Cardiovascular Risk Reduction among African Americans: A Call to Action

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African Americans are at greater risk for cardiovascular morbidity and mortality than European Americans or Asians. They also bear a disproportionately greater burden from type-2 diabetes mellitus. Not as much access to healthcare and less intensive use of available therapies may explain some of these disparities. However, the high prevalence of potentially modifiable risk factors, particularly hypertension and dyslipidemia, in African Americans also provides great opportunity for the prevention and treatment of cardiovascular disease in this population. In addition to lifestyle approaches, achieving aggressive goals for blood pressure (≤130/80 mmHg) and low-density lipoprotein cholesterol (<100 mg/dL, or <70 mg/dL for patients at very high cardiovascular risk, including those with diabetes) will necessitate the use of effective pharmacologic therapies. Clinical trial data indicate that antihypertensive regimens, particularly those that include a diuretic, are as effective in African Americans as in other racial/ethnic groups. Moreover, potent statins have been shown to decrease low-density lipoprotein cholesterol to goal levels in African-American patients.

Key words: African Americans ■ cardiovascular disease ■ diabetes mellitus ■ hypertension ■ statins

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Heart disease is the leading cause of death among African Americans, accounting for 27% of total deaths in 2004.[1] Although heart disease is also the leading cause of death in European Americans, there appears to be a marked disparity between racial/ethnic groups in coronary heart disease (CHD) mortality rates. In 2001, for example, the proportion of premature deaths (<65 years) from heart disease was much greater among African Americans (31.5%) than European Americans (14.7).[2] Compared with other racial/ethnic groups in the United States, African Americans have the highest out-of-hospital coronary death rates, particularly at younger ages, indicating earlier onset of cardiovascular disease (CVD).[3-5] Moreover, while mortality rates from CHD have declined overall in the past five decades, disparities by race/ethnicity and socioeconomic status have widened.[5,6]

The reasons for excess CHD risk in African Americans are complex and have not been fully elucidated. Both sociologic and physiologic explanations have been postulated. The former include public health issues, such as limited or not as much access to healthcare for African Americans, inadequate treatment and delays in the recognition and treatment of high-risk individuals.[7,8] The increased burden of CHD among African Americans may also reflect the contribution of underlying genetic factors that, in combination with environmental factors, lead to higher rates of CVD risk factors such as hypertension, dyslipidemia and diabetes mellitus (Table 1).[3,7,9]

GENETIC AND PHYSIOLOGIC FACTORS

Data suggest that different physiologic mechanisms may come into play in the development of CVD in African Americans.

Subclinical and Peripheral Vascular Disease

Racial/ethnic disparities in subclinical atherosclerosis across vascular beds were found in subjects without a prior diagnosis of atherosclerotic disease, including those with diabetes. While African Americans had not as much coronary calcification, they had more peripheral arterial disease, as evidenced by a lower ankle-brachial index, compared with European Americans.[10-12] This finding is consistent with National Health and Nutrition Examination Survey (NHANES) data, showing that peripheral arterial disease disproportionately affects African Americans.[13] The reasons for these differences and their clinical implications are not clear, but some investigators have suggested that coronary calcification may not have the same prognostic value for African Americans as for European Americans.[11]

Obesity

Although rates of obesity are similar in African-American and European-American men, they are two-fold higher in African-American than European American women.[7] Since visceral adiposity, as measured by waist circumference, is considered the most metabolically active form of fat,[14] the increased waist circumference in African-American women compared with women of other racial/ethnic groups likely contributes to their increased CHD risk.[14]

Hypertension

In the 10 years between 1990 and 2000, the prevalence of hypertension increased across all racial/ethnic groups.[15] However, hypertension rates remain substantially higher among African Americans. By 2002, data from NHANES showed that the age-adjusted prevalence was 40.5% among non-Hispanic blacks compared with 27.4% among non-Hispanic whites.[15] In addition, hypertension is more severe, develops at a younger age and is associated with greater clinical sequelae in African Americans than in European Americans.[9,16] For example, hypertension in African Americans is associated with a 3–5 times higher cardiovascular mortality rate than in European Americans.[7] High blood pressure is a particularly strong predictor of CHD in African-American women, whose risk is increased almost five-fold compared with a two-fold increased risk in African American men.[17] African Americans also experience greater rates of renal damage for any level of blood pressure compared with European Americans, and the risk of hypertensive end-stage renal disease (ESRD) is almost five times higher in African Americans, even when differences in hypertension prevalence, severity and age of onset are taken into account.[7,18] Indeed, in African Americans aged 30–39, the rate of hypertensive ESRD is 15 times greater than in European Americans in the same age group.[19]

Dyslipidemia

Approximately 25% of adult African Americans have high-risk lipid profiles.[7] Total cholesterol levels are >240 mg/dL in 27% of African-American men and 39% of African American women.[20] Furthermore, while the prevalence of elevated total cholesterol, low-density lipoprotein (LDL) cholesterol and triglycerides in this population is similar to or lower than that in European Americans, lipoprotein(a) levels are 2–3 times higher in African Americans than in European Americans.[7] In addition, although African Americans, particularly men, have higher levels of high-density lipoprotein (HDL) cholesterol than European Americans, these levels do not appear to be as cardioprotective as in other populations.[20]

