We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

2

Ethnic Difference in Lipid Profiles

Lei Zhang[1,2,3,4], Qing Qiao[1,4] and Yanhu Dong[2,3]
[1]*Hjelt Institute, University of Helsinki, Helsinki,*
[2]*Qingdao Endocrine & Diabetes Hospital, Qingdao,*
[3]*Weifang Medical University, Weifang,*
[4] *National Institute for Health and Welfare, Helsinki,*
[1,4]*Finland*
[2,3]*China*

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 ≥ 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 Orgnization (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 wided 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

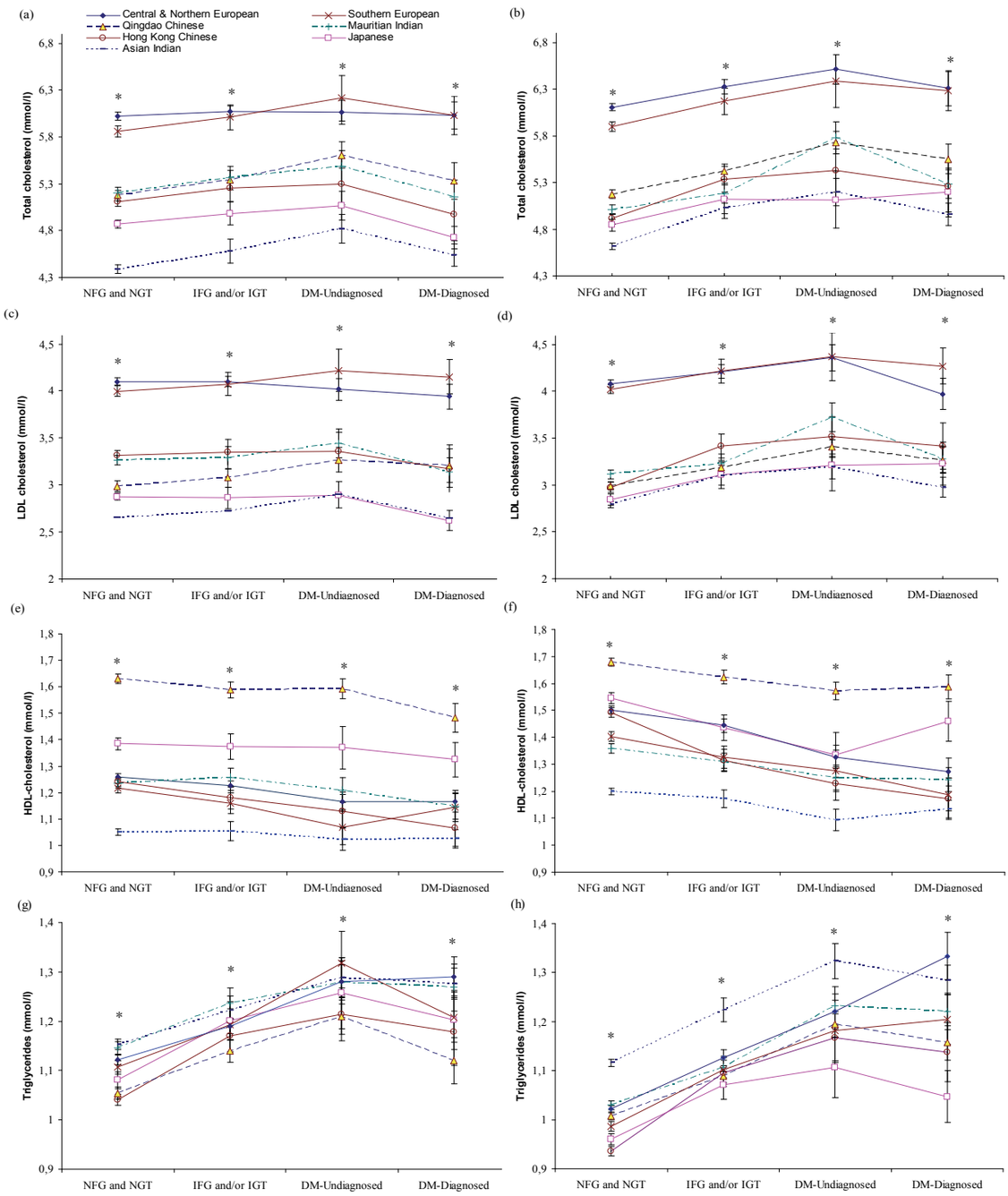


Fig. 1. Age-, study cohort- and body mass index-adjusted mean lipid (geometric means for triglycerides) and lipoprotein concentrations and 95% CIs (vertical bars) in men (figure 1-a, c, e and g) and women (figure 1-b, d, f and h) by ethnicities and glucose categories.* p for trend < 0.05 within each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

	HDL-C < 1.03 in men and < 1.29 in women (mmol/l)				TG ≥ 1.7 mmol/l				LDL-C ≥ 3 mmol/l			
	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												
Central & Northern 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												
Central & Northern 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 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 conbined 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)	phosphotumgstate.	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.

7. References

Abdel-Aal, N. M., A. T. Ahmad, E. S. Froelicher, A. M. Batieha, M. M. Hamza, et al. (2008). "Prevalence of dyslipidemia in patients with type 2 diabetes in Jordan." Saudi Med J 29(10): 1423-1428.

Adult Treatment panel III (2002). "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) final report." Circulation 106(25): 3143-3421.

Agarwal, A. K., S. Singla, R. Singla, A. Lal, H. Wardhan, et al. (2009). "Prevalence of coronary risk factors in type 2 diabetics without manifestations of overt coronary heart disease." J Assoc Physicians India 57: 135-142.

Ahmed, N., J. Khan and T. S. Siddiqui (2008). "Frequency of dyslipidaemia in type 2 diabetes mellitus in patients of Hazara division." J Ayub Med Coll Abbottabad 20(2): 51-54.

