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Determinants of serum triglycerides and high-density lipoprotein cholesterol in traditional Trobriand Islanders: the Kitava Study

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Objectives: To analyse variables explaining the variation between serum triglycerides (TGs) and high density lipoprotein cholesterol (HDL-C) in a non-western population characterized by unfavourable TG and HDL-C levels despite marked leanness, low blood pressure and low fasting serum insulin. The study subjects included yraditional Pacific Islanders from Kitava, Trobriand Islands, Papua New Guinea and a population in Sweden. *Methods*: The study was designed as a cross-sectional survey. Fasting serum lipoproteins and apolipoproteins, insulin, blood pressure and anthropometric measurements were analysed in 122 male and 47 female Kitavans aged 20–86 years and in a control population of 729 healthy men and women aged 20–66 from Uppsala. Main outcome measures were determinants of TG and HDL-C using a simple and multiple linear regression analysis. *Results*: A negative association was found between TGs and HDL-C in Kitava ($r = -0.38$, $p < 0.0001$) and Sweden ($r = -0.46$, $p < 0.0001$), while TGs were positively associated with non-HDL-C and ApoB in both groups. In contrast to what was found in the Swedish subjects, TG and HDL-C levels were not associated with body mass index, waist circumference, glucose, insulin or systolic blood pressure in the Kitavans. *Conclusion*: Despite an apparent absence of cardiovascular disease and the metabolic syndrome in the Kitavans, the relationship between TGs and HDL-C was similar to that observed in Caucasians, while neither of the variables was associated with markers of insulin sensitivity in the Kitavans. Whether the findings can be explained by normal physiology or partially reflect the high intake of carbohydrates and saturated fat in Kitava is uncertain.

Key words: Cardiovascular disease; diet; epidemiology; lipid transport; lipoproteins; metabolic syndrome; westernization

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INTRODUCTION

In westernized populations there is a gradual negative relationship between cardiovascular disease and serum high density lipoprotein cholesterol (HDL-C) and a similar positive one for fasting serum triglycerides (TGs) [1]. The two variables are negatively interrelated and the question of which of the two is more important is unresolved. Furthermore, there is uncertainty about whether HDL-C and TGs are causative factors of atherosclerotic disease or neutral bystanders in a cluster of metabolic disturbances linked to abdominal overweight and insulin resistance. This may be studied by comparing populations with different degrees of overweight and risk for cardiovascular diseases.

In populations where carbohydrates provide more than 50% of dietary energy (en%) and protein intake is low (<15 en%), TGs are typically high, especially when saturated fatty acids dominate over unsaturated fatty acids [2–7]. This seems to be true even when much of the carbohydrate is obtained from nutrient-rich foods such as tubers and vegetables. One such population is found in Kitava, Trobriand Islands, Papua New Guinea, Western Pacific. Tubers such as yams and sweet potato are dietary staples supplemented mainly by fruit, fish, coconut and leafy vegetables, and the intake of Western food is negligible [8]. According to our estimates, the percentages of energy (en%) from protein, fat and carbohydrates are 10, 21 and 69 en% in Kitava compared with 12, 37 and 48 en% in Sweden [9, 10]. The estimated intakes of saturated, monounsaturated and polyunsaturated fatty acids (PUFAs) in Kitava are 17, 2 and 2 en% compared with 16, 16 and 5 en% in Sweden. The n-3 fatty acids are the predominant PUFAs in Kitava while n-6 PUFAs predominate in Sweden [10]. However, although the Kitavans are apparently free from overweight, hypertension, hyperinsulinaemia, hyperleptinaemia, cardiovascular disease and malnutrition [8, 11–14], the population means of TGs and HDL-C are not favourable in comparison with those of Caucasians [11, 15].