It is possible that what may appear to be lower rates of dyslipidemia in African Americans merely indicate that the thresholds at which elevated triglycerides and LDL cholesterol and low levels of HDL cholesterol contribute to CHD risk may be lower for this group than for other racial/ethnic groups.[21] In addition, because African Americans are less likely to be treated and controlled to goal lipid levels, the impact of dyslipidemia in this population may, in fact, be greater than in European Americans.[8]

Table 1. Possible reasons for disproportionate coronary heart disease (CHD) in African Americans

- High prevalence of CHD risk factors
- Pre-eminence of hypertension and its consequences
- Excess cardiovascular risk associated with diabetes mellitus
- Heterogeneity of acute coronary syndromes
- Delays in identification of high-risk individuals
- Limited access to cardiovascular care

Adapted from Clark LT, et al.[7]

Metabolic Syndrome

Beyond the increased frequency of individual risk factors in African Americans, this group also experiences more frequent clustering of CVD risk factors (Figure 1).[22,23] NHANES data indicate that African Americans are twice as likely as other groups to have four or five CVD risk factors.[23] Metabolic syndrome, which represents a clustering of specific cardiovascular risk factors [abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, insulin resistance, elevated fasting glucose, as defined by the National Cholesterol Education Program (NCEP)[3]], is more common in African-American women than in European-American women and is about 57% higher in prevalence in African-American women than in their male counterparts.[24]

NCEP criteria for the metabolic syndrome may not adequately take into account the differences among racial and ethnic subpopulations that have been identified in recent research. For example, increased waist circumference, considered a strong predictive screening factor in metabolic syndrome,[25] can be found more often in African Americans with body mass index values considered normal,[14,26] and African-American women with normal body weight are more likely to have a waist circumference greater than the 35-inch cutoff considered normal.[26] These groups may therefore be at greater risk than is suggested by current guidelines. Moreover, a waist circumference greater than that considered within the normal range by NCEP is likely to be atherogenic in European Americans, but it is more likely to indicate risk for diabetes in African Americans.[27] The racial/ethnic disparities in metabolic and cardiovascular parameters are already evident in childhood and young adulthood, adding further insight into the causes of elevated risk of CHD in adult African Americans and the finding

that CHD develops at earlier ages in this group.[20]

Diabetes Mellitus

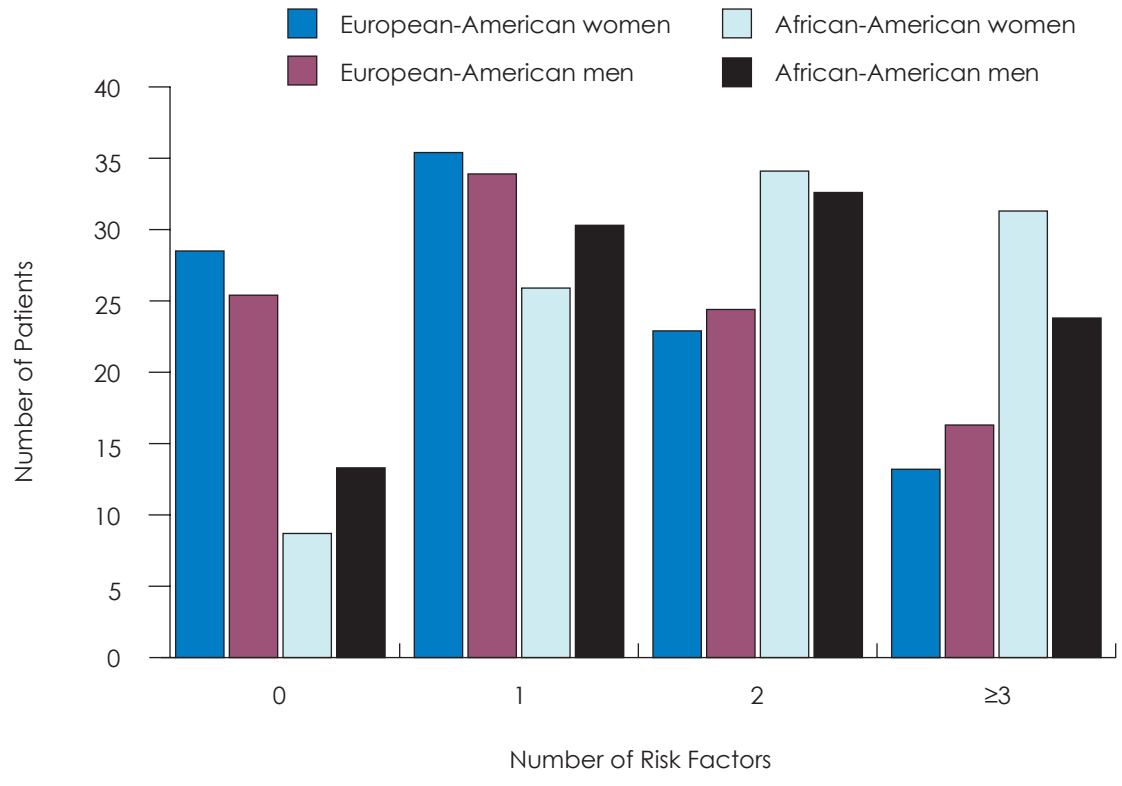
One of the most significant contributors to the elevated CHD risk in African Americans is the higher incidence of type-2 diabetes mellitus in this population.[21] Diabetes is one of the most powerful risk factors for heart disease and has been designated as a CHD "risk equivalent" in the Third Adult Treatment Panel guidelines of the NCEP (NCEP ATP III).[3] Approximately 70% of deaths in patients with diabetes are related to heart disease; CVD also accounts for the vast majority of hospitalizations in these patients.[28] Data from both the Atherosclerosis Risk in Communities (ARIC) study and NHANES III show that African Americans bear a disproportionately higher burden from type-2 diabetes.[29,30] In ARIC, the incidence of diabetes was 1.5 times higher in African-American men and 2.4 times higher in African-American women compared with their European American counterparts.[29] Adiposity accounted for almost half the increased risk of diabetes in African-American women, but not in African-American men.[29]

