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Ethnic Difference in Lipid Profiles

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1. Introduction

Dyslipidaemia is a major cardiovascular disease (CVD) risk factor that plays an important role in the progress of atherosclerosis, the underlying pathology of CVD. To keep lipids and lipoproteins levels within ideal range has been recommended by different national, regional, or global (2001; Graham et al. 2007; World Health Organization 2007) guidelines on the prevention and management of CVD. The prevalence and pattern of lipid disorder, however, differ between ethnicities and populations.

As a component of the metabolic syndrome, dyslipidaemia often coexists with diabetes, the coronary heart disease (CHD) risk equivalent. An atherogenic lipid profiles consists of high triglycerides (TG) and small dense low-density lipoprotein cholesterol (LDL-C) and low high-density lipoprotein cholesterol (HDL-C). The importance of dyslipidaemia on risk of CVD in patients with diabetes has been extensively studied in numerous studies. Reduced HDL-C is well documented as an independent predictor of CVD events (Wilson et al. 1988; Cooney et al. 2009). In contrast, the role of TG as an independent risk factor for CVD is more controversial (Patel et al. 2004; Psaty et al. 2004; Barzi et al. 2005; Sarwar et al. 2007; Wang et al. 2007). Recently, the interest to use novel parameters such as total cholesterol (TC) to HDL ratio (TC/HDL-C), non-HDL-cholesterol (non-HDL-C), apolipoprotein B (apoB) and apolipoprotein A (apoA) to assess CVD risk has increased (Barzi et al. 2005; Pischon et al. 2005; Charlton-Menys et al. 2009). As a CVD risk predictor, the non-HDL-C has been considered to be superior to LDL-C (Cui et al. 2001; Schulze et al. 2004; Liu et al. 2005; Ridker et al. 2005). However, there are racial and geographic disparities in lipid profiles not only in general populations but also in individuals with different glucose categories. The Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP-ATP III) has recommended that certain factors be recognized when clinicians evaluate the lipid profile of different population groups (Adult Treatment Panel III 2002). Although management of lipids using NCEP-ATP III guidelines is applicable to all populations, unique aspects of risk factor profile call for special attention to certain features in different racial/ethnic groups.

2. Ethnic differences in lipid profiles in general populations

The prevalence of dyslipidaemia varies depending on the population studied, geographic location, socioeconomic development and the definition used (Wood et al. 1972; Mann et al. 1988; Onat et al. 1992; Berrios et al. 1997; Ezenwaka et al. 2000; Foucan et al. 2000; Hanh et al. 2001; Zaman et al. 2001; Azizi et al. 2003; Florez et al. 2005; Li et al. 2005; Hertz et al. 2006; Pang et al. 2006; Pongchaiyakul et al. 2006; Tekes-Manova et al. 2006; Zhao et al. 2007; Erem et al. 2008; Steinhagen-Thiessen et al. 2008). Caucasians generally have higher mean TC concentrations than do populations of Asian or African origin (Fuentes et al. 2003; Tolonen et al. 2005). In general populations, the highest prevalence of hypercholesterolaemia (TC \geq 6.5mmol/l) has been seen in Malta (up to 50% in women) and the lowest in China (2.7% in men) in the World Health Organization (WHO) Inter-Health Programme (Berrios et al. 1997). However, inhabitants of the developing world now have had access to more fats in their diets and more sedentary lives; therefore the disease is becoming an increasing problem there.

Ethnic differences in the risk of CVD and type 2 diabetes have consistently been identified, with the most studies comparing the risk between African-Americans and Whites. African-Americans usually display a more favorable lipid profile compared with Whites, despite having the highest overall mortality rates from CVD. In general, African-American men have similar or lower LDL-C and TG but higher HDL-C levels compared with White men. There is evidence that the difference in HDL-C between African-American and White men may be due to a relatively lower hepatic lipase activity in African-Americans (Vega GL 1998). The difference in TG may be related to increased activity of lipoprotein lipase in African-Americans (Sumner AE 2005). However, compared with Whites, Hispanics and Asians, African-Americans have less favorable levels of lipoprotein(a) (Lp[a]), which is structurally similar to LDL-C, with an additional disulfide linked glycoprotein termed ApoA. A number of studies have suggested that Lp(a) may be an important risk factor for CVD (Danesh J 2000; The Emerging Risk Factors C 2009).

Compared to non-Hispanic Whites, Hispanics, specifically Mexican-Americans, have demonstrated lower HDL-C and higher TG levels (Sundquist J 1999). Data from the Dallas Heart Study and a smaller cross-sectional analysis of healthy individuals confirm that levels of Lp(a) are likely similar or even lower in Hispanics compared with Whites (Tsimikas S 2009). Although Lp(a) levels have been associated with endothelial dysfunction in Hispanics, the relationship with coronary artery disease in this population is less clear.

Asian Indians exhibit a higher prevalence of diabetes mellitus than Chinese and Malays (Tan et al. 1999). They also have higher serum TG concentrations and lower HDL-C concentrations than Chinese (Gupta M 2006). In the HeartSCORE and IndiaSCORE studies (Mulukutla et al. 2008) where lipids were measured with the same assay procedures for Asian Indians as for Whites and Blacks, Asian Indians had lowest TC and HDL-C and highest TG among all the ethnic groups studied. In another multi-ethnic study of the 1992 Singapore National Health Survey (Tan et al. 1999), Asian Indians appeared to have lower HDL-C but higher TG levels compared with the Chinese group. Data in other racial/ethnic groups are somewhat limited. Mean total cholesterol and LDL-C levels are lower in American Indians compared with the US average, and levels of Lp(a) are reported to be lower than in Whites (Wang W 2002). East Asians tend to have lower LDL-C, HDL-C and TG as compared with non-Asians (Karthikeyan et al. 2009). East Asians have been reported to have low Lp(a) levels, whereas south Asians have higher mean Lp(a) levels (Geethanjali FS 2003; Berglund L 2004).

