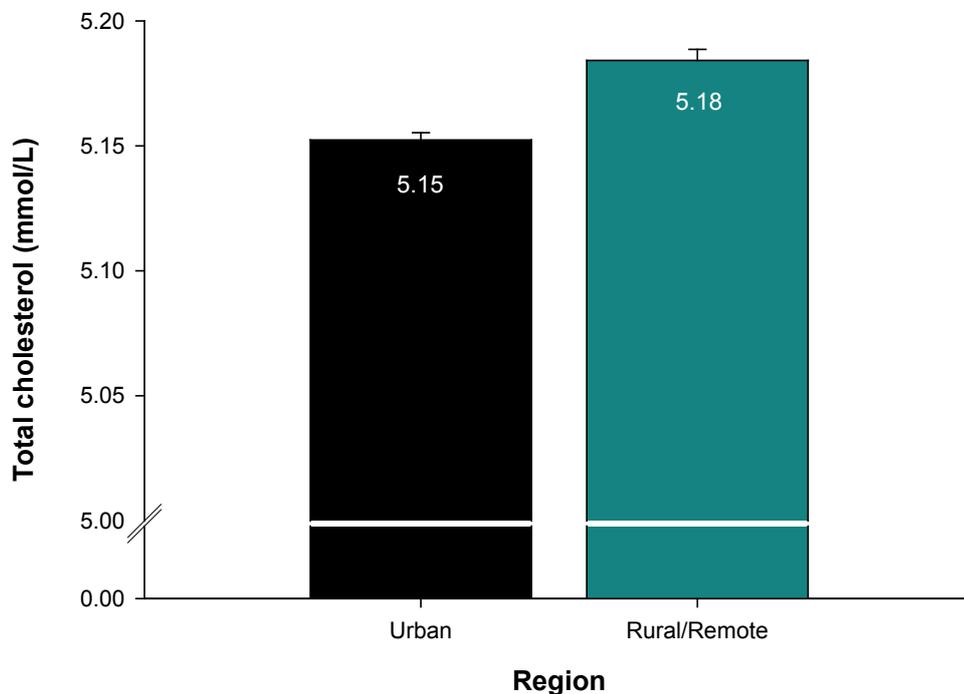


Figure 22. Average TC levels in urban and rural residents, 2004 to mid-2009

Consistent with observed trends in TC between regions, **Figure 23** shows that LDL-C was almost identical and above recommendations of 3.0 mmol/L in patients from urban and rural/remote areas during the study period. Furthermore, the proportion of patients from urban and rural/remote areas with a recorded LDL-C level greater than 3.0 mmol/L was almost identical (55% vs. 54% respectively).

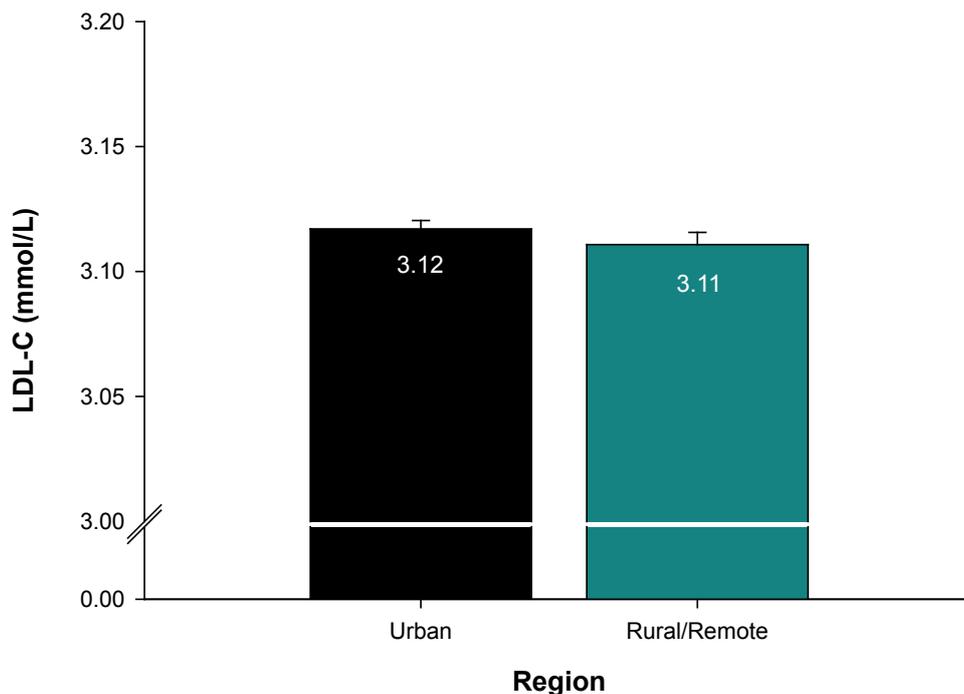


Figure 23. Average LDL-C levels in urban and rural residents, 2004 to mid-2009

Figure 24 shows that HDL-C levels were no different in urban compared to rural/remote residents and, on average, above 1 mmol/L during 2004 to mid-2009. However, the proportion of patients with a HDL-C measurement recorded below 1 mmol/L who were located in an urban region (36%) was marginally higher than for those who were located in a rural/remote region (33%).

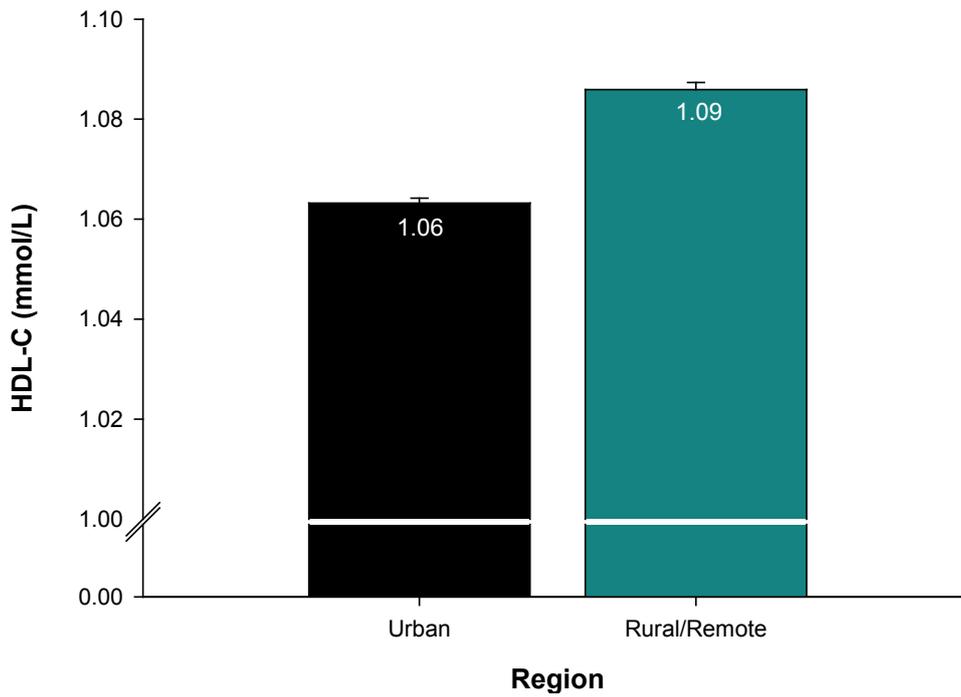


Figure 24. Average HDL-C levels in urban and rural residents, 2004 to mid-2009

Treatment differences in lipid profiles

There were five times more people not on treatment (n=165,863) in contrast to those prescribed lipid-modulating pharmacological therapy (n=33,468) when the first recorded lipid level was analysed. **Figure 25** shows that the TC levels of adults not on lipid-modulating therapy was higher (difference 0.54 mmol/L) than the TC levels of people who were prescribed lipid-modulating therapy. Whilst on average, TC levels were below 5.5 mmol/L for people not on treatment, based on national recommendations, 41% had an elevated TC above 5.5 mmol/L (AIHW 2010). For patients on lipid-modulating treatment, TC levels were on average above the treatment target for higher risk individuals of 4.0 mmol/L and a disappointing 76% were not achieving this level (National Heart Foundation of Australia 2001, Tonkin *et al.* 2005).

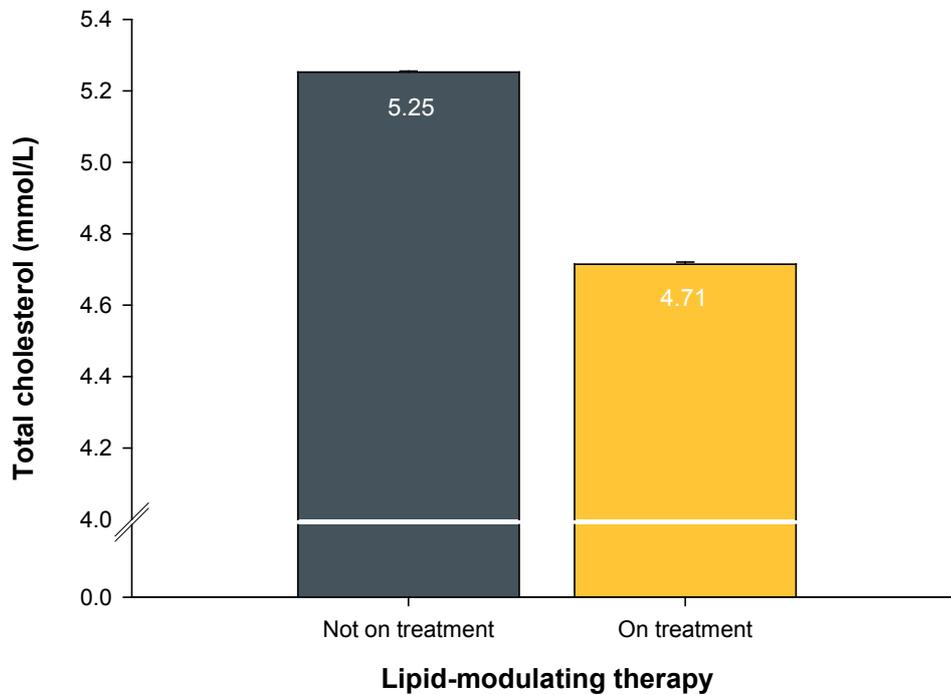


Figure 25. Average TC levels in adults on lipid-modulating therapy and not on treatment, 2004 to mid-2009

Note: Standard error bars are contained within a data point due to the very small variability.

Similar to TC, **Figure 26** shows that the LDL-C levels of those not on treatment was 0.64 mmol/L higher than the LDL-C of patients prescribed lipid-modulating therapy. There were 61% of people not on treatment who had undesirable LDL-C levels above 3.0 mmol/L and 77% of people on lipid-modulating treatment who were not meeting LDL-C recommended treatment targets of 2.0 mmol/L. Overall, average LDL-C levels were above 3.0 mmol/L for people not on treatment and above 2.0 for people on lipid-modulating treatment.

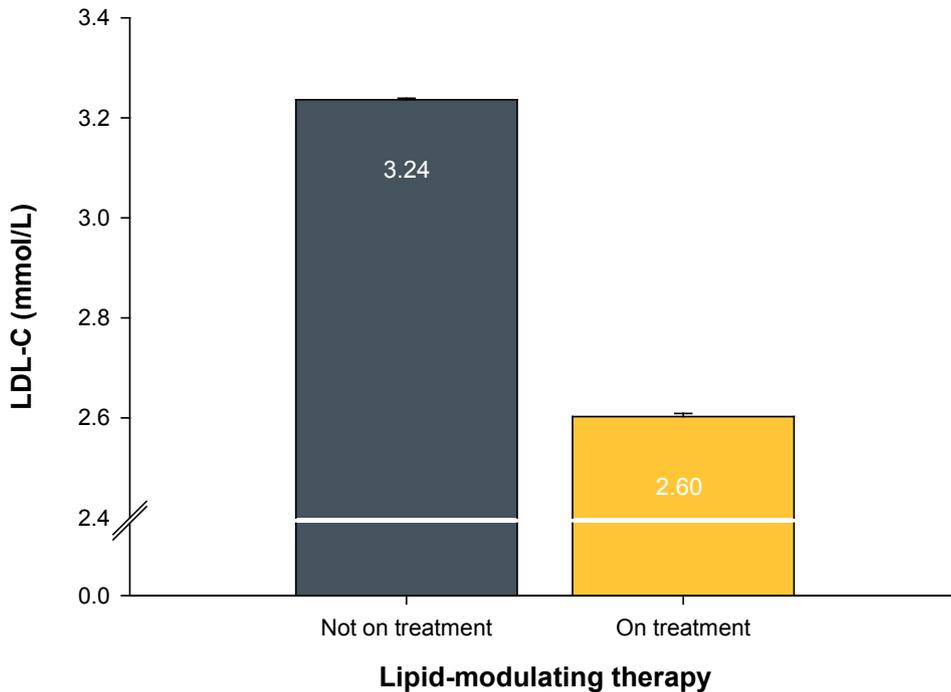


Figure 26. Average LDL-C levels in adults on lipid-modulating therapy and not on treatment, 2004 to mid-2009

Note: Standard error bars are contained within a data point due to the very small variability.

