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Repositioning of the global epicentre of non-optimal cholesterol

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Repositioning of the global epicentre of non-optimal cholesterol

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NCD Risk Factor Collaboration (NCD-RisC)*

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High blood cholesterol is typically considered a feature of wealthy western countries^{1,2}. However, dietary and behavioural determinants of blood cholesterol are changing rapidly throughout the world³ and countries are using lipid-lowering medications at varying rates. These changes can have distinct effects on the levels of high-density lipoprotein (HDL) cholesterol and non-HDL cholesterol, which have different effects on human health^{4,5}. However, the trends of HDL and non-HDL cholesterol levels over time have not been previously reported in a global analysis. Here we pooled 1,127 population-based studies that measured blood lipids in 102.6 million individuals aged 18 years and older to estimate trends from 1980 to 2018 in mean total, non-HDL and HDL cholesterol levels for 200 countries. Globally, there was little change in total or non-HDL cholesterol from 1980 to 2018. This was a net effect of increases in low- and middle-income countries, especially in east and southeast Asia, and decreases in high-income western countries, especially those in northwestern Europe, and in central and eastern Europe. As a result, countries with the highest level of non-HDL cholesterol—which is a marker of cardiovascular risk—changed from those in western Europe such as Belgium, Finland, Greenland, Iceland, Norway, Sweden, Switzerland and Malta in 1980 to those in Asia and the Pacific, such as Tokelau, Malaysia, The Philippines and Thailand. In 2017, high non-HDL cholesterol was responsible for an estimated 3.9 million (95% credible interval 3.7 million–4.2 million) worldwide deaths, half of which occurred in east, southeast and south Asia. The global repositioning of lipid-related risk, with non-optimal cholesterol shifting from a distinct feature of high-income countries in northwestern Europe, north America and Australasia to one that affects countries in east and southeast Asia and Oceania should motivate the use of population-based policies and personal interventions to improve nutrition and enhance access to treatment throughout the world.

Blood cholesterol is one of the most important risk factors for ischaemic heart disease (IHD) and ischaemic stroke^{4–6}. Consistent and comparable information on cholesterol levels and trends in different countries can help to benchmark national performance in addressing non-optimal cholesterol, investigate the reasons behind differential trends and identify countries in which interventions are needed the most.

A previous global analysis⁷ reported trends in total cholesterol from 1980 to 2008, but did not analyse important lipid fractions—including HDL and non-HDL cholesterol—that are key to understanding the cardiovascular disease risk associated with non-optimal cholesterol. Dietary and behavioural determinants of cholesterol have changed throughout the world in the past decades, including a worldwide rise in adiposity^{8,9}, divergent global trends in alcohol use¹⁰, a rise in the intake of animal-source foods in middle-income countries (especially in east Asia)^{3,11}, and a replacement of saturated fats and trans fats with unsaturated fats in some high-income countries^{3,11,12}. There is also considerable variation in how much different

countries have adopted lipid-lowering medications¹³. These changes are likely to have influenced cholesterol levels substantially in the decade since the last estimates were made. Furthermore, HDL and non-HDL cholesterol, which have opposite associations with cardiovascular diseases^{4,5}, respond differently to diet and treatment, and may therefore have different geographical patterns and trends over time¹⁴. Information on these major lipid fractions, which were not included in the previous global estimates, is essential for priority setting and intervention choice.

Here we pooled 1,127 population-based studies that measured blood lipids in 102.6 million individuals aged 18 years and older (Extended Data Figs. 1, 2 and Supplementary Table 1) and used a Bayesian hierarchical model to estimate trends from 1980 to 2018 in mean total, non-HDL and HDL cholesterol levels for 200 countries. We also estimated the number of deaths caused by IHD and ischaemic stroke that were attributable to high levels of non-HDL cholesterol using information on its hazards from epidemiological studies.

*A list of participants and their affiliations appears in the online version of the paper.

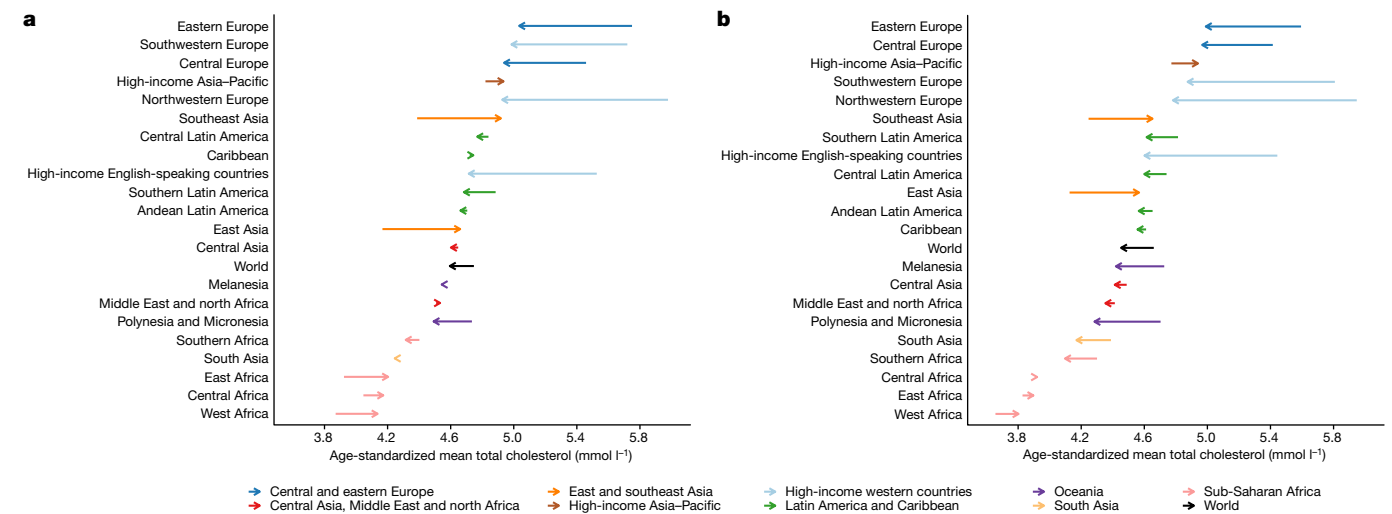


Fig. 1 | Change in age-standardized mean total cholesterol between 1980 and 2018 by region for women and men. a, Age-standardized mean total cholesterol in women. b, Age-standardized mean total cholesterol in men.

The start of the arrow shows the level in 1980 and the head indicates the level in 2018. See Extended Data Fig. 3 for age-standardized mean HDL cholesterol. One mmol l^{-1} is equivalent to 38.61 mg dl^{-1} .

Trends in total cholesterol

In 2018, global age-standardized mean total cholesterol was 4.6 mmol l^{-1} (95% credible interval, 4.5–4.7) for women and 4.5 mmol l^{-1} (4.3–4.6) for men. Global age-standardized mean total cholesterol changed little over these nearly four decades, decreasing by 0.03 mmol l^{-1} per decade (-0.02 – 0.08) in women and 0.05 mmol l^{-1} per decade (0.00 – 0.11) in men (posterior probability of the observed declines being true declines = 0.90 for women and 0.98 for men) (Fig. 1). Regionally, total cholesterol decreased the most in high-income western regions and in central and eastern Europe. The decrease was the largest (around 0.3 mmol l^{-1} per decade; posterior probability >0.9999) in northwestern Europe, where mean total cholesterol levels had been the highest in 1980. The decrease in total cholesterol in high-income western regions and central and eastern Europe was largely due to a decline in non-HDL cholesterol (Extended Data Fig. 4), which among women was offset partly by an increase in mean HDL cholesterol levels. Mean total cholesterol changed little in most of the other regions, with the notable exception of east and southeast Asia, where it increased by more than 0.1 mmol l^{-1} per decade in both women and men (posterior probability ≥ 0.95). The increase in east and southeast Asia was largely due to an increase in non-HDL cholesterol.

Trends in non-HDL and HDL cholesterol

In 2018, global age-standardized mean non-HDL cholesterol was 3.3 mmol l^{-1} (3.2–3.4) for women and 3.3 mmol l^{-1} (3.3–3.4) for men; global age-standardized mean HDL cholesterol was 1.3 mmol l^{-1} (1.2–1.3) for women and 1.1 mmol l^{-1} (1.1–1.2) for men. Global age-standardized mean non-HDL cholesterol remained almost unchanged from 1980 to 2018, decreasing by only 0.02 mmol l^{-1} per decade (-0.02 – 0.06 ; posterior probability = 0.80) in women and 0.01 mmol l^{-1} per decade (-0.03 – 0.06 ; posterior probability = 0.72) in men. Global age-standardized mean HDL cholesterol remained unchanged for women and decreased slightly for men (by 0.02 mmol l^{-1} per decade, posterior probability = 0.91).

Regionally, non-HDL cholesterol decreased substantially in high-income western regions and central and eastern Europe. The largest decrease occurred in northwestern Europe ($>0.3 \text{ mmol l}^{-1}$ per decade; posterior probability >0.9999) (Fig. 2). By contrast, it increased in east and southeast Asia, parts of sub-Saharan Africa and Melanesia. The increase was the largest in southeast Asia, increasing by

approximately 0.2 mmol l^{-1} per decade (posterior probability >0.9999). Mean HDL cholesterol increased in the high-income Asia–Pacific region, by as much as 0.1 mmol l^{-1} per decade in women (posterior probability >0.9999) but decreased in Melanesia, Polynesia and Micronesia (Extended Data Fig. 3).

Belgium, Finland, Greenland, Iceland, Norway, Sweden, Switzerland and Malta had some of the highest non-HDL cholesterol levels in 1980 ($>4.5 \text{ mmol l}^{-1}$ in women and $>4.7 \text{ mmol l}^{-1}$ in men) but experienced some of the largest declines (Figs. 3, 4). At the extreme, mean non-HDL cholesterol declined by around 0.45 mmol l^{-1} per decade or more in Belgian and Icelandic women and men, changing their ranks from being in the top 10 countries in terms of non-HDL cholesterol in 1980 to being ranked in the lower half of the countries in 2018—below countries in southwestern Europe such as France and Italy. The largest increases were found in east Asian countries (for example, China) and southeast Asian countries (for example, Indonesia, Thailand, Malaysia, Cambodia and Lao PDR). In these countries, age-standardized mean non-HDL cholesterol increased by as much as 0.23 mmol l^{-1} per decade. As a result of these opposite trends, countries with the highest age-standardized mean non-HDL cholesterol levels in 2018 were all outside northwestern Europe: Tokelau, Malaysia, The Philippines and Thailand, all of which had mean non-HDL cholesterol around or above 4 mmol l^{-1} . China, which had one of the lowest mean non-HDL cholesterol levels in 1980, reached or surpassed non-HDL cholesterol levels of many high-income western countries in 2018. Sub-Saharan African countries had the lowest mean non-HDL cholesterol in 2018, as low as 2.6 mmol l^{-1} in some countries, as they had in 1980. Not only did high-income countries benefit from decreasing non-HDL cholesterol levels, they had higher mean HDL cholesterol than low- and middle-income countries (Extended Data Fig. 6).

Deaths attributable to non-optimal cholesterol

In 2017, high non-HDL cholesterol was responsible for an estimated 3.9 million (3.7–4.2 million) worldwide deaths from IHD and ischaemic stroke (Fig. 5), accounting for a third of deaths from these causes. From 1990 to 2017, the number of deaths caused by IHD and ischaemic stroke that were attributable to high non-HDL cholesterol increased by around 910,000 globally. This increase was a net result of a large decrease in western countries, from 950,000 (890,000–990,000) to 480,000 (430,000–530,000), and a large increase throughout Asia. In particular, the number of deaths attributable to high non-HDL cholesterol more

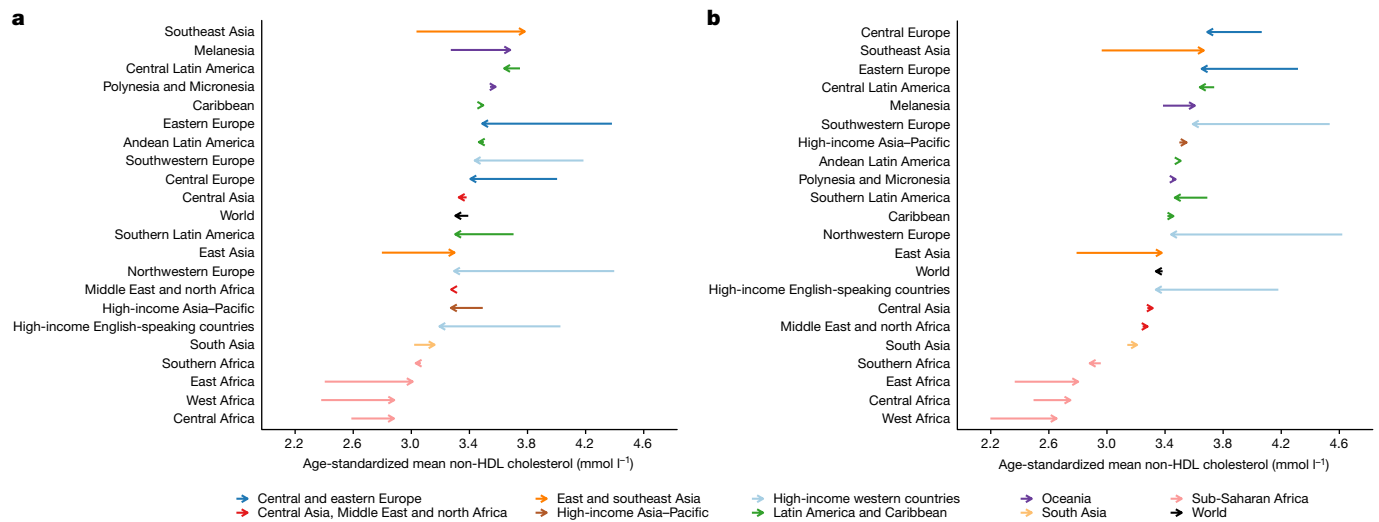


Fig. 2 | Change in age-standardized mean non-HDL cholesterol between 1980 and 2018 by region for women and men. a, Age-standardized mean non-HDL cholesterol in women. b, Age-standardized mean non-HDL

cholesterol in men. The start of the arrow shows the level in 1980 and the head indicates the level in 2018. See Extended Data Fig. 3 for age-standardized mean HDL cholesterol. One mmol l^{-1} is equivalent to 38.61 mg dl^{-1} .

than tripled in east Asia, from 250,000 (230,000–270,000) to 860,000 (770,000–940,000), and more than doubled in southeast Asia, from 110,000 (100,000–120,000) to 310,000 (290,000–330,000). As a

result, by 2017 east, southeast and south Asia accounted for half of all deaths attributable to high non-HDL cholesterol, compared with a quarter in 1990.

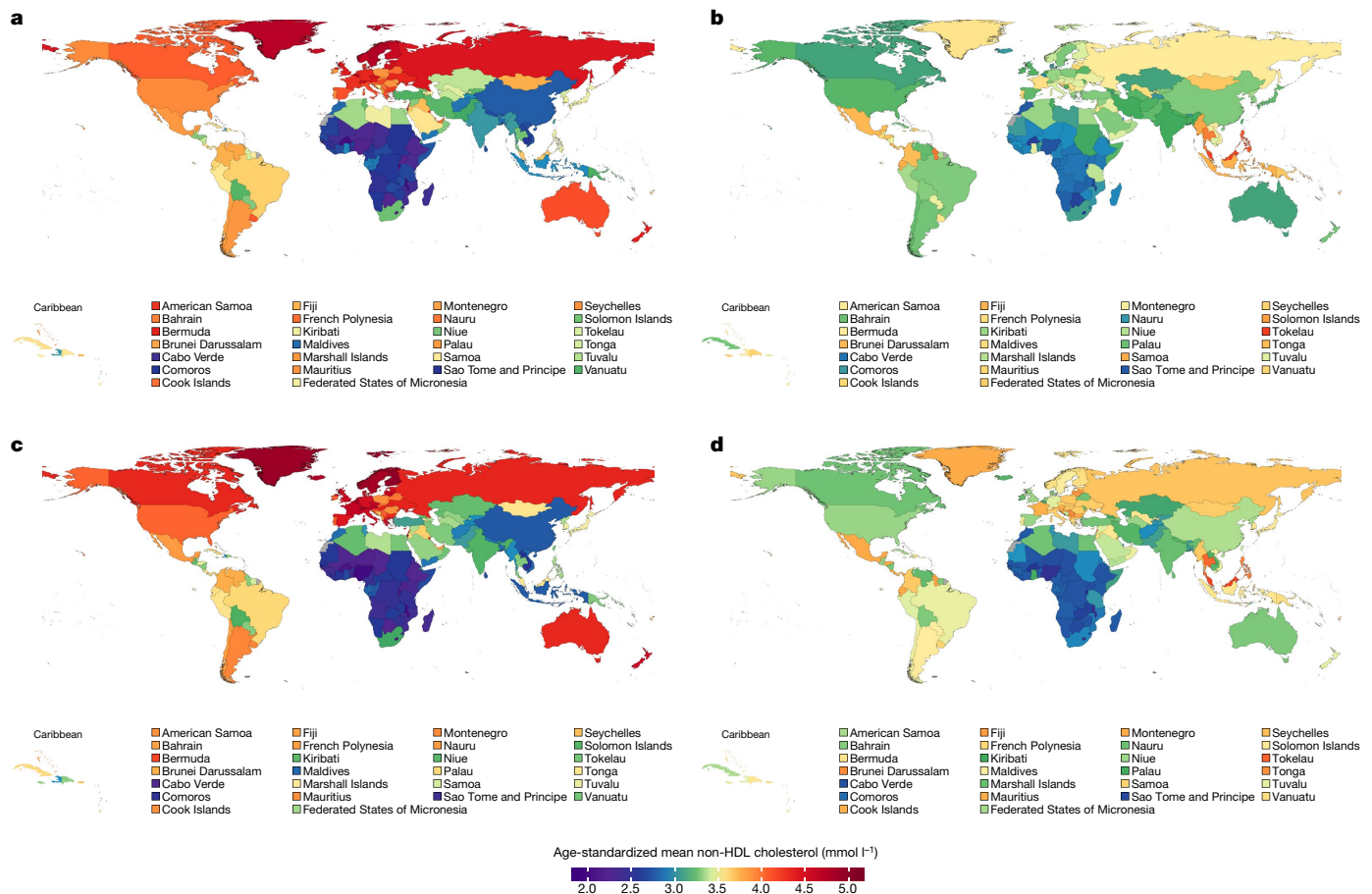


Fig. 3 | Age-standardized mean non-HDL cholesterol by country in 1980 and 2018 for women and men. a, Age-standardized mean non-HDL cholesterol in women in 1980. b, Age-standardized mean non-HDL cholesterol in women in 2018. c, Age-standardized mean non-HDL cholesterol in men in 1980. d, Age-standardized mean non-HDL cholesterol in men in 2018. See Extended

Data Fig. 5 for age-standardized mean total cholesterol and Extended Data Fig. 6 for age-standardized mean HDL cholesterol. One mmol l^{-1} is equivalent to 38.61 mg dl^{-1} .