Almdal, T., H. Scharling, J. S. Jensen and H. Vestergaard (2004). "The independent effect of type 2 diabetes mellitus on ischemic heart disease, stroke, and death: a population-based study of 13,000 men and women with 20 years of follow-up." Arch Intern Med 164(13): 1422-1426.

American Diabetes Association (2009). "Standards of medical care in diabetes--2009." Diabetes Care 32 Suppl 1: S13-61.

American Diabetes Association (2010). "Standards of medical care in diabetes--2010." Diabetes Care 33 Suppl 1: S11-61.

Anand, S. S., S. Yusuf, V. Vuksan, S. Devanesen, K. K. Teo, et al. (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(9226): 279-284.

Arslan, E., T. Yakar and I. Yavasoglu (2008). "The effect of smoking on mean platelet volume and lipid profile in young male subjects." Anadolu Kardiyol Derg 8(6): 422-425.

Azizi, F., M. Rahmani, A. Ghanbarian, H. Emami, P. Salehi, et al. (2003). "Serum lipid levels in an Iranian adults population: Tehran Lipid and Glucose Study." Eur J Epidemiol 18(4): 311-319.

Bangou-Bredent, J., V. Szmidt-Adjide, P. Kangambega-Nouvier, L. Foucan, A. Campier, et al. (1999). "Cardiovascular risk factors associated with diabetes in an Indian community of Guadeloupe. A case control study." Diabetes Metab 25(5): 393-398.

Barrett-Connor, E., S. M. Grundy and M. J. Holdbrook (1982). "Plasma lipids and diabetes mellitus in an adult community." Am J Epidemiol 115(5): 657-663.

Barzi, F., A. Patel, M. Woodward, C. M. Lawes, T. Ohkubo, et al. (2005). "A comparison of lipid variables as predictors of cardiovascular disease in the Asia Pacific region." Ann Epidemiol 15(5): 405-413.

Batic-Mujanovic, O., A. Beganlic, N. Salihefendic, N. Pranjic and Z. Kusljugic (2008). "Influence of smoking on serum lipid and lipoprotein levels among family medicine patients." Med Arh 62(5-6): 264-267.

Berg, A., M. Halle, I. Franz and J. Keul (1997). "Physical activity and lipoprotein metabolism: epidemiological evidence and clinical trials." Eur J Med Res 2(6): 259-264.

Berglund L, R. R. (2004). "Lipoprotein(a): an elusive cardiovascular risk factor." Arterioscler. Thromb. Vasc. Biol 24(12): 2219-2226.

Berrios, X., T. Koponen, T. Huiguang, N. Khaltaev, P. Puska, et al. (1997). "Distribution and prevalence of major risk factors of noncommunicable diseases in selected countries: the WHO Inter-Health Programme." Bull World Health Organ 75(2): 99-108.

Bhalodkar, N. C., S. Blum, T. Rana, R. Kitchappa, A. N. Bhalodkar, et al. (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(5): 247-251.

Boden, W. E. (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(12A): 19L-22L.

Brown, C. D., M. Higgins, K. A. Donato, F. C. Rohde, R. Garrison, et al. (2000). "Body mass index and the prevalence of hypertension and dyslipidemia." Obes Res 8(9): 605-619.

Bruckert, E., M. Baccara-Dinet and E. Eschwege (2007). "Low HDL-cholesterol is common in European Type 2 diabetic patients receiving treatment for dyslipidaemia: data from a pan-European survey." Diabet Med 24(4): 388-391.

Cade, J. and B. Margetts (1989). "Cigarette smoking and serum lipid and lipoprotein concentrations." Bmj 298(6683): 1312.

Chait, A. and J. D. Brunzell (1990). "Acquired hyperlipidemia (secondary dyslipoproteinemias)." Endocrinol Metab Clin North Am 19(2): 259-278.

Chandalia, M., V. Mohan, B. Adams-Huet, R. Deepa and N. Abate (2008). "Ethnic difference in sex gap in high-density lipoprotein cholesterol between Asian Indians and Whites." J Investig Med 56(3): 574-580.

Charlton-Menys, V., D. J. Betteridge, H. Colhoun, J. Fuller, M. France, et al. (2009). "Apolipoproteins, cardiovascular risk and statin response in type 2 diabetes: the Collaborative Atorvastatin Diabetes Study (CARDS)." Diabetologia 52(2): 218-225.

Chen, L. K., M. H. Lin, Z. J. Chen, S. J. Hwang, S. T. Tsai, et al. (2006). "Metabolic characteristics and insulin resistance of impaired fasting glucose among the middle-aged and elderly Taiwanese." Diabetes Res Clin Pract 71(2): 170-176.

Cohen, J. C., Z. Wang, S. M. Grundy, M. R. Stoesz and R. Guerra (1994). "Variation at the hepatic lipase and apolipoprotein AI/CIII/AIV loci is a major cause of genetically determined variation in plasma HDL cholesterol levels." J Clin Invest 94(6): 2377-2384.

Colhoun, H. M., D. J. Betteridge, P. N. Durrington, G. A. Hitman, H. A. Neil, et al. (2004). "Primary prevention of cardiovascular disease with atorvastatin in type 2 diabetes in the Collaborative Atorvastatin Diabetes Study (CARDS): multicentre randomised placebo-controlled trial." Lancet 364(9435): 685-696.

Collins, R., J. Armitage, S. Parish, P. Sleigh and R. Peto (2003). "MRC/BHF Heart Protection Study of cholesterol-lowering with simvastatin in 5963 people with diabetes: a randomised placebo-controlled trial." Lancet 361(9374): 2005-2016.

Cooney, M. T., A. Dudina, D. De Bacquer, L. Wilhelmsen, S. Sans, et al. (2009). "HDL cholesterol protects against cardiovascular disease in both genders, at all ages and at all levels of risk." Atherosclerosis 206(2): 611-616.

