Black/White Differences in Risk Factors for Arteriographically Documented Coronary Artery Disease in Men

- By: David S. Freedman, PhD, <u>Harvey W. Gruchow, PhD</u>, Jack C. Manley, MD, FACC, Alfred J. Anderson, MS, Kathleen A. Sobocinski, MS, and Joseph J. Barboriak, ScD
- Freedman DS, Gruchow HW, Manley JC, Anderson AJ, Sobocinski KA, Barboriak JJ. Black/white differences in risk factors for arteriographically-documented coronary artery disease in men. American Journal of Cardiology 62:214-219, 1988.

Made available courtesy of Elsevier: http://www.elsevier.com/

*** Note: Figures may be missing from this format of the document

Abstract:

Although the leading cause of death among black men in the United States is coronary artery disease (CAD), risk factors have not been well documented in black populations. Therefore, possible racial differences in the relation of several characteristics to the extent of CAD were assessed in 4,722 white and 169 black men who underwent arteriography. Associations between an occlusion score (ranging from 0 to 300), reflecting the severity of CAD, and levels of total and high-density lipoprotein (HDL) cholesterol, triglycerides, cigarette smoking, alcohol intake, relative weight, systemic hypertension and diabetes mellitus were examined. Most risk factors were significantly related to the extent of CAD in both races, but lipid levels showed stronger associations with CAD among blacks: correlations between CAD and total cholesterol were 0.16 (whites) vs 0.29 (blacks) and associations with HDL cholesterol were -0.22 (whites) vs -0.49 (blacks). In addition, at adverse levels of certain risk factors, blacks had more extensive CAD than did whites: mean occlusion scores were 148 (whites) and 238 (blacks) at HDL cholesterol levels <30 mg/dl. As assessed by multiple linear regression, however, only triglyceride levels were differentially related to CAD between whites ($\beta = 0$) and blacks ($\beta = 0.47$), p <0.01 for racial contrast. These results document the importance of risk factors in black men and indicate black/white differences in the relation of triglycerides to CAD.

Article:

Although blacks in the United States have one of the highest mortality rates due to coronary artery disease (CAD) in the world, risk factors among blacks have not been extensively studied.^{1,2} Even higher mortality rates among black men, due to a high prevalence of hypertension, may be mitigated by elevated levels of high-density lipoprotein (HDL) cholesterol.³ Levels of total cholesterol, along with the prevalence of diabetes mellitus and cigarette smoking, are similar in white men and black men.⁴

The Evans County Study, the first prospective study of risk factors in blacks, showed that elevated levels of total cholesterol and blood pressure, along with cigarette smoking,^{5,6} were predictive of CAD in black men. A subsequent report, however, suggested that black men with low (<170 mg/dl) levels of total cholesterol were also at increased risk for the clinical manifestations of CAD.⁷ Follow-up studies of black men in the Multiple Risk Factor Intervention Trial^{8,9} showed that levels of total and low-density lipoprotein cholesterol, but not HDL cholesterol, were predictive of subsequent clinical disease. Additional longitudinal studies of risk factors in blacks are needed, in part, to overcome the small number of events among blacks in Evans County (31 of 294) and in the Multiple Risk Factor Intervention Trial (16 of 465).

However, the relation of risk factors to anatomically defined CAD can be assessed using data collected during arteriography.¹⁰ Using this approach, Maynard et al¹¹ reported that diabetes mellitus, cigarette smoking and total cholesterol were associated with increased CAD among 571 blacks. Although the importance of triglyceride and HDL cholesterol levels was not assessed in this report, other cross-sectional studies have found that levels of HDL cholesterol are not significantly associated with CAD in black Men.^{12,13}

The relation of risk factors to the severity of anatomically defined CAD in whites has been previously described.^{10,14,15} The current study, involving 4,722 white and 169 black men, examines possible racial differences in the relation of risk factors to CAD. Because previous studies^{7,9,12,13} suggest that the association of total (or HDL) cholesterol with CAD may differ between whites and blacks, particular attention is given to lipid levels.