If the relation between TG and HDL-C were the same among the lean Kitavans as in Caucasians, it would add to the evidence that high levels of TGs and low HDL-C do not always represent an increased risk of cardiovascular disease or the metabolic syndrome. Therefore, in the present study we analysed the relationships between TGs and HDL-C and between each of these and a number of cardiovascular risk factors. These included age, body mass index (BMI), systolic and diastolic blood pressure, and serum levels of glucose, insulin total cholesterol (TC), non-HDL-C, apolipoprotein B (ApoB), apolipoprotein A1 (ApoA1) and lipoprotein(a) (Lp(a)). Of these variables, we found BMI, diastolic blood pressure, glucose and insulin to be particularly beneficial in the Kitavans, as compared with randomly selected Swedish subjects [11–13].

RESEARCH DESIGN AND METHODS

Descriptions of the Kitava study population and its lifestyle, our methods of age estimation, sampling procedures, and the examinations of non-attendants have been given in an earlier study [8]. Ages were estimated by using historic events as the reference. Fasting blood samples were obtained in the morning after an overnight fast from 122 males and 46 females aged 20–86 years. Electricity for the centrifuge was provided by a petrol-powered generating set. Samples were kept at −130°C in a liquid nitrogen vessel (Air Liquide GT35) until storage at −70°C in Sweden.

Conventional methods, validated for precision and accuracy, were used for serum analyses, as already described [11, 12, 14]. Serum TC, HDL-C and TGs were determined by using enzymatic methods. The concentrations of ApoA1 were determined by immunoturbidimetry, Lp(a) by the Pharmacia Apo(a) RIA method, serum glucose by the glucose oxidase method and serum insulin by radioimmunoassay (Phadeseph Insulin RIA; Pharmacia Diagnostics, Uppsala, Sweden). Standard methods were used for measurements of sitting blood pressure (mercury sphygmomanometer), weight (single beam balance scale), standing height (measuring rod) and waist circumference (tape measure). The same variables were measured in 729 healthy men and women aged 20–66 years, from Uppsala, constituting 78% of employees of a telephone company [16]. Among these, 21% of the men and 35% of the women were smokers.

Kitavan dietary habits were investigated by means of a diet history, by weighing ready-to-eat

foods and by sharing of their food habits by one of the authors (SL) who lived with the Kitavans for seven weeks [10]. Tubers (yams, sweet potatoes, taro and manioc), fruit, fish and coconuts were dietary staples and the intake of Western food was negligible. The estimated proportions of energy derived from total, saturated, monounsaturated fats and PUFAs were 21, 17, 2 and 2% of dietary energy (en%) compared with 37, 16, 16 and 5 en% in Sweden [17]. Median basal metabolic rate (BMR) as predicted from weight at age 18–30 years was 5.5 MJ/day in males and 4.9 MJ/day in females. The level of physical activity of Kitavans was roughly estimated at 1.7 multiples of BMR. Three out of of four Kitavans of both sexes were daily smokers and the rest were non-smokers. The difference in triceps skinfolds and serum lipoproteins and apolipoproteins between smokers and non-smokers was of the same magnitude as in Western populations [11]. Estimated life expectancy was 45 years at birth and an additional 25 years or more thereafter. Major causes of death were infections, trauma and complications of pregnancy, while atherothrombotic disorders were absent or rare.

Continuous variables were checked for or transformed into apparent normality by use of normal probability plots [18]. Pearson's correlation coefficients were calculated for univariate relations. Significant predictors from each of these analyses (excluding TC, ApoB, ApoA1 and ApoA1/HDL-C by predefined criteria) were introduced as independent variables in forward stepwise multiple linear regressions with log TG and HDL-C as dependent variables (F-to-enter = 4; F-to-remove = 3.996). Smoking status (No/Yes) was also introduced into the models, because smokers in Kitava had lower HDL-C and ApoA1 and higher TC and LDL-C than non-smokers, while TGs tended to be higher ($p = 0.07$) [11]. Standardized regression coefficients (std β) are given to show the relative impact, in the final step, of each independent variable. The value of std β demonstrates by how many standard deviations the dependent variable changes when the explanatory variable changes one standard deviation, given that the other entered variables are kept constant. Cumulative adjusted R^2 values are given to show, at each step, the percentage of variation of the dependent variable so far explained by the model. Standardized residuals in the stepwise regression models were plotted against standard z scores in normal plots, for quality control. The relationships did not differ essentially between males and females, therefore the two sexes were analysed together.

