# Distinct Ethnic Differences in Lipid Profiles across Glucose Categories

Lei Zhang, Qing Qiao, Jaakko Tuomilehto, Edward D. Janus, Tai Hing Lam, Ambady Ramachandran, Viswanathan Mohan, Coen D. A. Stehouwer, Yanhu Dong, Tomoko Nakagami, Altan Onat, and Stefan Söderberg, for the DECODE/DECODA Study Group

Department of Public Health (L.Z., Q.Q., J.T.), University of Helsinki, FIN-00014 Helsinki, Finland; Diabetes Prevention Unit (L.Z., Q.Q., J.T.), Department of Chronic Disease Prevention, National Institute for Health and Welfare, FIN-00271 Helsinki, Finland; Qingdao Endocrinology and Diabetes Hospital and Institute (L.Z., Y.D.), 266071 Qingdao, China; Department of Medicine, University of Melbourne (E.D.J.), Western Hospital, Footscray, VIC 3011, Australia; Department of Community Medicine (T.H.L.), School of Public Health, University of Hong Kong, Hong Kong, China; India Diabetes Research Foundation and Dr. A. Ramachandran's Diabetes Hospitals (A.R.), Egmore, Chennai 600 008, India; Madras Diabetes Research Foundation and Dr. Mohan's Diabetes Specialities Centre (V.M.), Chennai 600 086, India; Department of Internal Medicine and Cardiovascular Research Institute Maastricht (C.D.A.S.), Maastricht University Medical Centre, 6202 AZ Maastricht, The Netherlands; Diabetes Center (T.N.), Tokyo Women's Medical University School of Medicine, 162-8666 Tokyo, Japan; Turkish Society of Cardiology (A.O.), Istanbul University, Istanbul 34452, Turkey; Department of Cardiology (A.O.), Cerrahpaçsa Medical Faculty, Istanbul University, Istanbul 34384, Turkey; Department of Public Health and Clinical Medicine, Cardiology (S.S.), University of Umeå, SE-901 87 Umeå, Sweden; and Baker IDI Heart and Diabetes Institute (S.S.), Melbourne, VIC 8008, Australia

**Context:** Dyslipidemia coexists with hyperglycemia. However, little is known about the ethnic differences in lipid profiles at comparable glucose tolerance status.

**Objective:** The aim was to study ethnic differences in lipid profiles stratified by glucose levels.

**Design and Setting:** Data from 31 study cohorts of 12 countries, consisting of 24,760 men and 27,595 women aged 25–74 yr, were compared. The odds ratio for having dyslipidemia was estimated for each ethnic group stratified by glucose categories.

**Results:** Compared with central and northern Europeans, multivariable adjusted odds ratios (95% confidence intervals) for having lower high-density lipoprotein-cholesterol were 4.74 (4.19–5.37), 5.05 (3.88–6.56), 3.07 (2.15–4.40), and 2.37 (1.67–3.35) in Asian Indian men, but 0.12 (0.09–0.16), 0.07 (0.04–0.13), 0.11 (0.07–0.20), and 0.16 (0.08–0.32) in Chinese men who had normoglycemia, prediabetes, and undiagnosed and diagnosed diabetes, respectively. Similar results were obtained for women. The prevalence of low high-density lipoprotein-cholesterol remained higher in Asian Indians (62.8% of the nondiabetic and 67.4% of the diabetic) than in central and northern Europeans (20.3 and 37.3%), Japanese (25.7 and 34.1%), or Qingdao Chinese (15.7 and 17.0%), even in individuals with low-density lipoprotein-cholesterol of less than 3 mmol/liter.

**Conclusion:** There are distinct patterns of lipid profiles associated with ethnicity regardless of the glucose levels, suggesting that ethnic-specific strategies and guidelines on risk assessment and prevention of cardiovascular disease are required. **(*J Clin Endocrinol Metab* 95: 1793–1801, 2010)**

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Abbreviations: BMI, Body mass index; CI, confidence interval; C&N, central and northern; CVD, cardiovascular disease; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol; 2hPG, plasma glucose 2 h after 75-g glucose load; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; LDL-C, low-density lipoprotein cholesterol; NFG, normal fasting glucose; NGT, normal glucose tolerance; TC, total cholesterol; TG, triglycerides.

Dyslipidemia coexists not only with diabetes but also with impaired glucose tolerance (IGT) and/or impaired fasting glucose (IFG) (1, 2). A pattern of atherogenic dyslipidemia, consisting of high triglycerides (TGs) and small, dense, low-density lipoprotein cholesterol (LDL-C) and low high-density lipoprotein cholesterol (HDL-C), is closely linked to the progress of insulin resistance and atherosclerosis in the hyperglycemic state (3). It has already been shown that the prevalence of lipid or glucose abnormality differs between ethnic groups. A high prevalence of atherogenic dyslipidemia (4, 5) or diabetes (6) has been reported in Asian Indians compared with whites. However, little is known about the ethnic differences in lipid profiles at comparable glucose tolerance status. This study based on the collaborative data analysis of the DECODE (Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Europe) and DECODA (Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Asia) studies of Europeans and Asians addresses this issue.

## Subjects and Methods

### Study population

The study population and the recruitment of participants have been described in detail in previous DECODE/DECODA publications (7–10). Briefly, researchers who had carried out population-based or large occupational epidemiological studies on diabetes in Europe or Asia, using a standard 2-h 75-g oral glucose tolerance test, were invited to participate. Individual data on fasting plasma glucose (FPG) and 2-h plasma glucose (2hPG) concentrations as well as blood total cholesterol (TC), HDL-C, and TG and a number of other variables were sent to the Diabetes Prevention Unit of the National Institute for Health and Welfare in Helsinki, Finland, for collaborative data analysis. The Ethics Committee of the National Public Health Institute had approved the data analysis plans for both the DECODE and the DECODA studies. The inclusion criteria for the current data analysis were: 1) participants aged 25 to 74 yr; 2) baseline examination performed after 1980; and 3) data on TC, HDL-C, TG, FPG, 2hPG, and body mass index (BMI) available. A total of 52,355 subjects (24,760 men and 27,595 women with a mean age of 50 yr) from 31 (18 in DECODE and 13 in DECODA) study cohorts of 12 countries, including Asian Indian, Chinese, European, Japanese, and Mauritian Indian subjects, met the inclusion criteria and were included in the data analysis (Table 1). The Chinese were further divided into Qingdao and Hong Kong subgroups, and the Europeans into southern (Italy, Republic of Cyprus, and Spain) and central and northern (C&N) Europeans (Finland, Poland, Sweden, The Netherlands, and the United Kingdom) considering varying geographic locations, living environments, lifestyle habits, and socioeconomic status that may have a complex impact on metabolic characteristics.

### Measurements

In all cohorts, blood samples were collected after overnight fasting for the measurements of plasma glucose and lipids. Plasma glucose was measured in each of the studies with an oxidase or dehydrogenase method. Detailed information on lipid and lipoprotein assays in each study is shown in Supplemental Table 1 (published as supplemental data on The Endocrine Society's Journals Online web site at http://jcem.endojournals.org). Briefly, TC and TG were determined using enzymatic techniques (11–13) in all laboratories, and HDL-C was measured enzymatically after a precipitation of apolipoprotein B-containing lipoproteins with established methods (14) in most of the laboratories, except in the Qingdao 2006 study from China, the Funagata study from Japan, and two Indian studies in India applying direct method for HDL-C assay (Table 1). LDL-C was calculated among individuals with a TG value of less than 4.5 mmol/liter (n = 51,521) using the Friedewald formula (15) as follows: LDL-C (in mmol/liter) = TC − HDL-C − (0.45∗TG). Individuals with a TG of at least 4.5 mmol/liter (n = 834) were excluded from the analysis related to the LDL-C. The Mauritian samples were analyzed locally, with quality control analyzed in Newcastle, United Kingdom, as previously described (16, 17).