METHODS

Population and disease status: Our study included 4,891 men (3.5% black), ages 24 to 84 years, referred for diagnostic coronary arteriography between 1972 and 1986. Prior myocardial infarction was reported by 48% of patients and chest pain was present in 74%. Levels of total cholesterol and triglycerides were recorded for all patients.

Arteriograms were evaluated, without knowledge of risk factors, by a radiologist and cardiologist experienced in their interpretation. Disagreements were resolved by consensus after consultation with a second cardiologist. Percent reductions in lumen diameter (0%, 1 to 50%, 51 to 74%, 75 to 89%, 90 to 99% and 100%) due to the most severe stenosis in the left main, left anterior descending, circumflex and right coronary arteries were incorporated into an occlusion score reflecting the overall severity of CAD. A scale suggested by Rowe et al¹⁶ was modified, with a score of 0 representing no observed occlusion and a score of 300 denoting total occlusion of the major arteries.

Risk factor information: Medical records and questionnaires were used to obtain data concerning obesity, alcohol consumption, smoking, education, recent medication use and histories of hypertension, myocardial infarction, angina and diabetes mellitus. The dates of these clinical events were unknown. Patients were excluded if they reported use of thyroid or cholesterol-lowering medication, or had hypo- or hyperthyroidism. These restrictions eliminated 532 patients, leaving 4,891 subjects available for analysis.

Height and weight were used to calculate Quetelet index (kg/m²). A 5-point smoking scale, reflecting both frequency and duration of smoking (1 = never smoked; 5 = smoked \geq 2 packs daily for \geq 20 years), was calculated. Usual alcohol intake (including beer, wine and mixed drinks) was converted to ounces per week of absolute alcohol. Data concerning smoking history and alcohol intake were available for 4,367 (89%) patients. Following an overnight fast, blood samples were collected before arteriography. Plasma levels of total cholesterol and triglycerides were measured using automated procedures ¹⁷⁻¹⁹ in a laboratory standardized (and monitored) by the Centers for Disease Control. Beginning in 1977, levels of HDL cholesterol have been measured in 1,002 white and 32 black men using procedures followed by the Lipid Research Clinics.¹⁹ The ratio of total to HDL cholesterol was used as an estimate of the atherogenicity of each patient's lipoprotein profile.

Statistical analyses: Levels of selected characteristics were first compared between white men and black men using *t* and chi-square tests. Although several distributions were skewed, Wilcoxon tests yielded similar results. Following adjustment for age, quintiles of several risk factors were formed and racial comparisons of CAD were made within each risk-factor stratum. (These analyses used the same cutpoints in both races.) Analysis of covariance, also adjusted for age, was used to compare mean occlusion scores according to diabetes, hypertension, smoking status and educational achievement. Associations between risk factors and CAD were examined using partial correlations.

Multiple linear regression, including interaction terms (race X risk factor), was used to examine black/ white differences in the relation of risk factors to CAD. The statistical significance (at the 0.05 level) of each product term was assessed by a backward elimination procedure in a model that included all main effects.²⁰ Separate models were also fit for each race and regression coefficients were compared between whites and blacks using the standard error of their difference (i.e., the square root of the sum of the variances divided by the total sample size). Standardized regression coefficients, representing the change in occlusion predicted by a standard deviation change in each predictor variable, were used to compare the relative importance of each risk factor.

	Whites (n = 4,722)	Blacks (n = 169)		
Age (yrs)	55 (56) ± 9*	54 (55) ± 10*		
Occlusion score	$134(145) \pm 85^{\dagger}$	$112(115) \pm 95^{\dagger}$		
Number of coronary arteries narrowed ≥75% in diameter [‡]				
0	30)	44)		
1	31(§	22 (5		
2	25	19 ([°]		
3	14)	₁₄)		
Coronary artery narrowed (%)				
Left main (≥50% narrowing)	6	4		
Left anterior descending	47 [†]	36†		
Left circumflex	33	31		
Right	43	36		
Acute myocardial infarction (%)	48	47		
Chest pain (%)	73	78		

RESULTS

Mean levels of selected characteristics describing the extent of CAD are listed in Table I. Although the mean ages of black men and white men were almost identical, whites had more coronary artery occlusion primarily due to the large proportion of black men with minimal CAD (44% of blacks vs 31% of whites did not have significant narrowing of any vessel). This racial difference in stenotic disease was most pronounced in the left anterior descending artery. Although similar proportions of blacks and whites reported a previous myocardial infarction or chest pain, the increased CAD among whites existed irrespective of these characteristics.