HDL-C levels were higher by 0.06 mmol/L in patients prescribed lipid-modulating therapy compared to those not on treatment (**Figure 27**). Whilst HDL-C levels were on average above the recommended target of 1.0 mmol/L, there were a greater proportion of patients who were not on treatment that did not achieve recommended HDL-C levels compared to those prescribed lipid-modulating therapy (37% vs. 29% respectively).

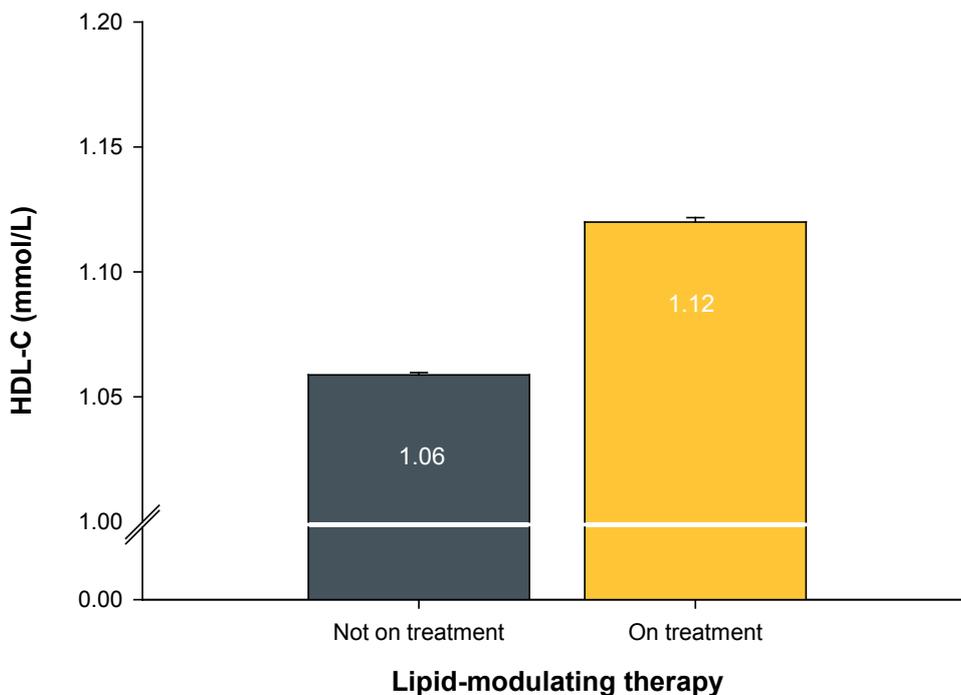


Figure 27. Average HDL-C levels in adults on lipid-modulating therapy and not on treatment, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Socio-economic differences in lipid profiles

Approximately one quarter of patients visited a GP clinic from a high income area (n=42,460) compared to three quarters of visits from a low income area (n=156,871). **Figure 28** shows that TC levels in low income areas were marginally higher (0.07 mmol/L) than in high income areas; on an individual level, this difference would have little or no clinical impact. On average, between 2004 to mid-2009, TC levels were below national recommendations and a similar number of patients from high and low income areas recorded TC levels over 5.5 mmol/L (36% vs. 38% respectively).

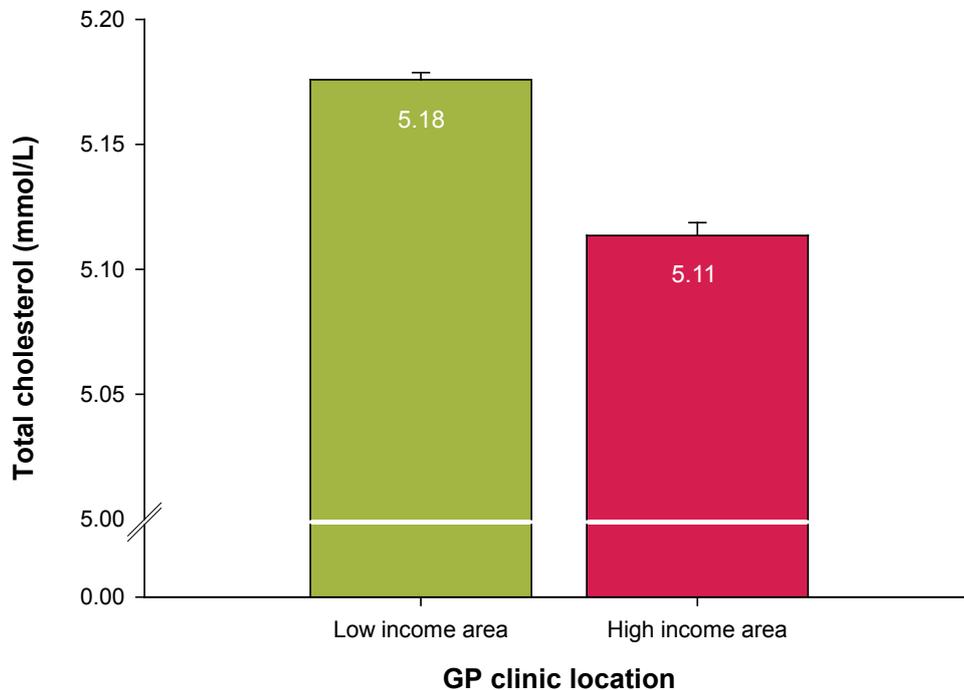


Figure 28. Average TC levels in low compared to high income areas, 2004 to mid-2009

Overall, adults from low income areas had similar LDL-C levels that were above recommended limits compared to those from high income areas, the difference being 0.06 mmol/L (**Figure 29**). A similar proportion of patients from low and high income areas recorded LDL-C levels over 3.0 mmol/L (55% vs. 53% respectively).

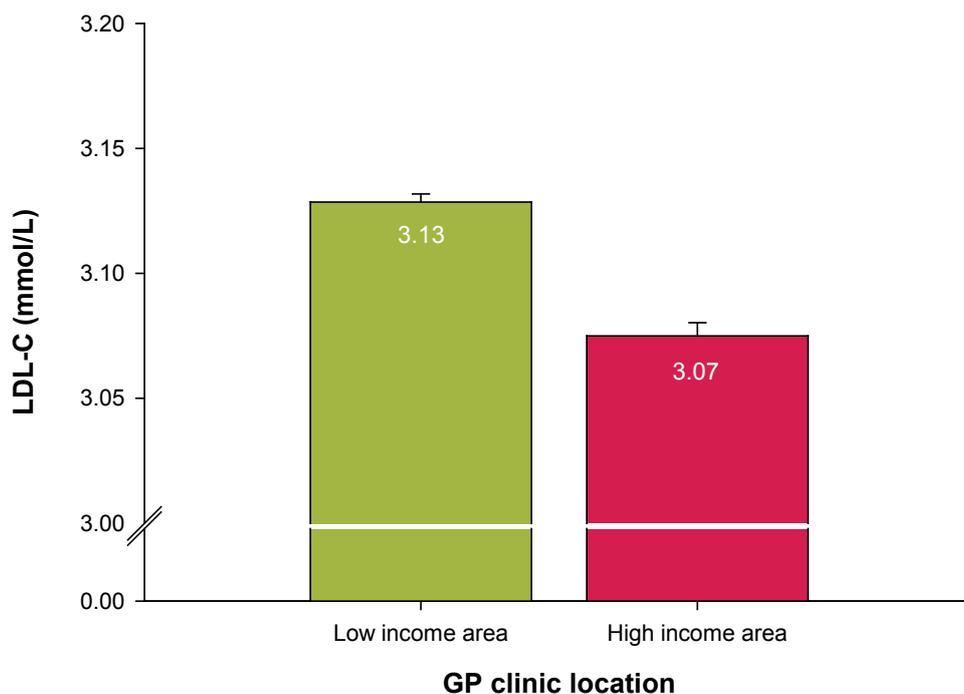


Figure 29. Average LDL-C levels in low compared to high income areas, 2004 to mid-2009

As shown in **Figure 30**, adults from low income areas had slightly higher HDL-C levels (by 0.04 mol/L) than those from high income areas. There were a greater proportion of people from high income areas with lower than recommended HDL-C levels of 1.0 mmol/L compared to people from low income areas (39% vs. 34% respectively).

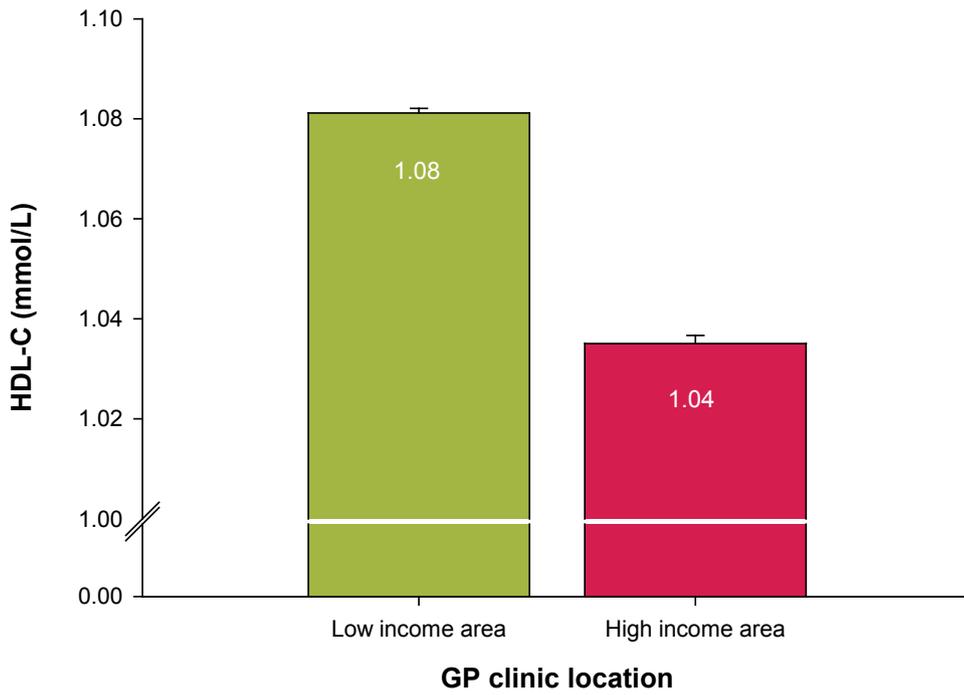


Figure 30. Average HDL-C levels in low compared to high income areas, 2004 to mid-2009

State differences in lipid profiles

Figure 31 shows that TC levels were highest in Tasmania and lowest in Queensland. For all States and Territories, TC levels were between 5.05 and 5.41 mmol/L from 2004 and mid-2009. Whilst recorded average TC levels were below 5.5 mmol/L in each State and Territory, the proportion of adults with a TC that exceeded 5.5 mmol/L was correspondingly highest in Tasmania (47%) and lowest in Queensland (33%).