Globalization of the western lifestyle contribute to worldwide increases of adiposity and type 2 diabetes not only in adults but also in children and adolescents (Kelishadi et al. 2006; Schwandt et al. 2010). In the BIG Study comparing the prevalence of the metabolic syndrome components in children and adolescents of European, Asian and South-American ethnicities, Iranian and Brazilian youths had considerably higher prevalence of dyslipidaemia than German youths. The most remarkable ethnic difference detected in this study is the high prevalence of low HDL-C levels in Iranian children and adolescents (38%) compared with German youths (7%) (Schwandt et al. 2010). Future longitudinal studies should seek the clinical importance of these ethnic differences.

3. Ethnic differences in lipid profiles in the state of hyperglycaemia

3.1 Lipid disorder and CVD risk in individuals with hyperglycaemia

Lipids and lipoproteins abnormalities are major metabolic disorders, commonly including elevated levels of TC, LDL-C, Lp(a) and TG and reduced levels of HDL-C. In patients with type 2 diabetes, a CHD equivalent (Juutilainen et al. 2005), it is most commonly characterized by elevated TG and reduced HDL-C (Goldberg, I. J. 2001; Krauss 2004; Kendall 2005). There is increasing evidence that the diabetic dyslipidaemia pattern is common not only in patients with overt diabetes (Barrett-Connor et al. 1982) but also in individuals with different glucose categories, i.e., impaired glucose tolerance (IGT) or impaired fasting glucose (IFG) (Meigs et al. 2002; Novoa et al. 2005; Chen et al. 2006; Pankow et al. 2007). These abnormalities can be present alone or in combination with other metabolic disorders. It is well known that the risk of morbidity and mortality from CVD is increased by two- to four-fold in diabetic patients compared with the general population (Kannel 1985; Morrish et al. 1991; Almdal et al. 2004). A number of studies have determined the association of dyslipidaemia with cardiovascular risk in people with hyperglycaemia, and most of them were conducted in patients with diabetes. There is a large body of evidence linking dyslipidaemia and cardiovascular risk in patients with diabetes against quite few negative reports (Vlajinac et al. 1992; Roselli della Rovere et al. 2003) on this issue. Cross-sectional studies have found positive associations of atherosclerotic vascular disease with TC (Ronnemaa et al. 1989; Jurado et al. 2009), LDL-C (Reckless et al. 1978; Agarwal et al. 2009; Jurado et al. 2009), non-HDL-C (Jurado et al. 2009), TG (Santen et al. 1972; Ronnemaa et al. 1989; Gomes et al. 2009), apoB (Ronnemaa et al. 1989) and Lp(a) (Mohan et al. 1998; Murakami et al. 1998; Smaoui et al. 2004), but inverse associations with HDL-C (Reckless et al. 1978; Ronnemaa et al. 1989; Smaoui et al. 2004; Grant and Meigs 2007; Gomes et al. 2009; Jurado et al. 2009) and apoA-I (Seviour et al. 1988; Ronnemaa et al. 1989).

Prospective data have provided with further evidence. The UKPDS study (Turner et al. 1998) has demonstrated that high LDL-C and low HDL-C are potentially modifiable risk factors for coronary artery disease (CAD) in patients with type 2 diabetes. TG, however, was not independently associated with CAD risk in this study, possibly because of its close inverse relationship with HDL-C. Results from the MRFIT (Stamler et al. 1993), in which 356,499 nondiabetic and 5163 diabetic men without CHD at baseline were followed for 12 years, indicated that serum cholesterol is an independent predictor of CHD mortality in men with diabetes. Rosengren et al. (Rosengren et al. 1989) showed similar results in a prospective study of 6897 middle aged diabetic men. Patients with TC > 7.3 mmol/l had a significantly higher incidence of CHD during the 7-year follow up than those with TC ≤ 5.5 mmol/l (28.3% vs. 5.4%, $p < 0.05$). Long term follow-up of the London cohort of the WHO

Multinational Study of Vascular Disease in Diabetics, consisting of 254 type 2 diabetic patients, has showed that TC was associated with incidence of MI (Morrish et al. 1991) and overall cardiovascular mortality (Morrish et al. 1990). The role of TC in predicting CHD was also confirmed in women patients with diabetes (Schulze et al. 2004).

3.2 Ethnic difference in lipid profiles across glucose categories

Although the ethnic variation in lipid patterns has been widely studied in general populations, the ethnic differences in lipid profiles given the same glucose levels have not been well investigated. This issue has been recently studied in the DECODE (Diabetes Epidemiology: Collaborative analysis Of Diagnostic criteria in Europe) and DECODA (Diabetes Epidemiology: Collaborative analysis Of Diagnostic criteria in Asia) study, which consisted of 64 cohorts of mainly population-based from 24 countries and regions around the world, with about 84 000 Europeans and 84 207 Asians of Chinese, Japanese, Indians, Mongolians and Filipinos.