The levels of risk factors compared between whites and blacks are listed in Table II. Plasma levels of total and HDL cholesterol showed no racial difference, but whites had higher triglyceride levels. Although a greater proportion of black men reported a history of diabetes mellitus, the prevalence of hypertension was similar in both races. Cumulative smoking histories were also similar, but black men were more likely to currently smoke cigarettes than were white men.

	Whites (n = 4,722)	Blacks (n = 169)
Total cholesterol (mg/dl) Triglycerides (mg/dl) HDL cholesterol (mg/dl) [‡] Total/HDL cholesterol (mg/dl) [‡] Quetelet index (kg/m ²) Alcohol intake (oz/wk) Diabetes mellitus (%) Hypertension (%) Antihypertensive medication (%) Smoking status (%) Never Past Current Education (%) < Grade 12 High school graduate 1–3 years of college College graduate	$233 (230) \pm 50^{*}$ $190 (162) \pm 128^{\dagger}$ $38 (36) \pm 10$ $6.1 (5.9) \pm 2$ $26.6 (26.3) \pm 3.5$ $5.5 (3.2) \pm 7.1$ 8^{\dagger} 39 25 $3.4 (4) \pm 1.4^{*}$ 19 63 8^{\dagger} 32 28 28 22 18 5	$235 (234) \pm 50^{4}$ $175 (144) \pm 93^{1}$ $38 (36) \pm 9$ $6.3 (6.5) \pm 2$ $26.5 (26.7) \pm 3.5$ $5.3 (2.6) \pm 8.2$ 13^{1} 40 22 $3.3 (3) \pm 1.4$ 19 52 29 5 46 25 20 9 5

Risk factors were then categorized into quintiles. Table III shows that the associations between lipids and CAD were stronger in blacks when compared with white men. For example, increases in mean occlusion scores associated with triglycerides (quintile 5-quintile 1) were 40 (whites) versus 90 (blacks). Although based on smaller numbers, the mean occlusion scores in blacks varied by >140 points across quintiles of both HDL and

total/HDL cholesterol. Similar black/white differences were also seen in age-adjusted correlations between risk factors and coronary artery occlusion. Correlations with levels of triglycerides were r = 0.10 (whites) versus 0.27 (blacks), and 0.25 versus 0.67 for the ratio of total to HDL cholesterol. In contrast to these fairly strong associations, alcohol intake showed a U-shaped association with occlusion in blacks.

Risk Factor	Race	Ouintile [†]					Difference Quintile 5	Correlation with
		1 (Low)	2	3	4	5 (High)	Quintile 1	Occlusion [‡]
Total cholesterol	Whites	112 ± 87	125 ± 84	137 ± 84	146 ± 82	151 ± 81	39	0.16
	Blacks	81 ± 87	104 ± 99	96 ± 77	121 ± 84	160 ± 105	79	0.29
Triglycerides	Whites	108 ± 89	132 ± 85	141 ± 84	142 ± 83	148 ± 78	40	0.10
0,	Blacks	81 ± 91	110 ± 93	118 ± 94	96 ± 77	171 ± 89	90	0.27
HDL cholesterol	Whites	148 ± 74	128 ± 80	140 ± 77	118 ± 80	100 ± 80	-48	-0.22
	Blacks	238 ± 25	199 ± 58	121 ± 102	117 ± 80	95 ± 93	-143	-0.49
Total/HDL cholesterol ¹	Whites	91 ± 82	122 ± 75	129 ± 80	140 ± 75	150 ± 74	59	0.25
	Blacks	81 ± 94	78 ± 103	129 ± 42	178 ± 56	227 ± 59	146	0.67
Alcohol consumption	Whites	139 ± 86	142 ± 84	140 ± 81	124 ± 86	123 ± 85	-16	-0.08
	Blacks	124 ± 97	116 ± 93	100 ± 94	108 ± 96	127 ± 94	3	0.07§
Smoking history [¶]	Whites	121 ± 87	128 ± 88	129 ± 88	138 ± 83	145 ± 79	24	0.09
	Blacks	116 ± 96	63 ± 84	96 ± 87	125 ± 92	145 ± 97	29	0.18 [§]
Quetelet index	Whites	135 ± 88	136 ± 83	132 ± 81	131 ± 85	134 ± 84	-1	-0.01§
	Blacks	103 ± 95	121 ± 95	95 ± 91	135 ± 97	117 ± 93	14	0.11 [§]