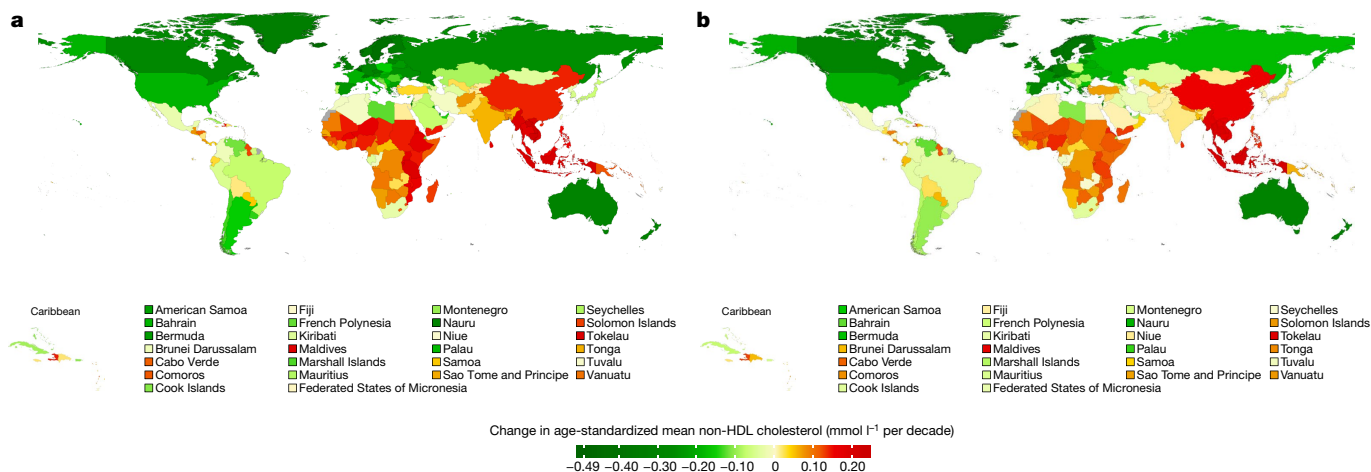


Fig. 4 | Change in age-standardized mean non-HDL cholesterol per decade by country for women and men. a, Change per decade in age-standardized mean non-HDL cholesterol in women. **b,** Change per decade in age-standardized mean non-HDL cholesterol in men. See Extended Data Fig. 7

for change per decade in age-standardized mean total cholesterol and Extended Data Fig. 8 for change per decade in age-standardized mean HDL cholesterol. One mmol l^{-1} is equivalent to 38.61 mg dl^{-1} .

Implications

Our results show that over the past nearly four decades, there has been a major global repositioning of lipid-related risk, with non-optimal cholesterol patterns shifting from being a distinct feature of high-income countries in northwestern Europe, north America and Australasia to one that affects middle-income countries in east and southeast Asia, as well as some countries in Oceania and central Latin America. This transition is especially noticeable for non-HDL cholesterol, which had not been quantified previously in a global analysis. This global repositioning has occurred as a consequence of opposing trends in high-income western countries and in Asia, which has led to some Asian countries having the highest worldwide non-HDL cholesterol levels in 2018.

The decrease in non-HDL cholesterol in western countries started in the 1980s, before statins were widely used^{15,16}. This indicates that changes in diet, especially the replacement of saturated with unsaturated fats^{3,17–21} and reduction in trans fats^{12,17,22}, are major contributors to this decline. Nonetheless, the increased use of statins from the late 1990s onwards^{15,16}, may explain up to one half of the decrease in those countries in which statins are widely used^{19,23,24}. In contrast to high-income western countries, the consumption of animal-source foods, refined carbohydrates and palm oil has increased substantially in east and southeast Asia^{3,25,26}, where statin use remains low^{13,27}. For example, the Pearson correlation coefficient between the change in non-HDL cholesterol and the change in a multi-dimensional score of animal-source foods and sugar³ was 0.69 for women and 0.67 for

men using data from high-income western countries and countries in east and southeast Asia, the two regions that experienced the largest decrease and increase, respectively, in non-HDL cholesterol levels. Finally, changes in diet, especially a decrease in carbohydrate and an increase in fat intake^{28–31}, may have contributed to the large increase in HDL cholesterol observed in the high-income Asia–Pacific region, where there was little increase in overweight and obesity relative to other regions^{8,9}. By contrast, the large increase in diabetes³² and adiposity⁸ in Oceania may have contributed to the decrease in HDL cholesterol in this region. The Pearson correlation coefficient between the change in HDL cholesterol and the change in body-mass index⁸ was -0.87 for women and -0.69 for men using countries in the high-income Asia–Pacific region and Oceania, the two regions that had the largest increase and decrease, respectively, in HDL cholesterol; the Pearson correlation coefficient for the change in HDL cholesterol and change in diabetes prevalence³² was -0.84 for women and -0.69 for men. In the same regions, the Pearson correlation coefficient between the change in non-HDL cholesterol and the change in body-mass index⁸ was 0.77 for women and 0.62 for men; for the change in non-HDL cholesterol and the change in diabetes prevalence³², the Pearson correlation coefficient was 0.54 for women and 0.40 for men.

Although it has previously been documented that the prevalence of adiposity^{8,9}, diabetes³² and high blood pressure³³ is now higher in low- and middle-income countries than in high-income countries, higher cholesterol is commonly considered to be a feature of affluent western nations^{1,2}. We show that, when focusing on non-HDL cholesterol,

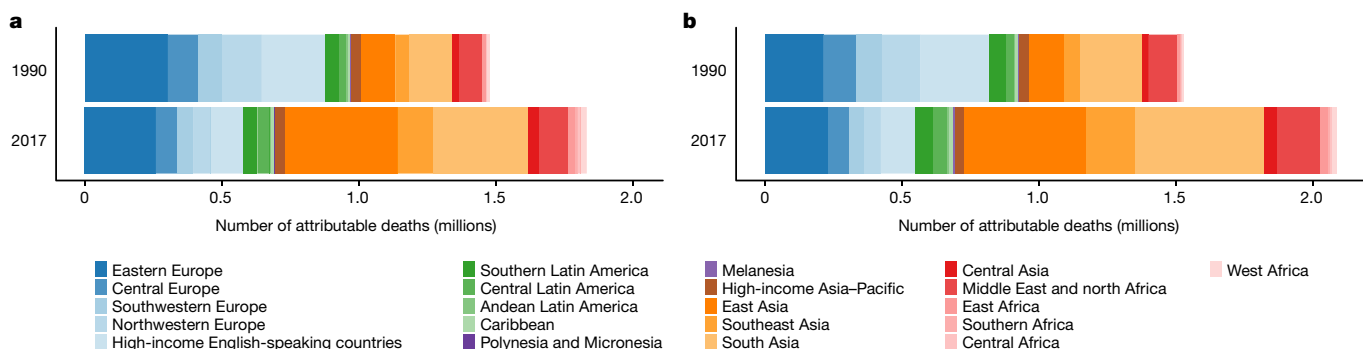


Fig. 5 | Deaths from IHD and ischaemic stroke attributable to high non-HDL cholesterol by region in 1990 and 2017 for women and men. a, Deaths in women attributable to high non-HDL cholesterol. **b,** Deaths in men attributable to high non-HDL cholesterol.


middle-income countries have emerged as the new global epicentre of non-optimal cholesterol as they did for other major cardiovascular disease risk factors, indicating that there is no such a thing as a western risk factor. At the same time, the populations of high-income countries would also benefit from further lowering non-HDL cholesterol. Therefore, population-based policies and personal interventions to improve nutrition and enhance treatment are now needed in all countries, especially as a part of the movement towards universal health coverage.

Online content

Any methods, additional references, Nature Research reporting summaries, source data, extended data, supplementary information, acknowledgements, peer review information; details of author contributions and competing interests; and statements of data and code availability are available at <https://doi.org/10.1038/s41586-020-2338-1>.

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Article

NCD Risk Factor Collaboration (NCD-RisC)

Cristina Taddei¹, Bin Zhou¹, Honor Bixby¹, Rodrigo M. Carrillo-Larco¹, Goodarz Danaei², Rod T. Jackson³, Farshad Farzadfar⁴, Marisa K. Sophiea⁴, Mariachiara Di Cesare⁵, Maria Laura Caminia Iurilli¹, Andrea Rodriguez Martinez⁶, Golaleh Ashgari⁶, Klodian Dhana⁷, Pablo Gulayin⁸, Sujay Kakarmath⁹, Marilina Santero⁸, Trudy Voortman¹⁰, Leanne M. Riley¹¹, Melanie J. Cowan¹¹, Stefan Savin¹¹, James E. Bennett¹, Gretchen A. Stevens¹¹², Christopher J. Paciorek¹³, Wichai Aekplakorn¹⁴, Renata Cifkova^{15,16}, Simona Giampaoli¹⁷, Andre Pascal Kengne¹⁸, Young-Ho Khang¹⁹, Kari Kuulasmaa²⁰, Avula Laxmaiah²¹, Paula Margozzini²², Prashant Mathur²³, Børge G. Nordestgaard²⁴, Dong Zhao²⁵, Mette Aadahl²⁶, Leandra Abarca-Gómez²⁷, Hanan Abdul Rahim²⁸, Niveen M. Abu-Rmeileh²⁹, Benjamin Acosta-Cazares³⁰, Robert J. Adams³¹, Imelda A. Agdeppa³², Javad Aghazadeh-Attari³³, Carlos A. Aguilar-Salinas³⁴, Charles Agyemang³⁵, Tarunveer S. Ahluwalia³⁶, Noor Ani Ahmad³⁷, Ali Ahmad³⁸, Naser Ahmadi⁴, Soheir H. Ahmed³⁹, Wolfgang Ahrens⁴⁰, Kamel Ajlouni⁴¹, Monira Alarouj⁴², Fadia AlBuhairan⁴³, Shahla AlDhukair⁴⁴, Mohamed M. Al¹¹, Abdullah Alkandari⁴², Ala'a Alkerwi⁴⁵, Eman Aly⁴⁶, Deepak N. Amarapurkar⁴⁷, Philippe Amouey^{48,49}, Lars Bo Andersen⁵⁰, Sigmund A. Anderssen⁵¹, Ranjit Mohan Anjana⁵², Alireza Ansari-Moghaddam⁵³, Hajer Aounallah-Skhiri⁵⁴, Joana Araujo⁵⁵, Inger Ariansen⁵⁶, Tahir Aris⁵⁷, Raphael E. Arku⁵⁷, Nimmathota Arlappa²¹, Krishna K. Aryal⁵⁸, Thor Aspelund⁵⁹, Maria Cecilia F. Assunção⁶⁰, Juha Auvinen^{61,62}, Mária Avdicova⁶³, Ana Azevedo⁶⁴, Fereidoun Azizi⁶⁵, Mehrdad Azmin⁴, Nagalla Balakrishna²¹, Mohamed Bamoshmooh⁶⁶, Maciej Banach⁶⁷, Piotr Bandosz⁶⁸, José R. Banegas⁶⁹, Carlo M. Barbagallo⁷⁰, Alberto Barceló⁷¹, Amina Barkat⁷², Iqbal Bata⁷³, Anwar M. Batieha⁷⁴, Assembekov Batyrbek⁷⁵, Louise A. Baur⁷⁶, Robert Beaglehole³, Antonisamy Belavendra⁷⁷, Habiba Ben Romdhane⁷⁸, Mikhail Benet⁷⁹, Marianne Benn²⁴, Salim Berkinbayev⁸⁰, Antonio Bernabe-Ortiz⁸¹, Gailute Bernotiene⁸², Heloisa Bettio⁸³, Santosh K. Bhargava⁸⁴, Yufang Bi⁸⁵, Asako Bienek⁸⁶, Mukharram Bikbov⁸⁷, Bihungum Bista⁸⁸, Peter Bjerregaard⁸⁹, Espen Bjertness³⁹, Marius B. Bjertness³⁹, Cecilia Björkelund⁹⁰, Katia V. Bloch⁹¹, Anneke Blokstra⁹², Simona Bo⁹³, Bernhard O. Boehm⁹⁴, Jose G. Boggia⁹⁵, Carlos P. Boissonnet⁹⁶, Marialaura Bonaccio⁹⁷, Vanina Bongard⁹⁸, Rossana Borchini⁹⁹, Herman Borghs¹⁰⁰, Pascal Bovet^{101,102}, Imperia Brajkovich¹⁰³, Juergen Breckenkamp¹⁰⁴, Hermann Brenner¹⁰⁵, Lizzy M. Brewster³⁵, Graziella Bruno⁹³, Anna Bugge¹⁰⁶, Markus A. Busch¹⁰⁷, Antonio Cabrera de León¹⁰⁸, Joseph Cacciatolo¹⁰⁹, Günay Can¹¹⁰, Ana Paula C. Cândido¹¹¹, Mario V. Capazana³², Eduardo Capuano¹¹², Vincenzo Capuano¹¹², Viviane C. Cardoso⁸³, Joana Carvalho¹¹³, Felipe F. Casanueva¹¹⁴, Laura Censi¹¹⁵, Charalambos A. Chadjiorgiou¹¹⁶, Snehalatha Chamukuttan¹¹⁷, Nish Chattervedi¹¹⁸, Chien-Jen Chen¹¹⁹, Fangfang Chen¹²⁰, Shuohua Chen¹²¹, Ching-Yu Cheng¹²², Bahman Chergaghian¹²³, Angela Chetrit¹²⁴, Shu-Ti Chiou¹²⁵, Maria-Dolores Chirlaque¹²⁶, Belong Cho¹²⁷, Yumi Cho¹²⁸, Jerzy Chudek¹²⁹, Frank Claessens¹³⁰, Janine Clarke¹³¹, Els Clays¹³², Hans Concin¹³³, Susana C. Confortin¹³⁴, Cyrus Cooper¹³⁵, Simona Costanzo⁹⁷, Dominique Cotte¹³⁶, Chris Cowell⁷⁶, Ana B. Crujeiras¹³⁷, Semánová Csilla¹³⁸, Liufu Cui¹²¹, Felipe V. Cureau¹³⁹, Graziella D'Arrigo¹⁴⁰, Eleonora d'Orsi¹⁴¹, Jean Dallongeville¹³⁶, Albertino Damasceno¹⁴², Rachel Dankner¹²⁴, Thomas M. Dantoft²⁶, Luc Dauchet^{48,49}, Kairat Davletov⁷⁵, Guy De Backer¹³², Dirk De Bacquer¹³², Giovanni de Gaetano⁹⁷, Stefaan De Henaux¹³², Paula Duarte de Oliveira⁶⁰, David De Ridder¹⁴³, Delphine De Smedt¹³², Mohan Deepa⁵², Alexander D. Deev¹⁴⁴, Abbas Dehghan¹, Hélène Delisle¹⁴⁵, Elaine Dennison¹³⁵, Valérie Deschamps¹⁴⁶, Meghnath Dhimal¹⁴⁸, Augusto F. Di Castelnuovo¹⁴⁷, Zivka Dika¹⁴⁸, Shirin Djalalinia¹⁴⁹, Annette J. Dobson¹⁵⁰, Chiara Donfrancesco¹⁷, Silvana P. Donoso¹⁵¹, Angela Döring¹⁵², Maria Dorobantu¹⁵³, Nico Dragano¹⁵⁴, Wojciech Drygas^{67,155}, Yong Du¹⁰⁷, Charmaine A. Duante³², Rosemary B. Duda¹⁵⁶, Vilnis Dzerve¹⁵⁷, Elzbieta Dzionkowska-Zaborszczyk⁶⁷, Ricky Eddie¹⁵⁸, Ebrahim Eftekhar¹⁵⁹, Robert Eggertsen⁹⁰, Sareh Eghtesad⁴, Gabriele Eiben¹⁶⁰, Ulf Ekelund⁶¹, Jalila El Atti¹⁶¹, Denise Eldemire-Shearer¹⁶², Marie Eliassen²⁶, Roberto Elosua¹⁶³, Rajiv T. Erasmus¹⁶⁴, Raimund Erbel¹⁶⁵, Cihangir Erem¹⁶⁶, Louise Eriksen⁶⁹, Johan G. Eriksson¹⁶⁷, Jorge Escobedo-de la Peña³⁰, Saeid Esлами¹⁶⁸, Ali Esmaeili¹⁶⁹, Alun Evans¹⁷⁰, David Faeh¹⁷¹, Caroline H. Fall¹³⁵, Elnaz Faramarzi¹⁷², Mojtaba Farjam¹⁷³, Mohammad Reza Fattahi¹⁷⁴, Francisco J. Felix-Redondo¹⁷⁵, Trevor S. Ferguson¹⁶², Daniel Fernández-Bergés¹⁷⁶, Daniel Ferrante¹⁷⁷, Marika Ferrari¹¹⁵, Caterina Ferreccio²², Jean Ferrieres⁹⁸, Bernhard Föger¹³³, Leng Huat Foo¹⁷⁸, Ann-Sofie Forsslund¹⁷⁹, Maria Franco¹⁷⁹, Heba M. Fouad¹⁸⁰, Damian K. Francis¹⁸², Maria do Carmo Franco¹⁸⁰, Oscar H. Franco¹⁰, Guillermo Frontera¹⁸¹, Yuki Fujita¹⁸², Matsuda Fumihiko¹⁸³, Takuro Furusawa¹⁸³, Zbigniew Gaciong¹⁸⁴, Fabio Galvano¹⁸⁵, Jingli Gao¹²¹, Manoli Garcia-de-la-Hera¹⁸⁶, Sarah P. Garnett⁷⁶, Jean-Michel Gaspoz¹⁴³, Magda Gasull¹⁸⁷, Andrea Gazzinelli¹⁸⁸, Johanna M. Geleijnse¹⁸⁹, Ali Ghanbari⁴, Erfan Ghasemi⁴, Oana-Florentina Gheorghe-Fronea¹⁵³, Anup Ghimire¹⁹⁰, Francesco Gianfagna^{147,191}, Tiffany K. Gill¹⁹², Jonathan Giovannelli^{48,49}, Glen Gironella³², Aleksander Givercman¹⁹³, David Goltzman¹⁹⁴, Helen Gonçalves⁶⁰, David A. Gonzalez-Chica¹⁹², Marcela Gonzalez-Gross¹⁹⁵, Juan P. González-Rivas¹⁹⁶, Clicerio González-Villalpando¹⁹⁷, Maria-Elena González-Villalpando¹⁹⁸, Angel R. Gonzalez¹⁹⁹, Frederic Gottrand⁴⁸, Sidsel Graff-Iversen⁵⁶, Dušan Grafnetter²⁰⁰, Ronald D. Gregor⁷³, Tomasz Grodzicki²⁰¹, Anders Grøntved²⁰², Giuseppe Grosso¹⁸⁵, Gabriella Gruden⁹³, Dongfeng Gu²⁰³, Pilar Guallar-Castillón⁶⁹, Ong Peng Guan²⁰⁴, Elias F. Gudmundsson²⁰⁵, Vilhelmur Gudnason⁵⁹, Ramiro Guerrero²⁰⁶, Idris Gueouss¹⁴³, Johanna Gunnlaugsdottir²⁰⁵, Rajeev Gupta²⁰⁷, Laura Gutierrez⁸, Felix Gutzwiller¹⁷¹, Seongjun Ha²⁰⁸, Farzad Hadaegh²⁰⁹, Rosa Haghsheenas⁴, Hamid Hakim¹⁶⁹, Ian R. Hambleton²¹⁰, Behrooz Hamzeh²¹¹, Sari Hantunen²¹², Rachakulla Hari Kumar²¹, Seyed Mohammad Hashemi-Shahri⁵³, Jun Hata²¹³, Teresa Haugsgjerd²¹⁴, Alison J. Hayes⁷⁶, Jiang He²¹⁵, Yuna He²¹⁶, Marleen Elisabeth Hendriks²¹⁷, Ana Henriques⁵⁵, Sauli Herralá⁶², Ramin Heshmat²¹⁸, Allan G. Hill¹³⁵, Sai Yin Ho²¹⁹, Suzanne C. Ho²²⁰, Michael Hobbs²²¹, Albert Hofman¹⁰, Reza Homayounfar¹⁷³, Wilma M. Hopman²²², Andrea R. V. R. Horimoto²²³, Claudia M. Hormiga²²⁴, Bernardo L. Horta⁶⁰, Leila Houti²²⁵, Christina Howitt²¹⁰, Thein Thein Htay²²⁶, Aung Soe Htet²²⁷, Maung Maung Than Htike²²⁷,

