

# Circulation

JOURNAL OF THE AMERICAN HEART ASSOCIATION



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*Circulation* 2006;113:2943-2946

DOI: 10.1161/CIRCULATIONAHA.106.176583

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 72514

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## Preventing Cardiovascular Disease and Diabetes A Call to Action From the American Diabetes Association and the American Heart Association

Robert H. Eckel, MD; Richard Kahn, PhD; Rose Marie Robertson, MD; Robert A. Rizza, MD

Excess body weight has become a major public health problem in the U.S., with nearly two-thirds of adults either overweight or obese.<sup>1</sup> The steady gain in the prevalence of obesity over the last 25 years has affected our entire population—no racial or ethnic group, no region of the country, and no socioeconomic group has been spared.<sup>2</sup> Perhaps most worrisome is the observation that the rise in the rate of obesity has been greatest in children and minorities, which suggests that future generations of Americans, and our fastest growing populations, may bear the ultimate burden of this condition.<sup>3</sup>

Overweight or obesity results in a wide range of elevated risk factors and many fatal and nonfatal conditions.<sup>4</sup> Paradoxically, although we have witnessed decades in which heart disease and stroke have steadily declined and cancer mortality has at worst remained stable,<sup>5</sup> the prevalence of diabetes has soared.<sup>6</sup> The increase in diabetes can largely be attributed to weight gain,<sup>7,8</sup> and it threatens the enormous advances in disease prevention we have seen.<sup>3,9,10</sup>

Among individuals with diabetes, cardiovascular disease (CVD) is the leading cause of morbidity and mortality<sup>9,11</sup>; adults with diabetes have a two- to fourfold higher risk of CVD compared with those without diabetes.<sup>12,13</sup> Diabetes is also accompanied by a significantly increased prevalence of hypertension and dyslipidemia.<sup>14</sup>

It is reasonable to postulate that in many individuals, excess weight gives rise to diabetes, hypertension, and dyslipidemia, thereby leading to frank CVD.<sup>15–17</sup> This seemingly simple algorithm is undoubtedly more complex because (1) many studies show that hyperglycemia at pre-diabetic levels is an independent risk factor for CVD,<sup>18–22</sup> (2) central obesity (i.e., intra-abdominal or visceral fat) may have a greater detrimental effect than overall weight/BMI,<sup>8,23,24</sup> and (3) there is a complex relationship between lipid metabolism and hyperglycemia.<sup>25,26</sup> Moreover, obesity in the absence of glucose intolerance is associated

with CVD, including coronary heart disease, stroke, and heart failure.<sup>27</sup>

The association among diabetes, hypertension, and dyslipidemia has been known for many decades, but the seminal paper by Reaven<sup>28</sup> ascribing much of the etiology of these risk factors to insulin resistance ushered in a new era of research and awareness<sup>29</sup> and the call for a better appreciation of the impact of obesity on CVD. Also, the concept that these “metabolic” abnormalities can cluster in many individuals gave rise to the term “metabolic syndrome,” and this construct has been the subject of many thousands of publications and extensive reviews. Although the metabolic syndrome has been embraced by many individuals and organizations,<sup>29–33</sup> others have questioned its clinical utility.<sup>34–38</sup>

Unfortunately, some of the medical press have positioned the scientific issues related to the metabolic syndrome as a “battle”<sup>39,40</sup> between the American Diabetes Association and the American Heart Association, implicitly suggesting that CVD risk factor identification and treatment is now questionable. We are concerned that the presumed dispute will lead to a reduction in the favorable trend of many aspects of CVD risk factor reduction.<sup>41</sup>

The intent of this article is to clarify and reinforce the notion that our organizations remain unified and committed to reducing the burden of diabetes and CVD. The importance of identifying and treating a core set of risk factors (pre-diabetes, hypertension, dyslipidemia, and obesity) cannot be overstated, and our commitment is evidenced by other previous joint publications.<sup>42,43</sup> While unrelated to an underlying metabolic abnormality, tobacco use also deserves special attention. Moreover, since recent evidence suggests that risk assessment and adherence to national guidelines remains woefully suboptimal,<sup>44–46</sup> we call for a renewed effort to prevent and treat these conditions.

### Risk Assessment

Although there are many approaches for estimating the risk of diabetes and CVD,<sup>47–49</sup> virtually none have been validated

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This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on April 24, 2006. A single reprint is available by calling 800-242-8721 (US only) or writing the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596. Ask for reprint No. 71-0366. To purchase additional reprints: Up to 999 copies, call 800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 410-528-4121, fax 410-528-4264, or e-mail kelle.ramsay@wolterskluwer.com. To make photocopies for personal or educational use, call the Copyright Clearance Center, 978-750-8400.