In the collaborative analysis of seven ethnic groups of European and Asian populations (studies included see Appendix 1), considerable ethnic differences in lipid profiles were observed 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 ethnic groups. 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. Age-, cohort- and BMI adjusted mean TC, LDL-C and TG increased while the mean HDL-C decreased with 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 normoglycaemia, mean lipid and lipoprotein concentrations differed among the ethnic groups. The Europeans had highest TC (Fig. 1 a-b) and LDL-C (Fig. 1 c-d), while Qingdao Chinese had highest HDL-C levels among all ethnic groups (Fig. 1 e-f). In contrast, Asian Indians had the lowest TC (Fig. 1 a-b), LDL-C (Fig. 1 c-d) and HDL-C (Fig. 1 e-f) but the highest TG (Fig. 1 g-h) among the ethnic groups ($p < 0.05$ for all comparisons). These ethnic differences were consistently found in all glucose categories.

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 compared with Central & North (C&N) Europeans, across all glucose categories from normal to diabetes (Table 1). Asian Indians and Mauritian Indians tended to have higher but Southern Europeans 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 the HeartSCORE and IndiaSCORE studies (Mulukutla et al. 2008) where lipids were measured with the same assay procedures for Asian Indians as for whites and blacks, Asian Indians had lowest TC and HDL-C and highest TG among all the ethnic groups studied. In another multi-ethnic study of the 1992 Singapore National Health Survey (Tan et al. 1999), 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 controlled in the previous studies.

Similar to others (Harris and Eastman 2000; Hadaegh et al. 2008), we observed a worse lipid profile in individuals with undiagnosed diabetes than that of previously diagnosed patients in most of the ethnic groups, indicating individuals with undiagnosed diabetes are at increased CVD risk and need to be identified and treated early. On the other hand, glycaemic control is shown to be an important determinant of diabetic dyslipidaemia (Ismail et al. 2001). 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 (HbA1c), a surrogate measure for average blood glucose. However, to what extent the levels of HbA1c have contributed to the differences is unknown due to the lack of information in the current study. In addition, the data on lipid-lowering treatment is 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 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 2, 71% non-diabetic and 57.6% diabetic Asian Indians had low LDL-C (< 3.0 mmol/l), while 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 2). The results were confirmed in the same analysis conducted separately for men and women.

There is a large body of evidence showing that diabetes is associated with a high prevalence of dyslipidaemia (Kannel 1985; Cowie et al. 1994; 1997; Jacobs et al. 2005; Bruckert et al. 2007; Abdel-Aal et al. 2008; Ahmed et al. 2008; Okafor et al. 2008; Surana et al. 2008; Agarwal et al. 2009; Jurado et al. 2009; Papazafiropoulou et al. 2009; Roberto Robles et al. 2009; Temelkova-Kurktschiev et al. 2009; Zhang et al. 2009; Seyum et al. 2010). In the Framingham Heart Study (Kannel 1985), the prevalence of low HDL-C (21% vs. 12% in men and 25% vs. 10% in women, respectively) and high TG levels (19% vs. 9% in men and 17% vs. 8% in women, respectively) in people with diabetes was almost twice as high as the prevalence in non-diabetic individuals. By contrast, TC and LDL-C levels did not differ from those of non-diabetic counterparts. A similar pattern of lipid profiles was observed in the UK Prospective Diabetes Study (UKPDS) (1997). In this study, the plasma TG levels were substantially increased whereas HDL-C levels were markedly reduced in both men and women with diabetes compared with the non-diabetic controls. Higher prevalence has been reported in other studies. Data from a primary care-based 7692 patients with type 2 diabetes in the United States showed nearly half of the patients had low HDL-C (Grant and Meigs 2007). The figure was even worse in an urban Indian cohort of 5088 type 2 diabetes patients, with more than half having low HDL-C (52.3%) or high TG (57.9%) (Surana et al. 2008). In addition to the traditional lipid measurement, increased levels of apoB were also seen in patients with diabetes compared with non-diabetic individuals (Bangou-Bredent et al. 1999). It has been shown that the prevalence of lipid and/or glucose abnormality differs between ethnic groups. It is clear that certain ethnic groups have differences in lipid profiles in general. Elevated TG and reduced HDL-C, as the components of the metabolic syndrome and atherogenic dyslipidaemia, was seen more common in Asian Indians than in the Whites (Anand et al. 2000; Razak et al. 2005; Chandalia et al. 2008; Mulukutla et al. 2008), Chinese (Tan et al. 1999; Anand et al. 2000; Razak et al. 2005; The DECODA Study Group 2007; Karthikeyan et al. 2009), Japanese (The DECODA Study Group 2007; Karthikeyan et al. 2009) or Africans (Mulukutla et al. 2008). In a nationally representative sample of seven

ethnic groups in the UK (Zaninotto et al. 2007), the prevalence of low HDL-C was highest in south Asian groups such as Bangladeshi, Indian and Pakistani, followed by Chinese, Irish and those from the general population living in private households; In contrast, the lowest prevalence was seen in Black Caribbean. Similar finding was reported in another study where the comparison was made between non-South-Asians and South Asians (France et al. 2003). In addition, African Americans have been reported to have less adverse lipid profiles than Whites or Hispanics despite the presence of diabetes (Werk et al. 1993; Cowie et al. 1994; Sharma and Pavlik 2001). The causes of ethnic difference in levels of CVD risk factor are complex and may include genetic, environmental and cultural factors (Zaninotto et al. 2007). However, little is known about such ethnic differences in lipid profiles at comparable glucose tolerance status.