José María Huerta²²⁸, Ilpo Tapani Huhtaniemi¹, Martijn Huisman²²⁹, Monica L. Hunsberger⁹⁰, Abdullatif S. Hussein²⁹, Inge Huybrechts²³⁰, Nahla Hwalla²³¹, Licia Iacoviello^{97,191}, Anna G. Iannone¹¹², Mohsen M. Ibrahim²³², Norazizah Ibrahim Wong³⁷, Iris Iglesias²³³, Nayu Ikeda²³⁴, M. Arfan Ikram¹⁰, Violeta Iotova²³⁵, Vilma E. Irazola⁸, Takafumi Ishida²³⁶, Muhammad Islam²³⁷, Aziz al-Safi Ismail⁷⁸, Masanori Iwasaki²³⁸, Jeremy M. Jacobs²³⁹, Hashem Y. Jaddou²⁴, Tazeen Jafar¹²², Kenneth James¹⁶², Konrad Jamrozik^{192,448}, Imre Janszky²⁴⁰, Edward Janus²⁴¹, Marjo-Riitta Jarvelin^{161,62}, Grazyna Jasienska²⁰¹, Ana Jelakovic²⁴², Bojan Jelakovic²⁴³, Garry Jennings²⁴⁴, Gorm B. Jensen²⁴, Seung-lyeal Jeong²⁰⁸, Anjani Kumar Jha⁸⁸, Chao Qiang Jiang²⁴⁵, Ramon O. Jimenez²⁴⁶, Karl-Heinz Jöckel¹⁶⁵, Michel Joffres²⁴⁷, Jari J. Jokelainen⁶², Jost B. Jonas²⁴⁸, Torben Jørgensen²⁶, Pradeep Joshi²⁴⁹, Farahnaz Joukar²⁵⁰, Jacek Józwiak²⁵¹, Anne Juolevi²⁰, Anthony Kafatos²⁵², Eero O. Kajantie²⁰, Ofra Kalter-Leibovici¹²⁴, Nor Azmi Kamaruddin²⁵³, Pia R. Kamstrup²⁴, Khem B. Karki²⁵⁴, Joanne Katz²⁵⁵, Jussi Kauhanen²¹², Prabhdeep Kaur²⁵⁶, Maryam Kavousi¹⁰, Gylli Kazakbaeva⁸⁷, Ulrich Keil²⁵⁷, Sirkka Keinänen-Kiukaanniemi⁶², Roya Kelishadi²⁵⁸, Maryam Keramati¹⁶⁸, Alina Kerimkulova²⁵⁹, Mathilde Kersting²⁶⁰, Yousef Saleh Khader²⁷, Davood Khalil⁶⁵, Mohammad Khatieb⁴¹, Motahareh Kheradmand²⁶¹, Alireza Khosravi²⁶², Ursula Kiechl-Kohlendorfer²⁶³, Stefan Kiechl²⁶³, Japhet Killewo²⁶⁴, Hyeon Chang Kim²⁶⁵, Jeongseon Kim²⁶⁶, Yeon-Yong Kim²⁰⁸, Jurate Klumbiene⁶², Michael Knoflach²⁶³, Stephanie Ko⁸⁶, Hans-Peter Kohler²⁶⁷, Iliana V. Koohe²⁶⁷, Elin Kolle⁵¹, Patrick Kolsteren¹³², Jürgen König²⁶⁸, Rajja Korpelainen^{61,269}, Paul Korrovtis²⁷⁰, Jelena Kos²⁴², Seppo Koskinen²⁰, Katsuyasu Kouda²⁷¹, Sudhir Kowlessur²⁷², Wolfgang Kratzer²⁷³, Susi Kriemler¹⁷¹, Peter Lund Kristensen²⁰², Steiner Krokstad²⁴⁰, Daan Kromhout²⁷⁴, Urho M. Kujala²⁷⁵, Pawel Kurjata¹⁶⁵, Catherine Kyobutungi²⁷⁶, Fatima Zahra Laamir²⁷⁷, Tiina Laatikainen²⁰, Carl Lachat³², Youcef Laid²⁷⁸, Tai Hing Lam²¹⁹, Christina-Paulina Lambrinou²⁷⁹, Vera Lanska²⁸⁰, Bagher Larijani²⁸¹, Tint Swe Latt²⁸², Lars E. Laugsand²⁴⁰, Maria Lazo-Porras⁸¹, Jeannette Lee²⁸³, Jeonghee Lee²⁸⁶, Nils Lehmann¹⁶⁵, Terho Lehtimäki^{284,285}, Naomi S. Levitt²⁸⁶, Yanping Li², Christina L. Lilly²⁸⁷, Wei-Yen Lim²⁸⁷, M. Fernanda Lima-Costa²⁸⁸, Hsien-Ho Lin²⁸⁹, Xu Lin²⁹⁰, Yi-Ting Lin²⁹¹, Lars Lind²⁹¹, Allan Linneberg²⁶, Lauren Lissner⁹⁰, Jing Liu²⁵, Helle-Mai Loit²⁹², Esther Lopez-Garcia⁶⁹, Tania Lopez²⁹³, Paulo A. Lotufo⁸³, José Eugenio Lozano²⁹⁴, Dalia Luksiene⁸², Annamari Lundqvist²⁰, Robert Lundqvist²⁹⁵, Nuno Lunet¹¹³, Guansheng Ma²⁹⁶, George L. L. Machado-Coelho²⁹⁷, Aristides M. Machado-Rodrigues²⁹⁸, Suka Machi²⁹⁹, Ahmed A. Madar³⁹, Stefania Magg³⁰⁰, Dianna J. Magliano³⁰¹, Emmanuella Magriplis³⁰², Gowri Mahasampath⁷⁷, Bernard Maire³⁰³, Marcia Makdisse³⁰⁴, Fatemeh Malekzadeh¹⁷⁴, Reza Malekzadeh⁴, Kodavanti Mallikharjuna Rao²¹, Yannis Manios²⁷⁹, Jim I. Mann³⁰⁵, Fariborz Mansour-Ghanaei²⁵⁰, Enzo Manzato³⁰⁶, Pedro Marques-Vidal³⁰⁷, Reynaldo Martorell³⁰⁸, Luis F. Mascarenhas³⁰⁹, Ellisiv B. Mathiesen³¹⁰, Tandi E. Matsha³¹¹, Christina Mavrogiani²⁷⁹, Shelly R. McFarlane¹⁶², Stephen T. McGarvey³¹², Stella McLachlan³¹³, Rachael M. McLean³⁰⁵, Scott B. McLean¹³¹, Breige A. McNulty³¹⁴, Sounnia Mediene-Benchechor²²⁵, Parinaz Mehdipour⁴, Kirsten Mehlig⁹⁰, Amir Houshang Mehrparvar³¹⁵, Aline Meirhaeghe³¹⁶, Christa Mense¹⁵², Ana Maria B. Menezes⁶⁰, Geetha R. Menon³¹⁷, Shahin Merat⁴, Alibek Mereke⁷⁵, Indrapal I. Meshram²¹, Patricia Metcalfe³, Haakon E. Meyer³⁹, Jie Mi¹²⁰, Nathalie Michels¹³², Jody C. Miller³⁰⁵, Cláudia S. Minderico³¹⁸, G. K. Mini³¹⁹, Juan Francisco Miquel²², J. Jaime Miranda⁸¹, Mohammad Reza Mirjalili³¹⁵, Erkin Mirrahimov²⁵⁹, Pietro A. Modesti³²⁰, Sahar Saedi Moghadam⁴, Bahram Mohajer⁴, Mostafa K. Mohamed³²¹, Kazem Mohammad³, Zahra Mohammad³²², Noushin Mohammadi³²², Reza Mohammadpourhodki¹⁶⁸, Viswanathan Mohan⁵², Salim Mohanna⁸¹, Muhammad Fadhli Mohd Yusoff³⁷, Iraj Mohebbi³²³, Farnam Mohebi⁴, Marie Moitry^{323,324}, Line T. Møllehave²⁶, Niels C. Møller²⁰², Dénes Molnár³²⁵, Amirabbas Momen⁶, Charles K. Mondo³²⁶, Eric Monterubbio-Flores¹⁹⁷, Mahmood Moosazadeh²⁶¹, Alain Morejon³²⁷, Luis A. Moreno²³³, Karen Morgan³²⁸, Suzanne N. Morin¹⁹⁴, George Moschonis³²⁹, Malgorzata Mossakowska³³⁰, Aya Mostafa³²¹, Jorge Mota¹¹³, Mohammad Esmael Mottagh¹²³, Jorge Motta³³¹, Keliás P. Msyamboza³³², Maria L. Muiésan³³³, Martina Müller-Nurasyid¹³², Jaako Mursu²¹², Norlaila Mustafa³³⁴, Iraj Nabipour³³⁴, Shohreh Naderimagham⁴, Gabriele Nagel³³⁵, Balkish M. Naidu³⁷, Farid Najafi²¹¹, Harunobu Nakamura³³⁶, Jana Námesná⁶³, El Ei K. Nang²⁸³, Vinay B. Nangia³³⁷, Matthias Nauck³³⁸, William A. Neal²⁸⁷, Azim Nejatizadeh¹⁶⁹, Ilona Nenko²⁰¹, Flavio Nervy²², Nguyen D. Nguyen³³⁹, Quang Ngoc Nguyen³⁴⁰, Ramfis E. Nieto-Martinez³⁴¹, Thomas Nihai¹⁷⁷, Teemu J. Niiranen^{20,342}, Guang Ning⁶⁵, Toshiharu Niomiya²¹⁵, Marianna Noale³⁰⁰, Oscar A. Noboa⁹⁵, Davide Noto⁷⁰, Mohannad Al Nsour³⁴³, Irfan Nuhoğlu¹⁶⁶, Terence W. O'Neill³⁴⁴, Dermot O'Reilly¹⁷⁰, Angélica M. Ochoa-Avilés¹⁵¹, Kyungwon Oh¹²⁸, Rytarou Ohtsuka³⁴⁵, Örn Olafsson²⁰⁵, Valérie Olié¹⁴⁶, Isabel O. Oliveira⁶⁰, Mohd Azahadi Omar³⁷, Altan Onat^{346,448}, Sok King Ong²⁴⁷, Pedro Orduñez²¹, Rui Ornelas³⁴⁸, Pedro J. Ortiz³¹, Clive Osmond³⁴⁹, Sergej M. Ostojic³⁵⁰, Afshin Ostovar⁴, Johanna A. Otero²²⁴, Ellis Owusu-Dabo³⁵¹, Fred Michel Paccou³⁵², Elena Pahomova⁵⁷, Andrzej Pajak²⁰¹, Luigi Palmieri¹⁷, Wen-Harn Pan¹¹⁹, Songhomitra Panda-Jonas²⁴⁸, Francesco Panza³⁵³, Winsome R. Parnell³⁰⁵, Nikhil D. Patel³⁵⁴, Nasheda Peir³⁵⁵, Sergio Viana Peixoto²⁸⁸, Markku Peltonen²⁰, Alexandre C. Pereira²²³, Annette Peters¹⁵², Astrid Petersmann³³⁸, Janina Petkeviciene⁸², Niloofar Peykari¹⁴⁹, Son Thai Pham³⁵⁶, Rafael N. Pichardo³⁵⁷, Iris Pigeon³⁵⁸, Aida Pilav³⁵⁹, Lorenza Pilotto³⁶⁰, Aleksandra Piwonka¹⁵⁵, Andrea N. Pizarro¹¹³, Pedro Plans-Rubió³⁶¹, Silvia Plata³⁶², Hermann Pohlabein³⁵⁸, Miguel Porta¹⁶³, Marileen L. P. Portegies¹⁰, Anil Poudyal¹⁸⁸, Farhad Pourfarzi³⁶³, Hossein Poustchi⁴, Rajendra Pradeepa⁵², Jacqueline F. Price³¹³, Rui Providencia¹¹⁸, Jardena J. Puder³⁰⁷, Soile E. Puhakka^{61,269}, Margus Punab²⁷⁰, Mostafa Qorbani³⁶⁴, Tran Quoc Bao³⁶⁵, Ricardas Radisauskas⁸², Salar Rahimkazeroni¹⁷⁴, Olli Raitakari³⁴², Sudha Ramachandra Rao²⁶⁵, Ambady Ramachandran³⁶⁶, Elisabete Ramos⁶⁴, Rafel Ramos³⁶⁷, Lekhraj Rampa³⁶⁸, Sanjay Rampa³⁶⁹, Josep Redon³⁷⁰, Paul Ferdinand M. Reganiti³⁷¹, Luis Revilla²⁹³, Abbas Rezaianzadeh¹⁷⁴, Robespierre Ribeiro^{372,448}, Adrian Richter³³⁸, Fernando Rigo³⁷³, Tobias F. Rinke de Wit³⁷⁴, Fernando Rodríguez-Artalejo⁶⁹, María del Cristo Rodríguez-Perez³⁷⁵, Laura A. Rodríguez-Villamizar³⁷⁶, Ulla Roggenbuck¹⁶⁵, Rosalba Rojas-Martinez¹⁹⁷, Dora Romaguera¹³⁷, Elisabetta L. Romeo³⁷⁷, Annika Rosengren^{90,378}, Joel G. R. Roy¹³¹, Adolfo Rubinstein⁸,