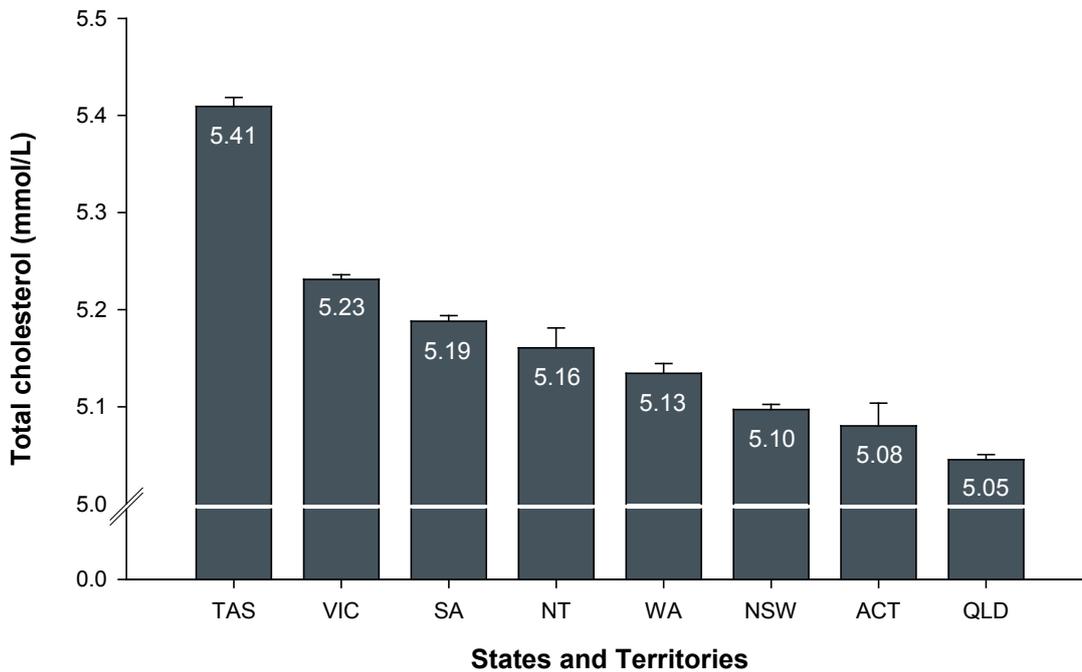


Figure 31. Average TC levels in each Australian State and Territory, 2004 to mid-2009

Overall, **Figure 32** indicates that LDL-C levels were equally highest in Tasmania (highest TC levels overall) and South Australia and lowest in New South Wales, the Northern Territory and Western Australia. In general for all States and Territories, LDL-C levels fluctuated between 3.03 and 3.23 mmol/L between 2004 and mid-2009 but remained equal to or above the primary prevention threshold of 3.0 mmol/L. The proportion of adults with a LDL-C level that exceeded 3.0 mmol/L was highest in South Australia (59%) and lowest in New South Wales (51%).

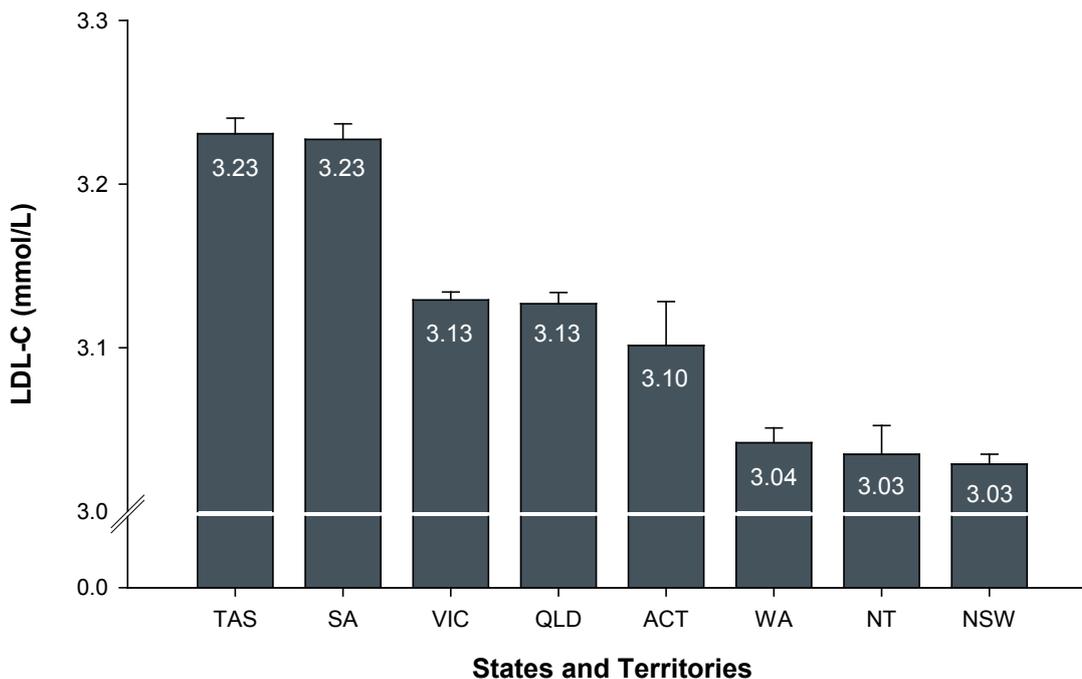


Figure 32. Average LDL-C levels in each Australian State and Territory, 2004 to mid-2009

There was high consistency amongst states and territories in HDL-C levels. The highest levels were recorded in Tasmania (highest also in TC and LDL-C levels) and the lowest, yet still within the optimal range, were recorded in the Northern Territory (**Figure 33**). Correspondingly, the proportion of patients with a recorded HDL-C level below 1 mmol/L was lowest in Tasmania (32%) and highest in the Northern Territory (51%).

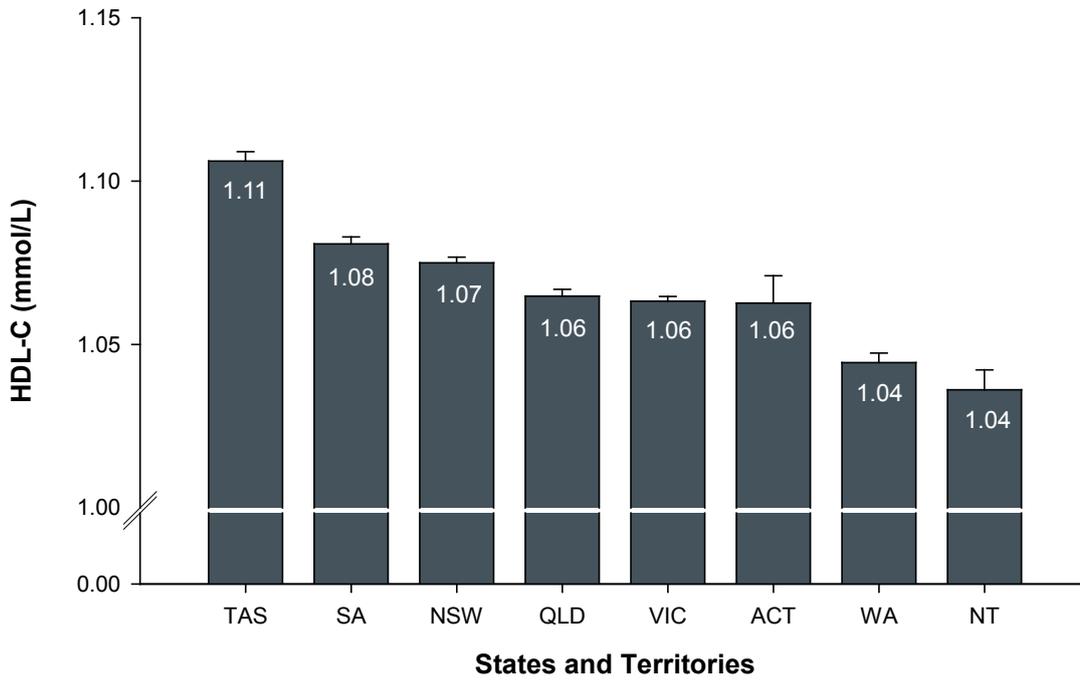


Figure 33. Average HDL-C levels in each Australian state and territory, 2004 to mid-2009

Part II. Frequency of visits and impact on lipid profiles

Trends in lipid levels across contiguous annual visits

Figure 34 shows that TC levels decreased across annual contiguous visits between 2004 and mid-2009 by almost 0.50 mmol/L, from an average of 5.22 mmol/L at baseline to 4.76 mmol/L at the final recorded visit. However, TC levels remained below national recommendations of 5.5 mmol/L over this time.

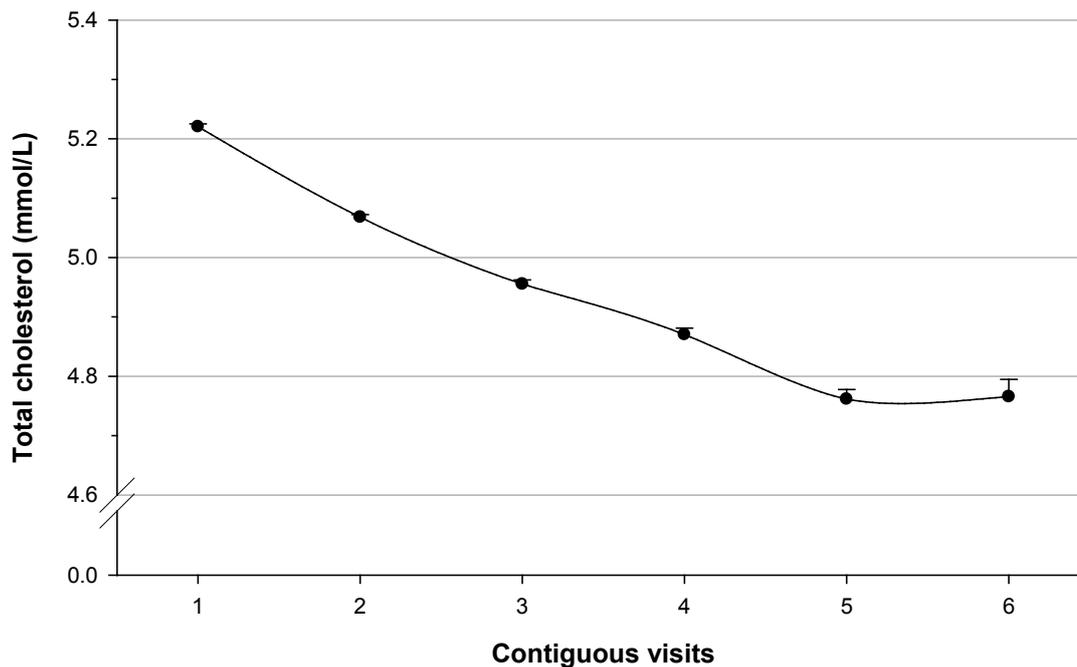


Figure 34. Average TC levels of contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Compared to baseline, there were larger reductions in TC levels in those who had more frequent GP visits (Table 2). TC levels decreased by 0.15 mmol/L after two visits, yet there were progressively greater improvements of up to 0.48 mmol/L when five or six GP visits were undertaken.

Table 2. Change (Δ) in lipid levels (mmol/L) between baseline and contiguous patient visits, 2004 to mid-2009

Lipid	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
TC	-0.15 (n = 60,245)	-0.26 (n = 24,453)	-0.37 (n = 10,516)	-0.48 (n = 4,546)	-0.48 (n = 1,452)
LDL-C	-0.14 (n = 28,348)	-0.23 (n = 9,921)	-0.31 (n = 3,561)	-0.36 (n = 1,344)	-0.31 (n = 611)
HDL-C	-0.02 (n = 39,568)	-0.04 (n = 15,440)	-0.07 (n = 6,800)	-0.09 (n = 3,121)	-0.06 (n = 1,139)

Figure 35 indicates there was an overall decrease of 0.27 mmol/L in repeat annual LDL-C measurements, from an average of 3.14 mmol/L at the first recorded visit to 2.87 at the final recorded visit. LDL-C fell below recommended limits of 3.0 mmol/L after two annual clinic visits.