These racial differences in associations between lipids and CAD existed irrespective of a previous myocardial infarction. For example, age-adjusted correlations between triglycerides and CAD were r = 0.12 (whites) versus 0.33 (blacks) among patients who did not report a previous myocardial infarction (data not shown). Among patients who had a myocardial infarction, corresponding correlations with triglycerides were 0.05 and 0.26, respectively. Despite small numbers, the ratio of total to HDL cholesterol also remained strongly associated with occlusion among blacks in both the nonmyocardial infarction group (r = 0.66) and in patients who had had a myocardial infarction (r = 0.67).

Table III also allows black/white differences in occlusion to be examined among patients with similar risk-factor levels. At favorable levels of lipids and smoking history, blacks had lower occlusion scores than did whites. For example, at levels of total cholesterol \leq 190 mg/dl (quintile 1), the mean occlusion scores were 112 (whites) and 81 (blacks). However, at adverse risk-factor levels, the mean occlusion scores were either similar between the races (for total cholesterol and smoking history) or were higher among blacks (for triglycerides and HDL cholesterol). The 12 black men with HDL cholesterol levels <35 mg/dl (quintiles 1 and 2) had a mean occlusion score of 212.

•		Whites		Blacks	
Risk Factor		No.	(Mean \pm SD)	No.	(Mean ± SD
Diabetes mellitus	0	4,204	131 ± 85†	137	110 ± 95
	+	381	$162 \pm 78^{\dagger}$	21	115 ± 97
Hypertension	0	2,889	$132 \pm 87^{\ddagger}$	102	102 ± 92
	+	1,883	$138 \pm 83^{\ddagger}$	67	128 ± 100
Antihypertensive medication	0	3,554	133 ± 87	131	110 ± 92
	+	1,168	137 ± 82	38	119 ± 104
Smoking status [§]					
Never		903	124 ± 89	31	115 ± 99
Past		2,908	139 ± 83	85	119 ± 97
Current		827	130 ± 87	48	99 ± 91
Education§					
< grade 12		1,510	136 ± 85	78	101 ± 99
High school graduate		1,331	134 ± 86	42	118 ± 90
1-3 years college		1,034	132 ± 83	33	120 ± 93
College graduate		831	134 ± 87	15	140 ± 91

The mean levels of coronary artery occlusion, according to categories of other risk factors, are listed in Table IV. Increased occlusion scores were associated with both diabetes and hypertension, but not with smoking status (never/past/current). However, the increase in occlusive disease that was associated with diabetes tended to be greater in white men, whereas the effect of hypertension was slightly stronger among blacks. Educational achievement tended (3 = 0.12) to be related to the extent of CAD only among black men.

To statistically test black/white differences in associations with CAD, race X risk-factor product terms were then examined in regression analyses. After controlling for the effects of other risk factors (with the exception of HDL cholesterol), the relation of triglyceride levels to CAD differed between white men and black men. Although significantly related to CAD in both races, triglycerides showed a stronger association with occlusion among blacks ($\beta = 0.25$) than among whites ($\beta = 0.03$), p = 0.007 for racial contrast. No other risk factor showed a. black/white difference in its association with CAD. Although HDL cholesterol was measured in only 21% of the patients, when this lipoprotein fraction was included as a predictor variable, triglycerides remained significantly related to the extent of CAD in blacks ($\beta = 0.47$) but not whites ($\beta = 0$). HDL cholesterol was inversely related to the extent of CAD in both races.