According to the World Health Organization 1999 criteria (18), a person with a prior history of diabetes was classified with previously diagnosed diabetes regardless of the glucose levels, and those with no history of diabetes were classified based on both FPG and 2hPG levels. Previously undiagnosed diabetes was defined as an FPG of at least 7.0 mmol/liter and/or 2hPG of at least 11.1 mmol/liter; IFG and/or IGT, *i.e.* prediabetes, was defined as FPG of 6.1–6.9 mmol/liter or 2hPG of 7.8–11.0 mmol/liter; and normal fasting glucose (NFG) and normal glucose tolerance (NGT), *i.e.* normoglycemia, were defined as FPG less than 6.1 mmol/liter and 2hPG less than 7.8 mmol/liter, respectively. According to the International Diabetes Federation criteria for the metabolic syndrome (19), elevated TG was defined as TG of at least 1.7 mmol/liter; reduced HDL-C was defined as HDL-C less than 1.03 mmol/liter for men and less than 1.29 mmol/liter for women. High LDL-C was defined as LDL-C of at least 3.0 mmol/liter according to the European guidelines on cardiovascular disease (CVD) prevention (20).

### Statistical analysis

Analysis of covariance was used to estimate the ethnic- and sex-specific mean concentration of each lipid variable with 95% confidence interval (CI), adjusted for age, study cohort, and BMI. Within each glucose category, pairwise comparisons between ethnic groups were made with Bonferroni method to adjust for multiple comparisons. Logarithmic transformation was used for TG due to its skewed distribution. Logistic regression analysis was used to estimate the odds of having low HDL-C, high TG, or high LDL-C for each ethnic group as compared with that for C&N Europeans (reference group) at a given glucose category, adjusted for age, study cohort, BMI, systolic blood pressure, and smoking status. Waist was not adjusted in the multivariate analysis because it was not available for every study. The proportions of individuals with different dyslipidemia were compared between ethnic groups using the $\chi^2$ test. All statistical analyses were performed using SPSS 15.0 for Windows (SPSS Inc., Chicago, IL).

## Results

### Lipid distributions in relation to ethnicity and glucose categories

Age-, study cohort-, and BMI-adjusted mean TC, LDL-C, and TG increased, whereas the mean HDL-C decreased with

**TABLE 1.** Characteristics of study cohorts included in the study

| Ethnic group, country | Study cohort | Year of survey | n | Mean age (yr) | Diabetes (%) | | IFG and/or IGT (%) | BMI (kg/m$^2$) | TC (mmol/liter) | LDL-C (mmol/liter)[a] | HDL-C (mmol/liter) | TG (mmol/liter) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Undiagnosed | Diagnosed | | | | | | |
| Men | | | | | | | | | | | | |
| Chinese, Hong Kong, China | HK-wscvdrf[b] | 1991 | 831 | 38 ± 8 | 3.7 | 1.9 | 8.3 | 23.3 ± 2.9 | 5.3 ± 1.0 | 3.39 ± 0.90 | 1.25 ± 0.31 | 1.35 ± 0.88 |
| | HK-cvrfps[c] | 1995–1996 | 1361 | 46 ± 13 | 6.8 | 3.5 | 15.3 | 24.3 ± 3.4 | 5.1 ± 0.9 | 3.22 ± 0.87 | 1.16 ± 0.30 | 1.38 ± 0.87 |
| Chinese, Qingdao, China | Qingdao 2002 | 2002 | 697 | 53 ± 11 | 11.2 | 2.0 | 18.1 | 26.6 ± 3.4 | 5.6 ± 1.1 | 3.34 ± 0.95 | 1.50 ± 0.28 | 1.75 ± 1.32 |
| | Qingdao 2006 | 2006 | 1614 | 49 ± 10 | 13.8 | 6.9 | 25.1 | 25.8 ± 3.2 | 5.3 ± 1.0 | 2.99 ± 0.95 | 1.59 ± 0.44[d] | 1.49 ± 1.13 |
| Asian Indian, India | Chennai 94 | 1994–1995 | 255 | 49 ± 7 | 11.8 | 15.3 | 13.7 | 22.4 ± 3.9 | 5.1 ± 1.0 | 3.09 ± 0.82 | 1.04 ± 0.25 | 1.92 ± 1.26 |
| | Chennai 97 | 1996–1998 | 397 | 44 ± 12 | 6.0 | 10.3 | 8.6 | 22.2 ± 4.1 | 4.5 ± 1.0 | 2.82 ± 0.85 | 0.96 ± 0.22 | 1.50 ± 1.00 |
| | CURES | 2003–2004 | 959 | 42 ± 11 | 11.7 | 8.8 | 11.1 | 23.2 ± 3.8 | 4.7 ± 1.0 | 2.91 ± 0.86 | 1.04 ± 0.24[d] | 1.64 ± 1.12 |
| | Chennai 2006 | 2006 | 2624 | 41 ± 11 | 5.9 | 11.5 | 8.3 | 22.9 ± 4.2 | 4.3 ± 1.0 | 2.40 ± 0.78 | 1.07 ± 0.24[d] | 1.63 ± 1.11 |