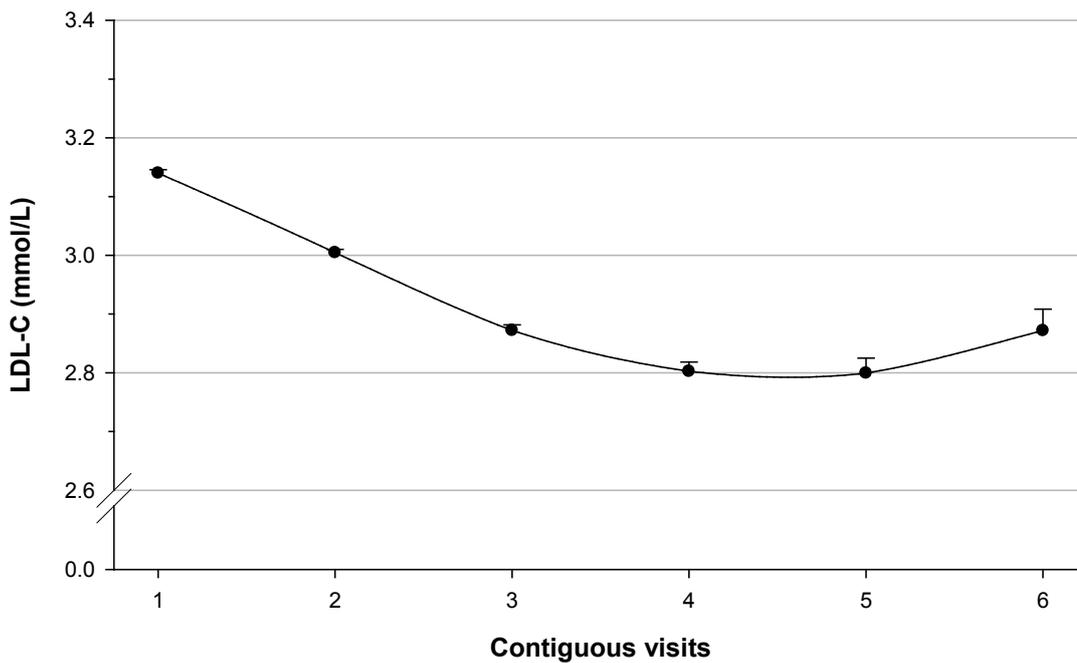


Figure 35. Average LDL-C levels of contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 2 indicates that there were greater reductions in LDL-C levels with more regular visits, ranging from a decrease of 0.14 mmol/L after two GP visits to a decrease of 0.36 mmol/L following five visits. There was no continued improvement seen in LDL-C levels at a sixth visit.

HDL-C levels essentially remained the same at around 1.1 mmol/L and always above national recommendations over annual contiguous visits between 2004 and mid-2009.

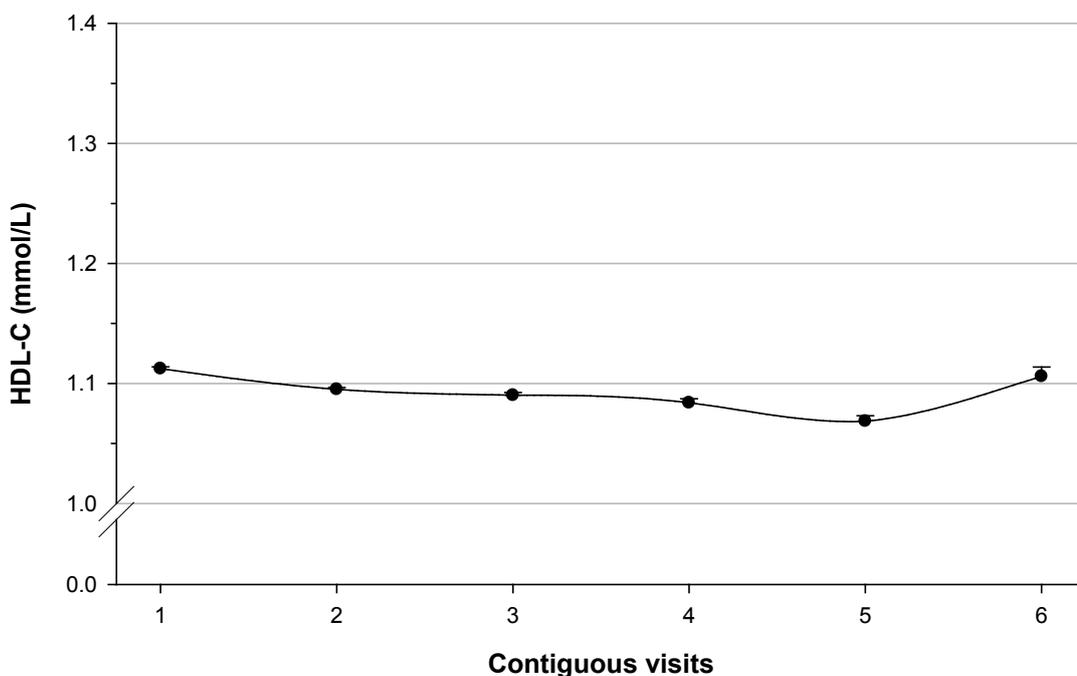


Figure 36. Average HDL-C levels of contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Compared to baseline HDL-C levels, **Table 2** shows there were unfavourable reductions in HDL-C levels with recurrent clinic visits, from 0.02 after two GP visits to a maximum of 0.09 mmol/L following five GP visits. There was a slight improvement in HDL-C levels pending a sixth visit.

Gender differences in lipid profiles across contiguous visits

TC levels decreased over annual contiguous visits between 2004 and mid-2009 by 0.5 mmol/L for men and 0.37 mmol/L for women, yet always remaining below 5.5 mmol/L for both sexes (**Figure 37**). After six clinic visits, TC levels were around 4.6 mmol/L for men compared to 5.0 mmol/L for women.

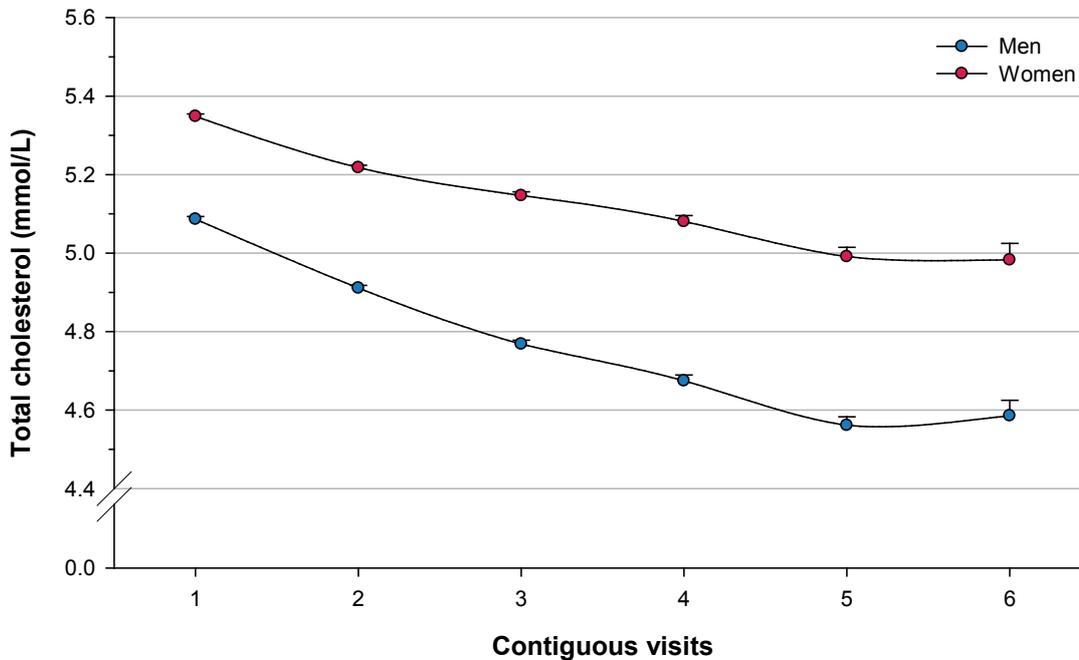


Figure 37. Average TC levels in men and women over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 3 shows that TC levels decreased from baseline by a greater amount in both men and women who had more regular follow-up. The reduction was greatest for men after five GP visits (maximum decrease of 0.52 mmol/L) and for women after six GP visits (maximum decrease of 0.47 mmol/L).

Table 3. Change (Δ) in TC levels (mmol/L) in men and women between baseline and contiguous patient visits, 2004 to mid-2009

Sex	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Men	-0.18 (n = 29,461)	-0.29 (n = 12,371)	-0.40 (n = 5,449)	-0.52 (n = 2,429)	-0.48 (n = 794)
Women	-0.13 (n = 30,784)	-0.23 (n = 12,082)	-0.35 (n = 5,067)	-0.44 (n = 2,117)	-0.47 (n = 658)

Figure 38 shows that the change in LDL-C measurements across annual contiguous visits between 2004 and mid-2009 was similar in men and women and decreased overall by 0.26 mmol/L. Following six clinic visits, LDL-C levels were 2.81 mmol/L for men and 2.95 mmol/L for women. Average LDL-C levels fell below national recommendations for men after two GP visits and for women after three visits.

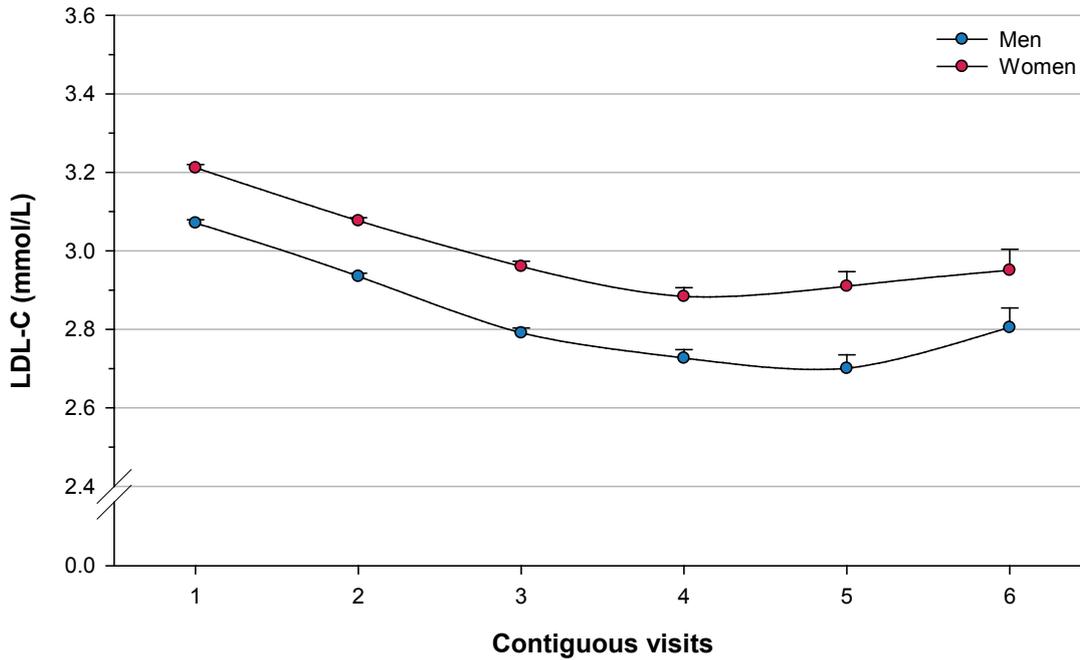


Figure 38. Average LDL-C levels in men and women over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

LDL-C levels decreased further below baseline levels with a greater frequency of follow-up visits in men and women alike (**Table 4**). The biggest change in men of 0.42 mmol/L was after five GP visits compared to the maximum change in women (approximately 0.3 mmol/L) following four to six GP visits.

Table 4. Change (Δ) in LDL-C levels (mmol/L) in men and women between baseline and contiguous patient visits, 2004 to mid-2009

Sex	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Men	-0.14 (n = 14,450)	-0.24 (n = 5,155)	-0.31 (n = 1,840)	-0.42 (n = 709)	-0.29 (n = 330)
Women	-0.14 (n = 13,898)	-0.22 (n = 4,766)	-0.32 (n = 1,721)	-0.30 (n = 635)	-0.32 (n = 281)

Figure 39 indicates that there were minimal changes in HDL-C levels from 2004 to mid-2009, which remained above the recommended level of 1.0 mmol/L over repetitive GP visits for both men and women. HDL-C levels recorded after six clinic visits were 1.06 mmol/L for men and 1.16 mmol/L for women.