4. Causes of ethnic differences

There are several factors that contribute to the development of dyslipidaemia (2001), including genetic factors (Cohen et al. 1994) and acquired factors (Chait and Brunzell 1990; Devroey et al. 2004; Ruixing et al. 2008) such as overweight and obesity (Denke et al. 1993; Denke et al. 1994; Brown et al. 2000), physical inactivity (Berg et al. 1997; Hardman 1999), cigarette smoking (Criqui et al. 1980; Cade and Margetts 1989; Umeda et al. 1998; Fisher et al. 2000; Wu et al. 2001; Maeda et al. 2003; Mammas et al. 2003; Venkatesan et al. 2006; Grant and Meigs 2007; Arslan et al. 2008; Batic-Mujanovic et al. 2008), high fat intake (Hennig et al. 2001; Millen et al. 2002; Tanasescu et al. 2004), very high carbohydrate diets (> 60 percent of total energy) (McNamara and Howell 1992) and certain drugs (Lehtonen 1985; Fogari et al. 1988; Roberts 1989; Middeke et al. 1990; Stone 1994) (such as beta-blockers, anabolic steroids, progestational agents, et al.). Excess alcohol intake is also documented as a risk factor (Umeda et al. 1998; Wu et al. 2001; Mammas et al. 2003) despite that moderate alcohol consumption may have a beneficial effect on improving HDL-C concentrations (De Oliveira et al. 2000; Shai et al. 2004). In addition, glycaemic control is an important determinant of dyslipidaemia in patients with diabetes (Ismail et al. 2001; Grant and Meigs 2007; Ahmed et al. 2008; Gatti et al. 2009). Among these acquired factors, overweight, obesity and physical inactivity appear to be most important (Denke et al. 1993; Denke et al. 1994; Berg et al. 1997; Hardman 1999; Brown et al. 2000). They are also the most important lifestyle variables that decrease insulin action and increase the risk of diabetes.

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 (Zaninotto et al. 2007). It is clear that the observed ethnic differences in lipid profiles cannot be explained by genetics alone and may be more indicative of lifestyle-related factors such as dietary pattern and physical activity (Ruixing et al. 2008; McNaughton et al. 2009; Sisson et al. 2009). To what extent is ethnic-specific lifestyle pattern associated with different lipid profiles deserves further investigation.

4.1 Genetic factors

An adverse lipid profile in Asian Indians has been reported to be associated with the greater susceptibility to insulin resistance (Tan et al. 1999; Anand et al. 2000; Bhalodkar et al. 2005; Palaniappan et al. 2007), and a higher percentage of body fat for the same BMI as compared with Whites (McKeigue et al. 1991), which may contribute to the high prevalence of CVD

(Kuller 2004) and diabetes (Ramachandran et al. 2008; Snehalatha and Ramachandran 2009) in this ethnic group. In addition, it may also reflect the genetic variation, for example, at the apoE locus (Tan et al. 2003) and an excess of other risk factors such as homocysteine, Lp(a) or dietary fat (France et al. 2003).

4.2 Environmental factors

As suggested by previous research, dietary factors may play a role in both lipid and insulin profiles, although these patterns may be mediated by body fat content (Ku CY 1998). Total fat (and saturated fat) intake has been shown to adversely affect total cholesterol concentrations in children, adolescents, and young adults (Post GB 1997). The difference in HDL-C concentrations between Qingdao and Hong Kong Chinese subgroups observed in the DECODA study cannot be simply explained by the difference in assay methods. It may largely attribute to the differences in dietary structure and preference, geographic and environmental factors. Shellfish and beer, for example, are commonly consumed all the year round in Qingdao. Nevertheless, whether other factors exist and contribute to the high HDL-C in Qingdao needs to be further investigated.

Mexican Americans have been previously reported to have greater adiposity, higher TG levels and lower HDL-C levels than Anglos. The relationship between behavioral variables (caloric balance, cigarette and alcohol consumption, exercise, post-menopausal estrogen or oral contraceptive use) and lipid pattern has been investigated in the San Antonio Heart Study (1979–1982) (n=2,102) to explain the ethnic difference in lipids and lipoproteins. Adjustment for caloric balance (as reflected by body mass index) narrowed the ethnic difference in TG and HDL-C levels for both sexes, while adjustment for smoking widened the ethnic difference. For females, the ethnic difference was also decreased by adjustment for alcohol and estrogen use. However, adjustment for these behavioral variables did not completely eliminate the ethnic difference in lipids and lipoproteins in either sex. Increased central adiposity, more characteristic of Mexican Americans than Anglos, was positively associated with triglycerides and negatively associated with HDL-C levels, especially in females. Fat patterning made a more important contribution to the prediction of TG and HDL-C levels than did the other behavioral variables (except for caloric balance) and, in general, eliminated ethnic differences in lipids and lipoproteins (Steven H 1986). Epidemiologists should consider the use of a centrality index to distinguish different types of adiposity since it is easy and inexpensive to measure.