Mortality rates among persons with diabetes are 27% higher in African Americans than in European Americans.[28] African Americans also have significantly higher rates of complications from diabetes, including myocardial infarction and ESRD, and numerically higher rates of stroke and congestive heart failure, compared with European Americans.[31] Based on data from 2004, rates of diabetic ESRD in African Americans have begun to stabilize in patients aged 20–29 but continue to rise in those aged 30–39.[19]

Many CVD risk factors, including elevated blood pressure, overweight/obesity and dyslipidemia, are more common in patients with diabetes,[32] and many of these risk factors are more common in diabetic African Americans than in diabetic European Americans. For example, both mean systolic and diastolic blood pressure and the rate of hypertension are higher in African Americans with diabetes than in European Americans with diabetes.[21,33]

Between 50–70% of African Americans with type-2 diabetes are insulin resistant, which has been shown to negatively alter lipid and lipoprotein profiles.[34,35] LDL cholesterol levels also tend to increase with increasing body mass index in African Americans with type-2 diabetes.[36] Although serum triglyceride levels appear to be lower in African Americans with diabetes than in European Americans with diabetes,[33] African Americans with

Figure 1. Percentage of subjects in the Atherosclerosis Risk in Communities study with 0, 1, 2 or ≥3 cardiovascular risk factors (obesity, hypertension, diabetes mellitus, smoking, hypercholesterolemia, hypertriglyceridemia or low high-density lipoprotein cholesterol)



Adapted from Hutchinson RG, et al.[22]

type-2 diabetes who are insulin resistant have higher total cholesterol, LDL cholesterol and triglycerides than those who are insulin sensitive.[36,37]

OPPORTUNITIES FOR INTERVENTION

The high prevalence of potentially modifiable risk factors provides great opportunities for prevention of CHD in African Americans. Control of cardiovascular risk factors is particularly important and particularly challenging in patients with diabetes. In addition to tight glycemic control, it is crucial that blood pressure and lipid goals be met in order to provide optimal global risk reduction.

Hypertension

Treatment of hypertension entails unique challenges in persons with diabetes. Blood pressure is disproportionately higher in patients with diabetes than in nondiabetic persons.[16] In addition, overall CVD risk is higher, so both the risks associated with hypertension and the benefits afforded by optimal blood pressure control have more clinical impact in patients with diabetes. A number of position papers on the treatment of hypertension in diabetic patients are available to guide the clinician. The most widely recognized of these are the 7th Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7),[16] the American Diabetes Association position statement on treatment of hypertension in adults with diabetes[38] and the consensus statement of the National Kidney Foundation Hypertension and Diabetes Executive Committees Working Group.[39] In addition, the Hypertension in African Americans Working Group of the International Society of Hypertension in Blacks has published a consensus statement on the management of high blood pressure in African Americans.[40] These diverse groups are all consistent in recommending a blood pressure goal of <130/80 mmHg for patients with diabetes, including African Americans.[16,38-40] Despite the higher rates of CVD morbidity and mortality in African Americans, no clinical trial data suggest that blood pressure goals should be lower in this population than in other racial/ethnic groups.[40]

Table 2. Therapeutic lifestyle changes to control high blood pressure in African Americans

- Normal weight for height
- Dietary goals:
  - Low fat
  - Low sodium
  - High potassium
  - Adequate calcium
- Limit alcohol
- Physical fitness
- No tobacco use

Adapted from Douglas JG, et al.[40]

Lifestyle modification remains the cornerstone of hypertension treatment; and obesity, high sodium intake and physical inactivity have been identified as particular obstacles to cardiovascular health in African Americans (Table 2).[40] Of note, it has also been demonstrated that the Dietary Approaches to Stop Hypertension (DASH) diet is particularly beneficial in African Americans with hypertension, reducing blood pressure substantially more than in non-Hispanic whites.[41] The added benefit of restricting sodium intake with the DASH diet, which consistently reduced mean blood pressure more than diet alone across all demographic groups, was also more pronounced in African Americans.[42,43]