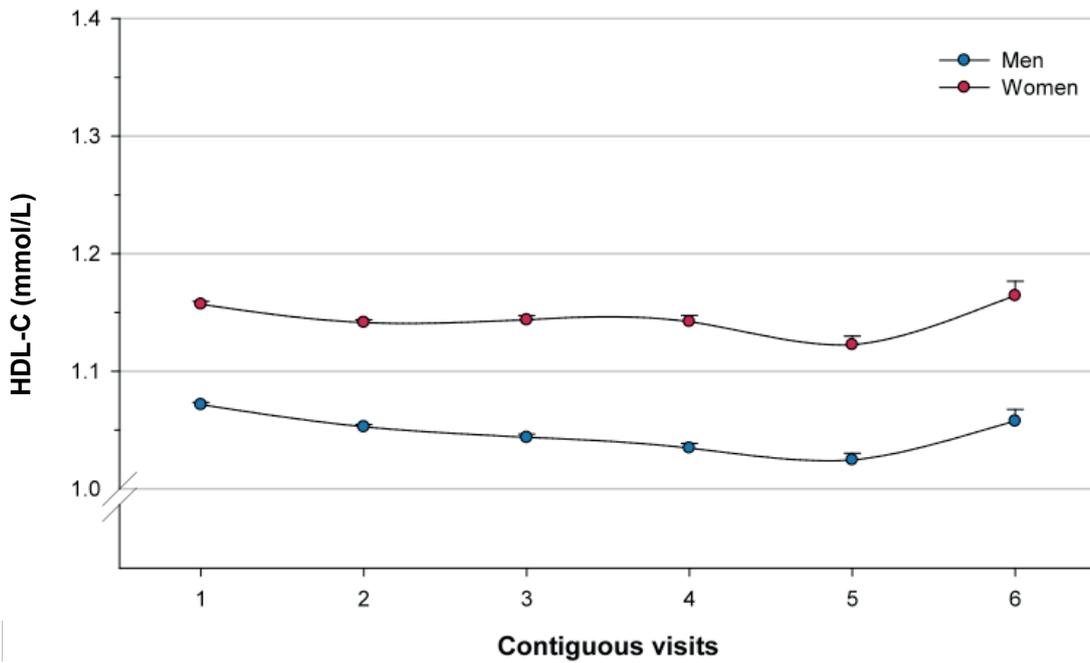


Figure 39. Average HDL-C levels in men and women over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

As shown in **Table 5**, there were minor decreases in HDL-C levels over contiguous annual visits compared to baseline levels. A maximum reduction of 0.1 mmol/L for both sexes was shown after five visits however.

Table 5. Change (Δ) in HDL-C levels (mmol/L) in men and women between baseline and contiguous patient visits, 2004 to mid-2009

Sex	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Men	-0.02 (n = 20,670)	-0.04 (n = 8,268)	-0.07 (n = 3,680)	-0.09 (n = 1,719)	-0.07 (n = 624)
Women	-0.02 (n = 18,898)	-0.04 (n = 7,172)	-0.07 (n = 3,120)	-0.10 (n = 1,402)	-0.06 (n = 515)

Gender differences in lipid profiles of middle-aged adults (45 to 64 years) across contiguous visits

Similar to the differences in TC between men and women of all ages, **Figure 40** shows that TC levels decreased across annual contiguous visits between 2004 and mid-2009 by 0.55 mmol/L for men and 0.36 mmol/L for women, finishing up after six visits at 4.7 mmol/L and 5.2 mmol/L, respectively. Average TC levels remained below 5.5 mmol/L across all continuous visits in middle-aged men but fell below this cut-off in middle-aged women following a second follow-up visit.

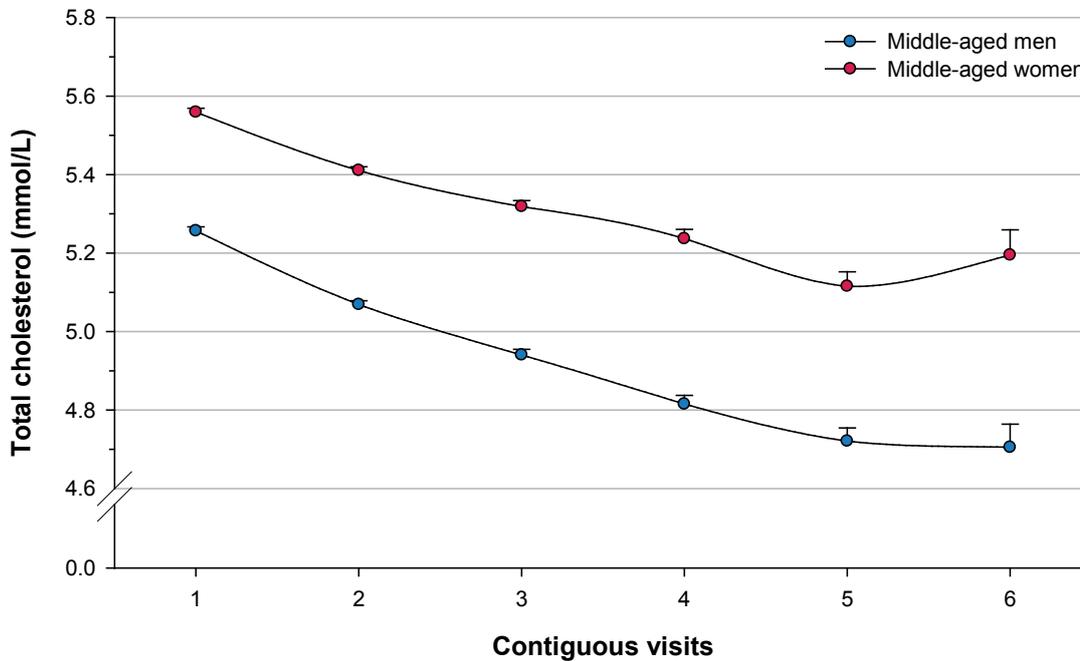


Figure 40. Average TC levels in middle-aged men and women over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

As shown in **Table 6**, TC levels decreased from baseline by a progressively greater amount with additional GP visits in middle-aged men and women. The change in TC was greatest after five or six visits for middle-aged men (0.53 mmol/L) and women (0.47 mmol/L).

Table 6. Change (Δ) in TC levels (mmol/L) in middle-aged men and women between baseline and contiguous patient visits, 2004 to mid-2009

Sex	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Middle-aged men	-0.19 (n = 13,895)	-0.30 (n = 5,805)	-0.43 (n = 2,499)	-0.53 (n = 1,080)	-0.52 (n = 370)
Middle-aged women	-0.15 (n = 13,021)	-0.25 (n = 5,024)	-0.38 (n = 2,110)	-0.46 (n = 899)	-0.47 (n = 282)

Figure 41 shows that the change in LDL-C levels across annual contiguous visits was almost identical in middle-aged men and women and decreased similarly by 0.27 mmol/L. After six GP visits, LDL-C levels were 2.90 mmol/L and 3.10 mmol/L for men and women, respectively. Average LDL-C levels did not fall below national recommendations of 3.0 mmol/L until after three contiguous annual visits for middle-aged men and remained above the primary prevention target across all follow-up GP visits for middle-aged women.

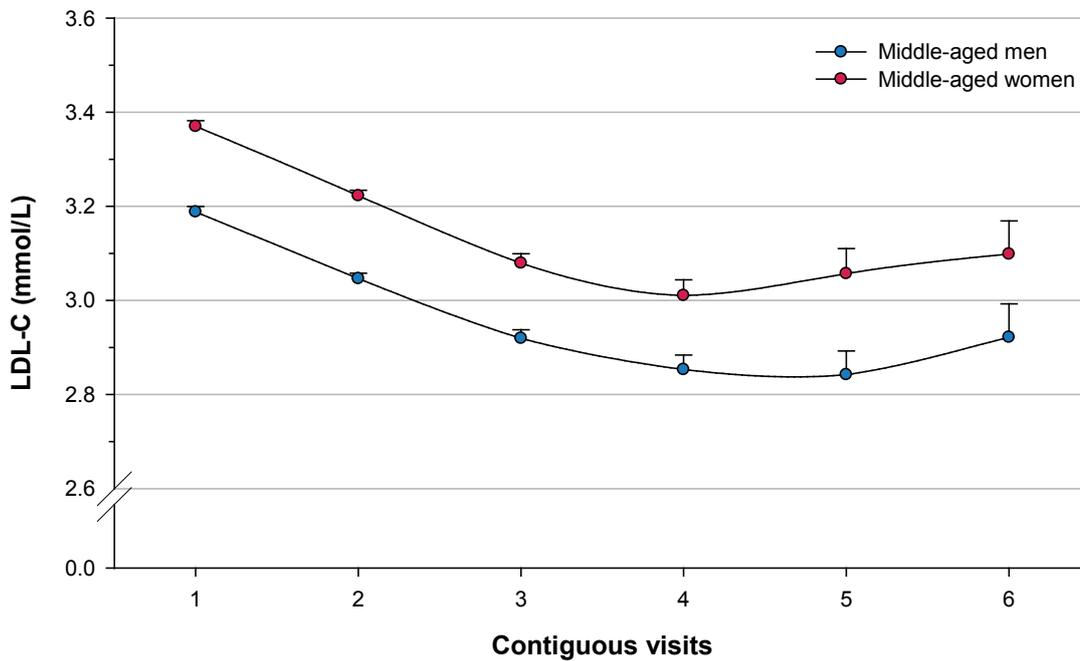


Figure 41. Average LDL-C levels in middle-aged men and women over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 7 shows that there were greater decreases from baseline in LDL-C levels with more frequent GP visits. The biggest change in middle-aged men of 0.44 mmol/L was after five GP visits compared to the largest change in middle-aged women of 0.35 mmol/L following four GP visits.

Table 7. Change (Δ) in LDL-C levels (mmol/L) in middle-aged men and women between baseline and contiguous patient visits, 2004 to mid-2009

Sex	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Middle-aged men	-0.14 (n = 7,282)	-0.24 (n = 2,619)	-0.32 (n = 914)	-0.44 (n = 369)	-0.33 (n = 178)
Middle-aged women	-0.15 (n = 6,588)	-0.26 (n = 2,211)	-0.35 (n = 815)	-0.29 (n = 322)	-0.33 (n = 153)

Consistent with observed trends in HDL-C for men and women of all ages, **Figure 42** shows that there was little change in HDL-C levels, which remained above 1.0 mmol/L for both sexes. After six clinic visits, HDL-C levels across contiguous visits were 1.08 mmol/L for middle-aged men and 1.18 mmol/L for middle-aged women.

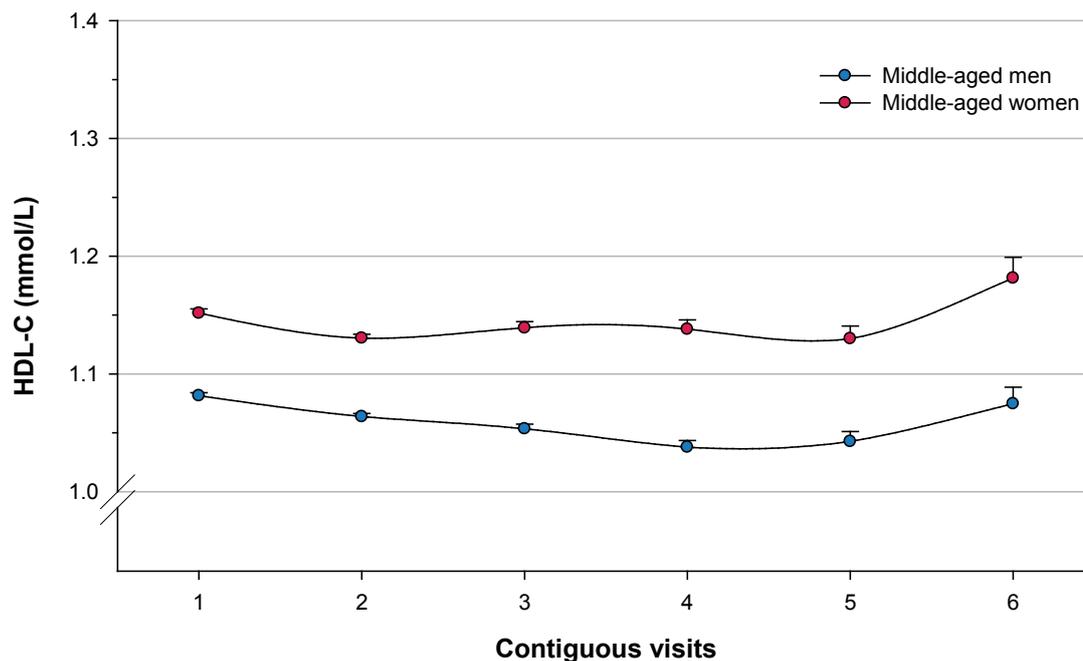


Figure 42. Average HDL-C levels in middle-aged men and women over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 8 shows that compared to baseline levels, there were very small decreases in HDL-C over contiguous annual visits. A maximum reduction of less than 0.1 mmol/L for both sexes was shown after five visits.

