

Person—low-density lipoprotein cholesterol level (calculated), total millimoles per litre N[N].N

Exported from METEOR (AIHW's Metadata Online Registry)

© Australian Institute of Health and Welfare 2024

This product, excluding the AIHW logo, Commonwealth Coat of Arms and any material owned by a third party or protected by a trademark, has been released under a Creative Commons BY 4.0 (CC BY 4.0) licence. Excluded material owned by third parties may include, for example, design and layout, images obtained under licence from third parties and signatures. We have made all reasonable efforts to identify and label material owned by third parties.

You may distribute, remix and build on this website's material but must attribute the AIHW as the copyright holder, in line with our attribution policy. The full terms and conditions of this licence are available at <https://creativecommons.org/licenses/by/4.0/>.

Enquiries relating to copyright should be addressed to info@aihw.gov.au.

Enquiries or comments on the METEOR metadata or download should be directed to the METEOR team at meteor@aihw.gov.au.

Person—low-density lipoprotein cholesterol level (calculated), total millimoles per litre N[N].N

Identifying and definitional attributes

Metadata item type:	Data Element
Short name:	Cholesterol—LDL (calculated)
METEOR identifier:	359262
Registration status:	Health , Standard 01/10/2008
Definition:	A person's calculated low-density lipoprotein cholesterol (LDL-C) in millimoles per litre.
Data Element Concept:	Person—low-density lipoprotein cholesterol level
Value Domain:	Millimoles per litre N[N].N

Value domain attributes

Representational attributes

Representation class:	Total	
Data type:	Number	
Format:	N[N].N	
Maximum character length:	3	
	Value	Meaning
Supplementary values:	99.9	Not stated/inadequately described
Unit of measure:	Millimole per litre (mmol/L)	

Data element attributes

Collection and usage attributes

Guide for use:	Formula: $\text{LDL-C} = (\text{plasma total cholesterol}) - (\text{high density lipoprotein cholesterol}) - (\text{fasting plasma triglyceride divided by 2.2}).$
Collection methods:	<p>The LDL-C is usually calculated from the Friedwald Equation (Friedwald et al. 1972), which depends on knowing the blood levels of the total cholesterol and HDL-C and the fasting level of the triglyceride.</p> <p>Note that the Friedwald equation becomes unreliable when the plasma triglyceride exceeds 4.5 mmol/L.</p> <p>Note also that while cholesterol levels are reliable for the first 24 hours after the onset of acute coronary syndromes, they may be unreliable for the subsequent 8 weeks after an event.</p> <ul style="list-style-type: none">• Measurement of lipid levels should be carried out by laboratories, or practices, which have been accredited to perform these tests by the National Association of Testing Authorities.• To be collected as a single venous blood sample, preferably following a 12-hour fast where only water and medications have been consumed.

Comments: High blood cholesterol is a key factor in heart, stroke and vascular disease, especially coronary heart disease (CHD).

Poor nutrition can be a contributing factor to heart, stroke and vascular disease as a population's level of saturated fat intake is the prime determinant of its level of blood cholesterol.

The majority of the cholesterol in plasma is transported as a component of LDL-C. Recent trials support a target LDL-C of <2.0 mmol/L for high risk patients with existing coronary heart disease.

Source and reference attributes

Submitting organisation: Cardiovascular Data Working Group

Origin: National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand, Lipid Management Guidelines - 2001, MJA 2001; 175: S57-S88.

National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand, Position Statement on Lipid Management - 2005, Heart, Lung and Circulation 2005; 14: 275-291.

Relational attributes

Related metadata references: Supersedes [Person—low-density lipoprotein cholesterol level \(calculated\), total millimoles per litre N\[N\].N](#)
[Health](#), Superseded 01/10/2008

Is formed using [Health service event—fasting indicator, code N](#)
[Health](#), Standard 21/09/2005

Is formed using [Person—cholesterol level \(measured\), total millimoles per litre N\[N\].N](#)
[Health](#), Superseded 01/10/2008

Is formed using [Person—high-density lipoprotein cholesterol level \(measured\), total millimoles per litre \[N\].NN](#)
[Health](#), Standard 01/03/2005

Is formed using [Person—triglyceride level \(measured\), total millimoles per litre N\[N\].N](#)
[Health](#), Superseded 01/10/2008

Implementation in Data Set Specifications: [Acute coronary syndrome \(clinical\) DSS](#)
[Health](#), Superseded 01/09/2012

[Acute coronary syndrome \(clinical\) DSS](#)
[Health](#), Superseded 02/05/2013

[Acute coronary syndrome \(clinical\) NBPDS 2013-](#)
[Health](#), Standard 02/05/2013

Implementation start date: 01/07/2013

[Cardiovascular disease \(clinical\) DSS](#)
[Health](#), Superseded 01/09/2012

DSS specific information:

Many studies have demonstrated the significance of blood cholesterol components as risk factors for heart, stroke and vascular disease.

Scientific studies have shown a continuous relationship between lipid levels and Coronary Heart Disease (CHD) and overwhelming evidence that lipid lowering interventions reduces CHD progression, morbidity and mortality.