RESULTS

Clinical characteristics of the Kitava study population are presented in Tables I and II and Pearson's correlation coefficients for TGs and HDL-C are listed in Tables III and IV. A negative association was found between TGs and HDL-C in Kitava and Sweden, while TGs were positively associated with non-HDL-C and ApoB in both groups. In contrast to what was found in the Swedish subjects, TGs and HDL-C were not associated with BMI, waist circumference, glucose, insulin or systolic blood pressure in the Kitavans. A positive association between diastolic blood pressure and HDL-C was found in Kitavans, whereas the association was negative among the Swedes. The expected associations between HDL-C and ApoA1 and between HDL-C and ApoA1/HDL-C were very similar between the two populations. HDL-C was correlated with TC only in Kitavans, and HDL-C was correlated with non-HDL-C and ApoB only in Swedes. A strong positive correlation between ApoA1 and HDL-C was

TABLE I. Clinical characteristics of Kitavan males (n = 122).

	Mean	(SD)	Min–max
Age, years	49	(18)	(20–86)
Weight, kg	53	(7)	(37–72)
Height, cm	162	(5)	(149–174)
Body mass index, kg/m^2	20	(2)	(15–26)
Systolic blood pressure, mmHg	116	(15)	(84–162)
Diastolic blood pressure, mmHg	70	(6)	(51–87)
S-cholesterol, mmol/L	4.7	(0.9)	(3.0–7.0)
HDL-C, mmol/L	1.1	(0.2)	(0.5–1.7)
Non-HDL-C, mmol/L	3.6	(1.0)	(1.2–7.4)
Triglycerides, mmol/L	1.7	(0.4)	(0.5–3.0)
–Median	1.0		
ApoB, g/L	1.0	(0.2)	(0.6–2.1)
ApoA1, g/L	1.0	(0.2)	(0.6–1.4)
ApoA1/HDL	0.9	(0.1)	(0.6–1.3)
Lp(a), U/L	237	(365)	(12–2268)
–Median	101		
S-glucose, mmol/L	3.7	(0.6)	(2.3–6.1)
Insulin, IU/mL	4.0	(2.9)	(0.7–26.2)
Smokers, %	77		

TABLE II. Clinical characteristics of Kitavan females (n = 47).

	Mean	(SD)	Min – max
Age, years	52	(15)	(20 – 83)
Weight, kg	40	(5)	(32 – 52)
Height, cm	150	(5)	(141 – 160)
Body mass index, kg/m^2	18	(2)	(15 – 23)
Systolic blood pressure, mmHg	121	(17)	(95 – 173)
Diastolic blood pressure, mmHg	70	(8)	(58 – 86)
S-cholesterol, mmol/L	5.8	(1.2)	(3.6 – 8.7)
HDL-C, mmol/L	1.2	(0.3)	(0.6 – 2.0)
Non-HDL-C, mmol/L	4.4	(1.2)	(2.6 – 7.2)
Triglycerides, mmol/L	1.3	(0.6)	(0.5 – 2.7)
– Median	1.0		
ApoB, g/L	1.1	(0.2)	(0.8 – 1.7)
ApoA1, g/L	1.1	(0.2)	(0.5 – 1.7)
ApoA1/HDL	0.9	(0.1)	(0.7 – 1.2)
Lp(a), U/L	300	(363)	(17 – 1648)
– Median	202		
S-glucose, mmol/L	3.9	(0.7)	(2.5 – 5.5)
Insulin, IU/mL	4.8	(4.7)	(1.8 – 30.3)
Smokers, %	78		

seen in the two populations. Furthermore, TGs were negatively associated with HDL-C and positively associated with non-HDL-C and the ApoA1/HDL-C ratio in both ethnic groups. These and other relationships between lipoproteins were very similar in the two populations, but only in Swedish subjects were the variations in TGs or HDL-C explained by glucose, insulin, BMI, waist and blood pressure. Non-HDL-C and ApoB were unrelated to HDL-C only in the Kitavans. Relationships between serum lipids were essentially unchanged in a forward stepwise multiple linear regression (Tables V and VI; highly collinear variables were not tested against each other).