Table 8. Change (Δ) in HDL-C levels (mmol/L) in middle-aged men and women between baseline and contiguous patient visits, 2004 to mid-2009

Sex	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Middle-aged men	-0.02 (n = 10,390)	-0.04 (n = 4,152)	-0.07 (n = 1,810)	-0.08 (n = 816)	-0.05 (n = 306)
Middle-aged women	-0.02 (n = 8,885)	-0.04 (n = 3,291)	-0.07 (n = 1,438)	-0.09 (n = 646)	-0.04 (n = 246)

Regional differences in lipid profiles across contiguous visits

There was an overall decrease in TC levels of 0.43 mmol/L and 0.55 mmol/L for urban and rural/remote patients, respectively (**Figure 43**). TC levels remained below 5.5 mmol/L in both locations and after six clinic visits was around 4.7 mmol/L for urban patients and 4.8 mmol/L for rural/remote patients.

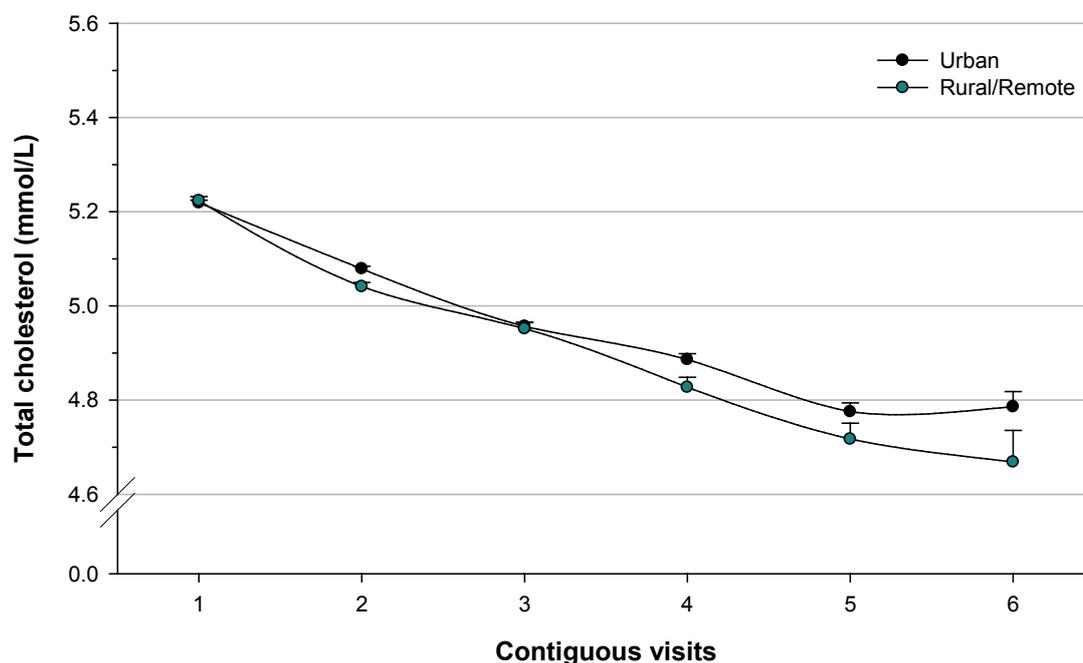


Figure 43. Average TC levels in urban and rural residents over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

TC levels decreased from baseline by a greater amount in those who had more regular follow-up, irrespective of location (**Table 9**). The reduction was greatest for rural/remote patients after five GP visits (0.52 mmol/L) and for people living in an urban location after either five or six GP visits (maximum decrease of 0.47 mmol/L).

Table 9. Change (Δ) in TC levels (mmol/L) in urban and rural residents between baseline and contiguous patient visits, 2004 to mid-2009

Region	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Urban	-0.14 (n = 42,790)	-0.26 (n = 17,800)	-0.36 (n = 7,684)	-0.47 (n = 3,447)	-0.46 (n = 1,200)
Rural/remote	-0.18 (n = 17,449)	-0.28 (n = 6,653)	-0.40 (n = 2,832)	-0.52 (n = 1,099)	-0.58 (n = 252)

Figure 44 shows that LDL-C levels decreased across annual contiguous visits by 0.27 mmol/L for urban residents and 0.41 mmol/L for rural/regional residents. Average LDL-C levels fell below 3.0 mmol/L after two GP visits for patients living in a rural/remote area and following three GP visits for patients living in an urban area. After six clinic visits, LDL-C levels were around 2.88 mmol/L and 2.72 mmol/L for urban and rural/regional residents, respectively.

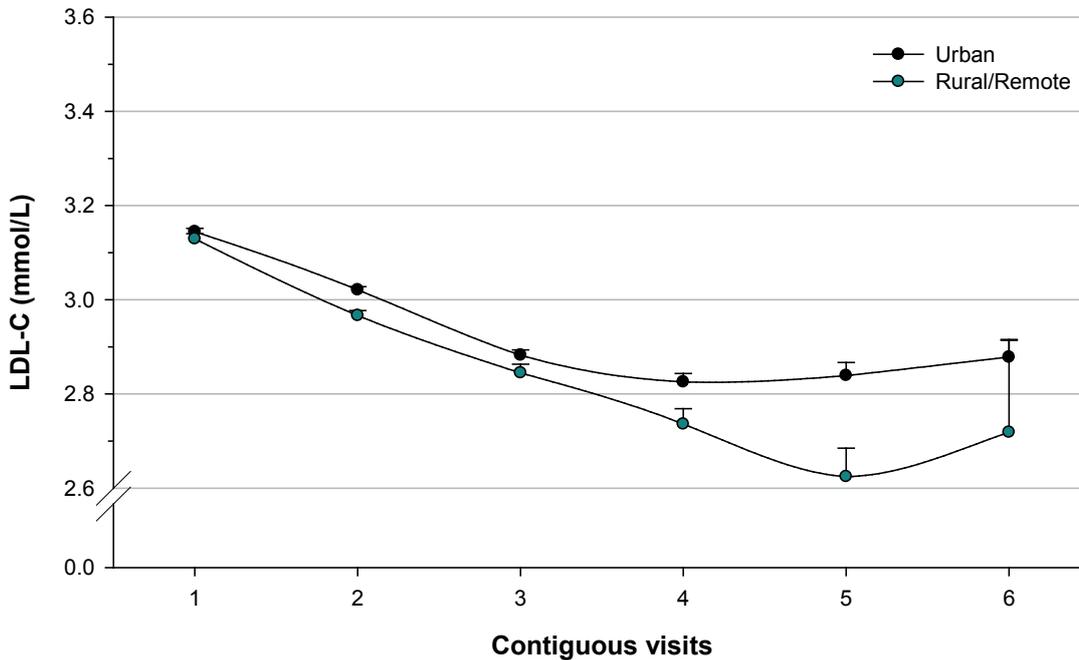


Figure 44. Average LDL-C levels in urban and rural residents over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 10 shows that LDL-C levels decreased further below baseline values with a greater frequency of follow-up visits. For urban residents, the biggest change of 0.3 mmol/L was after five or six GP visits whereas for rural/remote residents, the maximum change (0.64 mmol/L) was following five GP visits.

Table 10. Change (Δ) in LDL-C levels (mmol/L) in urban and rural residents between baseline and contiguous patient visits, 2004 to mid-2009

Region	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Urban	-0.12 (n = 19,637)	-0.22 (n = 7,182)	-0.27 (n = 2,645)	-0.30 (n = 1,096)	-0.30 (n = 586)
Rural/remote	-0.16 (n = 8,706)	-0.27 (n = 2,739)	-0.43 (n = 2,739)	-0.64 (n = 246)	-0.43 (n = 25)

Figure 45 indicates minor changes in HDL-C levels over annual GP visits which remained above 1.0 mmol/L for patients in both localities. HDL-C levels recorded after six clinic visits were 1.10 mmol/L for urban residents and 1.13 mmol/L for rural/remote residents.

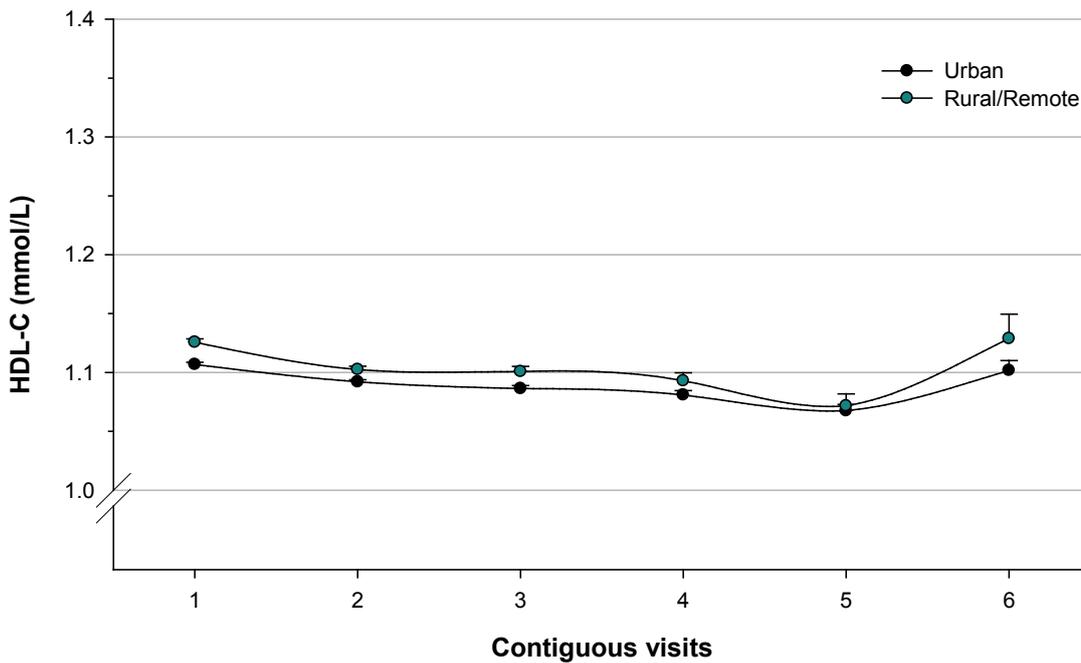


Figure 45. Average HDL-C levels in urban and rural residents over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

As shown in **Table 11**, there were small yet unfavourable decreases in HDL-C levels with contiguous annual visits compared to baseline levels for urban and rural/remote patients. A maximum reduction of 0.09 mmol/L was shown after five visits for both locations.

Table 11. Change (Δ) in HDL-C levels (mmol/L) in urban and rural residents between baseline and contiguous patient visits, 2004 to mid-2009

Region	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Urban	-0.01 (n = 27,852)	-0.04 (n = 11,233)	-0.07 (n = 5,033)	-0.09 (n = 2,405)	-0.07 (n = 960)
Rural/remote	-0.02 (n = 11,710)	-0.04 (n = 4,207)	-0.07 (n = 1,767)	-0.09 (n = 716)	-0.03 (n = 179)

Treatment differences in lipid profiles across contiguous visits

Figure 46 shows that there was a greater decrease in TC levels across contiguous visits of 0.60 mmol/L in those prescribed lipid-modulating therapy compared to a 0.08 mmol/L decrease for patients not on such treatment. Over time, there were fewer patients on lipid-modulating treatment for whom a TC measurement was recorded, yet there were more ongoing measurements for people on treatment than for patients not on such treatment (see Table 12). For patients prescribed lipid-modulating treatment, target goals recommended by the NHFA were not been met following the sixth annual GP visit and TC remained at 4.56 mmol/L. For those not prescribed lipid-modulating treatment, TC levels remained below the recommendation of 5.5 mmol/L at a level around 5.2 mmol/L.