Jean-Bernard Ruidavets²⁷⁹, Blanca Sandra Ruiz-Betancourt³⁰, Paola Russo³⁸⁰, Petra Rust²⁶⁸, Marcin Rutkowski⁶⁸, Charumathi Sabanayagam²⁰⁴, Harshpal S. Sachdev³⁸¹, Alireza Sadjad¹⁴, Ali Reza Safarpour¹⁷⁴, Saeid Safiri¹⁷², Olfa Saidi³⁸², Nader Saki¹²³, Benoit Salanave¹⁴⁶, Diego Salmerón²²⁸, Veikko Salomaa²⁰, Jukka T. Salonen¹⁶⁷, Massimo Salvetti³³³, Jose Sánchez-Abanto³⁸³, Susana Sans³⁸⁴, Alba M. Santaliestra-Pasías²³³, Diana A. Santos³⁸⁵, Maria Paula Santos¹¹³, Rute Santos¹¹³, Jouko L. Saramies³⁸⁶, Luis B. Sardinha³⁸⁵, Nizal Sarrafzadegan³⁸⁷, Kai-Uwe Saum¹⁰⁵, Savvas C. Savva¹¹⁶, Norie Sawada³⁸⁸, Mariana Sbaraini¹³⁹, Marcia Sczufca³⁸⁹, Beatriz D. Schaan¹³⁹, Herman Schargrodsy³⁹⁰, Christa Scheidt-Nave¹⁰⁷, Anja Schienkiewitz¹⁰⁷, Sabine Schip²³⁸, Carsten O. Schmidt³³⁸, Ben Schöttker¹⁰⁵, Sara Schramm¹⁶⁵, Sylvain Sebert⁶¹, Aye Aye Sein²²⁷, Abhijit Sen³⁹¹, Sadaf G. Sepanlou⁴, Jennifer Servais¹³¹, Ramin Shakeri⁴, Svetlana A. Shalnova¹⁴⁴, Teresa Shamah-Levy¹⁹⁷, Maryam Sharafkahn⁴, Sanjib K. Sharma¹⁹⁰, Jonathan E. Shaw³⁰¹, Amaneh Shayanrad⁴, Zumin Shi²⁸, Kenji Shibuya³⁹², Hana Shimizu-Furusawa³⁹³, Dong Wook Shin³⁹⁴, Youchan Shin²⁰⁴, Majid Shirani³⁸, Rahman Shiri³⁹⁵, Namuna Shrestha⁸⁸, Khairil Si-Ramlee³⁴⁷, Alfonso Siani³⁹⁰, Sorayyn Siant²⁰⁴, Abba M. Siba²³¹, Diego Augusto Santos Silva⁴¹, Mary Simon³⁸⁶, Judith Simons³⁹⁶, Leon A. Simons³⁹⁷, Michael Sjöström³⁹⁸, Tea Skaaby³⁹⁹, Jolanta Slowikowska-Hilczek⁶⁷, Przemyslaw Slusarczyk³⁹⁰, Liam Smeeth⁴⁰⁰, Marieke B. Snijder³⁵, Stefan Söderberg¹⁷⁹, Agustinus Soemantri⁴⁰¹, Reecha Sofat¹¹⁸, Vincenzo Solfrizzi⁴⁰², Mohammad Hossein Somi¹⁷², Emily Sonestedt¹⁹³, Thorkild I. A. Sørensen⁴⁰³, Carlos Sossa Jérôme⁴⁰⁴, Aïcha Soumaré⁴⁰⁵, Kaan Sozmen⁴⁰⁶, Karen Sparrenberger¹³⁹, Jan A. Staessen⁴⁰⁷, Maria G. Stathopoulou⁴⁰⁸, Bill Stavreski²⁴⁴, Jostein Steene-Johannessen⁵¹, Peter Stehle⁴⁰⁹, Aryeh D. Stein³⁰⁸, Jochanan Stessman²³⁹, Ranko Stevanović⁴¹⁰, Jutta Stieber^{152,448}, Doris Stöckl¹⁵², Jakub Stokwiszewski⁴¹¹, Karlen Stronks³⁵, Maria Wany Strubler¹⁸⁰, Ramón Suárez-Medina⁴¹², Chien-An Sun⁴¹³, Johan Sundström²⁹¹, Paibul Suriyawongpaisal¹⁴, Rody G. Sy²⁷¹, René Charles Sylva⁴¹⁴, Moyses Szklo²⁵⁵, E. Shyong Tai²⁸³, Abdonas Tamosiunas⁸², Eng Joo Tan⁷⁶, Mohammed Rasoul Tarawneh⁴¹⁵, Carolina B. Tarqui-Mamani³⁸³, Anne Taylor¹⁹², Julie Taylor¹¹⁸, Grethe S. Tel²¹⁴, Anita Tello⁸¹, K. R. Thankappan⁴¹⁶, Lutgarde Thijs⁴⁰⁷, Betina H. Thuesen²⁶, Ulla Toft²⁶, Hanna K. Tolonen²⁰, Janne S. Tolstrup⁸⁹, Murat Topbas¹⁶⁶, Roman Topór-Madry²⁰¹, María José Tormo⁴¹⁷, Michael J. Tornaritis¹¹⁶, Maties Torrent⁴¹⁸, Laura Torres-Collado¹⁸⁶, Pierre Traissac³⁰³, Oanh T. H. Trinh³³⁹, Julia Truthmann¹⁰⁷, Shoichiro Tsugane³⁸⁸, Marshall K. Tulloch-Reid⁶², Tomi-Pekka Tuomainen²¹², Jaakko Tuomilehto²⁰, Anne Tybjaerg-Hansen²⁴, Christophe Tzourio⁴⁰⁵, Peter Ueda³⁹⁸, Eunice Ugel⁴¹⁹, Hanno Ulmer²⁶³, Belgin Unal⁴²⁰, Hannu M. T. Uusitalo⁴²¹, Gonzalo Valdivia²², Damaskini Valvi⁴²², Rob M. van Dam²⁸³, Yvonne T. van der Schouw⁴²³, Koen Van Herck¹³², Hoang Van Minh⁴²⁴, Lenie van Rossem⁴²⁵, Natasja M. Van Schoor²²⁹, Irene G. M. van Valkengoed³⁵, Dirk Vanderschueren¹³⁰, Diego Vanuzzo³⁶⁰, Anette Varbo²⁴, Patricia Varona-Pérez⁴¹², Senthil K. Vasar¹³⁵, Lars Vatten²⁴⁰, Tomas Vega²⁹⁴, Toomas Veidebaum²⁹², Gustavo Velasquez-Melendez¹⁸⁸, Silvia J. Venero-Fernández⁴¹², Giovanni Veronesi¹⁹¹, W. M. Monique Verschuren⁹², Cesar G. Victora⁶⁰, Dhanasari Vidiawati⁴²⁶, Lucie Viet⁹², Salvador Villalpando¹⁹⁷, Jesus Vioque⁴²⁷, Jyrki K. Virtanen²¹², Sophie Visvikis-Siest⁴⁰⁸, Bharathi Viswanathan¹⁰¹, Tiina Vlasoff⁴²⁸, Peter Vollenweide³⁰⁷, Ari Voutilainen²¹², Alisha N. Wade⁴²⁹, Aline Wagner³²³, Janet Wong⁴³⁰, Wan Mohamad Wan Bekar¹⁷⁸, Wan Nazaimoon Wan Mohamad⁴³¹, Ming-Dong Wang⁴³², Ningli Wang⁴³³, Qian Wang⁴³⁴, Ya Xing Wang⁴³⁵, Ying-Wei Wang¹²⁵, S. Goya Wannamethee¹¹⁸, Niels Wedderkopp²⁰², Wenbin Wei⁴³⁵, Peter H. Whincup⁴³⁶, Kurt Widhalm⁴³⁷, Indah S. Widyahening⁴²⁶, Andrzej Wiecek¹²⁹, Alet H. Wijga⁹², Rainford J. Wilks¹⁶², Johann Willeit²⁶³, Peter Willeit²⁶³, Tom Wilsaard³¹⁰, Bogdan Wojtyniak⁴¹¹, Roy A. Wong-McClure²⁷, Andrew Wong¹¹⁸, Tien Yin Wong¹²², Jean Woo²²⁰, Mark Woodward^{397,438}, Frederick C. Wu³⁴⁴, Shouling Wu¹²¹, Haiquan Xu⁴³⁹, Liang Xu⁴³³, Weili Yan⁴⁴⁰, Xiaoguang Yang²¹⁶, Tabara Yasuharu¹⁸³, Xingwang Ye²⁹⁰, Toh Peng Yeow⁴⁴¹, Panayiotis K. Yiallourou⁴⁴², Moein Yoosof⁴, Akihiro Yoshihara²³⁸, San-Lin You⁴¹³, Novie O. Younger-Coleman¹⁶², Ahmad Fawzi Yusoff⁷, Ahmad A. Zainuddin³⁷, Seyed Rasoul Zakavi⁶⁸⁸, Mohammad Reza Zali⁸, Farhad Zamani⁴⁴³, Sabina Zambon³⁰⁶, Antonis Zampelas³⁰², Ko Ko Zaw²⁸², Tomasz Zdrojewski⁶⁸, Tajana Zeljkovic Vrkic²⁴², Zhen-Yu Zhang⁴⁰⁷, Wenhua Zhao²¹⁶, Shiqi Zhen⁴⁴⁴, Yingfeng Zheng⁴⁴⁵, Bekbolat Zholdin⁴⁴⁶, Baurzhan Zhussupov⁸⁰, Nada Zoghlimi⁵⁴, Julio Zuñiga Cisneros³³¹, Edward W. Gregg¹ & Majid Ezzati^{1,447,53}

¹Imperial College London, London, UK. ²Harvard T. H. Chan School of Public Health, Boston, MA, USA. ³University of Auckland, Auckland, New Zealand. ⁴Tehran University of Medical Sciences, Tehran, Iran. ⁵Middlesex University, London, UK. ⁶Shahid Beheshti University of Medical Sciences, Tehran, Iran. ⁷Rush University Medical Center, Chicago, IL, USA. ⁸Institute for Clinical Effectiveness and Health Policy, Buenos Aires, Argentina. ⁹Harvard Medical School, Boston, MA, USA. ¹⁰Erasmus Medical Center Rotterdam, Rotterdam, The Netherlands. ¹¹World Health Organization, Geneva, Switzerland. ¹²Independent researcher, Los Angeles, CA, USA. ¹³University of California Berkeley, Berkeley, CA, USA. ¹⁴Mahidol University, Nakhon Pathom, Thailand. ¹⁵Charles University in Prague, Prague, Czech Republic. ¹⁶Thomayer Hospital, Prague, Czech Republic. ¹⁷Istituto Superiore di Sanità, Rome, Italy. ¹⁸South African Medical Research Council, Cape Town, South Africa. ¹⁹Seoul National University, Seoul, Republic of Korea. ²⁰Finnish Institute for Health and Welfare, Helsinki, Finland. ²¹ICMR – National Institute of Nutrition, Hyderabad, India. ²²Pontificia Universidad Católica de Chile, Santiago, Chile. ²³ICMR – National Centre for Disease Informatics and Research, Bengaluru, India. ²⁴Copenhagen University Hospital, Copenhagen, Denmark. ²⁵Capital Medical University Beijing An Zhen Hospital, Beijing, China. ²⁶Bispebjerg and Frederiksberg Hospital, Copenhagen, Denmark. ²⁷Caja Costarricense de Seguro Social, San José, Costa Rica. ²⁸Qatar University, Doha, Qatar. ²⁹Birzeit University, Birzeit, Palestine. ³⁰Instituto Mexicano del Seguro Social, Mexico City, Mexico. ³¹Flinders University, Adelaide, South Australia, Australia. ³²Food and Nutrition Research Institute, Taigu, The Philippines. ³³Urmia University of Medical Sciences, Urmia, Iran. ³⁴Instituto Nacional de Ciencias Médicas y Nutrición, Mexico City,

Mexico. ³⁵University of Amsterdam, Amsterdam, The Netherlands. ³⁶Steno Diabetes Center Copenhagen, Gentofte, Denmark. ³⁷Ministry of Health Malaysia, Kuala Lumpur, Malaysia. ³⁸Shahrekord University of Medical Sciences, Shahrekord, Iran. ³⁹University of Oslo, Oslo, Norway. ⁴⁰University of Bremen, Bremen, Germany. ⁴¹National Center for Diabetes, Endocrinology and Genetics, Amman, Jordan. ⁴²Dasman Diabetes Institute, Kuwait City, Kuwait. ⁴³Aldara Hospital and Medical Center, Riyadh, Saudi Arabia. ⁴⁴King Abdullah International Medical Research Center, Riyadh, Saudi Arabia. ⁴⁵Luxembourg Institute of Health, Strassen, Luxembourg. ⁴⁶World Health Organization Regional Office for the Eastern Mediterranean, Cairo, Egypt. ⁴⁷Bombay Hospital and Medical Research Centre, Mumbai, India. ⁴⁸University of Lille, Lille, France. ⁴⁹Lille University Hospital, Lille, France. ⁵⁰Western Norway University of Applied Sciences, Sogndal, Norway. ⁵¹Norwegian Institute of Sport Sciences, Oslo, Norway. ⁵²Madras Diabetes Research Foundation, Chennai, India. ⁵³Zahedan University of Medical Sciences, Zahedan, Iran. ⁵⁴National Institute of Public Health, Tunis, Tunisia. ⁵⁵Institute of Public Health of the University of Porto, Porto, Portugal. ⁵⁶Norwegian Institute of Public Health, Oslo, Norway. ⁵⁷University of Massachusetts, Amherst, MA, USA. ⁵⁸Abt Associates, Kathmandu, Nepal. ⁵⁹University of Iceland, Reykjavik, Iceland. ⁶⁰Federal University of Pelotas, Pelotas, Brazil. ⁶¹University of Oulu, Oulu, Finland. ⁶²Oulu University Hospital, Oulu, Finland. ⁶³Regional Authority of Public Health, Banská Bystrica, Slovakia. ⁶⁴University of Porto Medical School, Porto, Portugal. ⁶⁵Research Institute for Endocrine Sciences, Tehran, Iran. ⁶⁶University of Science and Technology, Sana'a, Yemen. ⁶⁷Medical University of Lodz, Lodz, Poland. ⁶⁸Medical University of Gdansk, Gdansk, Poland. ⁶⁹Universidad Autónoma de Madrid/CIBERESP, Madrid, Spain. ⁷⁰University of Palermo, Palermo, Italy. ⁷¹Pan American Health Organization, Washington, DC, USA. ⁷²Mohammed V University of Rabat, Rabat, Morocco. ⁷³Dalhousie University, Halifax, Nova Scotia, Canada. ⁷⁴Jordan University of Science and Technology, Irbid, Jordan. ⁷⁵Al-Farabi Kazakh National University, Almaty, Kazakhstan. ⁷⁶University of Sydney, Sydney, New South Wales, Australia. ⁷⁷Christian Medical College, Vellore, India. ⁷⁸University Tunis El Manar, Tunis, Tunisia. ⁷⁹Cafam University Foundation, Bogota, Colombia. ⁸⁰Kazakh National Medical University, Almaty, Kazakhstan. ⁸¹Universidad Peruana Cayetano Heredia, Lima, Peru. ⁸²Lithuanian University of Health Sciences, Kaunas, Lithuania. ⁸³University of São Paulo, São Paulo, Brazil. ⁸⁴Sunder Lal Jain Hospital, Delhi, India. ⁸⁵Shanghai Jiao-Tong University School of Medicine, Shanghai, China. ⁸⁶Public Health Agency of Canada, Ottawa, Ontario, Canada. ⁸⁷Ufa Eye Research Institute, Ufa, Russia. ⁸⁸Nepal Health Research Council, Kathmandu, Nepal. ⁸⁹University of Southern Denmark, Copenhagen, Denmark. ⁹⁰University of Gothenburg, Gothenburg, Sweden. ⁹¹Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil. ⁹²National Institute for Public Health and the Environment, Bilthoven, The Netherlands. ⁹³University of Turin, Turin, Italy. ⁹⁴Nanyang Technological University, Singapore, Singapore. ⁹⁵Universidad de la República, Montevideo, Uruguay. ⁹⁶Centro de Educación Médica e Investigaciones Clínicas, Buenos Aires, Argentina. ⁹⁷IRCCS Neuromed, Pozzilli, Italy. ⁹⁸Toulouse University School of Medicine, Toulouse, France. ⁹⁹University Hospital of Varese, Varese, Italy. ¹⁰⁰University Hospital KU Leuven, Leuven, Belgium. ¹⁰¹Ministry of Health, Victoria, Seychelles. ¹⁰²University of Lausanne, Lausanne, Switzerland. ¹⁰³Universidad Central de Venezuela, Caracas, Venezuela. ¹⁰⁴Bielefeld University, Bielefeld, Germany. ¹⁰⁵German Cancer Research Center, Heidelberg, Germany. ¹⁰⁶University College Copenhagen, Copenhagen, Denmark. ¹⁰⁷Robert Koch Institute, Berlin, Germany. ¹⁰⁸Universidad de La Laguna, Tenerife, Spain. ¹⁰⁹University of Malta, Msida, Malta. ¹¹⁰Istanbul University – Cerrahpasa, Istanbul, Turkey. ¹¹¹Universidade Federal de Juiz de Fora, Juiz de Fora, Brazil. ¹¹²Gaetano Fucito Hospital, Mercato San Severino, Italy. ¹¹³University of Porto, Porto, Portugal. ¹¹⁴Santiago de Compostela University, Santiago, Spain. ¹¹⁵Council for Agricultural Research and Economics, Rome, Italy. ¹¹⁶Research and Education Institute of Child Health, Nicosia, Cyprus. ¹¹⁷Dr. A. Ramachandran's Diabetes Hospital, Chennai, India. ¹¹⁸University College London, London, UK. ¹¹⁹Academia Sinica, Taipei, Taiwan. ¹²⁰Capital Institute of Pediatrics, Beijing, China. ¹²¹Kailuan General Hospital, Tangshan, China. ¹²²Duke-NUS Medical School, Singapore, Singapore. ¹²³Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran. ¹²⁴The Gertner Institute for Epidemiology and Health Policy Research, Ramat Gan, Israel. ¹²⁵Ministry of Health and Welfare, Taipei, Taiwan. ¹²⁶Murcia Health Council, Murcia, Spain. ¹²⁷Seoul National University College of Medicine, Seoul, Republic of Korea. ¹²⁸Korea Centers for Disease Control and Prevention, Cheongju-si, Republic of Korea. ¹²⁹Medical University of Silesia, Katowice, Poland. ¹³⁰Katholieke Universiteit Leuven, Leuven, Belgium. ¹³¹Statistics Canada, Ottawa, Ontario, Canada. ¹³²Ghent University, Ghent, Belgium. ¹³³Agency for Preventive and Social Medicine, Bregenz, Austria. ¹³⁴Federal University of Maranhão, São Luís, Brazil. ¹³⁵University of Southampton, Southampton, UK. ¹³⁶Institut Pasteur de Lille, Lille, France. ¹³⁷CIBEROBN, Madrid, Spain. ¹³⁸University of Debrecen, Debrecen, Hungary. ¹³⁹Universidade Federal do Rio Grande do Sul, Porto Alegre, Brazil. ¹⁴⁰National Council of Research, Reggio Calabria, Italy. ¹⁴¹Federal University of Santa Catarina, Florianópolis, Brazil. ¹⁴²Eduardo Mondlane University, Maputo, Mozambique. ¹⁴³Geneva University Hospitals, Geneva, Switzerland. ¹⁴⁴National Research Centre for Preventive Medicine, Moscow, Russia. ¹⁴⁵University of Montreal, Montreal, Québec, Canada. ¹⁴⁶French Public Health Agency, St Maurice, France. ¹⁴⁷Mediterranea Cardiocentro, Naples, Italy. ¹⁴⁸University of Zagreb, Zagreb, Croatia. ¹⁴⁹Ministry of Health and Medical Education, Tehran, Iran. ¹⁵⁰University of Queensland, Brisbane, Queensland, Australia. ¹⁵¹Universidad de Cuenca, Cuenca, Ecuador. ¹⁵²Helmholtz Zentrum München, Munich, Germany. ¹⁵³Carol Davila University of Medicine and Pharmacy, Bucharest, Romania. ¹⁵⁴University Hospital Düsseldorf, Düsseldorf, Germany. ¹⁵⁵National Institute of Cardiology, Warsaw, Poland. ¹⁵⁶Beth Israel Deaconess Medical Center, Boston, MA, USA. ¹⁵⁷University of Latvia, Riga, Latvia. ¹⁵⁸Ministry of Health and Medical Services, Gizo, Solomon Islands. ¹⁵⁹Hormozgan University of Medical Sciences, Bandar Abbas, Iran. ¹⁶⁰University of Skövde, Skövde, Sweden. ¹⁶¹National Institute of Nutrition and Food Technology, Tunis, Tunisia. ¹⁶²The University of the West Indies, Kingston, Jamaica. ¹⁶³Institut