Regarding drug therapy, most patients require combination therapy with ≥2 antihypertensive agents to achieve goal blood pressure.[40] This fact has also been confirmed in clinical trials of hypertension treatment exclusively enrolling African Americans, such as the African American Study of Kidney Disease (AASK).[40] However, the response to individual antihypertensive agents may vary among ethnic groups. For example, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) in monotherapy appear to be slightly less effective in African Americans than in European Americans.[16] Adding a diuretic to any of these agents, however, generally abolishes any interracial differences in blood pressure-lowering effects. Beyond particular efficacy in reducing blood pressure, certain antihypertensive drug classes have been shown to provide benefits to patients with diabetes in terms of outcome reductions. These include diuretics, beta-blockers, ACE inhibitors, ARBs and calcium channel blockers.[16] In general, clinical trial data have shown that the benefits of antihypertensive therapy in reducing the risk of myocardial infarction, stroke and chronic kidney disease extend to patients with type-2 diabetes and hypertension.[44]

JNC 7 created a new classification of "prehypertension" for blood pressures between 120/80 and 139/89 mmHg, because this range identifies persons who are at high risk of developing hypertension.[16] The inclusion of a prehypertension category is particularly important for African Americans and other racial/ethnic groups with high rates of diabetes, as JNC 7 now recommends that antihypertensive drug treatment be considered in persons with diabetes and prehypertension if a trial of lifestyle modification fails to lower blood pressure to ≤130/80 mmHg.[16]

Dyslipidemia

Dyslipidemia is highly prevalent and largely untreated in persons with hypertension.[45] The dyslipidemia associated with diabetes is particularly atherogenic and typically consists of the combination of elevated triglycerides; small, dense LDL particles; and low HDL cholesterol.[44]

Overall, individuals with diabetes may have normal-appearing levels of LDL cholesterol, but these apparently normal levels (e.g., <100 mg/dL) may be associated with an increased CVD risk in this group.[46] The most common pattern of dyslipidemia among both African Americans and European Americans with diabetes, as reported in a recent study, is elevated levels of LDL cholesterol together with low levels of HDL cholesterol.[47] Thus, in high-risk patients, including those with diabetes, LDL cholesterol reduction remains the first priority of lipid management.[46] Moreover, although the goal of reducing LDL cholesterol levels to <100 mg/dL is strongly recommended, the NCEP provides the option of lowering levels even more aggressively (i.e., to <70 mg/dL) in high-risk patients.[46] For patients deemed to be at very high risk (e.g., because of the presence of established CHD and diabetes), a goal of <70 mg/dL should be considered.[46]

Although diet, weight loss and increased physical activity are critical to overall treatment in persons with diabetes and dyslipidemia, many patients will not be able to reach optimal LDL cholesterol levels without intensive pharmacologic therapy.[46] Clinical trials have shown LDL cholesterol-lowering therapy to be effective in persons with diabetes,[48-51] and patients with the highest CVD risk obtained the greatest benefit in terms of absolute risk reduction.[46,49,50] Because of their documented efficacy in lowering LDL cholesterol, statins are typically the agents of choice for patients with elevated LDL cholesterol and triglycerides.[3] The standard doses of currently available statins generally needed to lower LDL cholesterol levels by 30–40% are shown in Table 3.[52-57]

Data on differences in response to lipid-lowering therapies based on race/ethnicity are limited. While statins have been shown to be effective in African Americans,[58,59] mean LDL cholesterol reductions may be somewhat smaller (~3 mg/dL less) than those observed in European Americans.[59,60] In a study in which 247 African-American patients were randomized to ezetimibe plus simvastatin or placebo, the combined therapy proved to be both effective and well tolerated and provided greater improvement in atherogenic lipid profiles than simvastatin alone.[61] In another study, data from the NCEP Evaluation Project T Utilizing Novel E-Technology (NEPTUNE) II survey were used to assess ethnic differences in LDL cholesterol goal achievement among 4,885 patients receiving treatment for dyslipidemia.[62] Non-Hispanic whites comprised 79.7% of subjects and African Americans 8.4%; of these groups, 69% and 53.7%, respectively, achieved their LDL cholesterol goal (P<0.001). African-American patients were more likely to be in the highest-risk category. Despite this, they were less likely to be using lipid drug therapy or high-efficacy statins or to be receiving care from a subspecialist. However, the difference in goal achievement remained significant after adjustment for these predictors of treatment success (P<0.001).[62]

In the African-American Rosuvastatin Investigation of Efficacy and Safety (ARIES) study, aggressive statin treatment with open-label rosuvastatin was effective and well tolerated in hypercholesterolemic African-American adults.[59] Similar to populations of predominantly European-American patients with hypercholesterolemia,[63] African Americans in ARIES showed greater overall improvement in their lipid profile with rosuvastatin 10 mg or 20 mg than with equivalent doses of atorvastatin. In addition, greater proportions of patients achieved their LDL cholesterol goal on rosuvastatin than on atorvastatin (Figure 2).[59] Rosuvastatin 10 mg also increased HDL cholesterol significantly more than atorvastatin 20 mg (P<0.017).

In the lipid-lowering arm of the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT-LLT),[64] the only large-scale, randomized trial of statin therapy to enroll large numbers of African Americans, pravastatin lowered the cardiovascular event rate (fatal CHD plus nonfatal myocardial infarction) more effectively in African Americans than in non-African Americans, but overall mortality did not differ between groups.