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**Simultaneous publication:** This article is being simultaneously published in 2006 in *Diabetes Care* and *Circulation* by the American Diabetes Association and American Heart Association.

(*Circulation*. 2006;113:2943-2946.)

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*Circulation* is available at <http://www.circulationaha.org>

DOI: 10.1161/CIRCULATIONAHA.106.176583

much beyond the population from which they were constructed. There is one such tool, however (available free on the Internet at <http://www.diabetes.org/diabetesphd>), that has been extensively validated across many widely differing clinical trials, and it incorporates virtually all known CVD risk factors. Although it can be used to predict the risk of developing CVD/diabetes or the effects of treatment after developing diabetes/CVD, this tool and other risk-assessment algorithms are rarely used in clinical practice.

Conversely, emerging evidence suggests that simply ascertaining a person's blood glucose level, blood pressure, LDL cholesterol level, and tobacco use and noting the presence of obesity may be sufficient to initiate the appropriate interventions to prevent or identify diabetes and emerging CVD.<sup>22,24,50,51</sup> Even borderline abnormalities, especially if they are multiple, may well presage future problems and should be addressed. Certainly, a number of intriguing scientific questions remain regarding the relative impact of each risk factor, the hierarchy of risk factors, the inclusion of other risk factors, and the relationships among all of them; however, at the very least, we encourage providers to be cognizant of these key parameters.

### Risk Factor Management

Numerous studies have shown that attention to lifestyle modification can dramatically reduce progression to type 2 diabetes.<sup>52-54</sup> Weight loss of as little as 7% of body weight during the 1st year of intervention, with lesser amounts to follow, is extremely effective and well within the capability of most patients. Weight reduction also improves all cardiometabolic risk factors,<sup>55,56</sup> although there

has been no controlled clinical trial evidence documenting the effect of weight loss on CVD events. Current guidelines also recommend regular, moderate physical activity, and here too, all cardiometabolic risk factors improve with sustained physical activity.<sup>57</sup> Other strategies for the early detection and treatment of diabetes and CVD have been published by our organizations.<sup>43</sup>

### Summary

Both the American Heart Association and the American Diabetes Association remain jointly committed to a reduction in heart disease, stroke, and new-onset diabetes. We strongly recommend that all providers assess patients for their global risk for CVD and diabetes. Despite many unresolved scientific issues, a number of cardiometabolic risk factors have been clearly shown to be closely related to diabetes and CVD: fasting/postprandial hyperglycemia, overweight/obesity, elevated systolic and diastolic blood pressure, and dyslipidemia. Although pharmacologic therapy is often indicated when overt disease is detected, in the early stages of these conditions, lifestyle modification with attention to weight loss and physical activity may well be sufficient.

It must be remembered that obesity is far more than an unattractive appearance but can be prevented. Moreover, it is often a visible marker of other underlying risk factors that can be addressed. Thus, the overweight or obese patient deserves major clinical attention. The growing prevalence of this condition threatens to undermine all of our recent gains to prevent and control chronic disease.

### Writing Group Disclosures

Writing Group Member	Employment	Other					
		Research Grant	Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit.