Regression models were then fit separately for each race, with standardized regression coefficients shown in Figure 1. A large black/white difference in the relation of triglycerides to CAD was again seen, with standard-ized regression coefficients of 0.21 (blacks) and 0.05 (whites). A 1-unit standard deviation change in triglycerides was associated with a 24-point change in the occlusion score among blacks, but with only a 7-point change among whites. Associations of CAD with cholesterol, age, smoking history and Quetelet index were similar in blacks and whites. Although the results were suggestive of racial differences in the relation of alcohol intake, diabetes and hypertension to CAD, the contrasts were not statistically significant (p >0.20 for all differences).

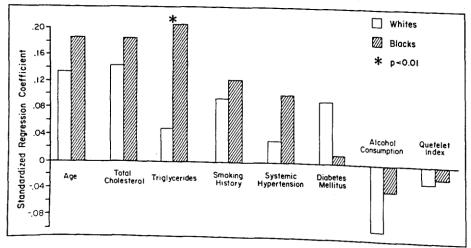


FIGURE 1. Standardized multiple linear regression coefficients related to coronary artery occlusion in males. Models were fit separately for each race. The relation of triglycerides to coronary artery disease was significantly different between whites and blacks.

DISCUSSION

These results indicate that risk factors predictive of clinical manifestations of CAD in whites are associated with anatomically defined disease in black men. Age, levels of total and HDL cholesterol, cigarette smoking, diabetes mellitus and hypertension were significantly related to the extent of CAD in both races. However, tri-glyceride levels were more strongly related to CAD among blacks than whites. Although the independent relation of triglyceride levels to disease is' controversial,^{15,21} these findings indicate that this association exists in black men even after controlling for HDL cholesterol.

CAD is the leading cause of death among black Americans,¹ but the importance of risk factors has been assessed in relatively few studies of black populations. Although cigarette smoking and blood pressure have consistently been predictive of clinical disease in black Men,^{6,8,22} the impact of diabetes and alcohol consumption on subsequent disease in blacks has not been examined.² (Diabetes mellitus, however, is associated with increased coronary atherosclerosis in blacks.²³) In contrast, generalized obesity in whites has often been only

weakly associated with both clinical disease²⁴ and anatomically defined CAD.¹⁰ In the present study, Quetelet index was not associated with the extent of CAD in either race.

However, results of previous studies have been suggestive of black/white differences in the relation of lipids to CAD. Although the relation of total cholesterol to CAD was similar between whites and blacks in the Coronary Artery Surgery Study,¹¹ results from Evans County indicate that mortality from ischemic heart disease among blacks is increased at levels of cholesterol <170 mg/dl.⁷ (Low levels of total cholesterol in blacks may reflect large decreases in the HDL fraction.) Furthermore, in contrast to the consistent, inverse relation seen in whites,²⁵ HDL cholesterol levels in black men were not predictive of clinical disease⁹ and have not been significantly related to documented CAD.^{12,13} Because of the small number of blacks in several of these studies, it has been recommended²⁶ that risk factors among blacks be further studied. Although triglyceride levels were not predictive of mortality among black men in Evans County, only 10 deaths occurred in the black cohort.²⁷

The current results show that CAD in black men is independently related to levels of triglycerides (positively) and HDL cholesterol (negatively). However, reasons for the stronger association between triglycerides and CAD in blacks, as compared with whites, are not clear. In the current study triglyceride levels were higher among white men. Furthermore, correlations between HDL cholesterol and triglycerides showed no racial difference (r = -0.38 vs -0.41) and, although the relation of triglycerides to CAD may be strongest at low levels of cholesterol,²⁸ blacks and whites had similar levels in the current study. Possibly, the elevated triglyceride levels in black men are more strongly related to additional atherogenic characteristics. Sniderman et al²⁹ found that among hypertriglyceridemics, patients who also have elevated levels of apolipoprotein B have increased CAD.