"RACE-BASED THERAPEUTICS"

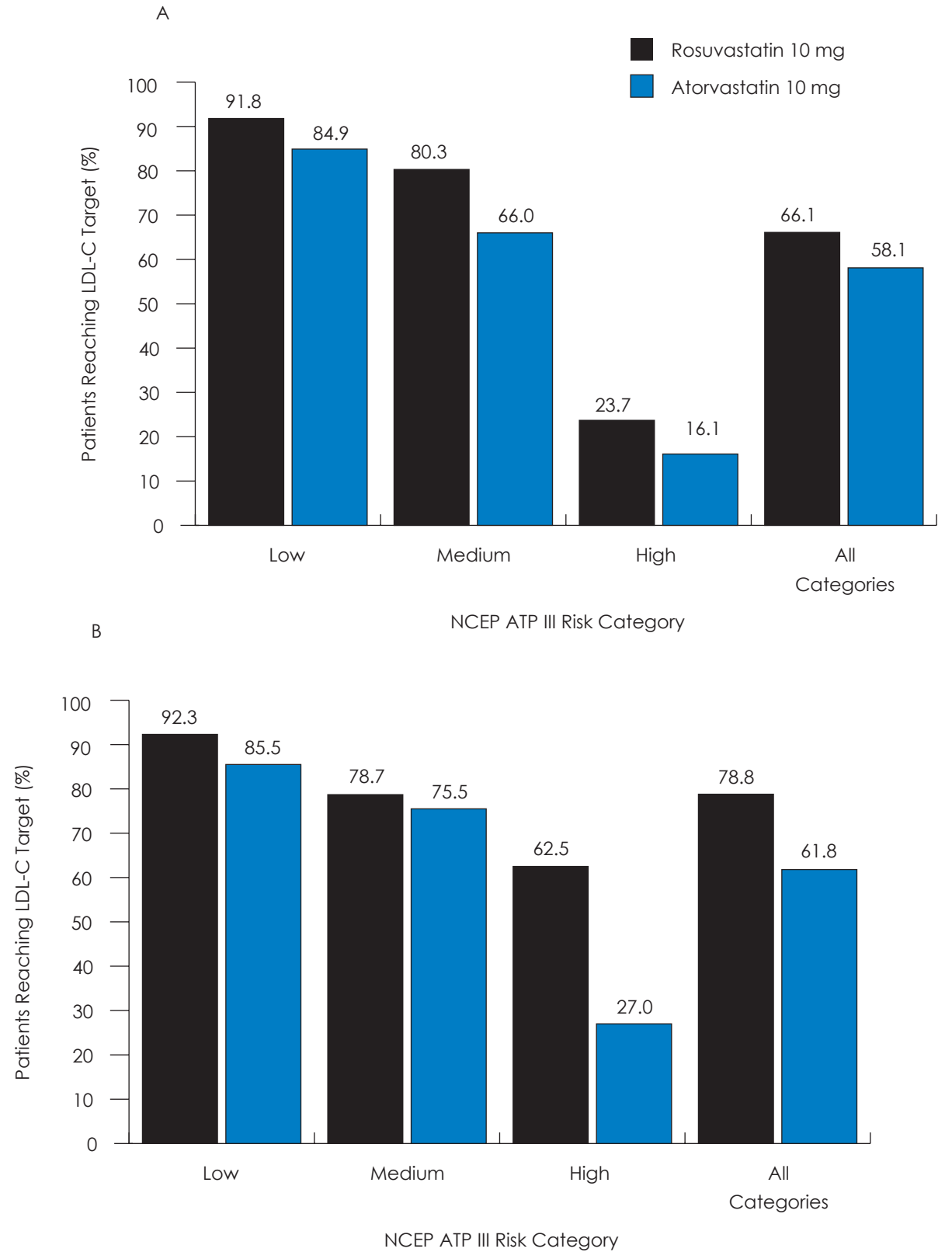
The development and use of so-called "race-based therapeutics" remains controversial. Results of some clinical trials indicate that racial/ethnic differences in vascular function may have implications for the treatment of CVD risk factors. African-American patients

Table 3. Standard doses of currently available statins that can achieve an approximate 30–40% reduction of low-density lipoprotein (LDL) cholesterol

Drug	Dose (mg/d)	LDL Cholesterol Reduction (%)
Atorvastatin	10*	39
Lovastatin	40*	31
Pravastatin	40*	34
Simvastatin	20–40*	38–41
Fluvastatin	40–80	25–36
Rosuvastatin	5–10†	45–52

* These drugs are available at doses up to 80 mg. Every doubling of the dose above the standard dose results in an approximate 6% additional decrease in LDL cholesterol; † Doses available up to 40 mg; Adapted from prescribing information for each statin.[52-57]

Figure 2. Percentage of patients treated with either rosuvastatin or atorvastatin who reached National Cholesterol Education Program (NCEP) ATP III low-density lipoprotein cholesterol (LDL-C) goals for low-risk (<160 mg/dL), medium-risk (<130 mg/dL) and high-risk (<100 mg/dL) patients and for all categories combined



Adapted from Ferdinand KC, et al.[59]

with diabetes and persistent microalbuminuria despite therapy with ACE inhibitors had significantly impaired flow-mediated and nitroglycerine-dependent vasodilation, independent of glycemic control, blood pressure and lipemic control.[65] Flow-mediated dilation was also significantly less than that in a group of age- and blood pressure-matched European Americans. There is also some evidence that race may affect the contribution of bradykinin to the antihypertensive actions of ACE inhibitors, at least under conditions of normal salt intake.[66] When salt is depleted, the response to vasodilator therapy with ACE inhibitors is similar among African Americans and European Americans.[66] Thus, African Americans may have a different endothelial response to ACE inhibition than European Americans.