## References

1. Flegal KM, Carroll MD, Ogden CL, Johnson CL: Prevalence and trends in obesity among US adults, 1999–2000. *JAMA* 288:1723–1727, 2002
2. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP: The continuing epidemics of obesity and diabetes in the United States. *JAMA* 286:1195–1200, 2001
3. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, Hayflick L, Butler RN, Allison DB, Ludwig DS: A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med* 352:1138–1145, 2005
4. Haslam DW, James WPT: Obesity. *Lancet* 366:1197–1209, 2005
5. Jemal A, Ward E, Hau Y, Thun M: Trends in the leading causes of death in the United States, 1970–2002. *JAMA* 294:1255–1259, 2005
6. Mokdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, Marks JS: Diabetes trends in the U.S.: 1990–1998. *Diabetes Care* 23:1278–1283, 2000
7. Ford ES, Williamson DF, Liu S: Weight change and diabetes incidence: findings from a national cohort of US adults. *Am J Epidemiol* 146:214–222, 1997
8. Koh-Banerjee P, Wang Y, Hu FB, Spiegelman D, Willett WC, Rimm EB: Changes in body weight and body fat distribution as risk factors for clinical diabetes in US men. *Am J Epidemiol* 159:1150–1159, 2004
9. Engelgau MM, Geiss LS, Saaddine JB, Boyle JP, Benjamin SM, Gregg EW, Tierney EF, Rios-Burrows N, Mokdad AH, Ford ES, Imperatore G, Narayan KM: The evolving diabetes burden in the United States. *Ann Intern Med* 140:945–950, 2004
10. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH: The disease burden associated with overweight and obesity. *JAMA* 282:1523–1529, 1999
11. Haffner SM, Lehto S, Ronnema T, Pyorala K, Laakso M: Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med* 339:229–234, 1998
12. Hu FB, Stampfer MJ, Solomon CG, Liu S, Willett WC, Speizer FE, Nathan DM, Manson JE: The impact of diabetes mellitus on mortality from all causes and coronary heart disease in women: 20 years of follow-up. *Arch Intern Med* 161:1717–1723, 2001
13. Fox CS, Coady S, Sorlie PD, Levy D, Meigs JB, D'Agostino RB Sr, Wilson PW, Savage PJ: Trends in cardiovascular complications of diabetes. *JAMA* 292:2495–2499, 2004
14. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS: Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA* 289:76–79, 2003
15. Jonsson S, Hedblad B, Engstrom G, Nilsson P, Berglund G, Janzon L: Influence of obesity on cardiovascular risk: twenty-three-year follow-up of 22,025 men from an urban Swedish population. *Int J Obes Relat Metab Disord* 8:1046–1053, 2002
16. Schulte H, Cullen P, Assmann G: Obesity, mortality and cardiovascular disease in the Munster Heart Study (PROCAM). *Atherosclerosis* 144:199–209, 1999
17. Thomas F, Bean K, Pannier B, Oppert JM, Guize L, Benetos A: Cardiovascular mortality in overweight subjects: the key role of associated risk factors. *Hypertension* 46:654–659, 2005
18. Haffner SM, Stern MP, Hazuda HP, Mitchell BD, Patterson JK: Cardiovascular risk factors in confirmed prediabetic individuals: does the clock for coronary heart disease start ticking before the onset of clinical diabetes? *JAMA* 263:2893–2898, 1990
19. Coutinho M, Gerstein HC, Wang Y, Yusuf S: The relationship between glucose and incident cardiovascular events: a metaregression analysis of published data from 20 studies of 95,783 individuals followed for 12.4 years. *Diabetes Care* 22:233–240, 1999
20. The DECODE Study Group, the European Diabetes Epidemiology Group: Glucose tolerance and cardiovascular mortality: comparison of fasting and 2-hour diagnostic criteria. *Arch Intern Med* 161:397–405, 2001
21. Meigs JB, Nathan DM, D'Agostino RB Sr, Wilson PW: Fasting and post-challenge glycemia and cardiovascular disease risk: the Framingham Offspring Study. *Diabetes Care* 25:1845–1850, 2002
22. Brunner EJ, Shipley MJ, Witte DR, Fuller JH, Marmot MG: Relation between blood glucose and coronary mortality over 33 years in the Whitehall Study. *Diabetes Care* 29:26–31, 2006
23. Carr DB, Utschneider KM, Hull RL, Kodama K, Retzlaff BM, Brunzell JD, Shofer JB, Fish BE, Knopp KH, Kahn SE: Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. *Diabetes* 53:2087–2094, 2004
24. Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM, Anand SS, the INTERHEART Study Investigators: Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 366:1640–1649, 2005
25. Boden G, Laakso M: Lipids and glucose in type 2 diabetes: what is the cause and effect? *Diabetes Care* 27:2253–2259, 2004
26. Savage DB, Petersen KF, Shulman GI: Mechanisms of insulin resistance in humans and possible links with inflammation. *Hypertension* 45:828–833, 2005
27. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer FX, Eckel RH, the American Heart Association, the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism: Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 113:898–918, 2006
28. Reaven GM: Banting Lecture 1988: Role of insulin resistance in human disease. *Diabetes* 37:1595–1607, 1988
29. 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* 285:2486–2497, 2001
30. World Health Organization: *Definition, Diagnosis, and Classification of Diabetes Mellitus and its Complications: Report of a WHO Consultation*. Geneva, World Health Org., 1999
31. Eckel RH, Grundy SM, Zimmet PZ: The metabolic syndrome. *Lancet* 365:1415–1428, 2005
32. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C, the American Heart Association, the National Heart, Lung, and Blood Institute: Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation* 109:433–438, 2004
33. Alberti KGMM, Zimmet P, Shaw J, the IDF Epidemiology Task Force Consensus Group: The metabolic syndrome: a new worldwide definition. *Lancet* 366:1059–1062, 2005
34. Service FJ: Mechanisms of metabolic mischief: meritorious or meretricious? *Endocr Pract* 9:101–102, 2003
35. Stern MP, Williams K, Gonzalez-Villalpando C, Hunt KJ, Haffner SM: Does the metabolic syndrome improve identification of individuals at risk of type 2 diabetes and/or cardiovascular disease? *Diabetes Care* 27:2676–2681, 2004 [erratum in *Diabetes Care* 28:238, 2005]
36. Reaven GM: The metabolic syndrome: requiescat in pace. *Clin Chem* 51:931–938, 2005
37. Greenland P: Critical questions about the metabolic syndrome. *Circulation* 112:3675–6, 2005
38. Kahn R, Buse J, Ferrannini E, Stern M: The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care* 28:2289–2304, 2005
39. Mitka M: Does the metabolic syndrome really exist? Diabetes and heart disease groups spar over issue. *JAMA* 294:2010–2013, 2005
40. Frantz S: Groups question existence of metabolic syndrome. *Nat Rev Drug Discov* 4:796–797, 2005
41. Gregg EW, Cheng YJ, Cadwell BL, Imperatore G, Williams DE, Flegal KM, Narayan KM, Williamson DF: Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *JAMA* 293:1868–1874, 2005 [erratum in *JAMA* 294:182, 2005]
42. Nesto RW, Bell D, Bonow RO, Fonseca V, Grundy SM, Horton ES, Le Winter M, Porte D, Semenkovich CF, Smith S, Young LH, Kahn R: Thiazolidinedione use, fluid retention, and congestive heart failure: a consensus statement from the American Heart Association and American Diabetes Association. *Diabetes Care* 27:256–263, 2004
43. Eyre H, Kahn R, Robertson RM, the ACS/ADA/AHA Collaborative Writing Committee: Preventing cancer, cardiovascular disease, and diabetes: a common agenda for the American Cancer Society, the American Diabetes Association, and the American Heart Association. *Diabetes Care* 27:1812–1824, 2004
44. Saydah S, Fradkin J, Cowie C: Poor control of risk factors for vascular disease among adults with previously diagnosed diabetes. *JAMA* 291:335–342, 2004
45. Bhatt DL, Steg PG, Ohman EM, Hirsch AT, Ikeda Y, Mas JL, Goto S, Liao CS, Richard AJ, Rother J, Wilson PW, the REACH Registry Investigators: International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. *JAMA* 295:180–189, 2006
46. Mosca L, Linfante AH, Benjamin EJ, Berra K, Hayes SN, Walsh BW, Fabunmi RP, Kwan J, Mills T, Simpson SL: National study of physician awareness and adherence to cardiovascular disease prevention guidelines. *Circulation* 111:499–510, 2005
47. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB: Prediction of coronary heart disease using risk factor categories. *Circulation* 97:1837–1847, 1998