Cowie, C. C., B. V. Howard and M. I. Harris (1994). "Serum lipoproteins in African Americans and whites with non-insulin-dependent diabetes in the US population." Circulation 90(3): 1185-1193.

Criqui, M. H., R. B. Wallace, G. Heiss, M. Mishkel, G. Schonfeld, et al. (1980). "Cigarette smoking and plasma high-density lipoprotein cholesterol. The Lipid Research Clinics Program Prevalence Study." Circulation 62(4 Pt 2): IV70-76.

Cui, Y., R. S. Blumenthal, J. A. Flaws, M. K. Whiteman, P. Langenberg, et al. (2001). "Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality." Arch Intern Med 161(11): 1413-1419.

Danesh J, C. R., Peto R. (2000). "Lipoprotein(a) and coronary heart disease: meta-analysis of prospective studies." Circulation 102(10): 1082-1085.

De Oliveira, E. S. E. R., D. Foster, M. McGee Harper, C. E. Seidman, J. D. Smith, et al. (2000). "Alcohol consumption raises HDL cholesterol levels by increasing the transport rate of apolipoproteins A-I and A-II." Circulation 102(19): 2347-2352.

Denke, M. A., C. T. Sempos and S. M. Grundy (1993). "Excess body weight. An underrecognized contributor to high blood cholesterol levels in white American men." Arch Intern Med 153(9): 1093-1103.

Denke, M. A., C. T. Sempos and S. M. Grundy (1994). "Excess body weight. An under-recognized contributor to dyslipidemia in white American women." Arch Intern Med 154(4): 401-410.

Devroey, D., N. De Swaef, P. Coigniez, J. Vandevoorde, J. Kartounian, et al. (2004). "Correlations between lipid levels and age, gender, glycemia, obesity, diabetes, and smoking." Endocr Res 30(1): 83-93.

Duffy, D. and D. J. Rader (2009). "Update on strategies to increase HDL quantity and function." Nat Rev Cardiol 6(7): 455-463.

Erem, C., A. Hacihasanoglu, O. Deger, M. Kocak and M. Topbas (2008). "Prevalence of dyslipidemia and associated risk factors among Turkish adults: Trabzon lipid study." Endocrine 34(1-3): 36-51.

Expert Panel on Detection, E., 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(19): 2486-2497.

Ezenwaka, C. E., N. Premanand and F. A. Orrett (2000). "Studies on plasma lipids in industrial workers in central Trinidad and Tobago." J Natl Med Assoc 92(8): 375-381.

Fisher, S. D., W. Zareba, A. J. Moss, V. J. Marder, C. E. Sparks, et al. (2000). "Effect of smoking on lipid and thrombogenic factors two months after acute myocardial infarction." Am J Cardiol 86(8): 813-818.

Florez, H., E. Silva, V. Fernandez, E. Ryder, T. Sulbaran, et al. (2005). "Prevalence and risk factors associated with the metabolic syndrome and dyslipidemia in White, Black, Amerindian and Mixed Hispanics in Zulia State, Venezuela." Diabetes Res Clin Pract 69(1): 63-77.

Fogari, R., A. Zoppi, C. Pasotti, L. Poletti, F. Tettamanti, et al. (1988). "Effects of different beta-blockers on lipid metabolism in chronic therapy of hypertension." Int J Clin Pharmacol Ther Toxicol 26(12): 597-604.

Foucan, L., P. Kangambega, D. Koumavi Ekouevi, J. Rozet and J. Bangou-Bredent (2000). "Lipid profile in an adult population in Guadeloupe." Diabetes Metab 26(6): 473-480.

France, M. W., S. Kwok, P. McElduff and C. J. Seneviratne (2003). "Ethnic trends in lipid tests in general practice." QJM 96(12): 919-923.

Fruchart J, S. F., Hermans M, Assmann, G, Brown W, Ceska R, Chapman M, Dodson P, Fioretto P, Ginsberg H (2008). "The residual risk reduction initiative: a call to action to reduce residual vascular risk in dyslipidaemic patients." Diabetes&Vascular Disease Research 5(4): 319-335.

Fuentes, R., T. Uusitalo, P. Puska, J. Tuomilehto and A. Nissinen (2003). "Blood cholesterol level and prevalence of hypercholesterolaemia in developing countries: a review of population-based studies carried out from 1979 to 2002." Eur J Cardiovasc Prev Rehabil 10(6): 411-419.

Gatti, A., M. Maranghi, S. Bacci, C. Carallo, A. Gnasso, et al. (2009). "Poor glycemic control is an independent risk factor for low HDL cholesterol in patients with type 2 diabetes." Diabetes Care 32(8): 1550-1552.

Geethanjali FS, L. K., Lingenhel A. (2003). "Analysis of the apo(a) size polymorphism in Asian Indian populations: association with Lp(a) concentration and coronary heart disease." Atherosclerosis 169(1): 121-130.

Goldberg, I. J. (2001). "Clinical review 124: Diabetic dyslipidemia: causes and consequences." J Clin Endocrinol Metab 86(3): 965-971.

Goldberg, R. B., M. J. Mellies, F. M. Sacks, L. A. Moye, B. V. Howard, et al. (1998). "Cardiovascular events and their reduction with pravastatin in diabetic and glucose-intolerant myocardial infarction survivors with average cholesterol levels: subgroup analyses in the cholesterol and recurrent events (CARE) trial. The Care Investigators." Circulation 98(23): 2513-2519.

Gomes, M. B., D. Giannella-Neto, M. Faria, M. Tambascia, R. M. Fonseca, et al. (2009). "Estimating cardiovascular risk in patients with type 2 diabetes: a national multicenter study in Brazil." Diabetol Metab Syndr 1(1): 22.

Graham, I., D. Atar, K. Borch-Johnsen, G. Boysen, G. Burell, et al. (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: E1-40.