5. Implications for management and prevention of dyslipidaemia

Epidemiological investigations of human populations have revealed a robust relationship between lipids and CVD risk. Furthermore, the benefit of lipid-modifying strategy on cardiovascular events has been demonstrated from a large number of randomized clinical trials (Thavendiranathan et al. 2006; Mills et al. 2008), especially from those using 3-hydroxy-3-methyl-glutaryl-CoA (HMG-CoA) reductase inhibitors (i.e., statins) (Goldberg, R. B. et al. 1998; Collins et al. 2003; Colhoun et al. 2004; Pyorala et al. 2004; Sever et al. 2005; Knopp et al. 2006; Shepherd et al. 2006). Intensive control of dyslipidaemia has been greatly emphasized in the prevention and management of CVD. Current guidelines from the National Cholesterol Education Program Adult Treatment Panel III (ATP III) (Adult Treatment Panel III 2002), the European Society of Cardiology (Graham et al. 2007) and the American Diabetes Association (American Diabetes Association 2009) consistently

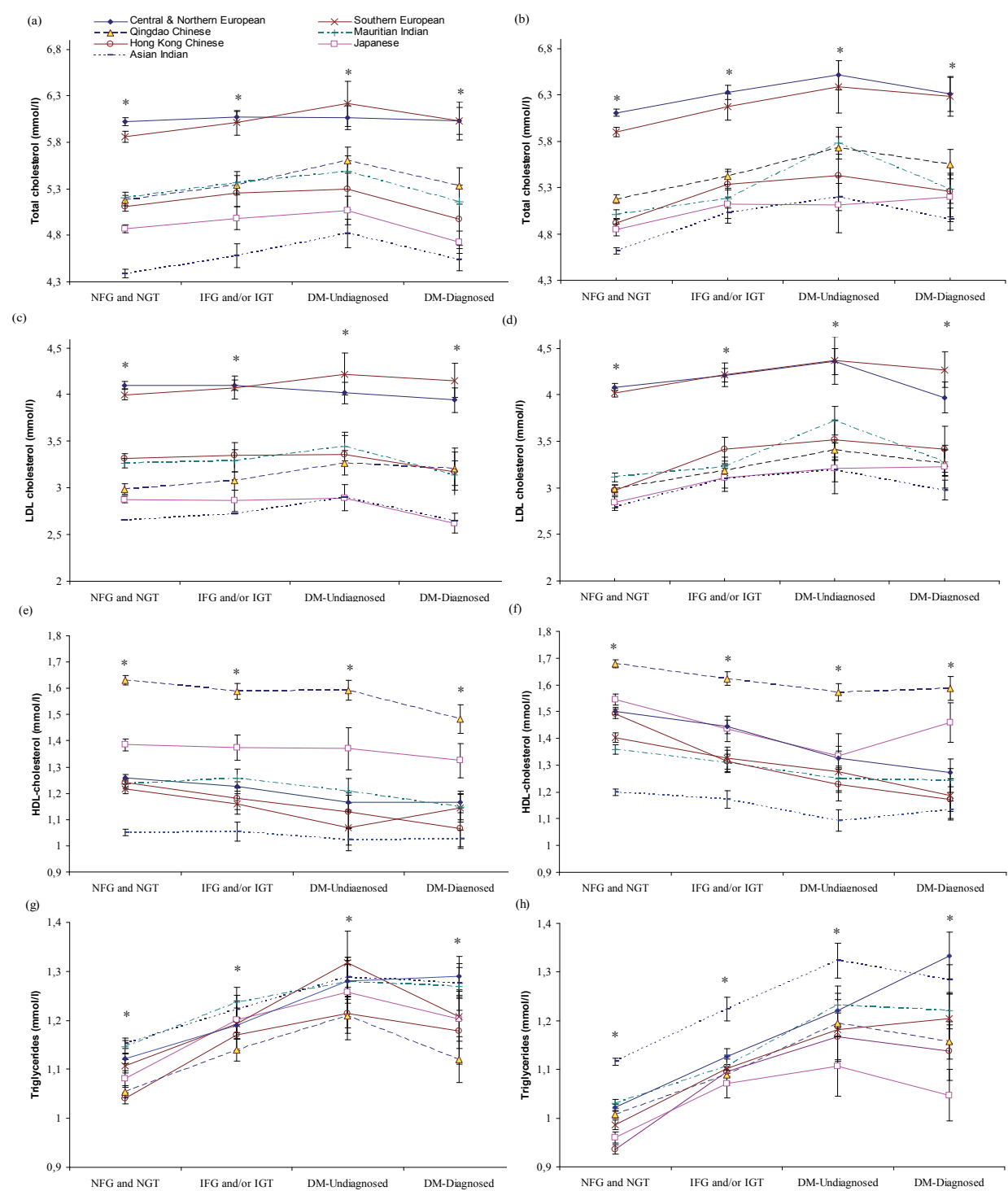


Fig. 1. Age-, study cohort- and body mass index-adjusted mean lipid (geometric means for triglycerides) and lipoprotein concentrations and 95% CIs (vertical bars) in men (figure 1-a, c, e and g) and women (figure 1-b, d, f and h) by ethnicities and glucose categories.* p for trend < 0.05 within each glucose category.

HDL-C < 1.03 in men and < 1.29 in women (mmol/l)				IG ≥ 1.7 mmol/l				LDL-C ≥ 3 mmol/l			
NFG and NGT	IFG and IGT	Undiagnosed diabetes	Diagnosed diabetes	NFG and NGT	IFG and IGT	Undiagnosed diabetes	Diagnosed Diabetes	NFG and NGT	IFG and IGT	Undiagnosed diabetes	Diagnosed diabetes
Men	1	1	1	1	1	1	1	1	1	1	1
Central & Northern European ^a											
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)	0.86 (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)	0.57 (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)	0.29 (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)	0.75 (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)	0.77 (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)	1.52 (0.99-2.31)
Women	1	1	1	1	1	1	1	1	1	1	1
Central & Northern European ^a											
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)	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)	0.67 (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)	0.41 (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)	0.85 (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)	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)	2.67 (1.62-4.42)

Model adjusted for age, study cohort, body mass index, systolic blood pressure and smoking status. NFG, normal fasting glucose; NGT, normal glucose tolerance. ^a Reference group

Table 1. Odds ratio (95% confidence interval) of having dyslipidaemia in relation to ethnicity by glucose categories.