| Mauritian Indian, Mauritius | Mauritius 87 | 1987 | 1518 | 42 ± 12 | 9.3 | 5.6 | 15.3 | 22.8 ± 3.7 | 5.6 ± 1.6 | 3.47 ± 1.46 | 1.27 ± 0.36 | 1.83 ± 1.35 |
| | Mauritius 92 | 1992 | 846 | 42 ± 11 | 9.7 | 7.7 | 20.8 | 24.0 ± 4.8 | 4.9 ± 0.8 | 2.90 ± 0.75 | 1.23 ± 0.33 | 1.62 ± 1.12 |
| | Mauritius 98 | 1998 | 355 | 43 ± 11 | 13.0 | 9.6 | 15.5 | 24.5 ± 4.1 | 5.1 ± 1.1 | 3.35 ± 0.99 | 0.90 ± 0.34 | 1.70 ± 1.00 |
| Japanese, Japan | Funagata | 1995–1997 | 802 | 56 ± 11 | 3.2 | 5.6 | 12.5 | 23.6 ± 3.0 | 5.2 ± 0.9 | 3.05 ± 0.84 | 1.41 ± 0.37[d] | 1.53 ± 1.47 |
| | Hisayama | 1988 | 978 | 55 ± 9 | 8.1 | 10.4 | 23.8 | 22.9 ± 2.9 | 5.1 ± 1.0 | 3.21 ± 1.00 | 1.26 ± 0.31 | 1.68 ± 1.49 |
| Italian, Italy | Cremona Study | 1990–1991 | 742 | 55 ± 8 | 3.0 | 8.2 | 10.5 | 26.8 ± 3.7 | 6.0 ± 1.1 | 3.98 ± 0.97 | 1.25 ± 0.38 | 1.67 ± 1.36 |
| Cypriot, Republic of Cyprus | Nicosia Diabetes Study | 2003–2004 | 542 | 49 ± 12 | 7.0 | 11.4 | 12.0 | 27.7 ± 4.1 | 5.5 ± 1.1 | 3.57 ± 0.95 | 1.15 ± 0.29 | 1.78 ± 1.56 |
| Spanish, Spain | The Guia Study | 1997 | 246 | 52 ± 13 | 8.5 | 9.3 | 19.5 | 27.5 ± 3.5 | 5.9 ± 1.1 | 3.73 ± 0.86 | 1.23 ± 0.32 | 1.75 ± 1.09 |
| | The Viva Study | 1995–1998 | 902 | 49 ± 8 | 4.2 | 2.3 | 13.6 | 27.6 ± 3.7 | 5.8 ± 1.1 | 3.89 ± 0.99 | 1.19 ± 0.32 | 1.54 ± 1.10 |
| Finnish, Finland | East-West men | 1989 | 156 | 71 ± 1 | 14.1 | 7.1 | 28.8 | 26.3 ± 3.9 | 5.8 ± 1.1 | 3.93 ± 0.99 | 1.16 ± 0.27 | 1.44 ± 0.65 |
| | FINMONICA | 1987–1992 | 877 | 54 ± 6 | 5.9 | 4.0 | 25.5 | 27.7 ± 3.8 | 6.0 ± 1.0 | 3.82 ± 0.92 | 1.24 ± 0.32 | 1.94 ± 1.24 |
| | National FINRISK Study 2002 | 2002 | 1799 | 58 ± 7 | 11.1 | 8.1 | 34.0 | 28.0 ± 4.0 | 5.8 ± 1.1 | 3.59 ± 0.96 | 1.34 ± 0.36 | 1.73 ± 1.08 |
| | Oulu Study | 1992 | 325 | 55 | 22.2 | 5.8 | 43.4 | 26.7 ± 3.4 | 5.6 ± 1.3 | 3.82 ± 1.21 | 1.17 ± 0.33 | 1.35 ± 0.98 |
| | Savitaipale | 1996–1999 | 584 | 53 ± 7 | 7.0 | 6.5 | 36.3 | 26.4 ± 3.9 | 5.6 ± 1.0 | 3.56 ± 0.87 | 1.35 ± 0.36 | 1.49 ± 1.11 |
| | Vantaa Study | 1990–1991 | 272 | 65 ± 0.4 | 4.4 | 11.4 | 30.1 | 26.9 ± 3.7 | 5.8 ± 1.0 | 3.83 ± 0.93 | 1.16 ± 0.30 | 1.58 ± 0.94 |
| Polish, Poland | POLMONICA | 1992–1993 | 168 | 58 ± 8 | 8.3 | 0 | 32.7 | 26.5 ± 4.1 | 5.5 ± 0.9 | 3.43 ± 0.85 | 1.39 ± 0.29 | 1.49 ± 0.98 |
| Swedish, Sweden | MONICA 1986 | 1986 | 218 | 44 ± 11 | 1.8 | 11.0 | 6.9 | 25.4 ± 3.7 | 5.9 ± 1.1 | 4.12 ± 1.00 | 1.13 ± 0.25 | 1.41 ± 0.96 |
| | MONICA 1990 | 1990 | 364 | 45 ± 11 | 1.9 | 2.7 | 11.3 | 25.8 ± 3.3 | 6.2 ± 1.2 | 4.14 ± 1.08 | 1.28 ± 0.31 | 1.66 ± 1.02 |
| | MONICA 1994 | 1994 | 497 | 50 ± 13 | 3.6 | 5.4 | 13.3 | 27.3 ± 3.7 | 6.2 ± 1.3 | 4.12 ± 1.09 | 1.22 ± 0.30 | 1.70 ± 1.24 |
| | ULSAM | 1991–1995 | 1176 | 70 ± 0.6 | 10.6 | 5.9 | 30.5 | 26.3 ± 3.4 | 5.8 ± 1.0 | 3.82 ± 0.88 | 1.28 ± 0.35 | 1.45 ± 0.77 |
| Dutch, The Netherlands | The Hoorn Study | 1989–1991 | 1080 | 60 ± 6 | 6.9 | 3.1 | 18.5 | 26.2 ± 3.0 | 6.4 ± 1.1 | 4.42 ± 1.02 | 1.18 ± 0.31 | 1.68 ± 1.02 |
| | Zutphen | 1990 | 211 | 71 ± 1 | 10.4 | 7.6 | 24.2 | 25.9 ± 2.9 | 6.2 ± 1.1 | 4.28 ± 1.03 | 1.15 ± 0.31 | 1.57 ± 0.87 |
| British, United Kingdom | ELY | 1990–1992 | 473 | 54 ± 8 | 8.2 | 0 | 34.2 | 26.1 ± 3.2 | 6.5 ± 1.2 | 4.43 ± 1.05 | 1.29 ± 0.32 | 1.49 ± 0.71 |
| | Newcastle Heart Project | 1993–1994 | 376 | 54 ± 12 | 8.5 | 2.7 | 31.4 | 26.3 ± 3.9 | 5.7 ± 1.0 | 3.61 ± 0.94 | 1.35 ± 0.39 | 1.51 ± 0.79 |
| | The Goodinge Study | 1990–1991 | 446 | 54 ± 10 | 9.4 | 0 | 43.0 | 25.5 ± 4.1 | 6.3 ± 1.2 | 4.17 ± 1.13 | 1.29 ± 0.38 | 1.70 ± 1.29 |
| Women | | | | | | | | | | | | |
| Chinese, Hong Kong, China | HK-wscvdrf[b] | 1991 | 551 | 40 ± 8 | 3.1 | 2.0 | 8.7 | 23.5 ± 3.5 | 4.8 ± 0.9 | 2.85 ± 0.82 | 1.52 ± 0.38 | 0.89 ± 0.64 |
| | HK-cvrfps[c] | 1995–1996 | 1409 | 45 ± 12 | 6.4 | 4.3 | 17.3 | 24.0 ± 3.8 | 5.0 ± 1.0 | 3.16 ± 0.91 | 1.35 ± 0.32 | 1.09 ± 0.71 |