Article

Hospital del Mar d'Investigacions Mèdiques, Barcelona, Spain. ¹⁶⁴University of Stellenbosch, Cape Town, South Africa. ¹⁶⁵University of Duisburg-Essen, Duisburg, Germany. ¹⁶⁶Karadeniz Technical University, Trabzon, Turkey. ¹⁶⁷University of Helsinki, Helsinki, Finland. ¹⁶⁸Mashhad University of Medical Sciences, Mashhad, Iran. ¹⁶⁹Rafsanjan University of Medical Sciences, Rafsanjan, Iran. ¹⁷⁰Queen's University of Belfast, Belfast, UK. ¹⁷¹University of Zurich, Zurich, Switzerland. ¹⁷²Tabriz University of Medical Sciences, Tabriz, Iran. ¹⁷³Fasa University of Medical Sciences, Fasa, Iran. ¹⁷⁴Shiraz University of Medical Sciences, Shiraz, Iran. ¹⁷⁵Centro de Salud Villanueva Norte, Badajoz, Spain. ¹⁷⁶Servicio Extremeño de Salud, Badajoz, Spain. ¹⁷⁷Ministry of Health, Buenos Aires, Argentina. ¹⁷⁸Universiti Sains Malaysia, Kelantan, Malaysia. ¹⁷⁹Umeå University, Umeå, Sweden. ¹⁸⁰Federal University of São Paulo, São Paulo, Brazil. ¹⁸¹Hospital Universitario Son Espases, Palma, Spain. ¹⁸²Kindai University, Osaka-Sayama, Japan. ¹⁸³Kyoto University, Kyoto, Japan. ¹⁸⁴Medical University of Warsaw, Warsaw, Poland. ¹⁸⁵University of Catania, Catania, Italy. ¹⁸⁶CIBER en Epidemiología y Salud Pública, Alicante, Spain. ¹⁸⁷CIBER en Epidemiología y Salud Pública, Barcelona, Spain. ¹⁸⁸Universidade Federal de Minas Gerais, Belo Horizonte, Brazil. ¹⁸⁹Wageningen University, Wageningen, The Netherlands. ¹⁹⁰B. P. Koirala Institute of Health Sciences, Dharan, Nepal. ¹⁹¹University of Insubria, Varese, Italy. ¹⁹²University of Adelaide, Adelaide, South Australia, Australia. ¹⁹³Lund University, Lund, Sweden. ¹⁹⁴McGill University, Montreal, Québec, Canada. ¹⁹⁵Universidad Politécnica de Madrid, Madrid, Spain. ¹⁹⁶St Anne's University Hospital, Brno, Czech Republic. ¹⁹⁷National Institute of Public Health, Cuernavaca, Mexico. ¹⁹⁸Centro de Estudios en Diabetes A.C., Mexico City, Mexico. ¹⁹⁹Universidad Autónoma de Santo Domingo, Santo Domingo, Dominican Republic. ²⁰⁰Institute for Clinical and Experimental Medicine, Prague, Czech Republic. ²⁰¹Jagiellonian University Medical College, Kraków, Poland. ²⁰²University of Southern Denmark, Odense, Denmark. ²⁰³National Center of Cardiovascular Diseases, Beijing, China. ²⁰⁴Singapore Eye Research Institute, Singapore, Singapore. ²⁰⁵Celtic Heart Association, Kopavogur, Iceland. ²⁰⁶Universidad Icesi, Cali, Colombia. ²⁰⁷Eternal Heart Care Centre and Research Institute, Jaipur, India. ²⁰⁸National Health Insurance Service, Wonju, Republic of Korea. ²⁰⁹Prevention of Metabolic Disorders Research Center, Tehran, Iran. ²¹⁰The University of the West Indies, Cave Hill, Barbados. ²¹¹Kermanshah University of Medical Sciences, Kermanshah, Iran. ²¹²University of Eastern Finland, Kuopio, Finland. ²¹³Kyushu University, Fukuoka, Japan. ²¹⁴University of Bergen, Bergen, Norway. ²¹⁵Tulane University, New Orleans, LA, USA. ²¹⁶Chinese Center for Disease Control and Prevention, Beijing, China. ²¹⁷Joep Lange Institute, Amsterdam, The Netherlands. ²¹⁸Chronic Diseases Research Center, Tehran, Iran. ²¹⁹University of Hong Kong, Hong Kong, China. ²²⁰The Chinese University of Hong Kong, Hong Kong, China. ²²¹University of Western Australia, Perth, Western Australia, Australia. ²²²Kingston Health Sciences Centre, Kingston, Ontario, Canada. ²²³Heart Institute, São Paulo, Brazil. ²²⁴Fundación Oftalmológica de Santander, Bucaramanga, Colombia. ²²⁵University Oran 1, Oran, Algeria. ²²⁶Independent Public Health Specialist, Nay Pyi Taw, Myanmar. ²²⁷Ministry of Health and Sports, Nay Pyi Taw, Myanmar. ²²⁸CIBER en Epidemiología y Salud Pública, Murcia, Spain. ²²⁹VU University Medical Center, Amsterdam, The Netherlands. ²³⁰International Agency for Research on Cancer, Lyon, France. ²³¹American University of Beirut, Beirut, Lebanon. ²³²Cairo University, Cairo, Egypt. ²³³University of Zaragoza, Zaragoza, Spain. ²³⁴National Institutes of Biomedical Innovation, Health and Nutrition, Tokyo, Japan. ²³⁵Medical University Varna, Varna, Bulgaria. ²³⁶The University of Tokyo, Tokyo, Japan. ²³⁷The Hospital for Sick Children, Toronto, Ontario, Canada. ²³⁸Niigata University, Niigata, Japan. ²³⁹Hadassah University Medical Center, Jerusalem, Israel. ²⁴⁰Norwegian University of Science and Technology, Trondheim, Norway. ²⁴¹University of Melbourne, Melbourne, Victoria, Australia. ²⁴²University Hospital Centre Zagreb, Zagreb, Croatia. ²⁴³University of Zagreb School of Medicine, Zagreb, Croatia. ²⁴⁴Heart Foundation, Melbourne, Victoria, Australia. ²⁴⁵Guangzhou 12th Hospital, Guangzhou, China. ²⁴⁶Universidad Eugenio María de Hostos, Santo Domingo, Dominican Republic. ²⁴⁷Simon Fraser University, Burnaby, British Columbia, Canada. ²⁴⁸Ruprecht-Karls-University of Heidelberg, Heidelberg, Germany. ²⁴⁹World Health Organization Country Office, Delhi, India. ²⁵⁰Guilan University of Medical Sciences, Rasht, Iran. ²⁵¹University of Opole, Opole, Poland. ²⁵²University of Crete, Heraklion, Greece. ²⁵³Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia. ²⁵⁴Maharajgunj Medical Campus, Kathmandu, Nepal. ²⁵⁵Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA. ²⁵⁶National Institute of Epidemiology, Chennai, India. ²⁵⁷University of Münster, Münster, Germany. ²⁵⁸Research Institute for Primordial Prevention of Non-communicable Disease, Isfahan, Iran. ²⁵⁹Kyrgyz State Medical Academy, Bishkek, Kyrgyzstan. ²⁶⁰Research Institute of Child Nutrition, Dortmund, Germany. ²⁶¹Mazandaran University of Medical Sciences, Sari, Iran. ²⁶²Hypertension Research Center, Isfahan, Iran. ²⁶³Medical University of Innsbruck, Innsbruck, Austria. ²⁶⁴Muhimbili University of Health and Allied Sciences, Dar es Salaam, Tanzania. ²⁶⁵Yonsei University College of Medicine, Seoul, Republic of Korea. ²⁶⁶National Cancer Center, Goyang-si, Republic of Korea. ²⁶⁷University of Pennsylvania, Philadelphia, PA, USA. ²⁶⁸University of Vienna, Vienna, Austria. ²⁶⁹Oulu Deaconess Institute Foundation, Oulu, Finland. ²⁷⁰Tartu University Clinics, Tartu, Estonia. ²⁷¹Kansai Medical University, Hirakata, Japan. ²⁷²Ministry of Health and Quality of Life, Port Louis, Mauritius. ²⁷³University Hospital Ulm, Ulm, Germany. ²⁷⁴University of Groningen, Groningen, The Netherlands. ²⁷⁵University of Jyväskylä, Jyväskylä, Finland. ²⁷⁶African Population and Health Research Center, Nairobi, Kenya. ²⁷⁷Higher Institute of Health Sciences of Settat, Settat, Morocco. ²⁷⁸Ministry of Health, Algiers, Algeria. ²⁷⁹Harokopio University, Athens, Greece. ²⁸⁰Sahlgrenska Academy, Gothenburg, Sweden. ²⁸¹Endocrinology and Metabolism Research Center, Tehran, Iran. ²⁸²University of Public Health, Yangon, Myanmar. ²⁸³National University of Singapore, Singapore, Singapore. ²⁸⁴Tampere University Hospital, Tampere, Finland. ²⁸⁵Tampere University, Tampere, Finland. ²⁸⁶University of Cape Town, Cape Town, South Africa. ²⁸⁷West Virginia University, Morgantown, WV, USA. ²⁸⁸Oswaldo Cruz Foundation Rene Rachou Research Institute, Belo Horizonte, Brazil. ²⁸⁹National Taiwan University, Taipei, Taiwan. ²⁹⁰University of Chinese Academy of Sciences, Shanghai, China.

²⁹¹Uppsala University, Uppsala, Sweden. ²⁹²National Institute for Health Development, Tallinn, Estonia. ²⁹³Universidad San Martín de Porres, Lima, Peru. ²⁹⁴Consejería de Sanidad Junta de Castilla y León, Valladolid, Spain. ²⁹⁵Norbotten County Council, Luleå, Sweden. ²⁹⁶Peking University, Beijing, China. ²⁹⁷Universidade Federal de Ouro Preto, Ouro Preto, Brazil. ²⁹⁸University of Coimbra, Coimbra, Portugal. ²⁹⁹The Jikei University School of Medicine, Tokyo, Japan. ³⁰⁰Institute of Neuroscience of the National Research Council, Padua, Italy. ³⁰¹Baker Heart and Diabetes Institute, Melbourne, Victoria, Australia. ³⁰²Agricultural University of Athens, Athens, Greece. ³⁰³French National Research Institute for Sustainable Development, Montpellier, France. ³⁰⁴Hospital Israelita Albert Einstein, São Paulo, Brazil. ³⁰⁵University of Otago, Dunedin, New Zealand. ³⁰⁶University of Padua, Padua, Italy. ³⁰⁷Lausanne University Hospital, Lausanne, Switzerland. ³⁰⁸Emory University, Atlanta, GA, USA. ³⁰⁹Universidade Estadual do Centro-Oeste, Guarapuava, Brazil. ³¹⁰UiT The Arctic University of Norway, Tromsø, Norway. ³¹¹Cape Peninsula University of Technology, Cape Town, South Africa. ³¹²Brown University, Providence, RI, USA. ³¹³University of Edinburgh, Edinburgh, UK. ³¹⁴University College Dublin, Dublin, Ireland. ³¹⁵Shahid Sadoughi University of Medical Sciences, Yazd, Iran. ³¹⁶Institut National de la Santé et de la Recherche Médicale, Lille, France. ³¹⁷ICMR–National Institute of Medical Statistics, New Delhi, India. ³¹⁸Lusófona University, Lisbon, Portugal. ³¹⁹Women's Social and Health Studies Foundation, Trivandrum, India. ³²⁰Università degli Studi di Firenze, Florence, Italy. ³²¹Ain Shams University, Cairo, Egypt. ³²²Isfahan Cardiovascular Research Center, Isfahan, Iran. ³²³University of Strasbourg, Strasbourg, France. ³²⁴Strasbourg University Hospital, Strasbourg, France. ³²⁵University of Pécs, Pécs, Hungary. ³²⁶Mulago Hospital, Kampala, Uganda. ³²⁷University of Medical Sciences of Cienfuegos, Cienfuegos, Cuba. ³²⁸Royal College of Surgeons in Ireland Dublin, Dublin, Ireland. ³²⁹La Trobe University, Melbourne, Victoria, Australia. ³³⁰International Institute of Molecular and Cell Biology, Warsaw, Poland. ³³¹Instituto Conmemorativo Gorgas de Estudios de la Salud, Panama City, Panama. ³³²World Health Organization Country Office, Lilongwe, Malawi. ³³³University of Brescia, Brescia, Italy. ³³⁴Bushehr University of Medical Sciences, Bushehr, Iran. ³³⁵Ulm University, Ulm, Germany. ³³⁶Kobe University, Kobe, Japan. ³³⁷Suraj Eye Institute, Nagpur, India. ³³⁸University Medicine of Greifswald, Greifswald, Germany. ³³⁹University of Medicine and Pharmacy at Ho Chi Minh City, Ho Chi Minh City, Vietnam. ³⁴⁰Hanoi Medical University, Hanoi, Vietnam. ³⁴¹Miami Veterans Affairs Healthcare System, Miami, FL, USA. ³⁴²University of Turku, Turku, Finland. ³⁴³Eastern Mediterranean Public Health Network, Amman, Jordan. ³⁴⁴University of Manchester, Manchester, UK. ³⁴⁵Japan Wildlife Research Center, Tokyo, Japan. ³⁴⁶Istanbul University, Istanbul, Turkey. ³⁴⁷Ministry of Health, Bandar Seri Begawan, Brunei. ³⁴⁸University of Madeira, Funchal, Portugal. ³⁴⁹MRC Lifecourse Epidemiology Unit, Southampton, UK. ³⁵⁰University of Novi Sad, Novi Sad, Serbia. ³⁵¹Kwame Nkrumah University of Science and Technology, Kumasi, Ghana. ³⁵²Institute for Social and Preventive Medicine, Ottawa, Ontario, Canada. ³⁵³RCCS Ente Ospedaliero Specializzato in Gastroenterologia S. de Bellis, Bari, Italy. ³⁵⁴Jivandeep Hospital, Anand, India. ³⁵⁵South African Medical Research Council, Durban, South Africa. ³⁵⁶Vietnam National Heart Institute, Hanoi, Vietnam. ³⁵⁷Clínica de Medicina Avanzada Dr. Abel González, Santo Domingo, Dominican Republic. ³⁵⁸Leibniz Institute for Prevention Research and Epidemiology – BIPS, Bremen, Germany. ³⁵⁹University of Sarajevo, Sarajevo, Bosnia and Herzegovina. ³⁶⁰Cardiovascular Prevention Centre, Udine, Italy. ³⁶¹Public Health Agency of Catalonia, Barcelona, Spain. ³⁶²Observatorio de Salud Pública de Santander, Bucaramanga, Colombia. ³⁶³Ardabil University of Medical Sciences, Ardabil, Iran. ³⁶⁴Alborz University of Medical Sciences, Karaj, Iran. ³⁶⁵Ministry of Health, Hanoi, Vietnam. ³⁶⁶India Diabetes Research Foundation, Chennai, India. ³⁶⁷Institut Universitari d'Investigació en Atenció Primària Jordi Gol, Girona, Spain. ³⁶⁸Universiti Putra Malaysia, Serdang, Malaysia. ³⁶⁹University of Malaya, Kuala Lumpur, Malaysia. ³⁷⁰University of Valencia, Valencia, Spain. ³⁷¹University of the Philippines, Manila, The Philippines. ³⁷²Minas Gerais State Secretariat for Health, Belo Horizonte, Brazil. ³⁷³CS S. Agustin Ibsalut, Palma, Spain. ³⁷⁴Amsterdam Institute for Global Health and Development, Amsterdam, The Netherlands. ³⁷⁵Canarian Health Service, Tenerife, Spain. ³⁷⁶Universidad Industrial de Santander, Bucaramanga, Colombia. ³⁷⁷Associazione Calabrese di Epatologia, Reggio Calabria, Italy. ³⁷⁸Sahlgrenska University Hospital, Gothenburg, Sweden. ³⁷⁹Toulouse University Hospital, Toulouse, France. ³⁸⁰Institute of Food Sciences of the National Research Council, Avellino, Italy. ³⁸¹Sitaram Bharti Institute of Science and Research, New Delhi, India. ³⁸²Faculty of Medicine of Tunis, Tunis, Tunisia. ³⁸³National Institute of Health, Lima, Peru. ³⁸⁴Catalan Department of Health, Barcelona, Spain. ³⁸⁵Universidade de Lisboa, Lisbon, Portugal. ³⁸⁶South Karelia Social and Health Care District, Lappeenranta, Finland. ³⁸⁷Cardiovascular Research Institute, Isfahan, Iran. ³⁸⁸National Cancer Center, Tokyo, Japan. ³⁸⁹University of São Paulo Clinics Hospital, São Paulo, Brazil. ³⁹⁰Hospital Italiano de Buenos Aires, Buenos Aires, Argentina. ³⁹¹Center for Oral Health Services and Research Mid-Norway, Trondheim, Norway. ³⁹²King's College London, London, UK. ³⁹³National Center for Global Health and Medicine, Tokyo, Japan. ³⁹⁴Sungkyunkwan University, Seoul, Republic of Korea. ³⁹⁵Finnish Institute of Occupational Health, Helsinki, Finland. ³⁹⁶St Vincent's Hospital, Sydney, New South Wales, Australia. ³⁹⁷University of New South Wales, Sydney, New South Wales, Australia. ³⁹⁸Karolinska Institutet, Stockholm, Sweden. ³⁹⁹Research Centre for Prevention and Health, Glostrup, Denmark. ⁴⁰⁰London School of Hygiene & Tropical Medicine, London, UK. ⁴⁰¹Diponegoro University, Semarang, Indonesia. ⁴⁰²University of Bari, Bari, Italy. ⁴⁰³University of Copenhagen, Copenhagen, Denmark. ⁴⁰⁴Institut Régional de Santé Publique, Ouidah, Benin. ⁴⁰⁵University of Bordeaux, Bordeaux, France. ⁴⁰⁶Izmir Katip Çelebi University, Izmir, Turkey. ⁴⁰⁷University of Leuven, Leuven, Belgium. ⁴⁰⁸Institut National de la Santé et de la Recherche Médicale, Nancy, France. ⁴⁰⁹Bonn University, Bonn, Germany. ⁴¹⁰Croatian Institute of Public Health, Zagreb, Croatia. ⁴¹¹National Institute of Public Health–National Institute of Hygiene, Warsaw, Poland. ⁴¹²National Institute of Hygiene, Epidemiology and Microbiology, Havana, Cuba. ⁴¹³Fu Jen Catholic University, Taipei, Taiwan. ⁴¹⁴National Statistic Office of Cabo Verde, Praia, Cabo Verde. ⁴¹⁵Ministry of Health, Amman, Jordan. ⁴¹⁶Central University of Kerala,