There are many large-scale, prospective population studies defining the relationship between plasma total (and Low-density Lipoprotein (LDL)) cholesterol and the future risk of developing CHD. The results of prospective population studies are consistent and support several general conclusions:

- the majority of people with CHD do not have markedly elevated levels of plasma total cholesterol or LDL-C,
- there is a continuous positive but curvilinear relationship between the concentration of plasma total (and LDL) cholesterol and the risk of having a

- coronary event and of dying from CHD,
- there is no evidence that a low level of plasma (or LDL) cholesterol predisposes to an increase in non-coronary mortality.

The excess non-coronary mortality at low cholesterol levels in the Honolulu Heart Study (Yano et al. 1983; Stemmermann et al. 1991) was apparent only in people who smoked and is consistent with a view that smokers may have occult smoking related disease that is responsible for both an increased mortality and a low plasma cholesterol.

It should be emphasised that the prospective studies demonstrate an association between plasma total cholesterol and LDL-C and the risk of developing CHD. (Lipid Management Guidelines - 2001, MJA 2001; 175: S57-S88 and Commonwealth Department of Health & Ageing and Australian Institute of Health and Welfare (1999) National Health Priority Areas Report: Cardiovascular Health 1998. AIHW Cat. No. PHE 9. HEALTH and AIHW, Canberra pgs 14-17).

In settings such as general practice where the monitoring of a person's health is ongoing and where a measure can change over time, the service contact date should be recorded.

[Cardiovascular disease \(clinical\) NBPDS](#)

[Health](#), Superseded 17/10/2018

DSS specific information:

Many studies have demonstrated the significance of blood cholesterol components as risk factors for heart, stroke and vascular disease.

Scientific studies have shown a continuous relationship between lipid levels and Coronary Heart Disease (CHD) and overwhelming evidence that lipid lowering interventions reduces CHD progression, morbidity and mortality.

There are many large-scale, prospective population studies defining the relationship between plasma total (and Low-density Lipoprotein (LDL)) cholesterol and the future risk of developing CHD. The results of prospective population studies are consistent and support several general conclusions:

- the majority of people with CHD do not have markedly elevated levels of plasma total cholesterol or LDL-C,
- there is a continuous positive but curvilinear relationship between the concentration of plasma total (and LDL) cholesterol and the risk of having a coronary event and of dying from CHD,
- there is no evidence that a low level of plasma (or LDL) cholesterol predisposes to an increase in non-coronary mortality.

The excess non-coronary mortality at low cholesterol levels in the Honolulu Heart Study (Yano et al. 1983; Stemmermann et al. 1991) was apparent only in people who smoked and is consistent with a view that smokers may have occult smoking related disease that is responsible for both an increased mortality and a low plasma cholesterol.

It should be emphasised that the prospective studies demonstrate an association between plasma total cholesterol and LDL-C and the risk of developing CHD. (Lipid Management Guidelines - 2001, MJA 2001; 175: S57-S88 and Commonwealth Department of Health & Ageing and Australian Institute of Health and Welfare (1999) National Health Priority Areas Report: Cardiovascular Health 1998. AIHW Cat. No. PHE 9. HEALTH and AIHW, Canberra pgs 14-17).

In settings such as general practice where the monitoring of a person's health is ongoing and where a measure can change over time, the service contact date should be recorded.

[Cardiovascular disease \(clinical\) NBPDS](#)

[Health](#), Standard 17/10/2018

DSS specific information:

Many studies have demonstrated the significance of blood cholesterol components as risk factors for heart, stroke and vascular disease.

Scientific studies have shown a continuous relationship between lipid levels and

Coronary Heart Disease (CHD) and overwhelming evidence that lipid lowering interventions reduces CHD progression, morbidity and mortality.

There are many large-scale, prospective population studies defining the relationship between plasma total (and Low-density Lipoprotein (LDL)) cholesterol and the future risk of developing CHD. The results of prospective population studies are consistent and support several general conclusions:

- the majority of people with CHD do not have markedly elevated levels of plasma total cholesterol or LDL-C,
- there is a continuous positive but curvilinear relationship between the concentration of plasma total (and LDL) cholesterol and the risk of having a coronary event and of dying from CHD,
- there is no evidence that a low level of plasma (or LDL) cholesterol predisposes to an increase in non-coronary mortality.

The excess non-coronary mortality at low cholesterol levels in the Honolulu Heart Study (Yano et al. 1983; Stemmermann et al. 1991) was apparent only in people who smoked and is consistent with a view that smokers may have occult smoking related disease that is responsible for both an increased mortality and a low plasma cholesterol.

It should be emphasised that the prospective studies demonstrate an association between plasma total cholesterol and LDL-C and the risk of developing CHD. (Lipid Management Guidelines - 2001, MJA 2001; 175: S57-S88 and Commonwealth Department of Health & Ageing and Australian Institute of Health and Welfare (1999) National Health Priority Areas Report: Cardiovascular Health 1998. AIHW Cat. No. PHE 9. HEALTH and AIHW, Canberra pgs 14-17).

In settings such as general practice where the monitoring of a person's health is ongoing and where a measure can change over time, the service contact date should be recorded.