(*Continued*)

**TABLE 1.** (*Continued*)

| Ethnic group, country | Study cohort | Year of survey | n | Mean age (yr) | Diabetes (%) | | IFG and/or IGT (%) | BMI (kg/m$^2$) | TC (mmol/liter) | LDL-C (mmol/liter)[a] | HDL-C (mmol/liter) | TG (mmol/liter) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Undiagnosed | Diagnosed | | | | | | |
| Chinese, Qingdao, China | Qingdao 2002 | 2002 | 1192 | 52 ± 11 | 9.2 | 2.5 | 18.0 | 26.3 ± 3.8 | 5.7 ± 1.1 | 3.51 ± 0.91 | 1.52 ± 0.28 | 1.48 ± 0.95 |
| | Qingdao 2006 | 2006 | 2368 | 49 ± 10 | 12.0 | 7.4 | 26.7 | 25.8 ± 3.6 | 5.3 ± 1.1 | 3.04 ± 0.92 | 1.65 ± 0.40[d] | 1.27 ± 0.86 |
| Asian Indian, India | Chennai 94 | 1994–1995 | 215 | 50 ± 8 | 12.6 | 19.1 | 19.5 | 24.0 ± 4.7 | 5.4 ± 1.1 | 3.34 ± 0.91 | 1.13 ± 0.30 | 1.82 ± 0.92 |
| | Chennai 97 | 1996–1998 | 566 | 44 ± 13 | 5.8 | 6.9 | 7.8 | 23.5 ± 4.5 | 4.7 ± 1.0 | 2.95 ± 0.88 | 1.10 ± 0.27 | 1.30 ± 0.74 |
| | CURES | 2003–2004 | 1116 | 40 ± 11 | 9.8 | 5.1 | 11.4 | 23.6 ± 4.0 | 4.8 ± 1.0 | 2.93 ± 0.83 | 1.18 ± 0.25[d] | 1.33 ± 0.80 |
| | Chennai 2006 | 2006 | 3250 | 40 ± 10 | 5.1 | 9.2 | 8.5 | 23.9 ± 5.0 | 4.4 ± 1.0 | 2.52 ± 0.82 | 1.16 ± 0.25[d] | 1.45 ± 0.88 |
| Mauritian Indian, Mauritius | Mauritius 87 | 1987 | 1692 | 42 ± 13 | 7.0 | 4.8 | 21.4 | 23.9 ± 4.7 | 5.3 ± 1.5 | 3.37 ± 1.39 | 1.31 ± 0.31 | 1.29 ± 0.87 |
| | Mauritius 92 | 1992 | 960 | 43 ± 12 | 8.0 | 12.2 | 17.7 | 25.6 ± 5.2 | 4.8 ± 0.9 | 2.84 ± 0.75 | 1.33 ± 0.29 | 1.26 ± 0.75 |
| | Mauritius 98 | 1998 | 436 | 42 ± 10 | 8.7 | 8.9 | 18.1 | 25.2 ± 4.8 | 4.7 ± 1.1 | 3.05 ± 0.93 | 0.99 ± 0.32 | 1.24 ± 0.62 |
| Japanese, Japan | Funagata | 1995–1997 | 1121 | 56 ± 11 | 2.2 | 3.8 | 12.7 | 23.9 ± 3.5 | 5.4 ± 0.9 | 3.31 ± 0.86 | 1.53 ± 0.36[d] | 1.15 ± 0.63 |
| | Hisayama | 1988 | 1312 | 55 ± 9 | 4.9 | 5.6 | 22.3 | 23.1 ± 3.2 | 5.6 ± 1.1 | 3.69 ± 0.99 | 1.34 ± 0.29 | 1.21 ± 0.76 |
| Italian, Italy | Cremona Study | 1990–1991 | 876 | 56 ± 8 | 2.4 | 5.6 | 8.3 | 26.4 ± 4.9 | 6.2 ± 1.2 | 4.13 ± 1.10 | 1.46 ± 0.36 | 1.25 ± 0.62 |
| Cypriot, Republic of Cyprus | Nicosia Diabetes Study | 2003–2004 | 558 | 50 ± 13 | 2.5 | 5.6 | 12.2 | 27.0 ± 5.9 | 5.5 ± 1.0 | 3.38 ± 0.92 | 1.48 ± 0.36 | 1.27 ± 0.89 |
| Spanish, Spain | The Guia Study | 1997 | 322 | 51 ± 12 | 4.0 | 11.2 | 18.6 | 28.6 ± 5.4 | 6.0 ± 1.0 | 3.91 ± 0.91 | 1.38 ± 0.33 | 1.41 ± 0.81 |
| | The Viva Study | 1995–1998 | 1107 | 49 ± 9 | 4.2 | 2.3 | 13.6 | 28.2 ± 4.9 | 5.7 ± 1.1 | 3.72 ± 0.96 | 1.40 ± 0.33 | 1.13 ± 0.61 |
| Finnish, Finland | FINMONICA | 1987–1992 | 1043 | 54 ± 6 | 4.0 | 3.3 | 14.5 | 27.2 ± 4.8 | 5.9 ± 1.1 | 3.75 ± 0.95 | 1.50 ± 0.34 | 1.41 ± 0.90 |
| | National FINRISK Study 2002 | 2002 | 2065 | 57 ± 8 | 6.2 | 5.9 | 23.7 | 27.9 ± 5.1 | 5.8 ± 1.0 | 3.48 ± 0.92 | 1.65 ± 0.43 | 1.35 ± 0.72 |
| | Oulu Study | 1992 | 411 | 55 | 13.1 | 2.2 | 42.8 | 26.5 ± 4.3 | 5.9 ± 1.3 | 3.89 ± 1.29 | 1.48 ± 0.43 | 1.05 ± 0.55 |
| | Savitaipale | 1996–1999 | 583 | 54 ± 7 | 5.8 | 4.8 | 37.9 | 26.6 ± 4.8 | 5.7 ± 1.1 | 3.46 ± 0.93 | 1.58 ± 0.41 | 1.26 ± 0.80 |
| | Vantaa Study | 1990–1991 | 337 | 65 ± 0.4 | 8.0 | 8.0 | 28.2 | 27.6 ± 4.6 | 6.3 ± 1.0 | 4.15 ± 0.99 | 1.40 ± 0.33 | 1.53 ± 0.97 |
| Polish, Poland | POLMONICA | 1992–1993 | 190 | 57 ± 8 | 5.8 | 2.6 | 28.9 | 29.3 ± 4.9 | 5.9 ± 1.0 | 3.69 ± 0.88 | 1.46 ± 0.26 | 1.54 ± 0.81 |
| Swedish, Sweden | MONICA 1986 | 1986 | 204 | 44 ± 11 | 1.1 | 4.4 | 9.8 | 24.4 ± 4.5 | 6.0 ± 1.3 | 4.09 ± 1.17 | 1.38 ± 0.41 | 1.08 ± 0.67 |
| | MONICA 1990 | 1990 | 410 | 44 ± 11 | 1.5 | 1.7 | 10.5 | 25.1 ± 4.4 | 6.1 ± 1.3 | 3.89 ± 1.19 | 1.55 ± 0.35 | 1.33 ± 0.70 |
| | MONICA 1994 | 1994 | 521 | 49 ± 14 | 4.4 | 3.6 | 11.9 | 25.9 ± 4.7 | 6.2 ± 1.4 | 3.96 ± 1.21 | 1.49 ± 0.40 | 1.44 ± 0.81 |
| Dutch, The Netherlands | The Hoorn Study | 1989–1991 | 1238 | 61 ± 6 | 6.0 | 3.4 | 15.3 | 26.8 ± 4.0 | 6.9 ± 1.2 | 4.66 ± 1.10 | 1.45 ± 0.37 | 1.55 ± 0.96 |
| British, United Kingdom | ELY | 1990–1992 | 619 | 53 ± 8 | 6.0 | 0 | 28.3 | 25.9 ± 5.0 | 6.6 ± 1.4 | 4.39 ± 1.25 | 1.58 ± 0.38 | 1.28 ± 0.68 |
| | Newcastle Heart Project | 1993–1994 | 354 | 53 ± 11 | 4.8 | 1.7 | 23.7 | 26.4 ± 5.0 | 5.9 ± 1.2 | 3.56 ± 1.07 | 1.61 ± 0.39 | 1.46 ± 0.78 |
| | The Goodinge Study | 1990–1991 | 559 | 54 ± 10 | 8.6 | 0 | 30.1 | 26.0 ± 5.3 | 5.6 ± 1.4 | 4.31 ± 1.24 | 1.54 ± 0.40 | 1.48 ± 0.83 |

Data are expressed as number, means ± SD, or percentage. Precipitation method for HDL-C and enzymatic method for TC and TG were applied in all studies, unless otherwise indicated.

[a] Calculated only in individuals with TG < 4.5 mmol/liter.

[b] Hong Kong Workforce Survey on CVD Risk Factors.

[c] Hong Kong Cardiovascular Disease Risk Factor Prevalence Study.

[d] Direct method for HDL-C assay.