Interestingly, individual response to the pleiotropic effects of statins, such as their beneficial effects on renal function independent of lipid lowering, may also be affected by race. In one study of short-term rosuvastatin treatment, estimated glomerular filtration rate increased by >3-fold in African-American patients compared with the overall study population.[67] The fact that African Americans and European Americans appear to exhibit differences in endothelial and vessel wall response suggests that alternative strategies may be needed to customize therapy appropriately for patients of different races/ethnicities. Recently, a proprietary combination of isosorbide dinitrate and hydralazine hydrochloride was approved for use in African Americans with heart failure based on results of the African-American Heart Failure Trial.[68] However, it is possible that this drug combination may work equally well in other racial/ethnic groups. More research in the area is needed.

SOCIOLOGIC ASPECTS OF CVD

Available data show that a gap exists in the United States in cardiovascular mortality between the poor and undereducated, and the wealthy and well educated, and that this gap continues to widen.[5] In the Multiple Risk Factor Intervention Trial (MRFIT) database, income and risk-factor levels differ substantially according to ethnicity, with greater concentrations of African-American men at the high and very high overall risk levels and in the lowest income quartile.[69] Low income may be associated with decreased access to or use of preventive healthcare services—resulting in a lack of awareness of risk and inability to modify health behaviors[70]—or with psychosocial factors, such as detrimental healthcare beliefs or behaviors.[71] Public health interventions that would promote healthful behaviors and make high-quality diagnostic and preventive services available to disadvantaged populations could help reduce the potentially lethal results of existing inequalities in healthcare.

CONCLUSION

Persons with diabetes are at higher risk for cardiovascular events than those without diabetes, and African Americans with diabetes have more severe manifestations than their European-American counterparts. The combination of multiple risk factors and less intensive use of available therapies may explain higher levels of morbidity and mortality in African Americans. For African Americans, achieving goal LDL cholesterol and blood pressure levels presents unique challenges. Despite the fact that treatment of dyslipidemia with statins and optimal control of blood pressure with antihypertensive therapy have proven to be highly effective in African Americans, African-American patients with dyslipidemia remain less likely to be treated with lipid-lowering drugs than European-American patients. Concerted efforts directed at minority populations to use targeted interventions and effective therapies designed to reach aggressive treatment goals may go a long way to closing the gap in cardiovascular health.

REFERENCES

1. National Center for Health Statistics. Health, United States, 2006 with Chartbook on Trends in the Health of Americans. Hyattsville, MD; 2006.

2. Centers for Disease Control and Prevention. Disparities in premature deaths from heart disease: 50 states and the District of Columbia, 2001. *Morb Mortal Wkly Rep*. 2004;53:121-125.

3. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. 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*. 2001;285:2486-2497.

4. Barnett E, Halverson J. Disparities in premature coronary heart disease mortality by region and urbanicity among black and white adults ages 35-64, 1985-1995. *Public Health Rep*. 2000;115:52-64.

5. Cooper R, Cutler J, Desvigne-Nickens P, et al. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: findings of the National Conference on Cardiovascular Disease Prevention. *Circulation*. 2000;102:3137-3147.

6. Centers for Disease Control and Prevention. Decline in deaths from heart disease and stroke: United States, 1900–1999. *Morb Mortal Wkly Rep*. 1999;48:649-656.

7. Clark LT, Ferdinand KC, Flack JM, et al. Coronary heart disease in African Americans. *Heart Dis*. 2001;3:97-108.

8. Goff DC Jr, Bertoni AG, Kramer H, et al. Dyslipidemia prevalence, treatment, and control in the Multi-Ethnic Study of Atherosclerosis (MESA): gender, ethnicity, and coronary artery calcium. *Circulation*. 2006;113:647-656.

9. Smith SC Jr, Clark LT, Cooper RS, et al. Discovering the full spectrum of cardiovascular disease. Minority Health Summit 2003: report of the Obesity, Metabolic Syndrome, and Hypertension Writing Group. *Circulation*. 2005;111:e134-e139.

10. Carnethon MR, Bertoni AG, Shea S, et al. Racial/ethnic differences in subclinical atherosclerosis among adults with diabetes. The Multiethnic Study of Atherosclerosis. *Diabetes Care*. 2005;28:2768-2770.

11. Bild DE, Detrano R, Peterson D, et al. Ethnic differences in coronary calcification. The Multi-Ethnic Study of Atherosclerosis (MESA). *Circulation*. 2005;111:1313-1320.

12. Budoff MJ, Nasir K, Mao S, et al. Ethnic differences of the presence and severity of coronary atherosclerosis. *Atherosclerosis*. 2006;187:343-350.

13. Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999-2000. *Circulation*. 2004;110:738-743.

14. Okosun IS, Choi ST, Boltri JM, et al. Trends in abdominal adiposity in white, black, and Mexican-American adults, 1988-2000. *Obes Res*. 2003;11:1010-1017.

15. Centers for Disease Control and Prevention. Racial/ethnic disparities in prevalence, treatment, and control of hypertension: United States, 1999–

2002. *Morb Mortal Wkly Rep*. 2005;54:7-9.

16. Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension.* 2003;42:1206-1252.

17. Jones DW, Chambless LE, Folsom AR, et al. Risk factors for coronary heart disease in African Americans: the Atherosclerosis Risk in Communities Study, 1987-1997. *Arch Intern Med*. 2002;162:2565-2571.

18. Whittle JC, Whelton PK, Seidler AJ, et al. Does racial variation in risk factors explain black-white differences in the incidence of hypertensive end-stage renal disease? *Arch Intern Med*. 1991;151:1359-1364.

19. U.S. Renal Data System. Excerpts from the USRDS 2006 Annual Data Report. *Am J Kidney Dis*. 2007;49:S1-S296.

20. Bonow RO, Bohannon N, Hazzard W. Risk stratification in coronary artery disease and special populations. *Am J Med*. 1996;101(suppl 4A):417S-424S.

21. Hall, WD, Clark LT, Wanger NK, et al. The metabolic syndrome in African Americans: a review. *Ethn Dis*. 2003;13:414-428.

22. Hutchinson RG, Watson RL, Davis CE, et al, for the ARIC Study Group. Racial differences in risk factors for atherosclerosis: the ARIC study. *Angiology*. 1997;48:279-290.

23. Sharma S, Malarcher AM, Giles WH, et al. Racial, ethnic and socioeconomic disparities in the clustering of cardiovascular disease risk factors. *Ethn Dis*. 2004;14:43-48.

24. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Nutritional Examination Survey. *JAMA*. 2002;287:356-359.

25. Palaniappan L, Carnethon MR, Wang Y, et al, for the Insulin Resistance Atherosclerosis Study. Predictors of the incident metabolic syndrome in adults: the Insulin Resistance Atherosclerosis Study. *Diabetes Care*. 2004;27:788-793.

26. Patt MR, Yanek LR, Moy TF, et al. Assessment of global coronary heart disease risk in overweight and obese African-American women. *Obes Res*. 2003;11:660-667.

27. Bacha F, Saad R, Gungor N, et al. Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: race differential in diabetogenic and atherogenic risk factors. *J Clin Endocrinol Metab*. 2003;88:2534-2540.

28. Gu K, Cowie CC, Harris MI. Mortality in adults with and without diabetes in a national cohort of the US population, 1971–1993. *Diabetes Care*. 1998;21:1138-1145.

29. Brancati FL, Kao WHL, Folsom AR, et al. Incident type 2 diabetes mellitus in African American and white adults. The Atherosclerosis Risk in Communities Study. *JAMA*. 2000;283:2253-2259.

30. Harris MI, Flegal KM, Cowie CC, et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: the Third National Health and Nutrition Examination Survey, 1988–1994. *Diabetes Care*. 1998;21:518-524.

31. Karter AJ, Ferrara A, Liu JY, et al. Ethnic disparities in diabetic complications in an insured population. *JAMA*. 2002;287:2519-2527.

32. Harris MI. Health care and health status and outcomes for patients with type 2 diabetes. *Diabetes Care*. 2000;23:754-758.

33. Sharma MD, Pavlik VN. Dyslipidaemia in African Americans, Hispanics, and whites with type 2 diabetes mellitus and hypertension. *Diabetes Obes Metab*. 2001;3:41-45.

34. Chaiken RL, Banerji MA, Huey H, et al. Do blacks with NIDDM have an insulin-resistant syndrome? *Diabetes*. 1993;42:444-449.

35. Banerji MA, Lebovitz HE. Insulin action in black Americans with NIDDM. *Diabetes Care*. 1992;15:1295-1302.

36. Chaiken RL, Banerji MA, Pasmantier R, et al. Patterns of glucose and lipid abnormalities in black NIDDM subjects. *Diabetes Care*. 1991;14:1036-1042.

37. Banerji MA, Lebovitz HE. Coronary heart disease risk factor profiles in black patients with non-insulin-dependent diabetes mellitus: paradoxic patterns. *Am J Med*. 1991;91:51-58.

38. American Diabetes Association. Hypertension management in adults with diabetes. *Diabetes Care*. 2004;27(suppl 1):S65-S67.

39. Bakris GL, Williams M, Dworkin L, et al, for the National Kidney Foundation Hypertension and Diabetes Executive Committees Working Group. Preserving renal function in adults with hypertension and diabetes: a consensus approach. *Am J Kidney Dis*. 2000;36:646-661.

40. Douglas JG, Bakris GL, Epstein M, et al. Management of high blood pressure in African Americans: consensus statement of the Hypertension in African Americans Working Group of the International Society on Hypertension in Blacks. *Arch Intern Med*. 2003;163:525-541.

41. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med*. 1997;336:1117-1124.

42. Sacks FM, Svetkey LP, Vollmer WM, et al, for the DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med*. 2001;344:3-10.

43. Vollmer WM, Sacks FM, Ard J, et al. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-Sodium Trial. *Ann Intern Med*. 2001;135:1019-1028.

44. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/ National Heart, Lung, and Blood Institute Scientific Statement. *Circulation*. 2005;112:2735-2752.