	LDL-C < 3 mmol/l				LDL-C ≥ 3 mmol/l			
	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, %
Non-diabetic 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
Central & Northern 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
Central & Northern 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

a < 1.03 mmol/l in men and < 1.29 mmol/l in women
b ≥ 1.70 mmol/l

Table 2. Proportions (%) of individuals according to lipid levels stratified by diabetic status in each ethnic group.

recommend that LDL-C should be the primary target of therapy not only in patients with CHD or diabetes but also in individuals with increased cardiovascular risk. In addition, non-HDL-C is set by ATP III as a secondary target of therapy and HDL-C and TG as potential target. The Current guideline, mainly based on the data of Whites, consistently recommend that LDL-C < 2.6 mmol/l should be the primary target of therapy in patients with diabetes. As shown in our study and others' (Mulukutla et al. 2008; Karthikeyan et al. 2009), the Asian Indian population had significantly lower TC and LDL-C than did Whites. The threshold of LDL-C for treatment target for Whites may be too high for Asian Indians. Further studies are warranted to verify this hypothesis and determine the threshold applicable to this ethnic group.

In contrast to LDL-C, HDL-C has been either dropped from (Graham et al. 2007) or set as a secondary (American Diabetes Association 2010) or tertiary (Expert Panel on Detection 2001) target in the major guidelines despite the strong evidence of reduced HDL-C as an independent risk factor for CVD (Boden 2000). This may change if more therapy choices developed to increase HDL-C levels and improve HDL function are shown to prevent CVD (Singh et al. 2007; Duffy and Rader 2009; Sorrentino et al. 2010) or reduce the residual cardiovascular risk (Fruchart J 2008). Most recently, the ARBITER 6-HALTS (Arterial Biology for the Investigation of the Treatment Effects of Reducing Cholesterol 6-HDL and LDL Treatment Strategies in Atherosclerosis) trial has shown a significant improvement in serum HDL-C levels and regression of carotid intima-media thickness when ERN was combined with statin therapy in patients with CHD or CHD equivalent (Taylor et al. 2009; Villines et al. 2010). Considering the high proportion of Asian Indians with adverse HDL-C levels, appropriate approaches to increasing HDL-C and/or improving HDL function may become an important treatment target in Asian Indians in order to reduce their excess CVD risks.

6. Appendix 1

Countries and studies	Blood sample	Total cholesterol	High-density lipoprotein cholesterol	Triglycerides
China				
Hong Kong Cardiovascular DiseaseRisk Factor Prevalence Study	Plasma	Cholesterol oxidase (CHOD) method; Hitachi 717 analyser (Hitachi Instruments, California, USA).	Measured after precipitation of very-low density lipoprotein (VLDL) and low-density lipoprotein (LDL) by polyethylene glycol PEG 6000.	Lipase/glycerol kinase method;
Hong Kong Workforce Survey on CVD Risk Factors	Venous Plasma	Enzymatic method, with reagents (Baker Instruments Corporation, Allentown, PA 18103, USA) with Cobas Mira analyzer (Hoffman-La Roche and Co., Basle Switzerland).	Enzymatic method after precipitation with dextran sulphate-MgCl ₂ on Cobas Mira analyzer (Hoffman-La Roche and Co., Basle Switzerland)	Enzymatic method, with reagents (Baker Instruments Corporation, Allentown, PA 18103, USA) with Cobas Mira analyzer (Hoffman-La Roche and Co., Basle Switzerland)
Qingdao Diabetes Survey 2002	Venous Plasma	Enzymatic method (AMS Analyzer Medical System, SABA-18, Rome, Italy)	Enzymatic method after precipitation (AMS Analyzer Medical System, SABA-18, Rome, Italy)	Enzymatic method (AMS Analyzer Medical System, SABA-18, Rome, Italy)
Qingdao Diabetes Study 2006	Serum	Enzymatic method (Olympus reagent) With OLYMPUS-AU640 Automatic Analyzers (Olympus Optical. Tokyo, Japan)	Direct method (Olympus reagent) with OLYMPUS-AU640 Automatic Analyzers (Olympus Optical. Tokyo, Japan)	Enzymatic method (Olympus reagent) with OLYMPUS-AU640 Automatic Analyzers (Olympus Optical. Tokyo, Japan)

Finland				
East-West men	Serum	Enzymatic techniques (Monotest, Boehringer Mannheim GmbH, FRG) Olli C3000 photometer (Kone Oy, Finland)	Enzymetic method after precipitation of VLDL and LDL by means of dextran-magnesium-chloride, with Olli C3000 photometer (Kone Oy, Finland)	Enzymatic techniques (Monotest, Boehringer Mannheim GmbH, FRG) Olli C3000 photometer (Kone Oy, Finland)
National FINRISK Study 87, 92	Serum	Enzymatic techniques (Cholesterol oxidase-peroxidase-amidopyrine, CHOD-PAP, Boehringer-Mannheim, Mannheim, Germany)	Enzymatic method after dextran sulfate magnesium chloride precipitation of apolipoprotein B (apoB)-containing lipoproteins	Enzymatic techniques (CHOD-PAP, Boehringer-Mannheim, Mannheim, Germany)
National FINRISK Study 2002	Serum	Enzymatic method (CHOD-PAP; Thermo Elektron Oy, Finland);	Enzymatic method (CHOD-PAP; Thermo Elektron Oy, Finland) after precipitation by the PTA-precipitation method	Enzymatic techniques (Glycerol phosphate oxidase-peroxidase-amidopyrine, GPO-PAP; Thermo Elektron Oy)
Oulu Study	Serum	Enzymatic method (CHOD-PAP, Boehringer Mannheim, Mannheim, Germany).	Enzymatic CHOD-PAP method after precipitation of LDL and VLDL with a reagent containing phosphotungstic acid and MgCl ₂ (Boehringer Mannheim)	Enzymatic method (CHOD-PAP, Boehringer Mannheim, Mannheim, Germany)
Savitaipale Study	Plasma	Enzymatic colorimetric method (CHOD-PAP) Cobas Integra 400/700 analyzer	Enzymatic colorimetric method (CHOD-PAP) Cobas Integra 400/700 analyzer	Enzymatic colorimetric method (CHOD-PAP) Cobas Integra 400/700 analyzer
Vantaa Study	Serum	Enzymatic techniques (Boehringer-Mannheim)	Enzymatic method after precipitation with polyethylenglycol	Enzymatic techniques (Boehringer-Mannheim)
India				
Chennai 94	Serum	Enzymatic method; Hitachi 704 autoanalyser, using Boehringer Mannheim (Mannheim, Germany) reagents	Phosphotungstate-magnesium precipitation method. Hitachi 704 autoanalyser, using Boehringer Mannheim (Mannheim, Germany) reagents	Enzymatic method. Hitachi 704 autoanalyser, using Boehringer Mannheim (Mannheim, Germany) reagents

Chennai 97	Venous Plasma	CHOD-PAP method (Boehringer Mannheim, Germany); Corning Express Plus Auto Analyser (Corning, medfied, MA, USA)	Phosphotungstic acid method after precipitation of LDL and chylomicrons (Boehringer Mannheim, Germany); Corning Express Plus Auto Analyser (Corning, medfied, MA, USA)	GPO-PAP method (Boehringer Mannheim, Germany); Corning Express Plus Auto Analyser (Corning, medfied, MA, USA)
CURES	Serum	CHOD-PAP method with Hitachi-912 Autoanalyser (Hitachi, Mannheim, Germany) using kits supplied by Roche Diagnostics (Mannheim, Germany).	Direct method (polyethylene glycol-pretreated enzymes) with Hitachi-912 Autoanalyser (Hitachi, Mannheim, Germany) using kits supplied by Roche Diagnostics (Mannheim, Germany).	GPO-PAP method; Hitachi-912 Autoanalyser (Hitachi, Mannheim, Germany) using kits supplied by Roche Diagnostics (Mannheim, Germany).
Chennai 2006	Serum	Standard enzymatic procedures (Roche Diagnostics, Mannheim, Germany)	Direct assay method (Roche Diagnostics, Mannheim, Germany)	Standard enzymatic procedures (Roche Diagnostics, Mannheim, Germany)
Italy				
Cremona Study	Plasma	Enzymatic techniques (Boehringer-Mannheim, Mannheim, Germany) with CIBA Corning 550 Express Auto-analyser	Precipitation with PEG using a Colortest kit (Roche, Basel, Switzerland).	Enzymatic techniques (Boehringer-Mannheim, Mannheim, Germany) with CIBA Corning 550 Express Auto-analyser
Japan				
Funagata Study	Plasma	Cholesterol oxidase method (L-type Wako CHO-H [Wako Pure Chemical Industries, Osaka, Japan]) with TBA 80FR (Toshiba medical system corporation, Tokyo)	Direct method (Cholesterol N HDL [Daiichi Pure Chemicals, Tokyo, Japan]) with TBA 80FR (Toshiba medical system corporation, Tokyo)	GPO HDAOS method (Pureauto S TG-N [Daiichi Pure Chemicals, Tokyo, Japan]) with TBA 80FR (Toshiba medical system corporation, Tokyo)
Hisayama Study	Serum	Enzymatic techniques (TBA-80S; Toshiba Inc., Tokyo, Japan)	Enzymatic method after precipitation of of VLDL and LDL with dextran sulfate and magnesium (TBA-80S; Toshiba Inc., Tokyo, Japan)	Enzymatic techniques (TBA-80S; Toshiba Inc., Tokyo, Japan)
Mauritius				