TABLE III. Pearson correlation coefficients for serum triglycerides.

	Kitava		Sweden	
	r	p-value	r	p-value
Age	0.16	0.04	0.27	<0.0001
BMI	−0.04	ns*	0.42	<0.0001
Waist	−0.03	Ns	0.49	<0.0001
Systolic BP	0.15	Ns	0.28	<0.0001
Diastolic BP	0.12	Ns	0.31	<0.0001
Cholesterol	0.33	<0.0001	0.47	<0.0001
Non-HDL-C	0.43	<0.0001	0.60	<0.0001
HDL-C	−0.38	<0.0001	−0.46	<0.0001
ApoB	0.37	<0.0001	0.44	<0.0001
ApoA1	−0.02	Ns	−0.03	ns
ApoA1/HDL	0.68	<0.0001	0.69	<0.0001
Lp(a)	0.18	0.03**	−0.03	ns
Glucose	0.01	Ns	0.20	<0.0001
Insulin	0.00	Ns	0.40	<0.0001

*Non-significant ($p > 0.05$).
**Non-significant after age adjustment ($p > 0.05$).
BMI = Body mass index.

TABLE IV. Pearson correlation coefficients for HDL cholesterol.

	Kitava		Sweden	
	r	p-value	r	p-value
Age	−0.15	0.05	−0.03	Ns
BMI	−0.11	ns*	−0.32	<0.0001
Waist	0.02	ns	−0.42	<0.0001
Systolic BP	0.04	ns	−0.16	<0.0001
Diastolic BP	0.17	0.04	−0.20	<0.0001
Cholesterol	0.27	0.0007	0.03	Ns
Non-HDL-C	0.02	Ns	−0.30	<0.0001
ApoB	−0.05	Ns	−0.28	<0.0001
ApoA1	0.81	<0.0001	0.78	<0.0001
ApoA1/HDL	−0.68	<0.0001	−0.82	<0.0001
Lp(a)	−0.11	Ns	0.00	Ns
Glucose	0.06	Ns	−0.11	0.0007
Insulin	0.00	Ns	−0.27	<0.0001

ns = Non-significant ($p > 0.05$).

DISCUSSION

This study shows that TG is related to HDL-C and ApoA1/HDL-C in a similar manner in Kitava as in Sweden. The relation is thus seen in a non-Western population with apparent absence of cardiovascular disease and several markers of the metabolic syndrome. Evaluation of TGs and HDL-C as cardiovascular risk factors must thus be restricted to the study population, and the relationship between TG and HDL parameters is a fundamental feature of lipoprotein metabolism.

A comprehensive hypothetical explanation of the correlations, that may be applicable to the Kitavans as well as some other populations, is the following: The diet is relatively high in carbohydrate and in saturated fatty acids. The abundance of carbohydrates will decrease postprandial oxidation of fatty acids, thereby increasing their recirculation in very low density lipoprotein triglycerides (VLDL-TGs). This recirculation may be higher for the saturated fatty acids, because of the lower degree of acylation of palmitic and myristic acid into the large pools of tissue phospholipids, than for the unsaturated fatty acids. With increasing

TABLE V. Variables explaining variations in serum triglycerides in a forward stepwise, multiple, linear regression analysis.

Explanatory variables	STD β in final step	p-value	Cumulative adj. R^2	p-value
Kitava (n = 169)				
Non-HDL-C	0.37	0.0001	0.17	0.0001
HDL-C	−0.46	0.0001	0.33	0.0001
Sweden (n = 729)				
Non-HDL-C	0.44	<0.0001	0.34	<0.0001
HDL-C	−0.25	<0.0001	0.43	<0.0001
Insulin	0.18	<0.0001	0.47	<0.0001
Smoker (no, yes)	0.11	<0.0001	0.49	<0.0001
Lp(a)	−0.10	<0.0001	0.52	<0.0001
BMI	0.10	0.0008	0.52	<0.0001

Non-significant tested independent variables: Kitava: Age, Lp(a); Sweden: Age, waist, systolic BP, diastolic BP, glucose.