Kasaragod, India. ⁴¹⁷Health Service of Murcia, Murcia, Spain. ⁴¹⁸Institut d'Investigacio Sanitaria Illes Balears, Menorca, Spain. ⁴¹⁹Universidad Centro-Occidental Lisandro Alvarado, Barquisimeto, Venezuela. ⁴²⁰Dokuz Eylul University, Izmir, Turkey. ⁴²¹University of Tampere Tays Eye Center, Tampere, Finland. ⁴²²Icahn School of Medicine at Mount Sinai, New York City, NY, USA. ⁴²³Utrecht University, Utrecht, The Netherlands. ⁴²⁴Hanoi University of Public Health, Hanoi, Vietnam. ⁴²⁵University Medical Center Utrecht, Utrecht, The Netherlands. ⁴²⁶Universitas Indonesia, Jakarta, Indonesia. ⁴²⁷Instituto de Investigación Sanitaria y Biomédica de Alicante, Alicante, Spain. ⁴²⁸North Karelian Center for Public Health, Joensuu, Finland. ⁴²⁹University of the Witwatersrand, Johannesburg, South Africa. ⁴³⁰Cork Institute of Technology, Cork, Ireland. ⁴³¹Institute for Medical Research, Kuala Lumpur, Malaysia. ⁴³²Health Canada, Ottawa, Ontario,

Canada. ⁴³³Beijing Institute of Ophthalmology, Beijing, China. ⁴³⁴Xinjiang Medical University, Urumqi, China. ⁴³⁵Capital Medical University, Beijing, China. ⁴³⁶St George's, University of London, London, UK. ⁴³⁷Medical University of Vienna, Vienna, Austria. ⁴³⁸University of Oxford, Oxford, UK. ⁴³⁹Institute of Food and Nutrition Development of Ministry of Agriculture and Rural Affairs, Beijing, China. ⁴⁴⁰Children's Hospital of Fudan University, Shanghai, China. ⁴⁴¹Penang Medical College, Penang, Malaysia. ⁴⁴²University of Cyprus, Nicosia, Cyprus. ⁴⁴³Iran University of Medical Sciences, Tehran, Iran. ⁴⁴⁴Jiangsu Provincial Center for Disease Control and Prevention, Nanjing, China. ⁴⁴⁵Sun Yat-sen University, Guangzhou, China. ⁴⁴⁶West Kazakhstan State Medical University, Aktobe, Kazakhstan. ⁴⁴⁷University of Ghana, Accra, Ghana. ⁴⁴⁸Deceased: Konrad Jamrozik, Altan Onat, Robespierre Ribeiro, Jutta Stieber. ⁵²e-mail: majid.ezzati@imperial.ac.uk

Methods

Our aim was to estimate trends in mean total, HDL and non-HDL cholesterol for 200 countries and territories (Supplementary Table 2). We used non-HDL cholesterol rather than low-density lipoprotein (LDL) cholesterol because most studies in our analysis had measured total cholesterol and HDL cholesterol, from which non-HDL cholesterol can be calculated through subtraction. By contrast, LDL cholesterol was directly measured in only around 14% of studies. When LDL cholesterol is not directly measured, its calculation requires data on triglycerides, which were available in approximately 64% of the studies. Furthermore, the most commonly used estimation method—that is, the Friedewald equation—can be inaccurate, particularly at high levels of triglycerides³⁴. Non-HDL and LDL cholesterol were highly correlated (Pearson correlation coefficient = 0.94) in studies with data on both variables (Extended Data Fig. 9), because LDL cholesterol constitutes most of non-HDL cholesterol. Furthermore, non-HDL cholesterol predicts IHD risk at least as well as LDL cholesterol^{15,35}, and can be measured at a lower cost than LDL cholesterol, which is relevant for how widely it can be used in low- and middle-income countries. Although non-HDL cholesterol is now commonly used in clinical guidelines^{36–38}, LDL cholesterol continues to be a key target for treatment^{36,37}, possibly because the interpretation of non-HDL cholesterol is more complex than LDL cholesterol alone. Specifically, an increase in non-HDL cholesterol could be due to the increase in LDL cholesterol or very-low-density lipoprotein cholesterol³⁹. Furthermore, there is some evidence that triglyceride levels are high in Asian populations, compared to levels seen in high-income western countries⁴⁰. Therefore, data on non-HDL cholesterol can motivate dietary interventions to both reduce LDL cholesterol (for example, reducing saturated and trans fat intake) and triglyceride levels (for example, reducing refined carbohydrates and increasing omega-3 fatty acids) as well as treatments that lower LDL cholesterol (statins), alongside those that lower triglycerides (for example, fibrates).

Data sources

We used a database of population-based data on cardiometabolic risk factors collated by the NCD Risk Factor Collaboration (NCD-RisC), a worldwide network of health researchers and practitioners that systematically monitors the worldwide trends and variations in non-communicable disease (NCD) risk factors. The database was collated through multiple routes for identifying and accessing data. We accessed publicly available population-based multi-country and national measurement surveys (for example, Demographic and Health Surveys and surveys identified through the Inter-University Consortium for Political and Social Research and European Health Interview & Health Examination Surveys Database). We requested, via the World Health Organization (WHO) and its regional and country offices, from ministries of health and other national health and statistical agencies to identify and access population-based surveys. Requests were also sent via the World Heart Federation to its national partners. We made a similar request to the co-authors of an earlier pooled analysis of cardiometabolic risk factors^{7,41–43}, and invited the co-authors of the analysis to reanalyse data from their studies and join NCD-RisC. Finally, to identify major sources that were not accessed through the above routes, we searched and reviewed published studies as described in the Supplementary Information and invited all eligible studies to join NCD-RisC.

For each data source, we recorded the available information about the study population, start year and duration of measurement, sampling approach and measurement methods. The information about study population was used to establish that each data source was population-based, and to assess whether it covered the whole country, multiple subnational regions or one or a small number of communities, and whether it was rural, urban or combined.

We carefully checked all data sources in terms of how they met our inclusion and exclusion criteria listed below. We identified duplicate data sources by comparing studies from the same country and year. Additionally, all NCD-RisC members are asked periodically to review the list of sources from their country, to suggest additional sources not in the database, and to verify that the included data meet the inclusion criteria listed below and are not duplicates. The NCD-RisC database is continuously updated through the above routes and through regular contact with NCD-RisC members.

Anonymized individual record data from sources included in NCD-RisC were reanalysed according to a common protocol. Within each survey, we included participants aged 18 years and older who were not pregnant. We removed participants with implausible total cholesterol levels (defined as total cholesterol levels of $<1.75 \text{ mmol l}^{-1}$ or $>20 \text{ mmol l}^{-1}$, or total cholesterol values that were lower than HDL cholesterol values) ($<0.05\%$ of all participants with total cholesterol measurements) or HDL cholesterol levels (defined as HDL cholesterol levels of $<0.4 \text{ mmol l}^{-1}$ or $>5 \text{ mmol l}^{-1}$, or total cholesterol values that were lower than HDL cholesterol values) ($<0.15\%$ of all participants with HDL cholesterol measurements). When data on LDL cholesterol were also available, we removed individuals for whom the sum of LDL and HDL cholesterol level surpassed total cholesterol level by more than is plausible based on the limits to errors in their measurement (following the CDC Cholesterol Reference Method Laboratory Network (CRMLN) standards, these errors were set at 8.9% for total cholesterol, 13% for HDL cholesterol and 12% for LDL cholesterol) ($<0.06\%$ of all participants with total cholesterol and HDL cholesterol measurements)^{44–46}.

We calculated mean total cholesterol, mean HDL cholesterol and mean non-HDL cholesterol, and associated standard errors and sample sizes, by sex and age group (18–19 years, 20–29 years, followed by 10-year age groups and 80+ years). All analyses incorporated appropriate sample weights and complex survey design in calculating age–sex-specific means when applicable. To ensure summaries were prepared according to the study protocol, computer code was provided to NCD-RisC members who requested assistance. All submitted data were checked independently by at least two researchers. Questions and clarifications were discussed with NCD-RisC members and resolved before the data were incorporated in the database.

Finally, we obtained data not accessed through the above routes by extracting data from published reports of all additional national health surveys identified through the above-described strategies, as well as eight sites of the WHO Multinational MONItoring of trends and determinants in Cardiovascular disease (MONICA) project that were not deposited in the MONICA Data Centre. Data were extracted from published reports only when reported by sex and in age groups no wider than 20 years. We also used data from a previous pooling study⁷ when such data did not overlap with those accessed through the above routes.

Data inclusion and exclusion

Data sources were included in NCD-RisC database if: (1) measured data on total, LDL, HDL cholesterol and/or triglycerides were available; (2) study participants were 10 years of age or older; (3) data were collected using a probabilistic sampling method with a defined sampling frame; (4) data were from population samples at the national, subnational (covering one or more subnational regions, more than three urban communities or more than five rural communities) or community (one or a small number of communities) level; (5) data were collected in or after 1950; and (6) data were from the countries and territories listed in Supplementary Table 2.

We excluded all data sources that included only hypercholesterolaemia or dyslipidaemia diagnosis history or medication status without measurement of cholesterol levels. We also excluded data sources on population subgroups for which the lipid profile may differ systematically from the general population, including: (1) studies that had included or excluded people on the basis of their health status or

cardiovascular risk; (2) studies for which the participants were only from ethnic minorities; (3) studies that had recruited only specific educational, occupational or socioeconomic subgroups, with the exception noted below; and (4) studies that had recruited participants through health facilities, with the exception noted below.

We used school-based data in countries and for age–sex groups, for which secondary school enrolment was 70% or higher. We used data for which the sampling frame was health insurance schemes in countries in which at least 80% of the population was insured. Finally, we used data collected through general practice and primary-care systems in high-income and central European countries with universal insurance, because contact with the primary-care systems tends to be as good as or better than response rates for population-based surveys. We used data sources regardless of fasting status, because the differences between fasting and non-fasting measurements are negligible for total, non-HDL and HDL cholesterol³⁹, and therefore non-fasting lipid profiles are now widely endorsed for the estimation of cardiovascular risk^{36,37}.

Data used in the analysis

For this paper, we used data from the NCD-RisC database for years 1980 to 2018 and individuals aged 18 years and older. A list of the data sources that we used in this analysis and their characteristics is provided in Supplementary Table 1. The data comprised 1,127 population-based measurement surveys and studies that included measurements of blood lipids on 102.6 million participants aged 18 years and older. We had at least one data source for 161 of the 200 countries that we made estimates for, covering 92.4% of the world's population in 2018 (Extended Data Fig. 1); and at least two data sources for 104 countries (87.5% of the world population). Of these 1,127 sources, 409 (36.3%) sampled from national populations, 250 (22.2%) covered one or more subnational regions, and the remaining 468 (41.5%) were from one or a small number of communities. Regionally, data availability ranged from around 2 data sources per country in sub-Saharan Africa to approximately 35 sources per country in the high-income Asia–Pacific region. In total, 454 data sources (40.3%) were from years before 2000 and the remaining 673 (59.7%) were collected from 2001 onwards.

Adjusting for the differences in mean cholesterol between portable device and laboratory measurements

In 112 (10%) of the 1,127 data sources used in our analysis (11.5% and 5.8% of age–sex-specific data points for total and HDL cholesterol, respectively) lipids were measured using a portable device. Some portable devices have narrower analytical ranges than laboratory methods, which results in truncations of blood cholesterol data that are outside their range (Supplementary Table 3). This may in turn affect the population mean. Although cholesterol concentrations that fall outside the analytical range are displayed as 'high' (above the measurement range) or 'low' (below the measurement range) by these devices, different surveys record and code cholesterol concentrations outside the analytical range in different ways, for example using 'too low', 'too high' and 'error' codes; assigning the minimum or maximum value to individuals whose cholesterol was below or above the analytical range, respectively; setting values outside the analytical range to missing; and so on. We used an approach that treated surveys with such data consistently.

Specifically, we first dropped all participants with cholesterol levels below and at the minimum, and at and above the maximum, values of the analytical range of each portable device before calculating the mean cholesterol (Supplementary Table 3). We then developed conversion regressions to adjust the mean cholesterol levels measured using a portable device (calculated over the restricted range, Supplementary Table 3) to the levels expected using laboratory measurements. The dependent variable in each regression was mean total, non-HDL or HDL cholesterol for the full range, and the main independent variable was mean total, non-HDL or HDL cholesterol over the above-mentioned restricted cholesterol range of the portable devices. The regression

coefficients were estimated from data sources for which lipids were measured in a laboratory, and thus had the full range of measurement and could be used to calculate both dependent and independent variables. When estimating the regression coefficients, we constructed the dependent variable using the full data, and the independent variable by dropping the values outside the above-mentioned restricted cholesterol range of each device, mimicking those that would be expected if a portable device had been used. Separate models were developed according to the specific range of the different portable devices. All regressions included terms for age and sex, as well as interactions between predictors and age and sex, based on the Bayesian information criterion⁴⁷. The regressions for mean non-HDL cholesterol also included mean total cholesterol and mean HDL cholesterol because non-HDL cholesterol is calculated from total cholesterol and HDL cholesterol. We excluded data points for which there were fewer than 25 individuals for the purpose of estimating the coefficients of these regressions. All sources of uncertainty in the conversion—including the sampling uncertainty of the original data, the uncertainty of the regression coefficients and residuals—were carried forward by using repeated draws from their respective distributions. The regression coefficients and number of data points used to estimate the coefficients are shown in Supplementary Table 4.

Statistical analysis

We used a statistical model to estimate mean total, non-HDL and HDL cholesterol by country, year, sex and age using all of the available data. The model is described in detail in a statistical paper and related substantive papers^{8,32,33,48}; the computer code is available at <http://www.ncdrisc.org/>. In summary, we organized countries into 21 regions, mainly based on geography and national income; these regions were further aggregated into 9 'super-regions' (Supplementary Table 2). The model had a hierarchical structure in which estimates for each country and year were informed by its own data, if available, and by data from other years in the same country and from other countries, especially countries in the same region or super-region with data for similar time periods. The extent to which estimates for each country-year are influenced by data from other years and other countries depends on whether the country has data, the sample size of data, whether or not they are national, and the within-country and within-region data variability. The model incorporated nonlinear time trends comprising linear terms and a second-order random walk. The age association of blood lipids was modelled using a cubic spline to allow nonlinear age patterns, which might vary across countries. The model accounted for the possibility that blood lipids in subnational and community samples might systematically differ from nationally representative ones; and/or have larger variation. These features were implemented by including data-driven fixed-effect and random-effect terms for subnational and community data. The fixed effects adjust for systematic differences between subnational or community studies and national studies. The random effects allow national data to have larger influence on the estimates than subnational or community data with similar sample sizes. The model also accounted for rural–urban differences in blood lipids, through the use of data-driven fixed effects for rural-only and urban-only studies. These rural and urban effects were weighted by the difference between study-level and country-level urbanization in the year in which the study was done. The proportion of the national population living in urban areas was also included as a predictor (covariate) in the model. The model for mean non-HDL and HDL cholesterol also used age-standardized mean total cholesterol as a covariate.

We fitted the statistical model with the Markov chain Monte Carlo (MCMC) algorithm, and obtained 5,000 post-burn-in samples from the posterior distribution of model parameters, which were in turn used to obtain the posterior distributions of mean total, non-HDL and HDL cholesterol. We calculated average change in mean total, HDL and non-HDL cholesterol across the 39 years of analysis (reported as change

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per decade). Age-standardized estimates were generated by taking weighted averages of age–sex-specific estimates, using the WHO standard population. Estimates for regions and the world were calculated as population-weighted averages of the constituent country estimates by age group and sex. The reported credible intervals represent the 2.5–97.5th percentiles of the posterior distributions. We also report the posterior probability that an estimated increase or decrease represents a truly increasing or decreasing trend as opposed to a chance observation. We performed all analyses by sex, because blood lipids levels and trends are different in men and women.

Validation of statistical model

We tested how well our statistical model predicts missing data, known as external predictive validity, in two different tests. In the first test, we held out all data from 10% of countries with data (that is, created the appearance of countries with no data where we actually had data). The countries for which the data were withheld were randomly selected from the following three groups: data rich (5 or more data sources, with at least one data source after the year 2000), data poor (1 data source) and average data availability (2–4 data sources). In the second test, we assessed other patterns of missing data by holding out 10% of our data sources, again from a mix of data-rich, data-poor and average-data countries, as defined above. For a given country, we either held out a random half of the data of a country or all of the 2000–2018 data of the country to determine, respectively, how well we filled in the gaps for countries with intermittent data and how well we estimated in countries without recent data. In both tests, we then fitted the model to the remaining 90% of the countries (test 1) or data sources (test 2) and made estimates of the held-out observations. We repeated each test five times, holding out a different subset of data in each repetition. In both tests, we calculated the differences between the held-out data and the estimates. We also calculated the 95% credible intervals of the estimates; in a model with good external predictive validity, 95% of held-out values would be included in the 95% credible intervals.

Our statistical model performed well in the external validation tests, that is, in estimating mean cholesterol when data were missing. The estimates of mean total, non-HDL and HDL cholesterol were unbiased, as evidenced with median errors that were very close to zero globally for every outcome and test, and less than ± 0.30 mmol l⁻¹ in every subset of withheld data except for women in the high-income Asia–Pacific region in test 1 for non-HDL cholesterol (median error 0.47 mmol l⁻¹) and men in south Asia in test 2 for non-HDL cholesterol (median error -0.33 mmol l⁻¹) (Supplementary Table 5). The 95% credible intervals of estimated means covered 83–92% and 75–83% of true data globally in the first and second tests, respectively. In subsets, coverage ranged from 47% to 100%, but was mostly greater than 75%, with coverage generally lower in test 2 than test 1. Median absolute errors ranged from 0.07 to 0.23 mmol l⁻¹ globally for different outcomes and sexes, and were no more than 0.45 mmol l⁻¹ in all subsets of withheld data, except for women in the high-income Asia–Pacific region for non-HDL cholesterol in test 1 (median absolute error 0.47 mmol l⁻¹).