48. Stevens RJ, Kothari V, Adler AI, Stratton IM, the United Kingdom Prospective Diabetes Study (UKPDS) Group: The UKPDS risk engine: a model for the risk of coronary heart disease in type II diabetes (UKPDS 56). *Clin Sci (Lond)* 101:671–679, 2001 [erratum in *Clin Sci (Lond)* 102:679, 2002]
49. Assmann G, Cullen P, Schulte H: Simple scoring scheme for calculating the risk of acute coronary events based on the 10-year follow-up of the prospective cardiovascular Munster (PROCAM) study. *Circulation* 105:310–315, 2002 [erratum in *Circulation* 105:900, 2002]
50. Eberly LE, Prineas R, Cohen JD, Vazquez G, Zhi X, Neaton JD, Kuller LH, the Multiple Risk Factor Intervention Trial Research Group: Metabolic syndrome: risk factor distribution and 18-year mortality in the Multiple Risk Factor Intervention Trial. *Diabetes Care* 29:123–130, 2006
51. Wilson PW, D'Agostino RB, Parise H, Sullivan L, Meigs JB: Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. *Circulation* 112:3066–3072, 2005
52. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukkaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M, the Finnish Diabetes Prevention Study Group: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001
53. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM, the Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002
54. Wadden TA, Berkowitz RI, Womble LG, Sarwer DB, Phelan S, Cato RK, Hesson LA, Osei SY, Kaplan R, Stunkard AJ: Randomized trial of lifestyle modification and pharmacotherapy for obesity. *N Engl J Med* 353:2111–2120, 2005
55. National Institutes of Health: Clinical guidelines on the identification, evaluation, treatment of overweight and obesity in adults: the evidence report. *Obes Res* 6 (Suppl. 2):51S–209S, 1998
56. US Department of Health and Human Services: *Physical Activity and Health: A Report of the Surgeon General*. Atlanta, GA, National Centers for Disease Control and Prevention, 1996
57. Lakka TA, Laaksonen DE, Lakka HM, Mannikko N, Niskanen LK, Rauramaa R, Salonen JT: Sedentary lifestyle, poor cardiorespiratory fitness, and the metabolic syndrome. *Med Sci Sports Exerc* 35:1279–1286, 2003