Grant, R. W. and J. B. Meigs (2007). "Prevalence and treatment of low HDL cholesterol among primary care patients with type 2 diabetes: an unmet challenge for cardiovascular risk reduction." Diabetes Care 30(3): 479-484.

Gupta M, S. N., Verma S. (2006). "South Asians and cardiovascular risk: what clinicians should know." Circulation 113(25): E924-E929.

Hadaegh, F., M. R. Bozorgmanesh, A. Ghasemi, H. Harati, N. Saadat, et al. (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.

Hanh, T. T. M., T. Komatsu, N. T. Hung, V. N. Chuyen, Y. Yoshimura, et al. (2001). "Nutritional status of middle-aged Vietnamese in Ho Chi Minh City." J Am Coll Nutr 20(6): 616-622.

Hardman, A. E. (1999). "Physical activity, obesity and blood lipids." Int J Obes Relat Metab Disord 23 Suppl 3: S64-71.

Harris, M. I. and R. C. Eastman (2000). "Early detection of undiagnosed diabetes mellitus: a US perspective." Diabetes Metab Res Rev 16(4): 230-236.

Hennig, B., M. Toborek and C. J. McClain (2001). "High-energy diets, fatty acids and endothelial cell function: implications for atherosclerosis." J Am Coll Nutr 20(2 Suppl): 97-105.

Hertz, R. P., A. N. Unger and C. M. Ferrario (2006). "Diabetes, hypertension, and dyslipidemia in Mexican Americans and non-Hispanic whites." Am J Prev Med 30(2): 103-110.

Ismail, I. S., W. Nazaimoon, W. Mohamad, R. Letchuman, M. Singaraveloo, et al. (2001). "Ethnicity and glycaemic control are major determinants of diabetic dyslipidaemia in Malaysia." Diabet Med 18(6): 501-508.

Jacobs, M. J., T. Kleisli, J. R. Pio, S. Malik, G. J. L'Italien, et al. (2005). "Prevalence and control of dyslipidemia among persons with diabetes in the United States." Diabetes Res Clin Pract 70(3): 263-269.

Jurado, J., J. Ybarra, P. Solanas, J. Caula, I. Gich, et al. (2009). "Prevalence of cardiovascular disease and risk factors in a type 2 diabetic population of the North Catalonia diabetes study." J Am Acad Nurse Pract 21(3): 140-148.

Juutilainen, A., S. Lehto, T. Ronnemaa, K. Pyorala and M. Laakso (2005). "Type 2 diabetes as a "coronary heart disease equivalent": an 18-year prospective population-based study in Finnish subjects." Diabetes Care 28(12): 2901-2907.

Kannel, W. B. (1985). "Lipids, diabetes, and coronary heart disease: insights from the Framingham Study." Am Heart J 110(5): 1100-1107.

Karthikeyan, G., K. K. Teo, S. Islam, M. J. McQueen, P. Pais, et al. (2009). "Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians: an analysis from the INTERHEART Study." J Am Coll Cardiol 53(3): 244-253.

Kelishadi, R., G. Ardalan, R. Gheiratmand and A. Ramezani (2006). "Is family history of premature cardiovascular diseases appropriate for detection of dyslipidemic children in population-based preventive medicine programs? CASPIAN study." Pediatr Cardiol 27(6): 729-736.

Kendall, D. M. (2005). "The dyslipidemia of diabetes mellitus: giving triglycerides and high-density lipoprotein cholesterol a higher priority?" Endocrinol Metab Clin North Am 34(1): 27-48.

Knopp, R. H., M. d'Emden, J. G. Smilde and S. J. Pocock (2006). "Efficacy and safety of atorvastatin in the prevention of cardiovascular end points in subjects with type 2 diabetes: the Atorvastatin Study for Prevention of Coronary Heart Disease Endpoints in non-insulin-dependent diabetes mellitus (ASPEN)." Diabetes Care 29(7): 1478-1485.

Krauss, R. M. (2004). "Lipids and lipoproteins in patients with type 2 diabetes." Diabetes Care 27(6): 1496-1504.

Ku CY, G. B., Nagy TR, Goran MI. (1998). "Relationships between dietary fat, body fat, and serum lipid profile in prepubertal children." Obes Res(6): 400-407.

Kuller, L. H. (2004). "Ethnic differences in atherosclerosis, cardiovascular disease and lipid metabolism." Curr Opin Lipidol 15(2): 109-113.

Lehtonen, A. (1985). "Effect of beta blockers on blood lipid profile." Am Heart J 109(5 Pt 2): 1192-1196.

Li, Z., R. Yang, G. Xu and T. Xia (2005). "Serum lipid concentrations and prevalence of dyslipidemia in a large professional population in Beijing." Clin Chem 51(1): 144-150.

Liu, J., C. Sempos, R. P. Donahue, J. Dorn, M. Trevisan, et al. (2005). "Joint distribution of non-HDL and LDL cholesterol and coronary heart disease risk prediction among individuals with and without diabetes." Diabetes Care 28(8): 1916-1921.

Maeda, K., Y. Noguchi and T. Fukui (2003). "The effects of cessation from cigarette smoking on the lipid and lipoprotein profiles: a meta-analysis." Prev Med 37(4): 283-290.

Mammas, I. N., G. K. Bertsias, M. Linardakis, N. E. Tzanakis, D. N. Labadarios, et al. (2003). "Cigarette smoking, alcohol consumption, and serum lipid profile among medical students in Greece." Eur J Public Health 13(3): 278-282.

Mann, J. I., B. Lewis, J. Shepherd, A. F. Winder, S. Fenster, et al. (1988). "Blood lipid concentrations and other cardiovascular risk factors: distribution, prevalence, and detection in Britain." Br Med J (Clin Res Ed) 296(6638): 1702-1706.

McKeigue, P. M., B. Shah and M. G. Marmot (1991). "Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians." Lancet 337(8738): 382-386.