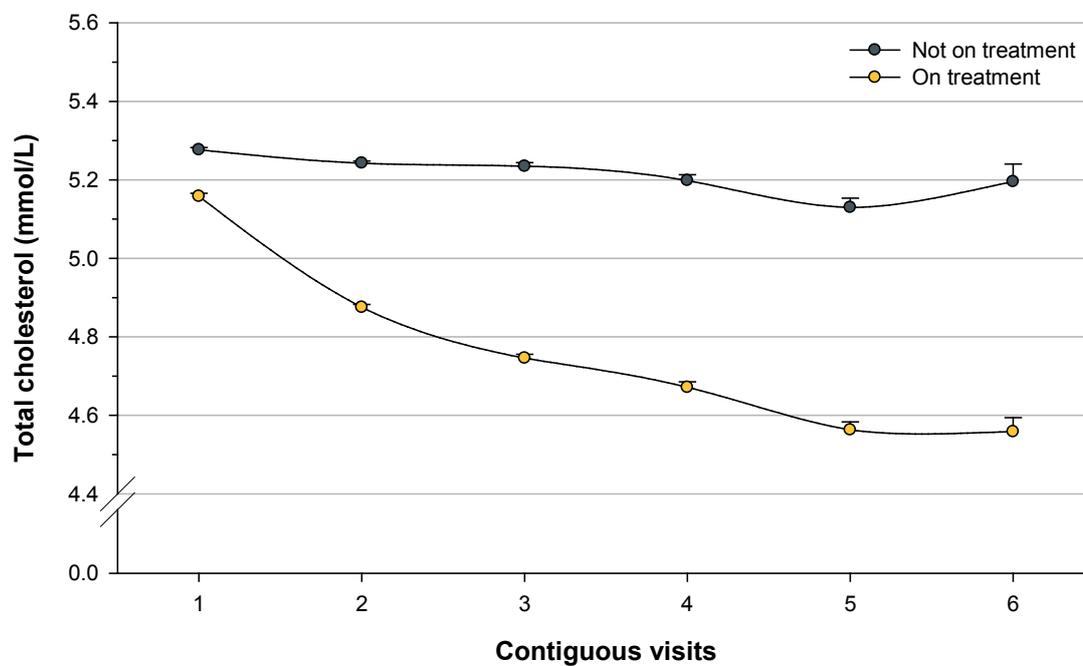


Figure 46. Average TC levels in adults on lipid-modulating therapy and not on treatment over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 12 shows that compared to baseline, there were significantly larger decreases in TC levels with subsequent GP visits which were seen sooner in those prescribed lipid-modulating therapy as opposed to patients not taking such treatment. The maximum reduction of 0.65 mmol/L and 0.17 mmol/L for patients who were taking or not taking lipid-modulating treatment, respectively, was shown after five GP visits.

Table 12. Change (Δ) in TC levels (mmol/L) in adults on lipid-modulating therapy and not on treatment between baseline and contiguous patient visits, 2004 to mid-2009

Lipid-modulating therapy	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
On treatment	-0.28 (n = 28,663)	-0.42 (n = 13,974)	-0.53 (n = 6,546)	-0.65 (n = 2,953)	-0.63 (n = 980)
Not on treatment	-0.03 (n = 31,582)	-0.05 (n = 10,479)	-0.11 (n = 3,970)	-0.17 (n = 1,593)	-0.15 (n = 472)

Similar to that seen for TC levels, **Figure 47** indicates that there was a greater decrease across contiguous visits in LDL-C levels of 0.34 mmol/L in those prescribed lipid-modulating therapy compared to 0.10 mmol/L for patients not on lipid-lowering treatment. Over time, there were less patients on lipid-modulating treatment who were re-tested annually according to guidelines. Target treatment goals advised by the NHFA were not being met following six GP visits, at which time LDL-C levels were on average 2.7 mmol/L. Among those not prescribed lipid-modulating treatment, LDL-C levels remained above the primary prevention ideal level of 3.0 mmol/L.

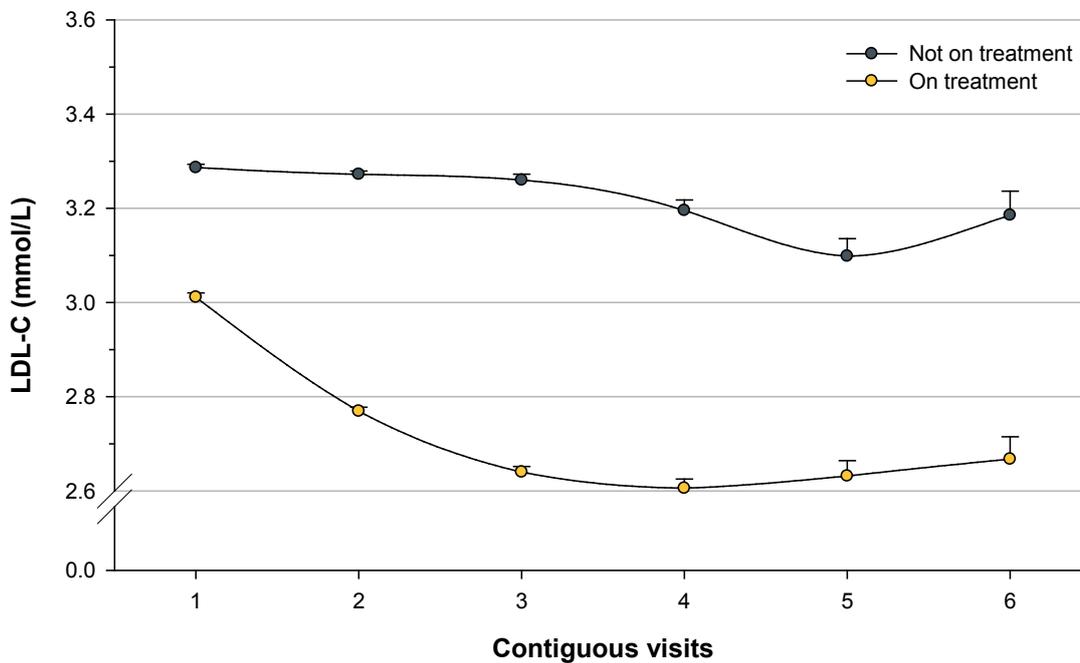


Figure 47. Average LDL-C levels in adults on lipid-modulating therapy and not on treatment over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

For those prescribed lipid-modulating therapy, there were greater decreases in LDL-C levels compared to baseline in those who had more frequent clinic visits. These changes were seen sooner (beginning after two GP visits) in contrast to patients not taking such treatment who had smaller and delayed reductions (**Table 13**). The maximum reduction of 0.51 mmol/L and 0.09 mmol/L for patients who were taking or not taking lipid-modulating treatment, respectively, was shown after five GP visits.

Table 13. Change (Δ) in LDL-C levels (mmol/L) in adults on lipid-modulating therapy and not on treatment between baseline and contiguous patient visits, 2004 to mid-2009

Lipid-modulating therapy	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
On treatment	-0.24 (n = 15,105)	-0.37 (n = 6,205)	-0.46 (n = 2,373)	-0.51 (n = 861)	-0.46 (n = 370)
Not on treatment	-0.01 (n = 13,243)	0.00 (n = 3,716)	(n = 2,373)	-0.09 (n = 483)	-0.07 (n = 241)

Figure 48 shows relatively minor changes in HDL-C levels across contiguous GP visits, irrespective of lipid-modulating treatment status. Over annual contiguous visits between 2004 and mid-2009, HDL-C levels showed a slight improvement (0.03 mmol/L) at the final recorded visit for those not prescribed lipid-modulating treatment compared to a small decrease in those taking lipid-modulating therapy. Overall, HDL-C levels remained higher than 1.0 mmol/L whether lipid lowering medication was prescribed or not.

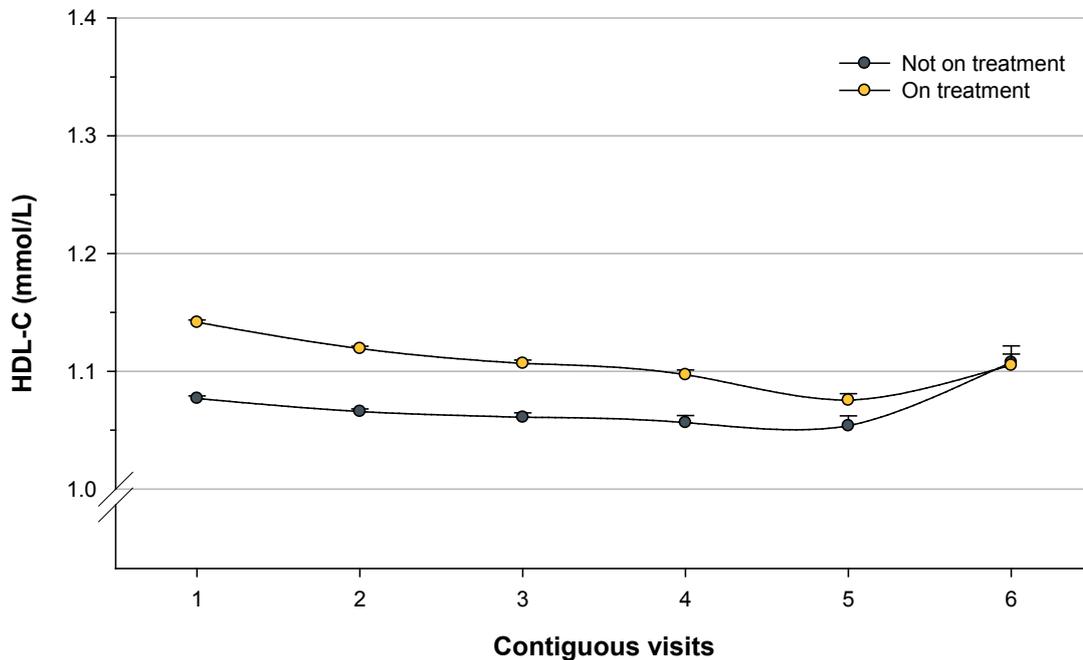


Figure 48. Average HDL-C levels in adults on lipid-modulating therapy and not on treatment over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Compared to baseline levels, there were small (unfavourable) decreases of around 0.10 mmol/L in HDL-C levels with contiguous annual visits which were shown following two visits for those not prescribed lipid-modulating treatment and after five GP visits in those on lipid-modulating treatment (**Table 14**).

Table 14. Change (Δ) in HDL-C levels (mmol/L) in adults on lipid-modulating therapy and not on treatment between baseline and contiguous patient visits, 2004 to mid-2009

Lipid-modulating therapy	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
On treatment	-0.02 (n = 21,675)	-0.05 (n = 9,873)	-0.07 (n = 4,596)	-0.10 (n = 2,124)	-0.08 (n = 775)
Not on treatment	-0.11 (n = 17,893)	-0.03 (n = 5,567)	-0.05 (n = 2,204)	-0.08 (n = 997)	-0.03 (n = 364)

Socio-economic differences in lipid profiles across contiguous visits

Figure 49 shows that TC levels decreased similarly across annual contiguous visits by 0.45 mmol/L irrespective of GP income area. Overall, average TC levels remained below the recommended target for primary prevention of 5.5 mmol/L for patients who visited a GP in both low and high income areas.

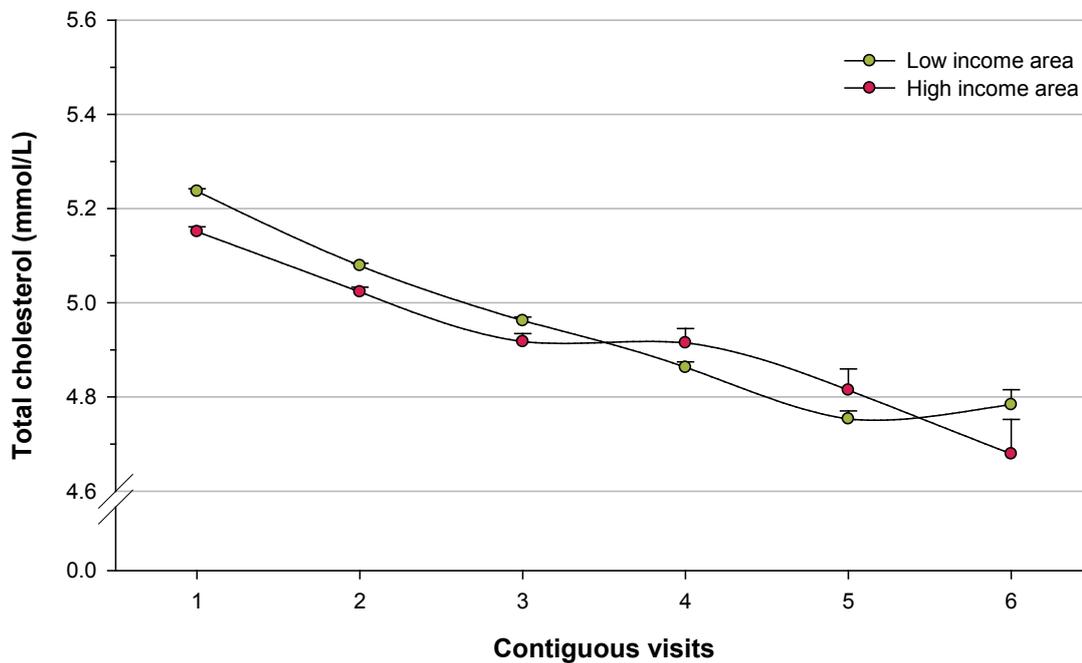


Figure 49. Average TC levels in low compared to high income areas over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Despite GP income area, Table 15 shows that TC levels decreased from baseline by a greater amount with more regular follow-up visits. The reduction was greatest for patients from a low income area after five clinic visits (0.51 mmol/L) and for patients from a high income area after six visits (0.42 mmol/L).