BMI = Body mass index.

TABLE VI. Variables explaining variations in serum HDL cholesterol in a forward stepwise multiple linear regression analysis.

Explanatory variables	std β	p-value	Adj. R^2	p-value
Kitava (n = 169)				
TGs	−0.40	<0.0001	0.14	<0.0001
Diastolic BP	0.24	0.002	0.19	<0.0001
Smoker (N, Y)	−0.18	0.02	0.23	<0.0001
Sweden (n = 729)				
TGs	−0.40	<0.0001	0.22	<0.0001
Waist	−0.25	<0.0001	0.29	<0.0001
Age	0.20	<0.0001	0.31	<0.0001
Smoker (N,Y)	0.07	0.02	0.32	<0.0001
Non-HDL-C	−0.07	0.05	0.32	<0.0001

TGs = Triglycerides; BMI = body mass index.

Non-significant tested independent variables: Kitava: Age, diastolic BP; Sweden: Age, BMI, systolic BP, diastolic BP, glucose, insulin.

concentrations of VLDL-TG, the transfer of cholesteryl esters from HDL to VLDL in exchange for TG will increase [19, 20]. In this way, HDL is depleted of cholesteryl ester, which explains the strong positive relationship between ApoA1/HDL-C and TG levels in both study populations. An increased transfer of cholesteryl ester to VLDL leads to conversion of VLDL into LDL, which, in turn, is catabolized mainly by the liver. A low HDL cholesterol may thus not reflect a decreased reverse cholesterol transport but rather the relative role of HDL and LDL in this process in individuals with different TG levels. Accordingly, the correlations may be understood from a simplistic view of lipid transport and it is not necessary to postulate any variation in cholesterol transport from peripheral tissues.

Knopp *et al.* found that lowering dietary fat below about 25 en% while increasing carbohydrate intake above 60 en% may not yield any further decrease in LDL-C and and may even be counterproductive to TG, HDL-C and ApoB levels [21]. The noted lack of benefit was suggested to be due to an enhanced endogenous synthesis of palmitic acid, as indicated by a slight increase in plasma palmitate, whereas stearate decreased slightly. This is in line with an earlier report from Kitava, where a high proportion of palmitate in serum cholesterol esters despite low intakes of palmitic acid and total fat suggested a high level of endogenous lipid synthesis [22, 23]. There is substantial evidence to support the notion that, at least in westernized populations, insulin resistance explains some of the relationship between TG levels and HDL-C, partly by increased hepatic production of VLDLs [24]. Our findings indicate that other mechanisms are important as well. The absence of overweight subjects and the low plasma levels of glucose, insulin, leptin and plasminogen activator inhibitor-1 activity in Kitavans [11–14] may explain the fact that none of these variables was related to TGs or HDL-C in this population and would argue against insulin resistance being a key factor behind the strong negative relationship between TGs and HDL-C in Kitava. However, this does not exclude the possibility that variation in reverse cholesterol transport from peripheral tissues to the liver is involved, since this process may largely be independent of insulin sensitivity [25].

In conclusion, despite the fact that cardiovascular disease seems to be absent in Kitava, TGs were related to non-HDL-C, HDL-C and ApoA1/HDL-C in an identical manner to that observed in Caucasians, while, conversely, neither the level of TGs nor HDL-C was associated with markers of insulin sensitivity. These findings support the notion that low HDL-C may result from high circulating levels of TGs, irrespective of whether these are endogenously synthesized or not, and irrespective of their pathogenetic significance. In addition, our findings lend no support to the concept that a very high intake of carbohydrates (>60 en%) increases the risk of cardiovascular disease.

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