McNamara, D. J. and W. H. Howell (1992). "Epidemiologic data linking diet to hyperlipidemia and arteriosclerosis." Semin Liver Dis 12(4): 347-355.

McNaughton, S. A., G. D. Mishra and E. J. Brunner (2009). "Food patterns associated with blood lipids are predictive of coronary heart disease: the Whitehall II study." Br J Nutr 102(4): 619-624.

Meigs, J. B., D. M. Nathan, R. B. D'Agostino, Sr. and P. W. Wilson (2002). "Fasting and postchallenge glycemia and cardiovascular disease risk: the Framingham Offspring Study." Diabetes Care 25(10): 1845-1850.

Middeke, M., W. O. Richter, P. Schwandt, B. Beck and H. Holzgreve (1990). "Normalization of lipid metabolism after withdrawal from antihypertensive long-term therapy with beta blockers and diuretics." Arteriosclerosis 10(1): 145-147.

Millen, B. E., P. A. Quatromoni, B. H. Nam, C. E. O'Horo, J. F. Polak, et al. (2002). "Dietary patterns and the odds of carotid atherosclerosis in women: the Framingham Nutrition Studies." Prev Med 35(6): 540-547.

Mills, E. J., B. Rachlis, P. Wu, P. J. Devereaux, P. Arora, et al. (2008). "Primary prevention of cardiovascular mortality and events with statin treatments: a network meta-analysis involving more than 65,000 patients." J Am Coll Cardiol 52(22): 1769-1781.

Mohan, V., R. Deepa, S. P. Haranath, G. Premalatha, M. Rema, et al. (1998). "Lipoprotein(a) is an independent risk factor for coronary artery disease in NIDDM patients in South India." Diabetes Care 21(11): 1819-1823.

Morrish, N. J., L. K. Stevens, J. H. Fuller, R. J. Jarrett and H. Keen (1991). "Risk factors for macrovascular disease in diabetes mellitus: the London follow-up to the WHO Multinational Study of Vascular Disease in Diabetics." Diabetologia 34(8): 590-594.

Morrish, N. J., L. K. Stevens, J. H. Fuller, H. Keen and R. J. Jarrett (1991). "Incidence of macrovascular disease in diabetes mellitus: the London cohort of the WHO Multinational Study of Vascular Disease in Diabetics." Diabetologia 34(8): 584-589.

Morrish, N. J., L. K. Stevens, J. Head, J. H. Fuller, R. J. Jarrett, et al. (1990). "A prospective study of mortality among middle-aged diabetic patients (the London Cohort of the WHO Multinational Study of Vascular Disease in Diabetics) II: Associated risk factors." Diabetologia 33(9): 542-548.

Mulukutla, S. R., L. Venkitachalam, O. C. Marroquin, K. C. Kip, A. Aiyer, et al. (2008). "Population variation in atherogenic dyslipidemia: A report from the HeartSCORE and IndiaSCORE Studies." Journal of Clinical Lipidology 2(6): 410-417.

Murakami, K., S. Ishibashi, Y. Yoshida, N. Yamada and Y. Akanuma (1998). "Lipoprotein(a) as a coronary risk factor in Japanese patients with Type II (non-insulin-dependent) diabetes mellitus. Relation with apolipoprotein(a) phenotypes." Diabetologia 41(11): 1397-1398.

Novoa, F. J., M. Boronat, P. Saavedra, J. M. Diaz-Cremades, V. F. Varillas, et al. (2005). "Differences in cardiovascular risk factors, insulin resistance, and insulin secretion in individuals with normal glucose tolerance and in subjects with impaired glucose regulation: the Telde Study." Diabetes Care 28(10): 2388-2393.

Okafor, C. I., O. A. Fasanmade and D. A. Oke (2008). "Pattern of dyslipidaemia among Nigerians with type 2 diabetes mellitus." Niger J Clin Pract 11(1): 25-31.

Onat, A., G. Surdum-Avci, M. Senocak, E. Ornek and Y. Gozukara (1992). "Plasma lipids and their interrelationship in Turkish adults." J Epidemiol Community Health 46(5): 470-476.

Palaniappan, L. P., A. C. Kwan, F. Abbasi, C. Lamendola, T. L. McLaughlin, et al. (2007). "Lipoprotein abnormalities are associated with insulin resistance in South Asian Indian women." Metabolism 56(7): 899-904.

Pang, R. W., S. Tam, E. D. Janus, S. T. Siu, O. C. Ma, et al. (2006). "Plasma lipid, lipoprotein and apolipoprotein levels in a random population sample of 2875 Hong Kong Chinese adults and their implications (NCEP ATP-III, 2001 guidelines) on cardiovascular risk assessment." Atherosclerosis 184(2): 438-445.

Pankow, J. S., D. K. Kwan, B. B. Duncan, M. I. Schmidt, D. J. Couper, et al. (2007). "Cardiometabolic risk in impaired fasting glucose and impaired glucose tolerance: the Atherosclerosis Risk in Communities Study." Diabetes Care 30(2): 325-331.

Papazafiropoulou, A., A. Sotiropoulos, E. Skliros, M. Kardara, A. Kokolaki, et al. (2009). "Familial history of diabetes and clinical characteristics in Greek subjects with type 2 diabetes." BMC Endocr Disord 9: 12.

Patel, A., F. Barzi, K. Jamrozik, T. H. Lam, H. Ueshima, et al. (2004). "Serum triglycerides as a risk factor for cardiovascular diseases in the Asia-Pacific region." Circulation 110(17): 2678-2686.

Pischon, T., C. J. Girman, F. M. Sacks, N. Rifai, M. J. Stampfer, et al. (2005). "Non-high-density lipoprotein cholesterol and apolipoprotein B in the prediction of coronary heart disease in men." Circulation 112(22): 3375-3383.