Table 15. Change (Δ) in TC levels (mmol/L) in low compared to high income areas between baseline and contiguous patient visits, 2004 to mid-2009

GP clinic location	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Low income area	-0.16 (n = 48,136)	-0.27 (n = 20,270)	-0.39 (n = 9,143)	0.51 (n = 3,939)	-0.49 (n = 1,198)
High income area	-0.13 (n = 11,509)	-0.23 (n = 4,052)	-0.28 (n = 1,330)	-0.33 (n = 605)	-0.42 (n = 254)

Figure 50 indicates that the change in LDL-C levels across annual contiguous visits decreased overall by 0.27 mmol/L in both low and high GP income areas. Following six clinic visits, LDL-C levels were 2.88 mmol/L for patients visiting a GP in a low income area and 2.82 mmol/L for patients visiting a GP in a high income area. Average LDL-C levels fell below the recommendation of 3.0 mmol/L after two visits in patients whose GP clinic resides in a high income area and after three visits for patients whose GP clinic resides in a low income area.

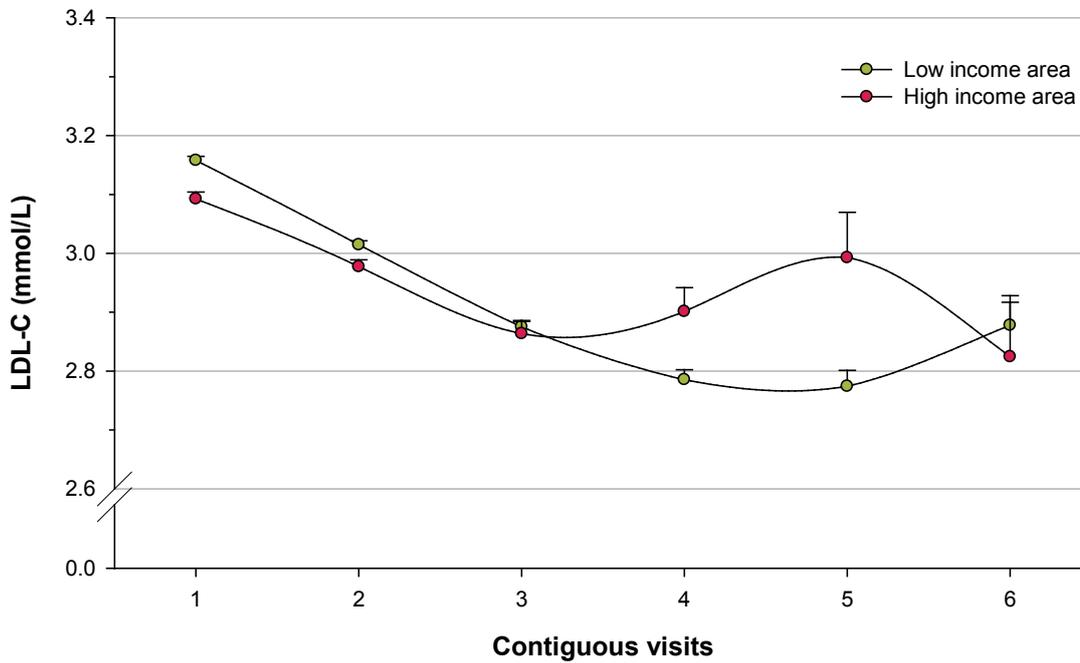


Figure 50. Average LDL-C levels in low compared to high income areas over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

Table 16 shows that for patients from a low income area, there were more sizeable decreases in LDL-C levels with more frequent GP visits compared to baseline levels. The biggest change in patients from a low income area of 0.40 mmol/L was after five GP visits compared to the maximum change in patients from a high income area (approximately 0.16 mmol/L) following two GP visits, but with similar progress at most follow-up visits.

Table 16. Change (Δ) in LDL-C levels (mmol/L) in low compared to high income areas between baseline and contiguous patient visits, 2004 to mid-2009

GP clinic location	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Low income area	-0.14 (n = 21,379)	-0.25 (n = 7,817)	-0.36 (n = 3,010)	-0.40 (n = 1,187)	-0.33 (n = 542)
High income area	-0.11 (n = 6,452)	-0.16 (n = 1,995)	-0.10 (n = 515)	-0.06 (n = 155)	-0.14 (n = 59)

There were negligible changes in HDL-C levels over annual contiguous visits (**Figure 51**). Over this time, HDL-C levels remained stable at around 1.10 mmol/L and hence above recommended levels, despite GP income area.

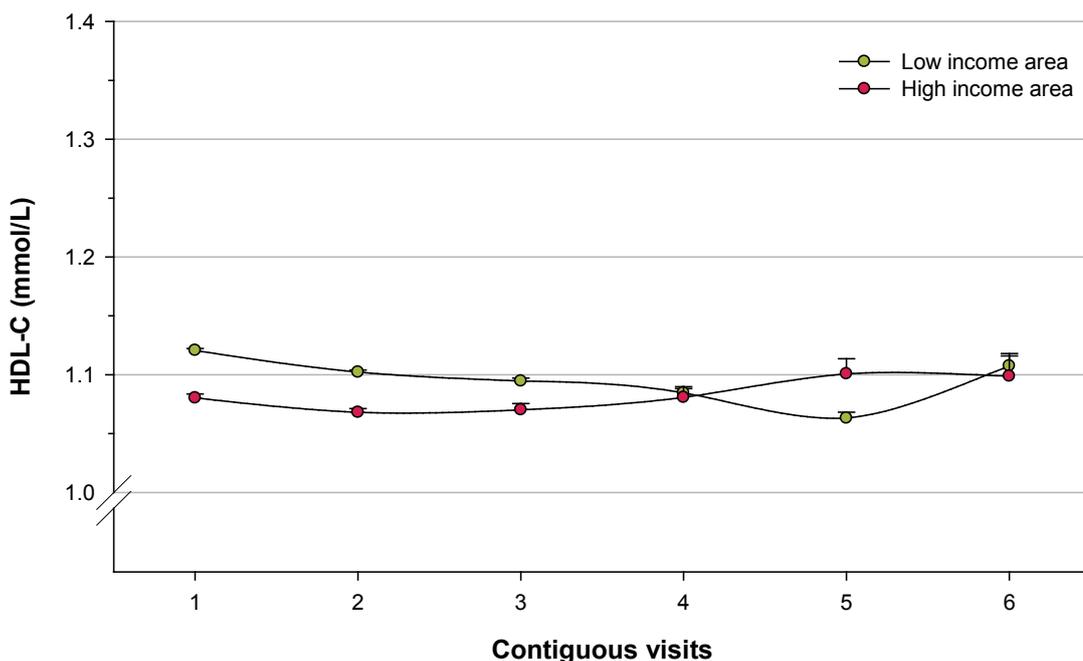


Figure 51. Average HDL-C levels in low compared to high income areas over contiguous patient visits, 2004 to mid-2009

Note: Standard error bars are contained within some data points due to the very small variability.

There were small (unfavourable) decreases in HDL-C levels over contiguous annual visits compared to baseline levels. The reduction (0.10 mmol/L) was greatest for patients from a low income area after five clinic visits and for patients from a high income area after four clinic visits (0.05 mmol/L).

Table 17. Change (Δ) in HDL-C levels (mmol/L) in low compared to high income areas between baseline and contiguous patient visits, 2004 to mid-2009

GP clinic location	Δ visit 1 to visit 2	Δ visit 1 to visit 3	Δ visit 1 to visit 4	Δ visit 1 to visit 5	Δ visit 1 to visit 6
Low income area	-0.02 (n = 31,056)	-0.04 (n = 12,500)	-0.07 (n = 5,779)	-0.10 (n = 2,679)	-0.07 (n = 945)
High income area	-0.01 (n = 7,943)	-0.02 (n = 2,812)	-0.05 (n = 979)	-0.04 (n = 440)	-0.04 (n = 194)

Conclusions

This report outlines the results of Australia's largest ever study of lipid levels. Focussing on nearly 200,000 patients being managed by nearly 600 GPs from throughout Australia during 2004 to mid-2009, it provides important insights into one of our most important and costly risk factors for ill health - dyslipidaemia. We observed both pleasing and worrying trends in respect to lipid profiles in this large cohort. Given the enormous investment in managing dyslipidaemia (at least \$1.4 billion per annum), we believe it is critical to apply continued surveillance to determine if we are maximising the potential of lipid-modulating treatments. If these data are a true indication of lipid management in primary care, we stand at the crossroads between sustaining reduced absolute lipid levels (specifically TC and LDL-C levels), and a rebound in elevated levels, that will leave many Australians at increased risk of highly preventable CVD.

Overall, mean TC levels modestly declined in both men and women between 2004 and 2008 before recording a small rise in mid-2009. Whether this upward inflection is a real phenomenon and reflects increased lipid levels in the wider Australian population is unknown. However, it is worth noting that at this time of the "global financial crisis" (beforehand Australia's overall wealth steadily increased), it was the fast food industry that reported a global "healthy" increase in sales; potentially this has "fed" the observed rebound in TC and LDL-C levels (The Associated Press 2008).

We also observed clear sex-based differences that remained constant when adjusting for age, with women recording higher TC, LDL-C and HDL-C levels overall. This is not a typical finding for population research but we have few data from a primary care perspective with which to compare our findings. Further studies are required to elucidate our observations in this regard.

Alternatively, although we expected differentials according to the geographic (i.e. urban vs. rural/regional) and socio-economic location of primary care management, we found few differences on this basis. In this cohort at least, lipid profiles did not discriminate according to an individual's social circumstances. We did, however, show quite large differences in lipid levels across Australia, with Tasmania faring worst overall.

Our initial analyses of those prescribed and not prescribed lipid-modulating therapy confirmed the efficacy of applying pharmacological lipid-modifying treatment to optimise lipid profiles. These positive data need to be tempered by the fact that a large proportion of patients did not fully achieve treatment targets (Tonkin *et al.* 2005). Further analyses of the relationship between contiguous GP management

and monitoring demonstrated clear benefits from a sustained approach to lipid management. Those patients who have up to six contiguous GP visits (characterised by prescribed treatment and serial lipid measurements) are able to achieve greater than 0.6 mmol/L and 0.5 mmol/L reductions in their TC and LDL-C levels, respectively. Those patients subject to continued surveillance but no treatment (potentially adopting lifestyle changes) achieved modest declines in their lipid levels, amounting to a 0.15 mmol/L decrease in TC over six contiguous GP visits. A proportion of the latter group would have undoubtedly benefited greater by initiating lipid-modulating therapy. Although it is possible that patients may have changed GPs, it is important to note that of almost 200,000 patients, we could only find approximately 60,000 patients in whom a regular pattern of surveillance (with and without the application of lipid-modulating therapy) was evident.

We can conclude therefore that while average lipid levels in primary care patients may appear acceptable from a public health perspective, there are still challenges ahead, particularly when the observed upward inflection in mid-2009 levels are considered. For example, considering the full study cohort in mid-2009, one in three adults had sub-optimal TC and HDL-C levels and a more disappointing one in every two adults had sub-optimal LDL-C levels, which was broadly equivalent in men and women. The key driver for improving the lipid profiles of those particularly at risk of CVD is also very clear from these data – continuous GP management and lipid-modulating therapy. Those without recorded prescriptions for a statin or other lipid lowering drug essentially maintained the same lipid levels across contiguous GP visits. Alternatively, treated patients made inroads to achieving optimal lipid levels after five years of (recorded) GP management. Therefore, consistent with our previous research, there is an element of cholesterol complacency amongst GPs and patients alike (Carrington *et al.* 2009). In order to achieve ideal lipid target levels, it is imperative that patients and GPs work together to consistently apply and monitor the efficacy of otherwise proven lipid-modulating treatment strategies.

In summary, this report has given us "food for thought" in respect to the lipid levels of Australians being managed in the primary care setting. Health professionals and the general public alike need to recognise that we stand at the crossroads between sustaining a continuous decrease, or an unwelcome reversal in cholesterol levels into the future. A renewed effort to collectively lower our lipid levels (using a combination of routine surveillance, lifestyle modification, lipid-modulating therapy and active GP management) is required to sustain our attack on Australia's number one cause of death and disability – CAD.

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