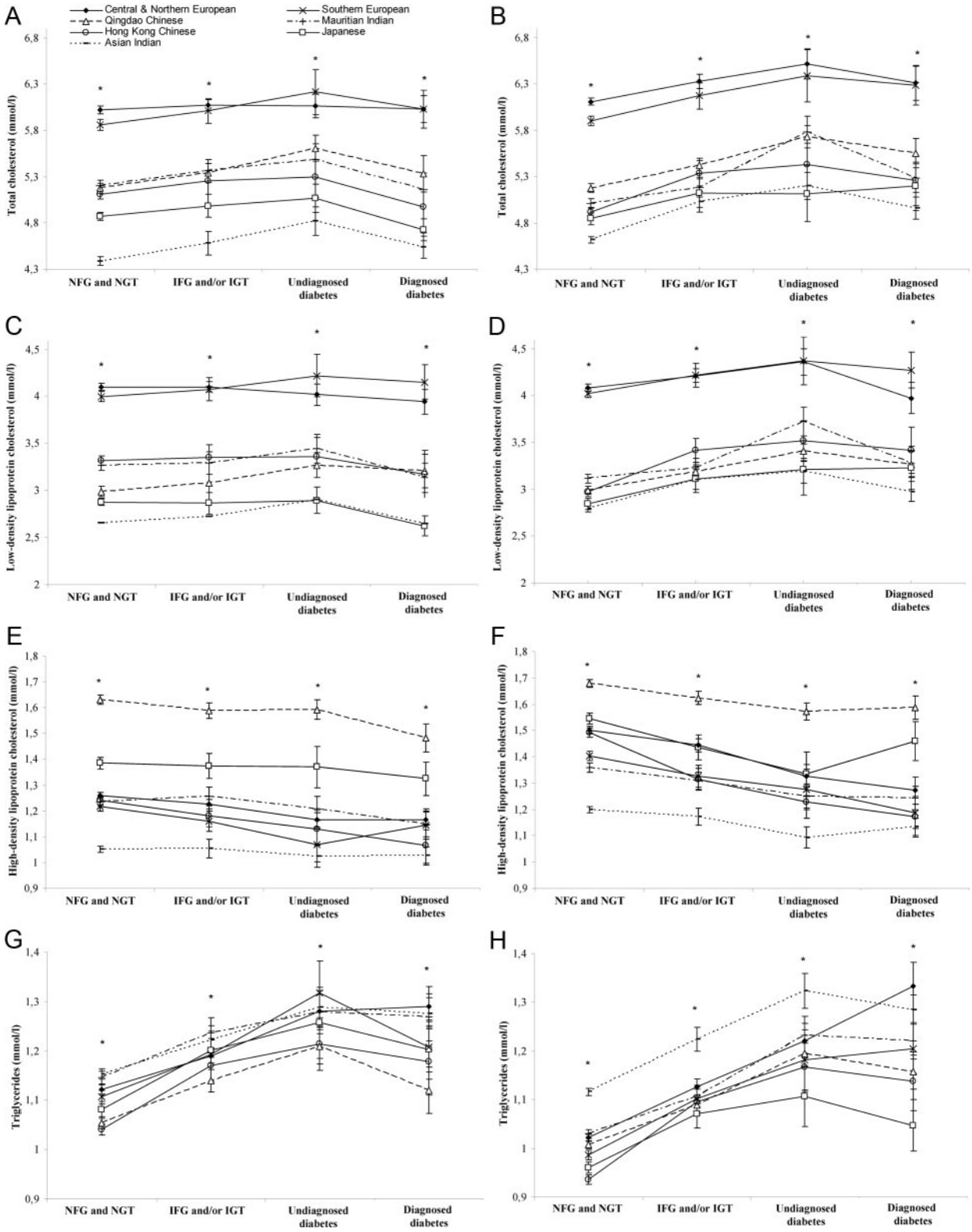


**FIG. 1.** Age-, study cohort-, and BMI-adjusted mean lipid (geometric means for TG) and lipoprotein concentrations and 95% CIs (*vertical bars*) in men (A, C, E, and G) and women (B, D, F, and H) by ethnicities and glucose categories. *, $P$ for trend $< 0.05$ within each glucose category.

more pronounced glucose intolerance in most of the ethnic groups in individuals without a prior history of diabetes (Fig. 1, A–H). Subjects with undiagnosed diabetes, however, had a worse lipid profile than those with known disease.

Within individuals with normoglycemia, mean lipid and lipoprotein concentrations differed among the ethnic groups. The Europeans had the highest TC (Fig. 1, A and B; $P < 0.05$) and LDL-C (Fig. 1, C and D; $P < 0.05$),

**TABLE 2.** Odds ratio (95% CI) of having dyslipidemia in relation to ethnicity by glucose categories

| | HDL-C < 1.03 mmol/liter in men and < 1.29 mmol/liter in women | | | | TG ≥ 1.7 mmol/liter | | | | LDL-C ≥ 3 mmol/liter | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NFG and NGT | IFG and/or IGT | Undiagnosed diabetes | Diagnosed diabetes | NFG and NGT | IFG and/or IGT | Undiagnosed diabetes | Diagnosed diabetes | NFG and NGT | IFG and/or IGT | Undiagnosed diabetes | Diagnosed diabetes |
| Men | | | | | | | | | | | | |
| C&N European[a] | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Hong Kong Chinese | 1.63 (1.41–1.87) | 2.75 (2.09–3.62) | 1.82 (1.20–2.76) | 2.57 (1.48–4.46) | 0.75 (0.64–0.87) | 1.16 (0.88–1.53) | 1.05 (0.70–1.58) | 0.63 (0.36–1.12) | 0.51 (0.44–0.58) | 0.61 (0.46–0.82) | 0.51 (0.33–0.78) | 0.86 (0.49–1.52) |
| Qingdao Chinese | 0.12 (0.09–0.16) | 0.07 (0.04–0.13) | 0.11 (0.07–0.20) | 0.16 (0.08–0.32) | 0.68 (0.58–0.79) | 0.81 (0.66–1.00) | 0.81 (0.61–1.09) | 0.40 (0.26–0.63) | 0.23 (0.20–0.26) | 0.30 (0.24–0.37) | 0.44 (0.32–0.60) | 0.57 (0.37–0.86) |
| Asian Indian | 4.74 (4.19–5.37) | 5.05 (3.88–6.56) | 3.07 (2.15–4.40) | 2.37 (1.67–3.35) | 1.40 (1.23–1.58) | 1.53 (1.19–1.97) | 1.24 (0.88–1.75) | 1.42 (1.01–2.00) | 0.12 (0.10–0.13) | 0.17 (0.13–0.22) | 0.23 (0.16–0.33) | 0.29 (0.20–0.41) |
| Mauritian Indian | 1.82 (1.58–2.09) | 2.04 (1.58–2.63) | 1.27 (0.89–1.81) | 1.16 (0.78–1.74) | 1.47 (1.28–1.69) | 1.55 (1.23–1.98) | 1.18 (0.85–1.65) | 1.06 (0.72–1.57) | 0.39 (0.34–0.45) | 0.38 (0.30–0.49) | 0.49 (0.34–0.70) | 0.75 (0.50–1.12) |
| Japanese | 0.87 (0.73–1.03) | 1.29 (0.98–1.70) | 0.73 (0.44–1.20) | 0.57 (0.36–0.90) | 0.99 (0.84–1.15) | 1.31 (1.02–1.68) | 1.36 (0.88–2.09) | 1.02 (0.68–1.53) | 0.26 (0.23–0.30) | 0.35 (0.27–0.44) | 0.36 (0.23–0.57) | 0.77 (0.51–1.16) |
| Southern European | 1.21 (1.06–1.37) | 1.49 (1.15–1.93) | 1.79 (1.19–2.70) | 1.13 (0.78–1.63) | 0.78 (0.69–0.88) | 0.83 (0.65–1.07) | 1.16 (0.77–1.75) | 0.58 (0.40–0.84) | 0.87 (0.75–1.00) | 0.99 (0.73–1.36) | 1.80 (1.01–3.23) | 1.52 (0.99–2.31) |
| Women | | | | | | | | | | | | |
| C&N European[a] | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| Hong Kong Chinese | 2.23 (1.93–2.57) | 3.79 (2.88–4.98) | 3.02 (1.88–4.85) | 3.03 (1.68–5.48) | 0.86 (0.69–1.08) | 1.16 (0.85–1.58) | 0.98 (0.61–1.57) | 0.69 (0.39–1.21) | 0.41 (0.35–0.47) | 0.64 (0.48–0.86) | 0.61 (0.36–1.04) | 1.21 (0.66–2.22) |
| Qingdao Chinese | 0.66 (0.57–0.76) | 0.52 (0.41–0.65) | 0.27 (0.19–0.38) | 0.20 (0.13–0.31) | 1.29 (1.11–1.50) | 1.06 (0.87–1.30) | 0.99 (0.73–1.36) | 0.57 (0.39–0.84) | 0.40 (0.36–0.45) | 0.45 (0.37–0.55) | 0.48 (0.33–0.69) | 0.67 (0.45–0.99) |
| Asian Indian | 10.91 (9.68–12.30) | 7.80 (5.99–9.94) | 8.64 (5.62–13.29) | 4.34 (2.93–6.44) | 2.76 (2.39–3.18) | 2.21 (1.71–2.87) | 3.13 (2.15–4.55) | 1.29 (0.90–1.85) | 0.22 (0.20–0.25) | 0.36 (0.28–0.47) | 0.36 (0.24–0.54) | 0.41 (0.28–0.60) |
| Mauritian Indian | 4.41 (3.88–5.02) | 3.80 (3.05–4.74) | 2.65 (1.82–3.88) | 2.26 (1.53–3.35) | 1.38 (1.16–1.65) | 1.15 (0.91–1.47) | 1.54 (1.07–2.23) | 0.81 (0.56–1.19) | 0.48 (0.42–0.55) | 0.50 (0.40–0.63) | 0.78 (0.51–1.21) | 0.85 (0.57–1.27) |
| Japanese | 2.40 (2.12–2.73) | 3.07 (2.44–3.87) | 2.65 (1.62–4.34) | 1.07 (0.67–1.72) | 0.92 (0.77–1.09) | 1.19 (0.93–1.53) | 0.72 (0.43–1.21) | 0.41 (0.25–0.68) | 0.58 (0.51–0.66) | 0.67 (0.52–0.87) | 0.56 (0.31–0.99) | 2.24 (1.27–3.93) |
| Southern European | 1.50 (1.34–1.68) | 1.62 (1.26–2.08) | 0.93 (0.56–1.52) | 1.70 (1.13–2.56) | 0.70 (0.60–0.81) | 0.80 (0.61–1.05) | 0.60 (0.36–1.01) | 0.53 (0.35–0.79) | 0.98 (0.87–1.11) | 1.39 (1.01–1.93) | 1.38 (0.70–2.72) | 2.67 (1.62–4.42) |