Pongchaiyakul, C., P. Hongsprabhas and V. Pisprasert (2006). "Rural-urban difference in lipid levels and prevalence of dyslipidemia: a population-based study in Khon Kaen province, Thailand." J Med Assoc Thai 89(11): 1835-1844.

Post GB, K. H., Twisk J, van Mechelen W. (1997). "The association between dietary patterns and cardiovascular disease risk indicators in healthy youngsters: results covering fifteen years of longitudinal development." Eur J Clin Nutr 51: 387-393.

Psaty, B. M., M. Anderson, R. A. Kronmal, R. P. Tracy, T. Orchard, et al. (2004). "The association between lipid levels and the risks of incident myocardial infarction, stroke, and total mortality: The Cardiovascular Health Study." J Am Geriatr Soc 52(10): 1639-1647.

Pyorala, K., C. M. Ballantyne, B. Gumbiner, M. W. Lee, A. Shah, et al. (2004). "Reduction of cardiovascular events by simvastatin in nondiabetic coronary heart disease patients with and without the metabolic syndrome: subgroup analyses of the Scandinavian Simvastatin Survival Study (4S)." Diabetes Care 27(7): 1735-1740.

Ramachandran, A., S. Mary, A. Yamuna, N. Murugesan and C. Snehalatha (2008). "High prevalence of diabetes and cardiovascular risk factors associated with urbanization in India." Diabetes Care 31(5): 893-898.

Razak, F., S. Anand, V. Vuksan, B. Davis, R. Jacobs, et al. (2005). "Ethnic differences in the relationships between obesity and glucose-metabolic abnormalities: a cross-sectional population-based study." Int J Obes (Lond) 29(6): 656-667.

Reckless, J. P., D. J. Betteridge, P. Wu, B. Payne and D. J. Galton (1978). "High-density and low-density lipoproteins and prevalence of vascular disease in diabetes mellitus." Br Med J 1(6117): 883-886.

Ridker, P. M., N. Rifai, N. R. Cook, G. Bradwin and J. E. Buring (2005). "Non-HDL cholesterol, apolipoproteins A-I and B100, standard lipid measures, lipid ratios, and CRP as risk factors for cardiovascular disease in women." Jama 294(3): 326-333.

Roberto Robles, N., S. Barroso, G. Marcos and J. F. Sanchez Munoz-Torrero (2009). "[Lipid control in diabetic patients in Extremadura (Spain)]." Endocrinol Nutr 56(3): 112-117.

Roberts, W. C. (1989). "Recent studies on the effects of beta blockers on blood lipid levels." Am Heart J 117(3): 709-714.

Ronnemaa, T., M. Laakso, V. Kallio, K. Pyorala, J. Marniemi, et al. (1989). "Serum lipids, lipoproteins, and apolipoproteins and the excessive occurrence of coronary heart disease in non-insulin-dependent diabetic patients." Am J Epidemiol 130(4): 632-645.

Roselli della Rovere, G., A. Lapolla, G. Sartore, C. Rossetti, S. Zambon, et al. (2003). "Plasma lipoproteins, apoproteins and cardiovascular disease in type 2 diabetic patients. A nine-year follow-up study." Nutr Metab Cardiovasc Dis 13(1): 46-51.

Rosengren, A., L. Welin, A. Tsipogianni and L. Wilhelmsen (1989). "Impact of cardiovascular risk factors on coronary heart disease and mortality among middle aged diabetic men: a general population study." Bmj 299(6708): 1127-1131.

Ruixing, Y., W. Jinzhen, H. Yaoheng, T. Jing, W. Hai, et al. (2008). "Associations of diet and lifestyle with hyperlipidemia for middle-aged and elderly persons among the Guangxi Bai Ku Yao and Han populations." J Am Diet Assoc 108(6): 970-976.

Santen, R. J., P. W. Willis, 3rd and S. S. Fajans (1972). "Atherosclerosis in diabetes mellitus. Correlations with serum lipid levels, adiposity, and serum insulin level." Arch Intern Med 130(6): 833-843.

Sarwar, N., J. Danesh, G. Eiriksdottir, G. Sigurdsson, N. Wareham, et al. (2007). "Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies." Circulation 115(4): 450-458.

Schulze, M. B., I. Shai, J. E. Manson, T. Li, N. Rifai, et al. (2004). "Joint role of non-HDL cholesterol and glycated haemoglobin in predicting future coronary heart disease events among women with type 2 diabetes." Diabetologia 47(12): 2129-2136.

Schwandt, P., R. Kelishadi and G. M. Haas (2010). "Ethnic disparities of the metabolic syndrome in population-based samples of german and Iranian adolescents." Metab Syndr Relat Disord 8(2): 189-192.

Schwandt, P., R. Kelishadi, R. Q. Ribeiro, G. M. Haas and P. Poursafa (2010). "A three-country study on the components of the metabolic syndrome in youths: the BIG Study." Int J Pediatr Obes 5(4): 334-341.

Sever, P. S., N. R. Poulter, B. Dahlof, H. Wedel, R. Collins, et al. (2005). "Reduction in cardiovascular events with atorvastatin in 2,532 patients with type 2 diabetes: Anglo-Scandinavian Cardiac Outcomes Trial--lipid-lowering arm (ASCOT-LLA)." Diabetes Care 28(5): 1151-1157.

Seviour, P. W., T. K. Teal, W. Richmond and R. S. Elkeles (1988). "Serum lipids, lipoproteins and macrovascular disease in non-insulin-dependent diabetics: a possible new approach to prevention." Diabet Med 5(2): 166-171.

Seyum, B., G. Mebrahtu, A. Usman, J. Mufunda, B. Tewolde, et al. (2010). "Profile of patients with diabetes in Eritrea: results of first phase registry analyses." Acta Diabetol 47(1): 23-27.

Shai, I., E. B. Rimm, M. B. Schulze, N. Rifai, M. J. Stampfer, et al. (2004). "Moderate alcohol intake and markers of inflammation and endothelial dysfunction among diabetic men." Diabetologia 47(10): 1760-1767.