Calculation of the number of deaths attributable to high cholesterol

We estimated the number of deaths from IHD and ischaemic stroke attributable to high non-HDL cholesterol. For each country, year, sex and age group, we first calculated the population attributable fractions—that is, the proportion of deaths from IHD and ischaemic stroke that would have been prevented if non-HDL cholesterol levels were at an optimal level (defined as a mean of 1.8–2.2 mmol l⁻¹) in the population^{6,49}. For these calculations, we used age-specific relative risks from meta-analyses of prospective cohort studies^{4,50}. The number of IHD and ischaemic stroke deaths attributable to high non-HDL cholesterol was calculated for each country–year–age–sex group by multiplying the cause-specific population attributable fractions by the cause-specific

deaths from the Global Burden of Disease study in 1990 and 2017 (the earliest and latest years with cause-specific mortality data).

Strengths and limitations

The strengths of our study include its scope in making consistent and comparable estimates of trends in blood cholesterol and its cardiovascular disease mortality burden, over almost four decades for all of the countries in the world, including global estimates of non-HDL and HDL cholesterol. We used a large amount of population-based data, which came from countries in which 92% of the global adult population lives. We used only data from studies that had measured blood lipids to avoid bias in self-reported data. Data were analysed according to a consistent protocol, and the characteristics and quality of data from each country were rigorously verified through repeated checks by NCD-RisC members. We pooled data using a statistical model that took into account the epidemiological features of cholesterol, including nonlinear time trends and age associations. Our statistical model used all available data while giving more weight to national data than to subnational and community sources.

Similar to all global analyses, our study is affected by some limitations. Despite our extensive efforts to identify and access worldwide population-based data, some countries had no or few data sources, especially those in sub-Saharan Africa, the Caribbean, central Asia and Melanesia. Estimates for these countries relied mostly or entirely on the statistical model, which shares information across countries and regions through its hierarchy. Data scarcity is reflected in wider uncertainty intervals of our estimates for these countries and regions, highlighting the need for national NCD-oriented surveillance. The distribution of lipids measured in a population using a portable device, which was used in 10% of our studies, may be truncated and may therefore affect the population mean. To overcome this issue, we developed conversion regressions to adjust mean cholesterol levels measured using a portable device to the levels expected in laboratory measurements; the conversion regressions used for this purpose had good predictive accuracy. Although most studies had measured cholesterol in serum samples, around 7% had used plasma samples. As cholesterol measured in plasma and serum samples differ⁵¹ by only about 3%, adjusting for plasma-serum differences would have little effect on our results, as seen in a previous analysis¹⁴. Although methods to measure total and HDL cholesterol have evolved over time, since the 1950s there have been systematic efforts to standardize lipid measurements that have resulted in increased comparability between different methods. In our analysis, 90% of studies measured lipids in a laboratory; of these studies more than 60% for total cholesterol and more than 70% for HDL cholesterol participated in a lipid standardization programme or quality control scheme. We did not analyse emerging lipid markers such as apolipoprotein B and apolipoprotein A-I, because they are neither commonly measured in population-based health surveys, nor routinely used in clinical practice³⁶.

Comparison with other studies

There are no global analyses on trends in lipid fractions for comparison with our results. Our findings for total cholesterol were largely consistent with the only other previous analysis⁷, but we estimated a larger decrease in mean total cholesterol in high-income western countries and central Europe, and a larger increase in southeast Asia, because we had an additional decade of data compared with the earlier global analysis. Therefore, although the highest mean total cholesterol levels reported previously⁷, for 2008, were still in high-income western countries, we estimated that in 2018 total cholesterol was equally high or higher in southeast Asia. Our findings on mean total cholesterol trends are also largely consistent with previous multi- and single-country reports^{14,15,17–21,52–73}. Differences from previous studies—for example, in Italy⁶¹, Lithuania⁶³, the Netherlands⁶⁵, Russian Federation⁶⁹ and in some countries that participated in the MONICA Project⁵²—mostly arise

because our study covered a longer period and used a larger number of data sources. Studies^{15,18,54,63,66,70,74–77} that have reported trends in lipid fractions for a period longer than 15 years have found changes in non-HDL cholesterol (or in LDL cholesterol for some studies) that were consistent with our results.

Reporting summary

Further information on research design is available in the Nature Research Reporting Summary linked to this paper.

Data availability

Estimates of mean total, non-HDL and HDL cholesterol by country, year and sex are available at <http://www.ncdrisc.org/>. Input data from publicly available sources can also be downloaded from <http://www.ncdrisc.org/>. For other data sources, contact information for data providers can be obtained from <http://www.ncdrisc.org/>.

Code availability

The computer code for the Bayesian hierarchical model used in this work is available at <http://www.ncdrisc.org/>.

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Author contributions M.E. and G.D. designed the study and oversaw research. C.T., B.Z., H.B. and R.C.L. led the data collection. The other authors contributed to study design; and collected, reanalysed, checked and pooled data. C.T. analysed pooled data and prepared results. C.T., E.G. and M.E. wrote the first draft of the manuscript with input from the other authors.

Competing interests M.E. reports a charitable grant from the AstraZeneca Young Health Programme, and personal fees from Prudential, Scor and Third Bridge, outside the submitted work. The other authors declare no competing interests.

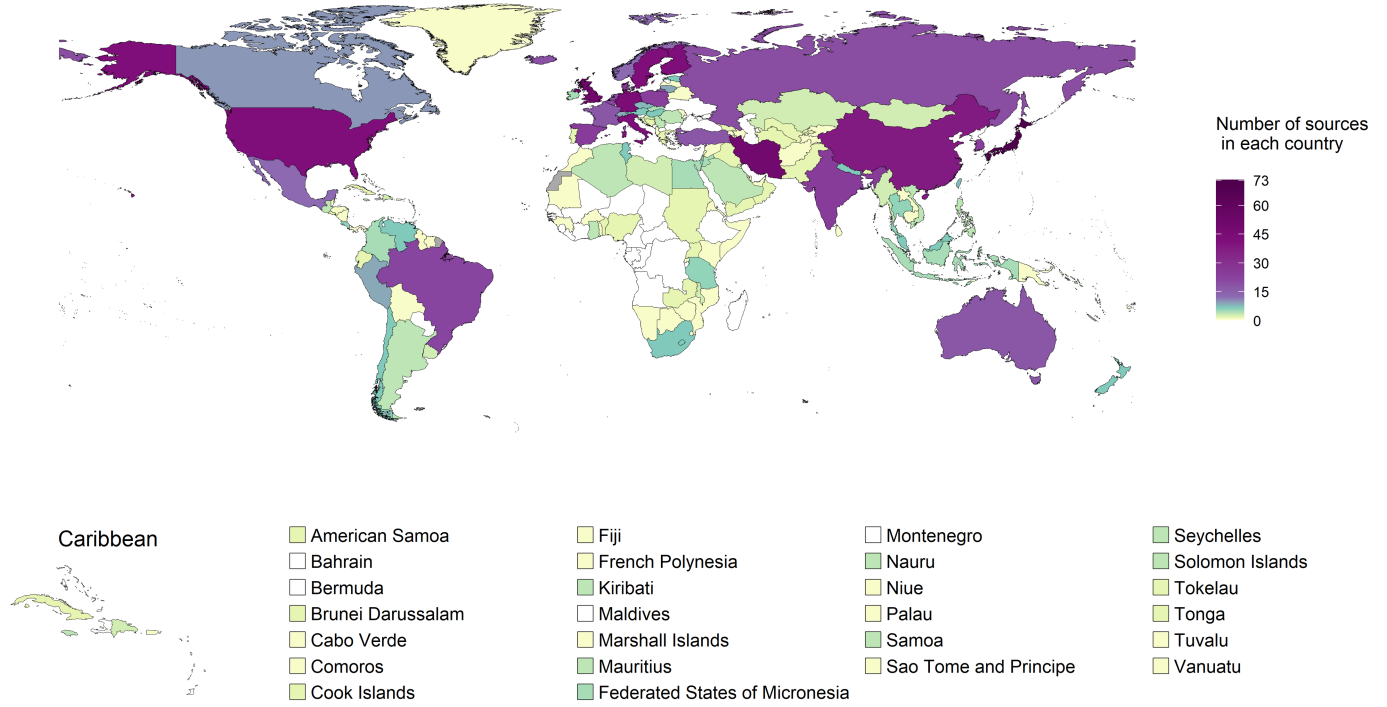
Additional information

Supplementary information is available for this paper at <https://doi.org/10.1038/s41586-020-2338-1>.

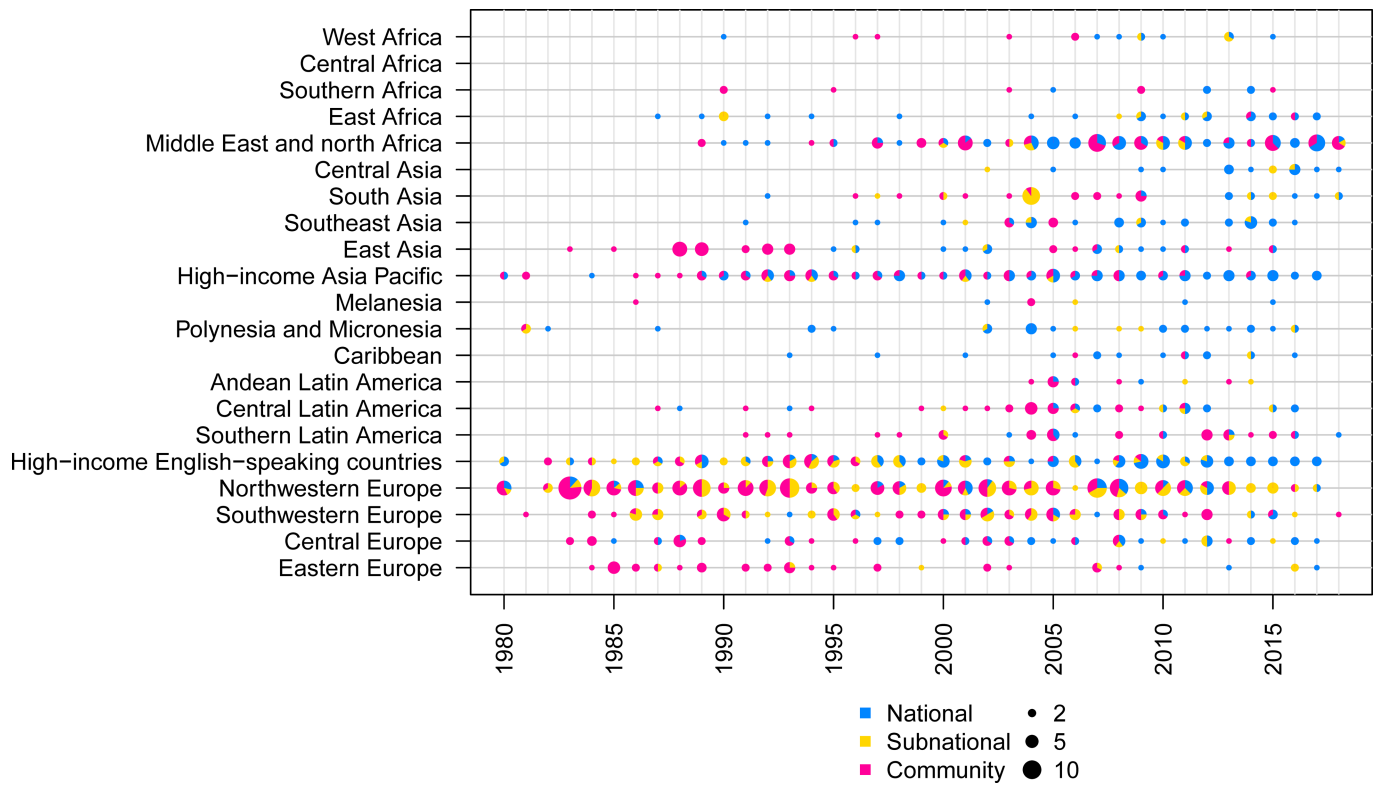
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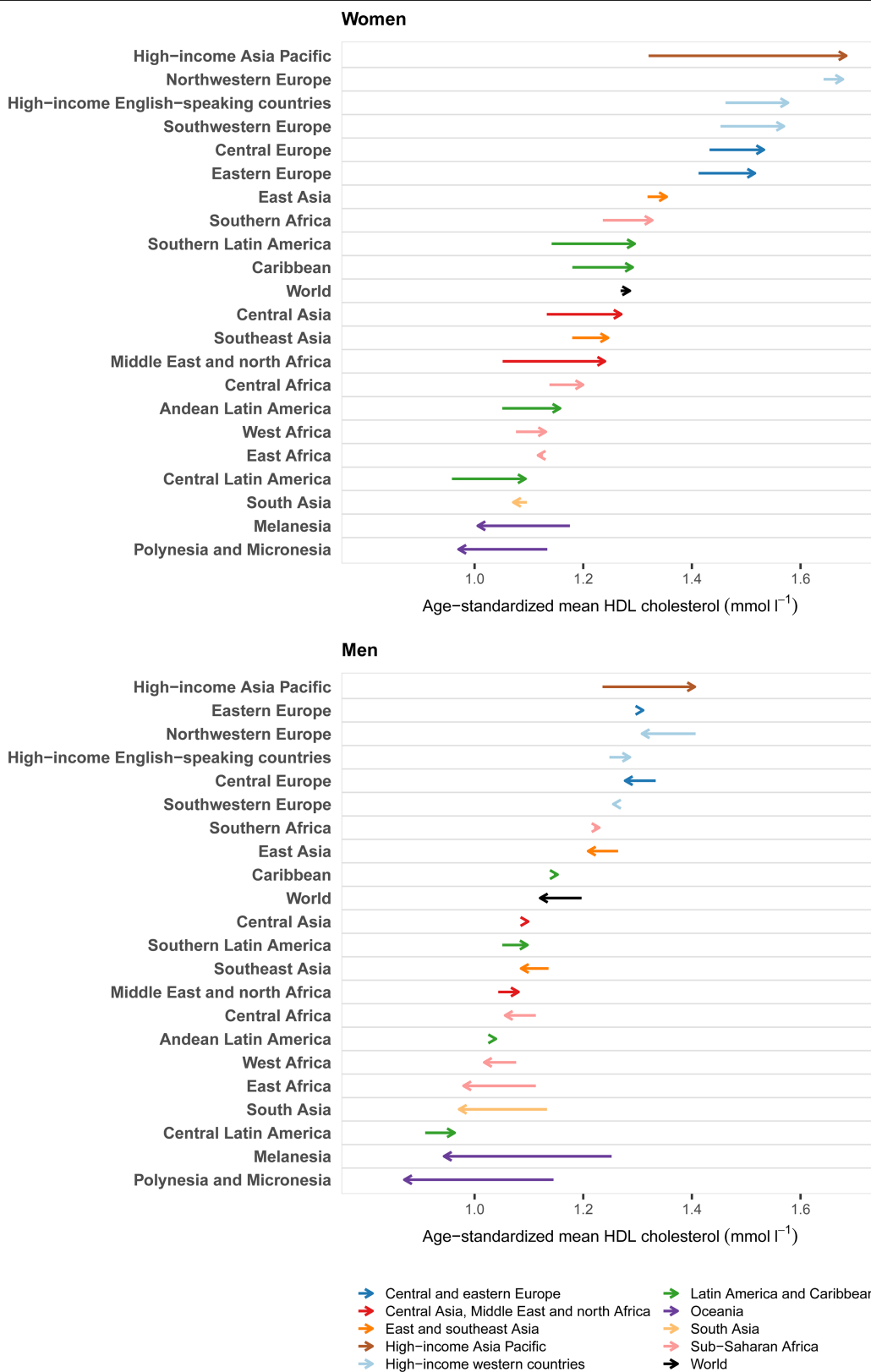
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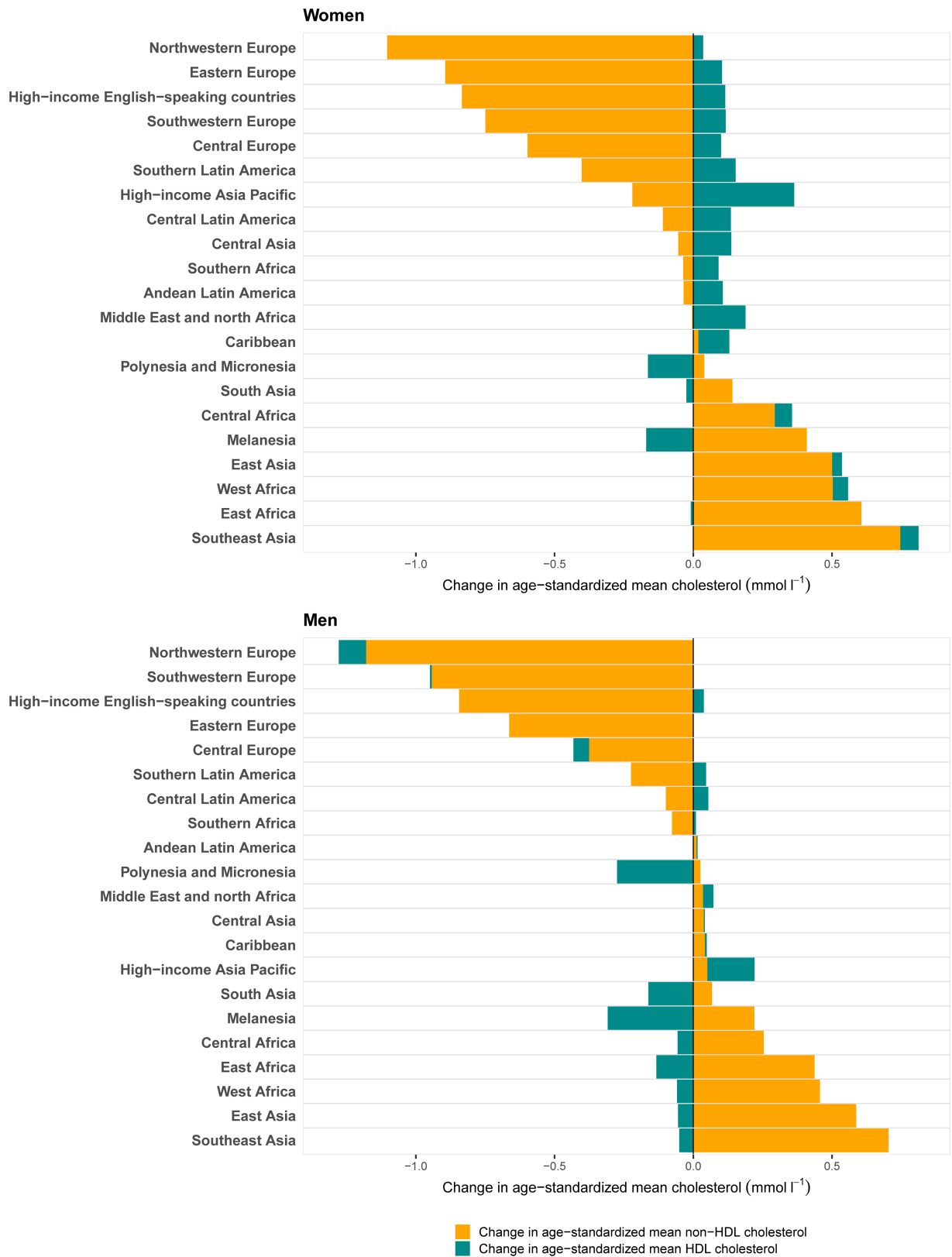
Extended Data Fig. 1 | Number of data sources by country. The colour indicates the number of data sources for each country used in the analysis. Countries and territories that were not included in the analysis are coloured in grey.