Model is adjusted for age, study cohort, BMI, systolic blood pressure, and smoking status.

[a] Reference group.

whereas Qingdao Chinese had the highest HDL-C levels (Fig. 1, E and F; $P < 0.05$) among all ethnic groups. In contrast, Asian Indians had the lowest TC (Fig. 1, A and B; $P < 0.05$), LDL-C (Fig. 1, C and D; $P < 0.05$), and HDL-C (Fig. 1, E and F; $P < 0.05$), but the highest TG (Fig. 1, G and H; $P < 0.05$) among the ethnic groups. These ethnic differences were consistently found in all glucose categories.

### Dyslipidemia in relation to ethnicity controlling for glucose levels

Compared with C&N Europeans, the multivariate-adjusted odds ratio (95% CI) of having low HDL-C was significantly higher for Asian Indians, Mauritian Indians, Hong Kong Chinese, and Southern Europeans but lower for Qingdao Chinese, across all glucose categories from normal to diabetes (Table 2). Asian Indians and Mauritian Indians tended to have higher odds ratios, but Southern Europeans had lower odds ratios for having high TG compared with the reference group. Unlike that for HDL-C or TG, the odds ratio for having high LDL-C was consistently lower in all Asian ethnic groups compared with the reference, across most of the glucose categories.

In contrast to the lower HDL-C and higher TG profiles, Asian Indians had considerably lower TC and LDL-C concentrations than others. As shown in Table 3, 71% of nondiabetic and 57.6% of diabetic Asian Indians had low LDL-C (<3.0 mmol/liter), whereas the corresponding figures were 19.2 and 24.6% ($P < 0.01$) for C&N Europeans and 46.6 and 38.8% ($P < 0.01$) for Qingdao Chinese. However, even within the low LDL-C category, there was still a higher proportion of Asian Indians having low HDL-C compared with others (Table 3). The results were confirmed in the same analysis conducted separately for men and women (data not shown).

## Discussion

This collaborative analysis of the data from the DECODE and the DECODA study cohorts showed considerable ethnic differences in lipid profiles within each glucose category. Asian Indians exhibited an adverse lipid pattern consisting of low HDL-C and high TG across all glucose categories as compared with other populations. Reduced HDL-C is prevalent even in Asian Indians with desirable LDL-C levels, regardless of the diabetic status. In addition, in most of the ethnic groups, individuals detected with undiagnosed diabetes had a worse lipid profile than did diagnosed cases.

The adverse lipid profiles associated with elevated glucose levels from normal to IFG and/or IGT and diabetes have been reported for both Europeans (1) and Asians (2),

**TABLE 3.** Proportions of individuals according to lipid levels stratified by diabetic status in each ethnic group

| | LDL-C < 3 mmol/liter | | | | LDL-C ≥ 3 mmol/liter | | | |
|---|---|---|---|---|---|---|---|---|
| | Normal HDL-C and normal TG | Low HDL-C[a] alone | High TG[b] alone | Both | Normal HDL-C and normal TG | Low HDL-C[a] alone | High TG[b] alone | Both |
| Nondiabetic population | | | | | | | | |
| Hong Kong Chinese | 29.3 | 9.9 | 1.6 | 4.2 | 32.1 | 12.9 | 3.7 | 6.2 |
| Qingdao Chinese | 31.0 | 5.4 | 8.3 | 1.9 | 40.5 | 2.4 | 9.8 | 0.7 |
| Asian Indian | 23.2 | 33.6 | 3.2 | 11.0 | 9.2 | 10.7 | 2.8 | 6.4 |
| Mauritian Indian | 23.9 | 15.8 | 5.0 | 4.7 | 23.2 | 14.7 | 5.7 | 7.0 |
| Japanese | 25.2 | 6.4 | 3.4 | 3.5 | 38.2 | 13.0 | 5.0 | 5.3 |
| C&N European | 13.3 | 2.3 | 2.0 | 1.6 | 48.6 | 9.7 | 12.6 | 10.0 |
| Southern European | 14.2 | 4.3 | 1.1 | 2.1 | 45.5 | 15.1 | 7.8 | 10.0 |
| Diabetic population | | | | | | | | |
| Hong Kong Chinese | 12.4 | 9.6 | 1.4 | 11.0 | 22.6 | 18.1 | 7.6 | 17.2 |
| Qingdao Chinese | 21.1 | 3.5 | 11.1 | 3.1 | 37.9 | 2.7 | 19.1 | 1.5 |
| Asian Indian | 12.8 | 17.4 | 6.0 | 21.4 | 8.1 | 12.4 | 7.2 | 14.7 |
| Mauritian Indian | 12.4 | 8.6 | 6.4 | 10.2 | 21.2 | 15.5 | 10.2 | 15.5 |
| Japanese | 14.3 | 6.0 | 7.1 | 5.1 | 34.3 | 11.6 | 12.2 | 9.4 |
| C&N European | 10.5 | 2.8 | 4.9 | 6.4 | 30.4 | 9.3 | 16.4 | 19.4 |
| Southern European | 7.5 | 3.3 | 6.0 | 10.2 | 24.4 | 11.2 | 12.8 | 14.8 |

Data are expressed as percentage.

[a] Less than 1.03 mmol/liter in men and < 1.29 mmol/liter in women.

[b] ≥1.70 mmol/liter.

but the ethnic differences in lipid profiles given the same glucose levels have not been well investigated. In the HeartSCORE and IndiaSCORE studies (21) where lipids were measured with the same assay procedures for Asian Indians as for whites and blacks, Asian Indians had the lowest TC and HDL-C and highest TG among all the ethnic groups studied. In another multiethnic study of the 1992 Singapore National Health Survey (22), Asian Indians appeared to have lower HDL-C but higher TG levels compared with Chinese. The findings of these previous studies are consistent with ours, although glucose status was not taken into account in the previous studies. The causes of ethnic difference in cardiovascular risk profile are complex. Possible contributors include genetic, environmental, psychosocial, cultural, and unmeasured factors, and many are not well clarified (23). An adverse lipid profile in Asian Indians has been reported to be associated with the greater susceptibility to insulin resistance (4, 22, 24, 25), and a higher percentage of body fat for the same BMI as compared with whites (26), which may contribute to the high prevalence of CVD (27) and diabetes (6, 28) in this ethnic group. In addition, it may also reflect the genetic variation, for example, at the apolipoprotein E locus (29) and an excess of other risk factors such as homocysteine, lipoprotein (a), or dietary fat (30).