Sharma, M. D. and V. N. Pavlik (2001). "Dyslipidaemia in African Americans, Hispanics and whites with type 2 diabetes mellitus and hypertension." Diabetes Obes Metab 3(1): 41-45.

Shepherd, J., P. Barter, R. Carmena, P. Deedwania, J. C. Fruchart, et al. (2006). "Effect of lowering LDL cholesterol substantially below currently recommended levels in patients with coronary heart disease and diabetes: the Treating to New Targets (TNT) study." Diabetes Care 29(6): 1220-1226.

Singh, I. M., M. H. Shishehbor and B. J. Ansell (2007). "High-density lipoprotein as a therapeutic target: a systematic review." Jama 298(7): 786-798.

Sisson, S. B., S. M. Camhi, T. S. Church, C. K. Martin, C. Tudor-Locke, et al. (2009). "Leisure time sedentary behavior, occupational/domestic physical activity, and metabolic syndrome in U.S. men and women." Metab Syndr Relat Disord 7(6): 529-536.

Smaoui, M., S. Hammami, R. Chaaba, N. Attia, K. B. Hamda, et al. (2004). "Lipids and lipoprotein(a) concentrations in Tunisian type 2 diabetic patients; Relationship to glycemic control and coronary heart disease." J Diabetes Complications 18(5): 258-263.

Snehalatha, C. and A. Ramachandran (2009). "Cardiovascular risk factors in the normoglycaemic Asian-Indian population--influence of urbanisation." Diabetologia 52(4): 596-599.

Sorrentino, S. A., C. Besler, L. Rohrer, M. Meyer, K. Heinrich, et al. (2010). "Endothelial-vasoprotective effects of high-density lipoprotein are impaired in patients with type 2 diabetes mellitus but are improved after extended-release niacin therapy." Circulation 121(1): 110-122.

Stamler, J., O. Vaccaro, J. D. Neaton and D. Wentworth (1993). "Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial." Diabetes Care 16(2): 434-444.

Steinhagen-Thiessen, E., P. Bramlage, C. Losch, H. Hauner, H. Schunkert, et al. (2008). "Dyslipidemia in primary care--prevalence, recognition, treatment and control: data from the German Metabolic and Cardiovascular Risk Project (GEMCAS)." Cardiovasc Diabetol 7: 31.

Steven H, M. M., Helen H, Marc R, J.AVA K. (1986). "The role of behavioral variables and fat patterning in explaining ethnic differences in serum lipids and lipoproteins." Am. J. Epidemiol 123(5): 830-839.

Stone, N. J. (1994). "Secondary causes of hyperlipidemia." Med Clin North Am 78(1): 117-141.

Sumner AE, V. G., Genovese DJ, Finley KB, Bergman RN, Boston RC. (2005). "Normal triglyceride levels despite insulin resistance in African Americans: role of lipoprotein lipase." Metabolism 54(7): 902-909.

Sundquist J, W. M. (1999). "Cardiovascular risk factors in mexican american adults: a transcultural analysis of NHANES III, 1988-1994." Am. J. Public Health 89(5): 723-730.

Surana, S. P., D. B. Shah, K. Gala, S. Susheja, S. S. Hoskote, et al. (2008). "Prevalence of metabolic syndrome in an urban Indian diabetic population using the NCEP ATP III guidelines." J Assoc Physicians India 56: 865-868.

Tan, C. E., S. C. Emmanuel, B. Y. Tan and E. Jacob (1999). "Prevalence of diabetes and ethnic differences in cardiovascular risk factors. The 1992 Singapore National Health Survey." Diabetes Care 22(2): 241-247.

Tan, C. E., E. S. Tai, C. S. Tan, K. S. Chia, J. Lee, et al. (2003). "APOE polymorphism and lipid profile in three ethnic groups in the Singapore population." Atherosclerosis 170(2): 253-260.

Tanasescu, M., E. Cho, J. E. Manson and F. B. Hu (2004). "Dietary fat and cholesterol and the risk of cardiovascular disease among women with type 2 diabetes." Am J Clin Nutr 79(6): 999-1005.

Taylor, A. J., T. C. Villines, E. J. Stanek, P. J. Devine, L. Griffen, et al. (2009). "Extended-release niacin or ezetimibe and carotid intima-media thickness." N Engl J Med 361(22): 2113-2122.

Tekes-Manova, D., E. Israeli, T. Shochat, M. Swartzon, S. Gordon, et al. (2006). "The prevalence of reversible cardiovascular risk factors in Israelis aged 25-55 years." Isr Med Assoc J 8(8): 527-531.

Temelkova-Kurktschiev, T. S., D. P. Kurktschiev, L. G. Vladimirova-Kitova, I. Vaklinova and B. R. Todorova (2009). "Prevalence and type of dyslipidaemia in a population at risk for cardiovascular death in Bulgaria." Folia Med (Plovdiv) 51(2): 26-32.

Thavendiranathan, P., A. Bagai, M. A. Brookhart and N. K. Choudhry (2006). "Primary prevention of cardiovascular diseases with statin therapy: a meta-analysis of randomized controlled trials." Arch Intern Med 166(21): 2307-2313.

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(1): 57-67.

The Emerging Risk Factors C (2009). "Lipoprotein(a) concentration and the risk of coronary heart disease, stroke, and nonvascular mortality." Jama 302(4): 412-423.

Tolonen, H., U. Keil, M. Ferrario and A. Evans (2005). "Prevalence, awareness and treatment of hypercholesterolaemia in 32 populations: results from the WHO MONICA Project." Int J Epidemiol 34(1): 181-192.

Tsimikas S, C. P., Brilakis ES. (2009). "Relationship of oxidized phospholipids on apolipoprotein B-100 particles to race/ethnicity, apolipoprotein(a) isoform size, and cardiovascular risk factors: results from the Dallas Heart Study." Circulation 119(13): 1711-1719.