Mauritius 1987	Venous plasma	Manual enzymatic colorimetric method (Coulter Minikem Spectrophotometer), (Boeringer Cat no 701912)	Manual enzymatic colorimetric method (Coulter Minikem Spectrophotometer), (Boeringer Cat no 701912) Precipitation method (Biomerieux)	Manual enzymatic colorimetric method(Coulter Minikem Spectrophotometer) (Boeringer Cat nr 400971)
Mauritius 1992	Venous plasma	Automated enzymatic method with Chemistry Profile Analyser Model LS (Coulter- France)	Automated enzymatic method, Chemistry Profile Analyser Model LS (Coulter- France) Precipitation method (Biomerieux)	Automated enzymatic method with Chemistry Profile Analyser Model LS (Coulter- France)
Mauritius 1998	Venous plasma	Automated enzymatic methods; Cobas Mira analyzer (Roche Diagnostics, France)	Automated enzymatic methods; Cobas Mira analyzer (Roche Diagnostics, France) Direct method (Biomerieux)	Automated enzymatic methods; Cobas Mira analyzer (Roche Diagnostics, France)
Poland POLMONICA	Serum	Direct Liebermann-Burchard method (Boehringer-Mannheim)	Determined in the supernatant after precipitation with heparin manganese (Boehringer-Mannheim)	Enzymatic method (Boehringer-Mannheim)
Republic of Cyprus Nicosia Diabetes Study	Whole Blood	Cobas Micra Plus Roche	Cobas Micra Plus Roche	Cobas Micra Plus Roche
Spain The Guía Study	Plasma	Standard enzymatic methods (Boehringer-Mannheim Hitachi 717 autoanalyser, Tokyo, Japan)	Phosphotungstate precipitation (Boehringer-Mannheim Hitachi 717 autoanalyser, Tokyo, Japan)	Standard enzymatic methods (Boehringer-Mannheim Hitachi 717 autoanalyser, Tokyo, Japan)
The Viva Study	Plasma	Enzymatic techniques (Boehringer-Mannheim)	Enzymatic techniques (Boehringer-Mannheim)	Enzymatic techniques (Boehringer-Mannheim)
Sweden MONICA	Serum	Enzymatic techniques (Boehringer-Mannheim GmbH, Germany)	Phosphotungstate-Mg ²⁺ precipitation method	Enzymatic method (CHOD-PAP, Boehringer-Mannheim GmbH, Germany)
The Uppsala Longitudinal Study of Adult	Serum	Enzymatic techniques using IL Test Cholesterol Trinders's	Separated by precipitation with magnesium chloride/	Enzymatic techniques using IL Test Cholesterol

Men (ULSAM)		Method and IL Test Enzymatic- colorimetric Method for use in a Monarch apparatus (Instrumentation Laboratories, Lexington, USA). (http://www.pubcare.uu.se/ULSAM/invest/70yrs/meth70.htm#09)	phosphotungstate.	Trinders's Method and IL Test Enzymatic- colorimetric Method for use in a Monarch apparatus (Instrumentation Laboratories, Lexington, USA).
The Netherlands The Hoorn Study	Serum	Enzymatic techniques (Boehringer- Mannheim, Mannheim, Germany);	Enzymatic techniques after precipitation of the low and very low-density lipoproteins (Boehringer- Mannheim, Mannheim, Germany)	Enzymatic techniques (Boehringer- Mannheim, Mannheim, Germany);
Zutphen	Serum	Enzymatic techniques (CHOD-PAP mono- test kit,Boehringer- Mannheim)	Enzymatic method after precipitation of apoB- containing particles by means of dextran magnesium sulphate.	Enzymatic techniques (CHOD- PAP mono-test kit,Boehringer- Mannheim)
U.K. Isle of ELY Diabetes Project	Plasma	Enzymatic techniques, RA 1000 (Bayer Diagnostics, Basingstoke, Hants, UK)	Enzymatic methods	Standard automated enzymatic method with the RA1000 (Bayer Diagnostics, Suffolk, U.K.),
Newcastle Heart Project	Plasma	Cholesterol oxidase/peroxidase method with Cobas Bio centrifugal analyzer (Roche Products Ltd, Welwyn Garden City, UK)	Measuring the supernatant cholesterol concentration after precipitation of apoB- containing lipoproteins with heparin and manganese. Cobas Bio centrifugal analyzer (Roche Products Ltd, Welwyn Garden City, UK)	Lipase/glycerol kinase method. Cobas Bio centrifugal analyzer (Roche Products Ltd, Welwyn Garden City, UK)
The Goodinge Study	Plasma	Cholesterol esterase method (Boehringer Mannheim, Lewes, Sussex, U.K.)	Enzymatic spectrophotometric method (Roche Diagnostics, Hatfield, Herts, U.K.) after precipitation of LDL by the addition of phosphotungstic acid in the presence of magnesium ions.	Enzymatic spectrophotometric method (Roche Diagnostics, Hatfield, Herts, U.K.).

Measures of lipid components in each study.

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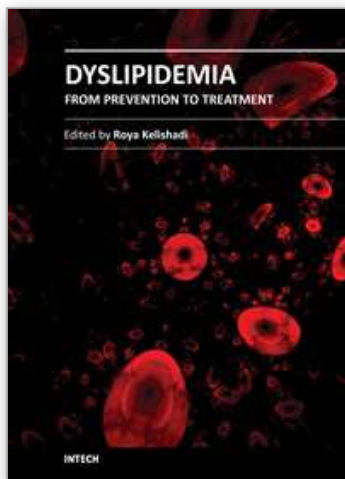
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Dyslipidemia has a complex pathophysiology consisting of various genetic, lifestyle, and environmental factors. It has many adverse health impacts, notably in the development of chronic non-communicable diseases. Significant ethnic differences exist due to the prevalence and types of lipid disorders. While elevated serum total- and LDL-cholesterol are the main concern in Western populations, in other countries hypertriglyceridemia and low HDL-cholesterol are more prevalent. The latter types of lipid disorders are considered as components of the metabolic syndrome. The escalating trend of obesity, as well as changes in lifestyle and environmental factors will make dyslipidemia a global medical and public health threat, not only for adults but for the pediatric age group as well. Several experimental and clinical studies are still being conducted regarding the underlying mechanisms and treatment of dyslipidemia. The current book is providing a general overview of dyslipidemia from diverse aspects of pathophysiology, ethnic differences, prevention, health hazards, and treatment.

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