Several limitations of the present study should also be considered in interpreting the results. The power of these analyses to detect racial differences in certain risk-factor associations (e.g., HDL cholesterol) is limited because of the relatively small number of black men. In addition, results of coronary arteriography do not always agree with histologic findings and observer variation exists in the interpretation of arteriograms. Furthermore, because arteriography is performed on symptomatic cases thought to be surgical candidates, patients are not representative of the total CAD population. Although it is likely that selection factors for arteriography differ between the races, possibly resulting in a biased estimate of the black/white difference in CAD, the increased stenotic disease among whites existed irrespective of a history of chest pain or myocardial infarction.

As reported in other arteriography studies,^{11,30} black men had lower levels of triglycerides than did white men, but were more likely to currently smoke and to be diabetic. Although these racial differences parallel those observed in the general population,^{2,4} racial comparisons of certain risk factors may be biased. For example, as opposed to findings from population-based studies, the prevalence of hypertension and levels of HDL cholesterol were similar between whites and blacks. Relatively affluent blacks, who are likely overrepresented in arteriography studies, tend to have decreased levels of HDL cholesterol.³¹ However, differences in the relation of risk factors to CAD are unlikely to be due to selection factors and similar racial differences were seen irrespective of a previous myocardial infarction. Risk factor associations in arteriography studies have generally confirmed the findings of population-based studies.¹⁰

Blacks in the US have one of the highest mortality rates due to CAD in the world' and the characteristics that have been established as risk factors in whites are prevalent among blacks. The present findings document that, in general, these risk factors are related to CAD in blacks. Although few black/white differences exist, the relation of triglyceride levels to CAD, a subject of much controversy,²¹ is stronger among blacks. Although further documentation of risk-factor associations is important if persons with moderately elevated cholesterol levels are to be treated,²⁶ the current results suggest that triglyceride levels should also be measured when assessing the risk of clinical complications of CAD in black men.

REFERENCES

1. Gillum RF. Coronary heart disease in black populations. L Mortality and morbidity. Am Heart J 1982; 104:839-851.

2. Gillum RF, Grant CT. Coronary heart disease in black populations. II. Risk factors. Am Heart J 1982; 104:852-864.

^{3.} Tyroler HA, Glueck CJ, Christensen B, Kwiterovich PO. Plasma high-density lipoprotein cholesterol comparisons in black and white populations. The Lipid Research Clinics Program Prevalence Study. Circulation 1980;62(suppl IV):IV99-107.

4.Curry CL, Oliver J, Mumtaz FB. Coronary artery disease in blacks: risk factors. Am Heart J 1984;108•653-657.

^{5.} Tyroler HA, Heyden S, Bartel A, Cassel J, Cornoni JC, Hames CG, Kleinbaum D. Blood pressure and cholesterol as coronary heart disease risk factors. Arch Intern Med 1971;128:907-914.

^{6.} Heyden S, Cassel JC, Bartel A, Tyroler HA, Hames CG, Cornoni JC. Body weight and cigarette smoking as risk factors. Arch Intern Med 1971;128:915919.

^{7.} Tyroler HA, Knowles MG, Wing SB, Logue EE, Davis CE, Heiss G, Heyden S, flames CG. Ischemic heart disease risk factors and twenty-year mortality in middle-age Evans County black males. Am Heart J 1984;108:738-747.

8. Neaton JD, Kuller LH, Wentworth D, Borhani NO. Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration and diastolic blood pressure among black and white males followed up for five years. Am Heart J 1984;108:759-769.

^{9.} Watkins LO, Neaton JD, Kuller LH. Racial differences in high-density lipoprotein cholesterol and coronary heart disease incidence in the usual-care group of the Multiple Risk Factor Intervention Trial. Am J Cardiol 1986;57: 538-545.

10. Pearson TA. Coronary arteriography in the study of the epidemiology of coronary artery disease. Epidemiol Rev 1984,6:140-166.

¹¹. Maynard C, Fisher LD, Passamani ER, Pullum T. Blacks in the Coronary Artery Surgery Study: risk factors and coronary artery disease. Circulation 1986;74:64-71.

^{12.} Cooper R, Sempos C, Ghali J, Ferlinz J. High-density lipoprotein cholesterol and angiographic coronary artery disease in black patients. Am Heart J 1985;110:1006-1011.

^{13.} Pearson TA, Bulkley BH, Kwiterovich PO, Achuff SC, Gordis L. Anatomically defined coronary disease in blacks: importance of hypertension as a risk factor (abstr). Circulation 1979;59-60(suppl II):11-14.

14. Barboriak JJ, Rimm AA, Anderson AJ, Tristani FE, Walker JA, Flemma RJ. Coronary artery occlusion and blood lipids. Am Heart J 1974;87:716-721.

15. Freedman DS, Gruchow HG, Anderson AJ, Rimm AA, Barboriak JJ. Relation of triglyceride levels to coronary artery disease: The Milwaukee Cardiovascular Data Registry. Am J Epidemiol 1988;127:1118-1130.
16. Rowe GG, Thomsen HI, Sternlund RR, McKenna DH, Sialer S, Corliss RJ. A study of hemodynamics and coronary blood flow in man with coronary artery disease. Circulation 1969;39:139-148.

17. Block WD, Jarrett KJ Jr, Levine JB. Use of a single color reagent to improve . the automated determination of serum total cholesterol. In: Skeggs LT Jr, ed. Automation in Analytical Chemistry. New York: Mediad, 1966:345-347.

18. Kessler G, Lederer H. Flurometric measurement of triglycerides. In: Skeggs LT Jr, ed. Automation in Analytical Chemistry. New York: Mediad, 1966:341344.

¹⁹ Manual of Laboratory Operation. Lipid Research Clinics Program. Vol. I. Bethesda: National Institutes of Health, 1974 (DHEW publication No. [N111] 75-628).

20. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiologic Research. Principles and Quantitative Methods. Belmont, California: Wadsworth, 1982 447- 456.

²¹. Hulley SB, Rosenman RH, Bawol RD, Brand RJ. Epidemiology as a guide to clinical decisions. The association between triglyceride and coronary heart disease. N Engl J Med 1980;302:1383-1389.

22. Garfinkel L. Cigarette smoking and coronary heart disease in blacks: comparison to whites in a prospective study. Am Heart J 1984;108:802-807.

23. Robertson WB, Strong JP. Atheroscleroisis in persons with hypertension and diabetes mellitus. Lab Invest 1968;18:538-551.

24. Bjorntorp P. Obesity and the risk of cardiovascular disease. Ann Clin Res 1985;17:3-9.

25. Castelli WP, Garrison RJ, Wilson PWF, Abbott RD, Kalousidian S, Kannel WB. Incidence of coronary heart disease and lipoprotein cholesterol levels. The Framingham Study. JAMA 1986;256:2835-2838.

26. Lowering blood cholesterol to prevent heart disease. Consensus conference. JAMA 1985;253:2080-2086.27. Heyden S, Heiss G, Flames CG, Bartel AG. Fasting triglycerides as predictors of total and CHD mortality in Evans County, Georgia. J Chron Dis 1980;33:275-282.

²⁸ Cambien F, Jacqueson A, Richard JL, Warnet JM, Ducimetiere P, Claude JR. Is the level of serum triglyceride a significant predictor of coronary death in "normocholesterolemic" subjects? The Paris Prospective Study. Am J Epidemiol 1986;124:624-632.

²⁹ Sniderman AD, Wolfson C, Teng B, Franklin FA, Bachorik PS, Kwiterovich PO. Association of hyperapobetalipoproteinemia with endogenous hypertriglyceridemia and atherosclerosis. Ann Intern Med 1982,97:833-839.

^{30.} Oberman A, Cutter G. Issues in the natural history and treatment of coronary heart disease in black populations: surgical treatment. Am Heart J 1984;108:688-694.

³¹. Wilson PWF, Savage DD, Castelli WP, Garrison RJ, Donahue RP, Feinleib M. HDL-cholesterol in a sample of black adults: The Framingham Minority Study. Metabolism 1983;32:328-332.