45. O'Meara JG, Kardia SL, Armon JJ, et al. Ethnic and sex differences in the prevalence, treatment, and contril of dyslipidemia among hypertensive adults in the GENOA study. *Arch Intern Med*. 2004;164:13130-1318.

46. Grundy SM, Cleeman JI, Bairey Merz CN, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation*. 2004;110:227-239.

47. Cook CB, Erdman DM, Ryan GJ, et al. The pattern of dyslipidemia among urban African-Americans with type 2 diabetes. *Diabetes Care*. 2000;23:319-324.

48. Haffner SM. The Scandinavian Simvastatin Survival Study (4S) subgroup analysis of diabetic subjects: implications for the prevention of coronary heart disease. *Diabetes Care*. 1997;20:469-471.

49. Goldberg RB, Mellies MJ, Sacks FM, et al. 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. *Circulation.* 1998;98:2513-2519.

50. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol-lowering with simvastatin in 5963 people with diabetes: a randomised placebo-controlled trial. *Lancet*. 2003;361:2005-2016.

51. Colhoun HM, Betteridge DJ, Durrington PN, et al, on behalf of the CARDS investigators. Primary prevention of cardiovascular disease with atorvastatin in type 2 diabetes in the Collaborative Atorvastatin Diabetes Study (CARDS): multicentre randomised placebo-controlled trial. *Lancet*. 2004;364:685-696.

52. Lipitor (atorvastatin calcium) [package insert]. New York, NY: Pfizer; September 2005.

53. Mevacor (lovastatin) [package insert]. Whitehouse Station, NJ: Merck & Co; November 2004.

54. Pravachol (pravastatin sodium) [package insert]. Princeton, NJ: Bristol-Myers Squibb; December 2004.

55. Zocor (simvastatin) [package insert]. Whitehouse Station, NJ: Merck & Co; November 2004.

56. Lescol (fluvastatin) [package insert]. East Hanover, NJ: Novartis; April 2006.

57. Crestor (rosuvastatin calcium) [package insert]. Wilmington, DE: AstraZeneca; January 2005.

58. Jacobson TA, Chin MM, Curry CL, et al. Efficacy and safety of pravastatin in African Americans with primary hypercholesterolemia. *Arch Intern Med*. 1995;155:1900-1906.

59. Ferdinand KC, Clark LT, Watson KE, et al, for the ARIES Study Group. Comparison of efficacy and safety of rosuvastatin versus atorvastatin in African-American patients in a six-week trial. *Am J Cardiol*. 2006;97:229-235.

60. Simon JA, Lin F, Hulley SB, et al. Phenotypic predictors of response to simvastatin therapy among African-Americans and Caucasians: the Cholesterol and Pharmacogenetics (CAP) study. *Am J Cardiol*. 2006;97:843-850.

61. Rodney RA, Sugimoto D, Wagman B, et al. Efficacy and safety of coad-

ministration of ezetimibe and simvastatin in African-American patients with primary hypercholesterolemia. *J Natl Med Assoc*. 2006;98:772-778.

62. Clark LT, Maki KC, Galant R, et al. Ethnic differences in achievement of cholesterol treatment goals. Results from the National Cholesterol Education Program Evaluation Project Utilizing Novel E-Technology II. *J Gen Intern Med*. 2006;21:320-326.

63. Jones PH, Davidson MH, Stein EA, et al. Comparison of the efficacy and safety of rosuvastatin versus atorvastatin, simvastatin, and pravastatin across doses (STELLAR Trial). *Am J Cardiol*. 2003;92:152-160.

64. The ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. Major outcomes in moderately hypercholesterolemic, hypertensive patients randomized to pravastatin vs usual care: the Antihypertensive and Lipid-Lowering to Prevent Heart Attack Trial (ALLHAT-LLT). *JAMA*. 2002;288:2998-3007.

65. Jawa A, Nachimuthu S, Pendergrass M, et al. Impaired vascular reactivity in African-American patients with type 2 diabetes mellitus and microalbuminuria or proteinuria despite angiotensin-converting enzyme inhibitor therapy. *J Clin Endocrinol Metab*. 2006;91:31-35.

66. Gainer JV, Morrow JD, Loveland A, et al. Effect of bradykinin-receptor blockade on the response to angiotensin-converting-enzyme inhibitor in normotensive and hypertensive subjects. *N Engl J Med*. 1998;339:1285-1292.

67. Vidt DG, Harris S, McTaggart F, et al. Effect of short-term rosuvastatin treatment on estimated glomerular filtration rate. *Am J Cardiol*. 2006;97:1602-1606.

68. Ferdinand KC. Isosorbide dinitrate and hydralazine hydrochloride: a review of efficacy and safety. *Expert Rev Cardiovasc Ther*. 2005;3:993-1001.

69. Thomas AJ, Eberly LE, Smith GD, et al, for the Multiple Risk Factor Intervention Trial Research Group. Race/ethnicity, income, major risk factors, and cardiovascular disease mortality. *Am J Public Health*. 2005;95:1417-1423.

70. Fiscella K, Franks P, Gold M, et al. Inequality in quality: addressing socioeconomic, racial, and ethnic disparities in health care. *JAMA*. 2000;289:2579-2584.

71. Strike PC, Steptoe A. Psychosocial factors in the development of coronary artery disease. *Prog Cardiovasc Dis*. 2004;46:337-347. ■

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