To date, intensive control of dyslipidemia has been greatly emphasized in the prevention and management of CVD. Current guidelines from the National Cholesterol Education Program Adult Treatment Panel III (31), the European Society of Cardiology (20), and the American Diabetes Association (32), mainly based on the data of whites, consistently recommend that LDL-C should be the primary target of therapy not only in patients with coronary heart disease or diabetes but also in persons with increased cardiovascular risk. Meanwhile, HDL-C has been either dropped from (20) or set as a secondary (32) or tertiary (31) target in the major guidelines despite the strong evidence of reduced HDL-C as an independent risk factor for CVD (33). This may change if more therapy choices developed to increase HDL-C levels and improve HDL function are shown to prevent CVD (34, 35). Our study showed that there are distinct patterns of lipid profiles between different ethnic groups. Considering the high proportion of Asian Indians with adverse HDL-C and TG profiles, appropriate approaches to increasing HDL-C may become an important treatment target in Asian Indians to reduce their excess CVD risks.

The difference in HDL-C concentrations between Qingdao and Hong Kong Chinese subgroups cannot be simply explained by the difference in assay methods. It may largely be attributed to the differences in dietary structure and preference, geographic, and environmental factors. Shellfish and beer, for example, are commonly consumed all year round in Qingdao. Nevertheless, whether other factors exist and contribute to the high HDL-C in Qingdao needs to be further investigated.

Similar to others (36, 37), we observed a worse lipid profile in individuals with undiagnosed diabetes than that of previously diagnosed patients in most of the ethnic groups, indicating that individuals with undiagnosed diabetes are at increased CVD risk and need to be identified and treated early. On the other hand, glycemic control is shown to be an important determinant of diabetic dyslipidemia (38). The better lipid profile in diagnosed diabetes

as compared with undiagnosed diabetes might imply a benefit of lifestyle intervention or drug treatment targeting favorable metabolic profiles and hemoglobin A1c, a surrogate measure for average blood glucose. However, to what extent the levels of hemoglobin A1c have contributed to the differences is unknown due to the lack of information in the current study and in literature. In addition, the data on lipid-lowering treatment are not available for most of the earlier studies conducted in the 1990s because the statins were not widely prescribed at that time. These deserve further investigation in future studies.

In this collaborative analysis of large populations of European and Asian origins, all studies were population-based with a random sampling approach except for the Hong Kong Workforce study and the Hisayama study (community-based). Populations of the same ethnicity were pooled to increase statistical power, and "study cohort" was also considered as a covariate in the data analysis. Although all blood samples for lipid assays were obtained in the fasting state (1, 10, 39), a limitation of this study was the lack of standardization in assay methods for lipids in different laboratories. This needs to be kept in mind when interpreting the results for, in particular, HDL-C, the measurement of which remains a major challenge over time. Except for the direct assays applied in a few of the studies, most of the studies have used chemical precipitation methods for HDL-C assays, including the heparin/$Mn^{2+}$, the dextran sulfate $MgCl_2$, the phosphotungstate $MgCl_2$, and the polyethylene glycol method. The observed differences in HDL-C among ethnicities, to our knowledge, are less likely biased by the laboratory assays. First, there is a good agreement and a similar accuracy between the results of most of the precipitation methods (40). Second, the direct method and the precipitation method are shown to be closely correlated (41). On average, the HDL-C concentration obtained from the direct method is about 0.1–0.2 mmol/liter higher than that from the precipitation method when TG is less than 4.6 mmol/liter (41, 42). The mean difference in the HDL-C concentration was about 0.4–0.5 mmol/liter higher in Qingdao Chinese (direct or precipitation method) than in Asian Indians (precipitation method), much greater than what could be attributed to the difference in assay methods. Most importantly, our observation is consistent with previous reports regarding the adverse lipid profiles in Asian Indians compared with Western or Chinese populations where the lipids were measured using the same assay procedure for Indians as for others (21, 22). Moreover, the mean lipid levels in our study were similar to others. The mean TC was 4.4 mmol/liter, HDL-C was 1.03–1.04 mmol/liter, and TG was 1.53–2.04 mmol/liter for Asian Indians in the HeartSCORE and IndiaSCORE study (21) and the Singapore National Health Survey (22). This further strengthens the validity of the study.

In summary, there are distinct patterns of lipid profiles associated with ethnicity, regardless of the glucose levels. Ethnic- and region-specific considerations are an important component for guidelines on risk assessment and prevention of CVD.

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Address all correspondence and requests for reprints to: Dr. Qing Qiao, Department of Public Health, University of Helsinki, PL41, Mannerheimintie 172, FIN-00014 Helsinki, Finland. E-mail: qing.qiao@helsinki.fi.

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Study cohorts and investigators included in this analysis are listed in Supplemental Appendix 1 (see supplemental data).

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## References

1. **Zhang L, Qiao Q, Tuomilehto J, Hammar N, Alberti KG, Eliasson M, Heine RJ, Stehouwer CD, Ruotolo G** 2008 Blood lipid levels in relation to glucose status in European men and women without a prior history of diabetes: The DECODE Study. Diabetes Res Clin Pract 82:364–377
2. **Zhang L, Qiao Q, Tuomilehto J, Hammar N, Janus ED, Söderberg S, Mohan V, Ramachandran A, Dong YH, Lam TH, Pang ZC** 2009 Blood lipid levels in relation to glucose status in 7 populations of Asian origin without a prior history of diabetes: the DECODA Study. Diabetes Metab Res Rev 25:549–557
3. **Avramoglu RK, Basciano H, Adeli K** 2006 Lipid and lipoprotein dysregulation in insulin resistant states. Clin Chim Acta 368:1–19
4. **Anand SS, Yusuf S, Vuksan V, Devanesen S, Teo KK, Montague PA, Kelemen L, Yi C, Lonn E, Gerstein H, Hegele RA, McQueen M** 2000 Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups (SHARE). Lancet 356:279–284
5. **Chandalia M, Mohan V, Adams-Huet B, Deepa R, Abate N** 2008 Ethnic difference in sex gap in high-density lipoprotein cholesterol between Asian Indians and Whites. J Investig Med 56:574–580
6. **Ramachandran A, Mary S, Yamuna A, Murugesan N, Snehalatha C** 2008 High prevalence of diabetes and cardiovascular risk factors associated with urbanization in India. Diabetes Care 31:893–898
7. **Qiao Q, Nakagami T, Tuomilehto J, Borch-Johnsen K, Balkau B, Iwamoto Y, Tajima N** 2000 Comparison of the fasting and the 2-h glucose criteria for diabetes in different Asian cohorts. Diabetologia 43:1470–1475
8. **Qiao Q, Hu G, Tuomilehto J, Nakagami T, Balkau B, Borch-Johnsen K, Ramachandran A, Mohan V, Iyer SR, Tominaga M, Kiyohara Y, Kato I, Okubo K, Nagai M, Shibazaki S, Yang Z, Tong Z, Fan Q, Wang B, Chew SK, Tan BY, Heng D, Emmanuel S, Tajima N, Iwamoto Y, Snehalatha C, Vijay V, Kapur A, Dong Y, Nan H, Gao W, Shi H, Fu F** 2003 Age- and sex-specific prevalence of diabetes and impaired glucose regulation in 11 Asian cohorts. Diabetes Care 26:1770–1780

9. **The DECODE Study Group** 2003 Age- and sex-specific prevalences of diabetes and impaired glucose regulation in 13 European cohorts. Diabetes Care 26:61–69
10. **The DECODA Study Group** 2007 Prevalence of the metabolic syndrome in populations of Asian origin. Comparison of the IDF definition with the NCEP definition. Diabetes Res Clin Pract 76:57–67
11. **Warnick GR, Remaley AT** 2001 Measurement of cholesterol in plasma and other body fluids. Curr Atheroscler Rep 3:404–411
12. **McGowan MW, Artiss JD, Strandbergh DR, Zak B** 1983 A peroxidase-coupled method for the colorimetric determination of serum triglycerides. Clin Chem 29:538–542
13. **Stein EA, Steiner PM** 1989 Triglyceride measurement and its relationship to heart disease. Clin Lab Med 9:169–185
14. **Langlois MR, Blaton VH** 2006 Historical milestones in measurement of HDL-cholesterol: impact on clinical and laboratory practice. Clin Chim Acta 369:168–178
15. **Friedewald WT, Levy RI, Fredrickson DS** 1972 Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 18:499–502
16. **Söderberg S, Zimmet P, Tuomilehto J, de Courten M, Dowse GK, Chitson P, Gareeboo H, Alberti KG, Shaw JE** 2005 Increasing prevalence of type 2 diabetes mellitus in all ethnic groups in Mauritius. Diabet Med 22:61–68
17. **Dowse GK, Gareeboo H, Alberti KG, Zimmet P, Tuomilehto J, Purran A, Fareed D, Chitson P, Collins VR** 1995 Changes in population cholesterol concentrations and other cardiovascular risk factor levels after five years of the non-communicable disease intervention programme in Mauritius. Mauritius Non-Communicable Disease Study Group. BMJ 311:1255–1259
18. **Alberti KG, Zimmet PZ** 1998 Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med 15:539–553
19. **Alberti KG, Zimmet P, Shaw J** 2005 The metabolic syndrome—a new worldwide definition. Lancet 366:1059–1062
20. **Graham I, Atar D, Borch-Johnsen K, Boysen G, Burell G, Cifkova R, Dallongeville J, De Backer G, Ebrahim S, Gjelsvik B, Herrmann-Lingen C, Hoes A, Humphries S, Knapton M, Perk J, Priori SG, Pyorala K, Reiner Z, Ruilope L, Sans-Menendez S, Op Reimer WS, Weissberg P, Wood D, Yarnell J, Zamorano JL, Walma E, Fitzgerald T, Cooney MT, Dudina A, Vahanian A, Camm J, De Caterina R, Dean V, Dickstein K, Funck-Brentano C, Filippatos G, Hellemans I, Kristensen SD, McGregor K, Sechtem U, Silber S, Tendera M, Widimsky P, Altiner A, Bonora E, Durrington PN, Fagard R, Giampaoli S, Hemingway H, Hakansson J, Kjeldsen SE, Larsen ML, Mancia G, Manolis AJ, Orth-Gomer K, Pedersen T, Rayner M, Ryden L, Sammut M, Schneiderman N, Stalenhoef AF, Tokgozoglu L, Wiklund O, Zampelas A** 2007 European guidelines on cardiovascular disease prevention in clinical practice: executive summary. Fourth Joint Task Force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of nine societies and by invited experts). Eur J Cardiovasc Prev Rehabil 14(Suppl 2):E1–E40
21. **Mulukutla SR, Venkitachalam L, Marroquin OC, Kip KC, Aiyer A, Edmundowicz D, Ganesh S, Varghese R, Reis SE** 2008 Population variation in atherogenic dyslipidemia: a report from the HeartSCORE and IndiaSCORE Studies. J Clin Lipidol 2:410–417
22. **Tan CE, Emmanuel SC, Tan BY, Jacob E** 1999 Prevalence of diabetes and ethnic differences in cardiovascular risk factors. The 1992 Singapore National Health Survey. Diabetes Care 22:241–247
23. **Zaninotto P, Mindell J, Hirani V** 2007 Prevalence of cardiovascular risk factors among ethnic groups: results from the Health Surveys for England. Atherosclerosis 195:e48–e57
24. **Bhalodkar NC, Blum S, Rana T, Kitchappa R, Bhalodkar AN, Enas EA** 2005 Comparison of high-density and low-density lipoprotein cholesterol subclasses and sizes in Asian Indian women with Caucasian women from the Framingham Offspring Study. Clin Cardiol 28:247–251
25. **Palaniappan LP, Kwan AC, Abbasi F, Lamendola C, McLaughlin TL, Reaven GM** 2007 Lipoprotein abnormalities are associated with insulin resistance in South Asian Indian women. Metabolism 56:899–904
26. **McKeigue PM, Shah B, Marmot MG** 1991 Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. Lancet 337:382–386
27. **Kuller LH** 2004 Ethnic differences in atherosclerosis, cardiovascular disease and lipid metabolism. Curr Opin Lipidol 15:109–113
28. **Snehalatha C, Ramachandran A** 2009 Cardiovascular risk factors in the normoglycaemic Asian-Indian population—influence of urbanisation. Diabetologia 52:596–599
29. **Tan CE, Tai ES, Tan CS, Chia KS, Lee J, Chew SK, Ordovas JM** 2003 APOE polymorphism and lipid profile in three ethnic groups in the Singapore population. Atherosclerosis 170:253–260
30. **France MW, Kwok S, McElduff P, Seneviratne CJ** 2003 Ethnic trends in lipid tests in general practice. QJM 96:919–923
31. **Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults** 2001 Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 285:2486–2497
32. **American Diabetes Association** 2009 Standards of medical care in diabetes—2009. Diabetes Care 32(Suppl 1):S13–S61
33. **Boden WE** 2000 High-density lipoprotein cholesterol as an independent risk factor in cardiovascular disease: assessing the data from Framingham to the Veterans Affairs High-Density Lipoprotein Intervention Trial. Am J Cardiol 86:19L–22L
34. **Singh IM, Shishehbor MH, Ansell BJ** 2007 High-density lipoprotein as a therapeutic target: a systematic review. JAMA 298:786–798
35. **Duffy D, Rader DJ** 2009 Update on strategies to increase HDL quantity and function. Nat Rev Cardiol 6:455–463
36. **Hadaegh F, Bozorgmanesh MR, Ghasemi A, Harati H, Saadat N, Azizi F** 2008 High prevalence of undiagnosed diabetes and abnormal glucose tolerance in the Iranian urban population: Tehran Lipid and Glucose Study. BMC Public Health 8:176
37. **Harris MI, Eastman RC** 2000 Early detection of undiagnosed diabetes mellitus: a US perspective. Diabetes Metab Res Rev 16:230–236
38. **Ismail IS, Nazaimoon W, Mohamad W, Letchuman R, Singaraveloo M, Hew FL, Shuguna C, Khalid BA** 2001 Ethnicity and glycaemic control are major determinants of diabetic dyslipidaemia in Malaysia. Diabet Med 18:501–508
39. **Zhang L, Qiao Q, Tuomilehto J, Hammar N, Ruotolo G, Stehouwer CD, Heine RJ, Eliasson M, Zethelius B** 2009 The impact of dyslipidaemia on cardiovascular mortality in individuals without a prior history of diabetes in the DECODE Study. Atherosclerosis 206:298–302
40. **Demacker PN, Hessels M, Toenhake-Dijkstra H, Baadenhuijsen H** 1997 Precipitation methods for high-density lipoprotein cholesterol measurement compared, and final evaluation under routine operating conditions of a method with a low sample-to-reagent ratio. Clin Chem 43:663–668
41. **Jensen T, Truong Q, Frandsen M, Dinesen B, Stender S** 2002 Comparison of a homogeneous assay with a precipitation method for the measurement of HDL cholesterol in diabetic patients. Diabetes Care 25:1914–1918
42. **Okazaki M, Sasamoto K, Muramatsu T, Hosaki S** 1997 Evaluation of precipitation and direct methods for HDL-cholesterol assay by HPLC. Clin Chem 43:1885–1890