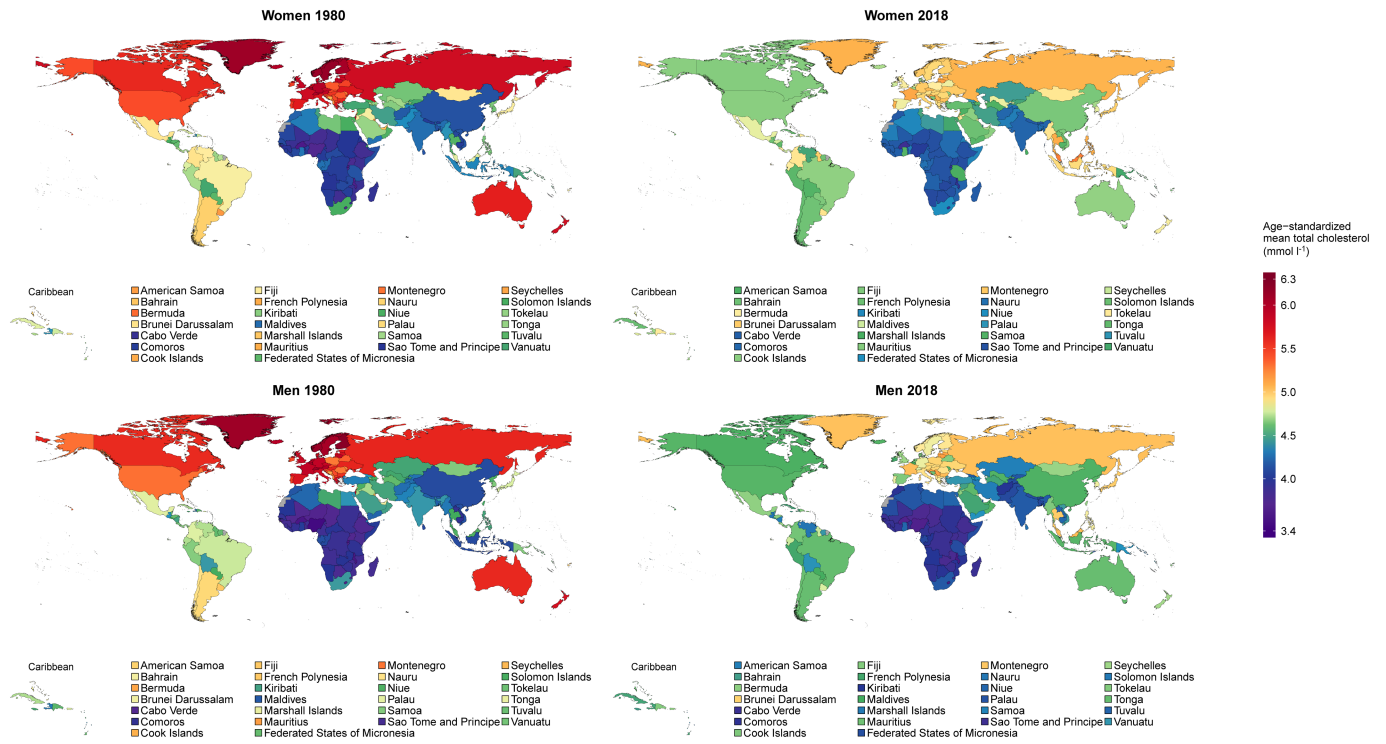
Extended Data Fig. 2 | Number of data sources by region and year. The size of each circle shows the number of data sources for each region and year, and the colours indicate the relative size of national, subnational and community data sources.



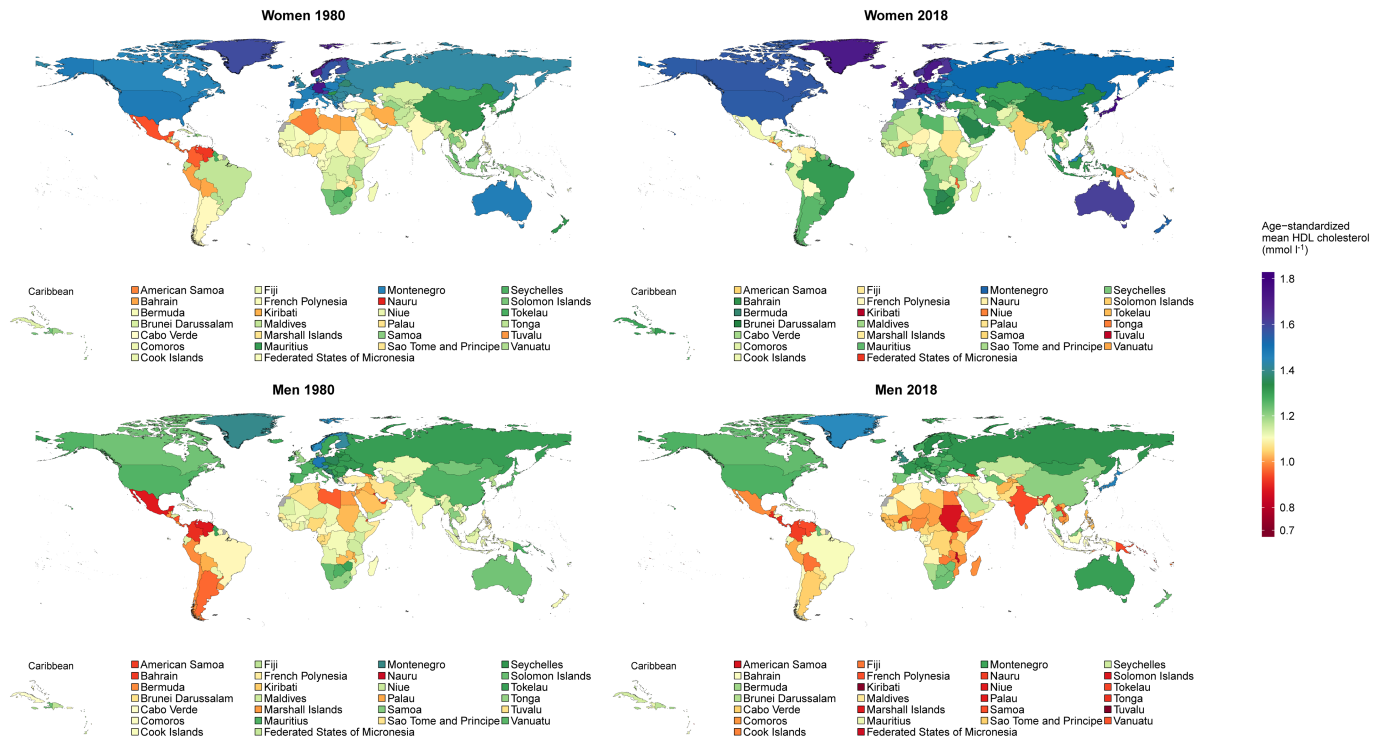
Extended Data Fig. 3 | Change in age-standardized mean HDL cholesterol between 1980 and 2018 by region for women and men. The start of the arrow shows the level in 1980 and the head shows the level in 2018. One mmol l⁻¹ is equivalent to 38.61 mg dl⁻¹.



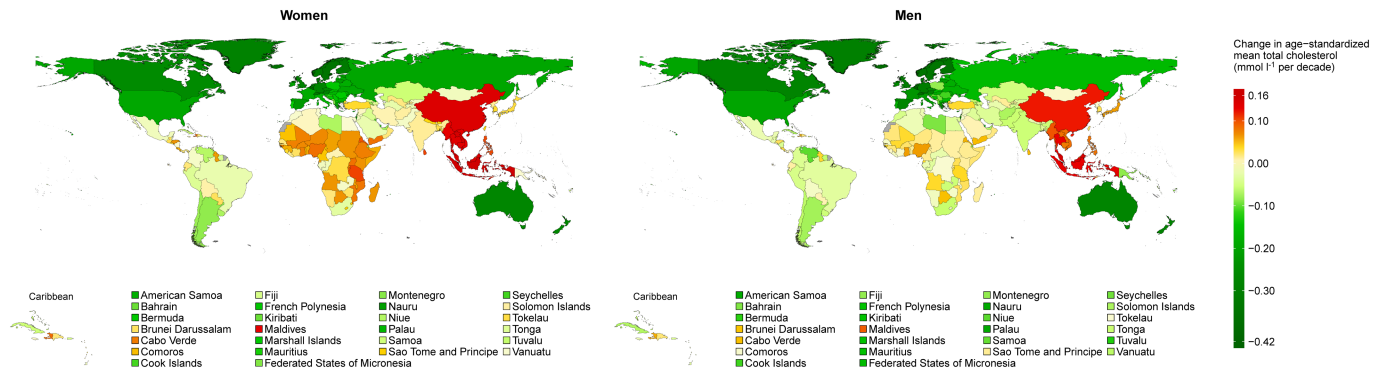
Extended Data Fig. 4 | Change in age-standardized mean HDL and non-HDL cholesterol between 1980 and 2018 by region for women and men. One mmol l⁻¹ is equivalent to 38.61 mg dl⁻¹.



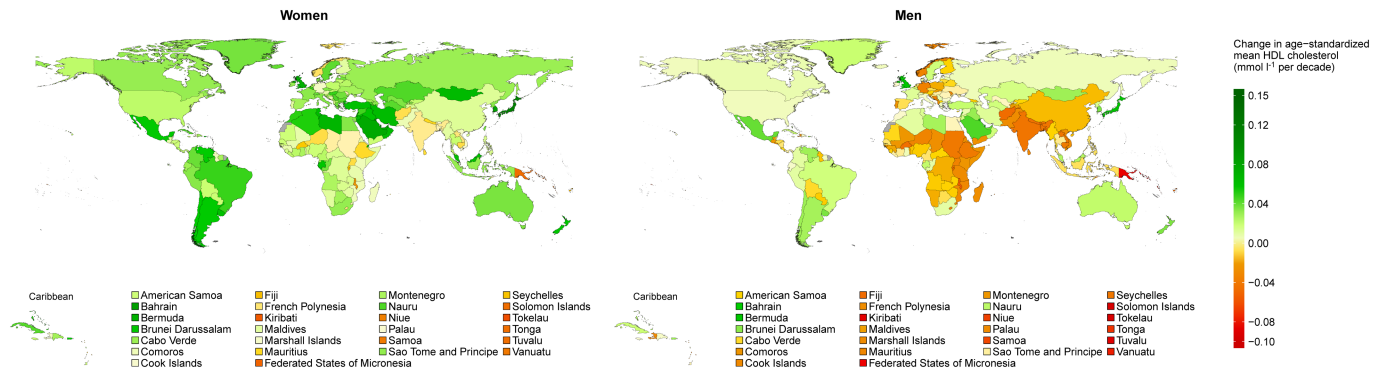
Extended Data Fig. 5 | Age-standardized mean total cholesterol by country in 1980 and 2018 for women and men. One mmol l⁻¹ is equivalent to 38.61 mg dl⁻¹.



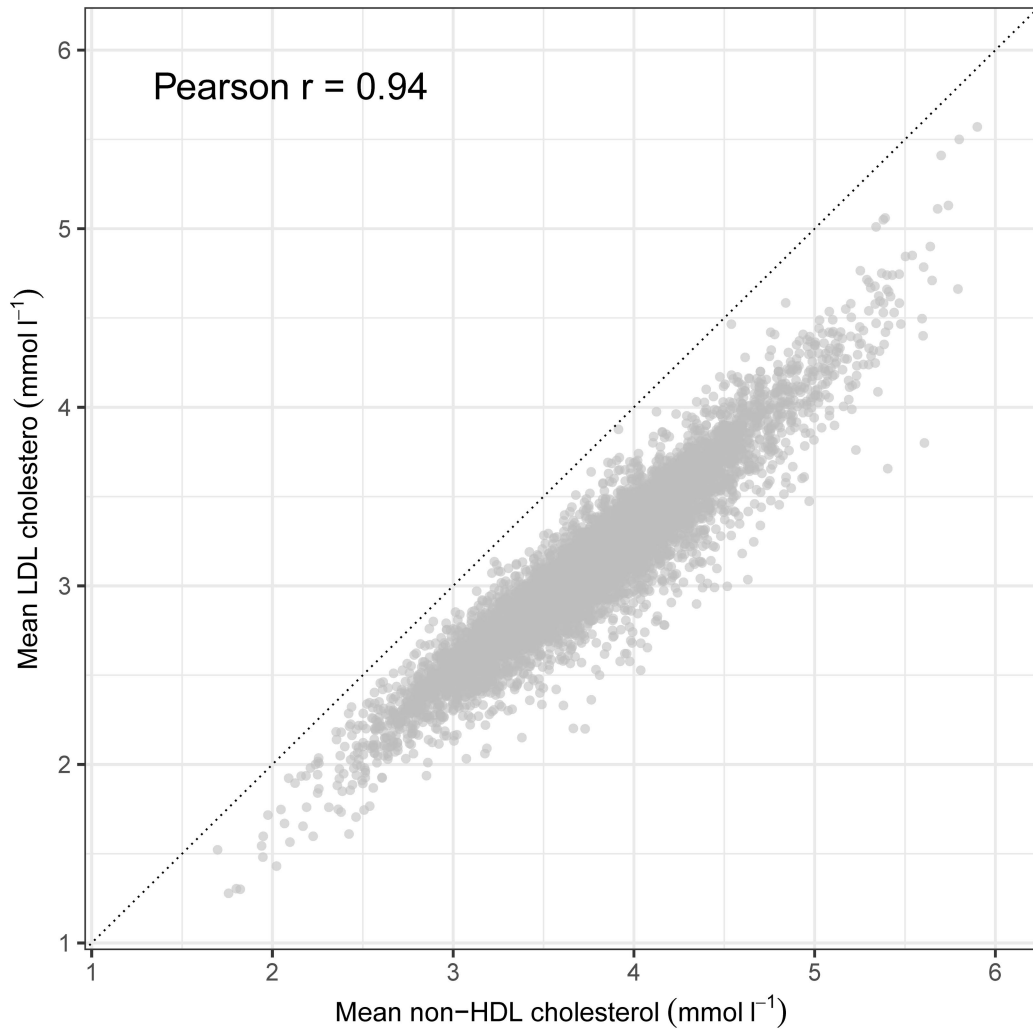
Extended Data Fig. 6 | Age-standardized mean HDL cholesterol by country in 1980 and 2018 for women and men. One mmol l⁻¹ is equivalent to 38.61 mg dl⁻¹.



Extended Data Fig. 7 | Change per decade in age-standardized mean total cholesterol by country for women and men. One mmol l⁻¹ is equivalent to 38.61 mg dl⁻¹.



Extended Data Fig. 8 | Change per decade in age-standardized mean HDL cholesterol by country for women and men. One mmol l⁻¹ is equivalent to 38.61 mg dl⁻¹.



Extended Data Fig. 9 | The association between mean LDL and non-HDL cholesterol in studies that measured lipids in a laboratory that had data for both variables. Each data point is one study-age-sex group ($n = 6,864$). One mmol l^{-1} is equivalent to 38.61 mg dl^{-1} .

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Data collection

Processing of secondary data was conducted using the statistical software R (version 3.6.0).

Data analysis

All analyses were conducting using the statistical software R (version 3.6.0). The code for estimation of mean risk factor trends is available at www.ncdrisc.org.

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This is a data-pooling study that brings together more than 1000 disparate data sources and uses a Bayesian hierarchical model to estimate population risk factor trends. Estimates of mean total, non-HDL and HDL cholesterol by country, year, and sex will be available from www.ncdrisc.org upon the publication of the paper. Some of the input data sources are publicly available, for which we will add links in the final version of the paper. Others are the property of specific research groups and agencies, for which we will provide contact information.

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Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	We pooled and re-analysed population-based data that had measured blood lipids in adults to estimate trends in mean total, non-HDL and HDL cholesterol from 1980 to 2018 for 200 countries and territories, using a Bayesian hierarchical model.
Research sample	We pooled data from 1,127 population-based studies of blood lipids conducted in 161 countries, with measurement of blood lipids in over 102 million adults aged 18 years and older. Studies were representative of a national, subnational or community population.
Sampling strategy	We included data collected using a probabilistic sampling method with a defined sampling frame. We therefore included studies with simple random and complex survey designs but excluded convenience samples.
Data collection	We used data on measured blood lipids to calculate mean total, non-HDL and HDL cholesterol. We excluded self-reported data.
Timing	We pooled data collected from 1980 to 2018. We also included national studies for the 3 years prior to 1980 (n=1), assigning them to 1980, so that they can inform the estimates in countries with slightly earlier national data.
Data exclusions	<p>We excluded all data sources that included only hypercholesterolemia or dyslipidaemia diagnosis history or medication status without measurement of cholesterol levels. We also excluded data sources on population subgroups whose lipid profile may differ systematically from the general population, including:</p> <ul style="list-style-type: none"> • studies that had included or excluded people based on their health status or cardiovascular risk; • studies whose participants were only ethnic minorities; • specific educational, occupational, or socioeconomic subgroups, with the exception noted below; • those recruited through health facilities, with the exception noted below. <p>We used school-based data in countries, and in age-sex groups, where secondary school enrollment was 70% or higher. We used data whose sampling frame was health insurance schemes in countries where at least 80% of the population were insured. Finally, we used data collected through general practice and primary care systems in high-income and central European countries with universal insurance, because contact with the primary care systems tends to be as good as or better than response rates for population-based surveys. Our exclusion criteria were established at the initiation of the study to ensure all data were representative.</p>
Non-participation	Our inclusion/exclusion criteria were designed to ensure participants of the surveys included were representative of the general population from which each sample was drawn.
Randomization	Our study is descriptive, and we did not carry out experiments.

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