Turner, R. C., H. Millns, H. A. Neil, I. M. Stratton, S. E. Manley, et al. (1998). "Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus: United Kingdom Prospective Diabetes Study (UKPDS: 23)." Bmj 316(7134): 823-828.

U.K. Prospective Diabetes Study Investigators (1997). "U.K. Prospective Diabetes Study 27. Plasma lipids and lipoproteins at diagnosis of NIDDM by age and sex." Diabetes Care 20(11): 1683-1687.

Umeda, T., S. Kono, Y. Sakurai, K. Shinchi, K. Imanishi, et al. (1998). "Relationship of cigarette smoking, alcohol use, recreational exercise and obesity with serum lipid atherogenicity: a study of self-defense officials in Japan." J Epidemiol 8(4): 227-234.

Vega GL, C. L., Tang A, Marcovina S, Grundy SM, Cohen JC. (1998). "Hepatic lipase activity is lower in African-American men than in white American men: effects of 5' flanking polymorphism in the hepatic lipase gene (LIPC)." J Lipid Res 39(1): 228-232.

Venkatesan, A., A. Hemalatha, Z. Bobby, N. Selvaraj and V. Sathiyapriya (2006). "Effect of smoking on lipid profile and lipid peroxidation in normal subjects." Indian J Physiol Pharmacol 50(3): 273-278.

Villines, T. C., E. J. Stanek, P. J. Devine, M. Turco, M. Miller, et al. (2010). "The ARBITER 6-HALTS Trial (Arterial Biology for the Investigation of the Treatment Effects of Reducing Cholesterol 6-HDL and LDL Treatment Strategies in Atherosclerosis) Final Results and the Impact of Medication Adherence, Dose, and Treatment Duration." J Am Coll Cardiol: doi:10.1016/j.jacc.2010.1003.1017.

Vlajinac, H., M. Ilic and J. Marinkovic (1992). "Cardiovascular risk factors and prevalence of coronary heart disease in type 2 (non-insulin-dependent) diabetes." Eur J Epidemiol 8(6): 783-788.

Wang, J., S. Ruotsalainen, L. Moilanen, P. Lepisto, M. Laakso, et al. (2007). "The metabolic syndrome predicts cardiovascular mortality: a 13-year follow-up study in elderly non-diabetic Finns." Eur Heart J 28(7): 857-864.

Wang W, H. D., Lee ET. (2002). "Lipoprotein(a) in American Indians is low and not independently associated with cardiovascular disease: the Strong Heart Study." Ann. Epidemiol. 12(2): 107-114.

Werk, E. E., Jr., J. J. Gonzalez and J. E. Ranney (1993). "Lipid level differences and hypertension effect in blacks and whites with type II diabetes." Ethn Dis 3(3): 242-249.

Wilson, P. W., R. D. Abbott and W. P. Castelli (1988). "High density lipoprotein cholesterol and mortality. The Framingham Heart Study." Arteriosclerosis 8(6): 737-741.

Wood, P. D., M. P. Stern, A. Silvers, G. M. Reaven and J. von der Groeben (1972). "Prevalence of plasma lipoprotein abnormalities in a free-living population of the Central Valley, California." Circulation 45(1): 114-126.

World Health Organization (2007). "Prevention of cardiovascular disease: guideline for assessment and management of cardiovascular risk." WHO Press.

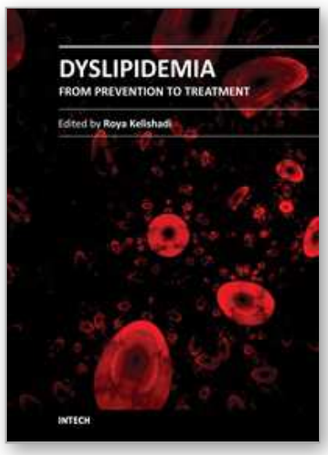
Wu, D. M., L. Pai, P. K. Sun, L. L. Hsu and C. A. Sun (2001). "Joint effects of alcohol consumption and cigarette smoking on atherogenic lipid and lipoprotein profiles: results from a study of Chinese male population in Taiwan." Eur J Epidemiol 17(7): 629-635.

Zaman, M. M., N. Yoshiike, M. A. Rouf, M. H. Syeed, M. R. Khan, et al. (2001). "Cardiovascular risk factors: distribution and prevalence in a rural population of Bangladesh." J Cardiovasc Risk 8(2): 103-108.

Zaninotto, P., J. Mindell and V. Hirani (2007). "Prevalence of cardiovascular risk factors among ethnic groups: results from the Health Surveys for England." Atherosclerosis 195(1): e48-57.

Zhang, X., Z. Sun, D. Zhang, L. Zheng, J. Li, et al. (2009). "Prevalence and association with diabetes and obesity of lipid phenotypes among the hypertensive Chinese rural adults." Heart Lung 38(1): 17-24.

Zhao, W. H., J. Zhang, Y. Zhai, Y. You, Q. Q. Man, et al. (2007). "Blood lipid profile and prevalence of dyslipidemia in Chinese adults." Biomed Environ Sci 20(4): 329-335.



Dyslipidemia - From Prevention to Treatment
Edited by Prof. Roya Kelishadi

ISBN 978-953-307-904-2
Hard cover, 468 pages
Publisher InTech
Published online 03, February, 2012
Published in print edition February, 2012

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.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Lei Zhang, Qing Qiao and Yanhu Dong (2012). Ethnic Difference in Lipid Profiles, Dyslipidemia - From Prevention to Treatment, Prof. Roya Kelishadi (Ed.), ISBN: 978-953-307-904-2, InTech, Available from: http://www.intechopen.com/books/dyslipidemia-from-prevention-to-treatment/ethnic-differences-in-lipid-profiles

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen