Published in final edited form as:

J Natl Med Assoc. 2008 May ; 100(5): 565–571.

The Relative Risk of Cardiovascular Death among Racial and Ethnic Minorities with Metabolic Syndrome: Data from the NHANES-II Mortality Follow-Up

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Abstract

The tendency for selected cardiovascular disease (CVD) risk factors to occur in clusters has led to the description of metabolic syndrome (MetS). The relative impact of the individual risk factor on the overall relative risk (RR) for cardiovascular death from metabolic syndrome is not well established and may differ across the different racial/ethnic groups.

Using data from the National Health and Nutrition Examination Survey (NHANES II) mortality follow-up (NH2MS), we determined the prevalence and RR of cardiovascular death for individual components in the overall population and across racial and ethnic groups.

The prevalence of MetS components varied significantly across gender and racial/ethnic groupings. The RR for CVD also varies for the number and different components of MetS. The adjusted RR for cardiovascular death was highest with diabetes (3.23; 95% CI: 2.70–3.88), elevated blood pressure (2.28; 95% CI: 1.94–2.67) and high triglycerides (1.63; 95% CI: 1.34–2.00). Although the RR for cardiovascular death differs significantly for some of the different components, the overall findings were similar across racial/ethnic groups. The two components that confer the highest risks for death are more prevalent in African Americans.

We concluded that the RR of cardiovascular death associated with the diagnosis of MetS varies depending on the number and components used to establish the diagnosis of MetS and the racial/ethnic characteristic of the participants.

Keywords

metabolic syndrome; cardiovascular; minority health; race/ethnicity

BACKGROUND AND SIGNIFICANCE

Cardiovascular disease (CVD) is a major cause of mortality and morbidity in the United States.¹ Hypertension and diabetes are among the leading risk factors for CVD. African Americans are known to exhibit a greater prevalence and severity of hypertension and diabetes compared to white Americans². The common co-occurrence of select CVD risk factors, including hypertension and diabetes, has led to the description of metabolic syndrome (MetS) in anticipation of a unifying pathogenesis and clinical prediction of CVD.^{3,4} Charts and programs based on the Framingham data are currently being used to estimate cardiovascular disease risk in the clinics. The Framingham Study population was drawn from a single location with mainly white residents (Framingham, MA) and may not be representative of the U.S. adult population⁵. The clinical value of MetS for long-term prediction of CVD is modest at best at the moment. However, the predictive and prognostic implications of a disproportionate distribution of any of the components of the syndrome will be significant especially for the African-American population. The isolation of the cardiovascular risk of the syndrome from that of its individual components will be important for subpopulations with disproportionate burden of the components of the syndrome. Currently, CVD risk attributable to individual components comprising the diagnosis of MetS is not clear. A recent review of existing prospective data concludes CVD risk associated with the diagnosis of MetS varies with the components employed in the diagnosis of the syndrome, and the diagnosis of MetS itself only modestly predicts [relative risk (RR)=1.65–1.93] CVD.^{6,7}

The overall age-adjusted prevalence of MetS in the United States is about 24% but varies across different gender and racial/ethnic groupings.^{8,9} The implication of these differences for the risk of CVD and the prognosis of the diagnosis of MetS is unknown. It is important in clinical practice to accurately identify high-risk patients for cardiovascular risk-reduction interventions.

The purpose of this study was to determine the RR of cardiovascular death for the different components and combinations of components that establish the diagnosis of MetS in the overall population and across racial and ethnic groups.

MATERIALS AND METHODS

Study Design

This study utilizes data from the Second National Health and Nutrition Examination Survey Mortality Study (NH2MS). The NH2MS is a prospective cohort study design which passively followed a subset of participants from the Second National Health and Nutrition Examination Survey (NHANES II).

NHANES II was a national probability survey conducted by the National Center for Health Statistics from February 1976 to February 1980. It included 27,801 non institutionalized people who were U.S. civilians between the ages of 6 months and 74 years; institutionalized individuals were excluded from the sample. Of the nearly 28,000 individuals identified for

the NHANES-II survey, 20,322 (73%) subjects participated in interviews, physical examinations and laboratory assessments.¹⁰

NH2MS was designed to investigate the association between factors measured at baseline during NHANES-II assessments and subsequent overall mortality or death from a specific cause. For this purpose, original participants of NHANES II who were ≥ 30 years of age at the time of their baseline examination were identified.

The status of subjects in the NH2MS follow-up was determined by a passive study design from 1979–1992; there was no attempt to recontact participants. Instead, a search was undertaken of two comprehensive computerized databases containing the dates and causes of death for all known deaths in the United States. The databases used were the National Death Index (NDI) compiled by the National Center for Health Statistics (NCHS); and the Social Security Administration (SSA) Death Master File, which pools data from different SSA files, including the SSA Master Beneficiary Record. Since the information in these databases is thought to represent a complete record of U.S. deaths, individuals from the NH2MS cohort who were not found in either the NDI or the SSA Death Master File were assumed to still be alive at the time the follow-up was undertaken in 1992. Using these validated national mortality records for the years 1979 (the end of the NHANES II survey) through 1992 (follow-up period of 12–16 years), NH2MS mortality data can be linked with the earlier baseline NHANES II data to examine the relationships between various factors and specific causes of death.¹¹

For the purposes of our study, we examined the RR of cardiovascular death for the individual components and combinations of components that establish the diagnosis of MetS across racial and ethnic groupings through a detailed analysis of publicly available NH2MS datasets.

Study Sample Population

Our study used the interview, examination and laboratory data from 9,066 adult participants aged 30–75 years, with available data for the assessment of MetS. The process by which the analysis sample was extracted from the available data is depicted in Figure 1. The NH2MS cohort at baseline search was composed of 12,102 adults aged 30–74 years who were interviewed (from NHANES II). Seventy-six percent (9,250) of the participants interviewed completed a physical examination.

Age at examination was used to identify those participants aged ≥ 30 for inclusion, with 75 years as the upper age limit for selection into the cohort. Because several weeks could have elapsed between the interview and examination, some participants who were 74 years of age during their interview (from NHANES II) turned 75 years of age between their interview and examination. Thus, the upper age limit for the NH2MS cohort selection was 75 years.

Racial/ethnic grouping for the purpose of our study was by self-identification as white and African American. The participants that self-identified as Hispanics and others were excluded from our analysis due to low sample size. The participants of the original NHANES II that were interviewed but not examined were excluded from the cohort. Also,

excluded were those <30 or >75 years and those with incomplete laboratory data. The participants that had incomplete personal identifying data from the original 9,252 participants were considered lost to follow-up. Thus, our final analysis was based the 9,066 participants with MetS and the 903 established cardiovascular deaths, 42.1% of all-cause mortality in the original NH2MS participants (2,145).

Study Variables

Our study employed the modified National Cholesterol Education Program expert panel on detection, evaluation and treatment of high blood cholesterol in adults (NCEP-ATP III)¹² and the World Health Organization (WHO) definitions of the metabolic syndrome¹³ (Table 1). We substituted body mass index (BMI) >30 kg/m² from the WHO definition for abdominal circumference in the (NCEP-ATP -III) definition of metabolic syndrome because abdominal circumference was not measured in the NHANES-II cohort. We defined MetS as the presence of any three of the following components: elevated blood pressure, elevated blood sugar, obesity, hypertriglyceridemia and low levels of high-density lipoprotein cholesterol (HDL-C).

Elevated blood sugar status was based on interview questions. Participants who reported having ever been told by a physician that they have diabetes mellitus, sugar diabetes, fasting blood sugar >110 mg/dl (6.1 mmol/L), or who had use of antidiabetic medication were classified as having elevated blood sugar. Serum glucose concentration was measured using an enzymatic reaction (Cobas Mitra assay; Roche, Basel, Switzerland).

Elevated blood pressure was defined by a systolic blood pressure of ≥135 mmHg and/or a diastolic blood pressure of ≥85 mmHg. Participants who reported use of antihypertensive medication or were told by a physician they have hypertension were also classified as participants with elevated blood pressure.

BMI was calculated as weight in kilograms divided by the square of height in meters. Obesity was defined as a BMI of ≥30 kg/m².

Serum triglyceride was measured enzymatically after hydrolysis (Hitachi 704 Analyzer; Hitachi, Tokyo, Japan). Hypertriglyceridemia was defined as serum triglyceride level ≥150 mg/dl (1.69 mmol/L).

HDL-C was measured following the precipitation of other lipoproteins with heparin manganese chloride mixture (Hitachi 704 Analyzer) at the Lipoprotein Analytical Laboratory at Johns Hopkins Hospital, Baltimore, MD, which is certified by the Lipid Standardization Program of the Centers for Disease Control and Prevention. A low level of HDL-C was defined as: HDL-C <40 mg/dl (1.04 mmol/L) in men and <50 mg/dl (1.29 mmol/L) in women.

Statistical Analysis

Demographic variables and the components of MetS were expressed by frequency counts and percentages. Tests for normality of distribution were not performed because all the

variables were categorical variables. Comparisons of categorical variables of the two groups were analyzed by Chi-squared test. A p value <0.05 was considered statistically significant.

Logistic regression analysis was used to determine the odds ratio (OR) of CVD death associated with the components of MetS for white and black groups as well as overall analysis sample, adjusting for age and gender. Adjusted ORs are significant if 95% confidence intervals (CIs) do not cross 1.0. To account for the complex sampling design of the NHANES II, all estimates presented here have been adjusted using weights provided by the NCHS.

The RR of cardiovascular death from MetS was adjusted for established CVD risk factors not included in the definition of MetS such as smoking and hyper-cholesterolemia. All analyses samples were weighted so that the prevalence estimates represented the noninstitutionalized civilian U.S. population aged 30–75 years. All data for this study were analyzed using the Statistical Analysis System® (version 8.0, 2000. SAS Institute, Cary, NC). We employed the SUDAAN version 8.0 software to adjust for the multistaged cluster sampling design and the oversampling of the elderly and minority participants in NHANES II.¹⁴

RESULTS

The demographic characteristics of the analysis sample and the distribution of the components of MetS in the analysis sample are as shown in Table 2. The analysis sample is mostly young (<65 years) and white with an even gender distribution. Elevated blood pressure was the most prevalent component of the MetS (60.2%), while elevated blood sugar was the lowest (9.5%).

The age- and gender-adjusted prevalence of the different components of MetS in the overall sample (Figure 2) and across racial/ethnic grouping is as shown in Table 3. The prevalence of elevated blood sugar, obesity and elevated blood pressure was higher among the black than white participants. The prevalence of low HDL-C and high triglycerides were higher among the white than black participants. Black females had a higher rate of diabetes and obesity than white females. The age and gender adjusted RR of cardiovascular death associated with the components of MetS in the overall sample and across racial/ethnic grouping is as shown in Table 4. Diabetes has the highest RR for CVD (3.23; 95% CI: 2.70–3.88), while obesity has the lowest RR for CVD (1.14; 95% CI: 0.94–1.38). There was a significant dose-response relationship between the number of components of MetS and the risk of CVD death in the overall analysis sample (Table 5). There were no sufficient cardiovascular deaths to accurately assess the results of the combined analyses using a multistage computational model with the 10 possible combinations of the ATP-III components needed for the clinical diagnosis of metabolic syndrome.

DISCUSSION

We found that the RR of cardiovascular death associated with the diagnosis of MetS varied with the individual components used to establish the diagnosis of the syndrome and the racial/ethnic characteristics of the participants. Many of the components of MetS are also

established cardiovascular risk factors, and the clinical challenge has always been the establishment of the additional risk posed by the diagnosis of metabolic syndrome above and beyond the sum of the cardiovascular risk factors employed in its diagnosis. The diagnosis of MetS is usually based on the clustering of these metabolic abnormalities more often than would be predicted by chance,¹⁵ but considerable debate exists about the independence of the RR of CVD and death associated with the diagnosis of MetS.^{16–19} The findings from our study are consistent with the existing body of literature on the age, gender and racial/ethnic differences in the prevalence of the defining components of MetS.^{20,21} Although the clustering of the metabolic abnormalities in MetS has been traditionally attributed in part to insulin resistance,²² other studies suggest that the prevalence of several of its components can be significantly affected by many psychosocial rather than physiologic factors.²¹ The little or no risk associated with obesity in this study lends support to the view that the CVD risk that is attributable to obesity is mediated through the traditional risk factors such as diabetes and hypertension.^{23–25} This study explored the implications of the racial/ethnic differences in the prevalence of the defining components of MetS for the RR of cardiovascular death among racial/ethnic minorities. Diabetes and hypertension account for most of the RR of cardiovascular death in this study, and both of these conditions are more prevalent in African Americans—highlighting the need for prevention and early treatment in this population.

This study provides valuable insight into the clinical implications of the diagnosis of MetS for a racial/ethnic minority population using the NHANES-II mortality follow-up data. Although NHANES II provided a wealth of information on the prevalence of health conditions and risk factors, there was no attempt to reestablish contact with the participants in the mortality follow-up study. Vital status was established using validated national mortality records. Participants not found to be deceased at time of analysis were assumed to be alive for analytic purposes with a potential for misclassification of vital status. The follow-up period in NH2MS ranged from 12–16 years. An apparent lag time of about 10–15 years has been reported to occur before the mortality curves for men with and without MetS begin to diverge in prospective studies.²⁶ The follow-up period in our study may have been too short for some of the components and combinations of the components of MetS in our study. In addition, we substituted BMI >30 kg/m² from the WHO definition for abdominal circumference in the (NCEP-ATP-III) definition of metabolic syndrome because abdominal circumference was not measured in the NHANES-II cohort. The RR of CVD reported in this study may have been inflated by this substitution

In spite the aforementioned limitations of this study, we believe that our findings represent the primary attempt to assess the implication of the racial/ethnic differences in the prevalence of the defining components of MetS for the RR of cardiovascular death among racial/ethnic minorities. It is our recommendation that the independence of the risk of cardiovascular death associated with the diagnosis of MetS be unequivocally established, particularly among racial/ethnic minority populations prior to the development of national health policies and treatment guidelines that could potentially obscure the need to control the established cardiovascular disease risk factors such as diabetes, hypertension and obesity, which are also defining components of MetS.

Acknowledgments

Financial support: This research was supported in part by RR03026, RR11145 and RR14616 (DM, NT, KN), and RR019234 (DP) from the National Center for the Research Resources, National Institutes of Health (NIH); MD00148 (KN, DM) from the DREW/UCLA Project EXPORT, National Center Minority Health and Health Disparities. Statistical support for this project was in part provided by NIH-NCRR/RCMI, DREW/UCLA Project EXPORT and NIH-NCMHD (MD00148).

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Figure 1.
The schematic representation of the selection of analysis sample

Figure 2. Overall relative risk of cardiovascular disease (CVD) mortality for different components of metabolic syndrome (MS)

Table 1

Definitions of metabolic syndrome

Components	NCEP-ATP-III Definitions ¹	WHO Definitions ²
Impaired glucose metabolism	Fasting plasma glucose >110 mg/dl (6.1 mmol/L)	Glucose intolerance, impaired fasting glucose, insulin resistance or diabetes mellitus
Hypertension	Blood pressure >130/85 mmHg or treatment for hypertension	Blood pressure >140/90 mmHg or treatment for hypertension
Dyslipidemia	Serum triglycerides ≥150 mg/dl (1.69 mmol/L) and HDL-C <40 mg/dl (1.04 mmol/L) in men and <50 mg/dl (1.29 mmol/L) in women.	Serum Triglyceride level ≥150 mg/dl (1.7 mmol/L) HDL-C <40 mg/dl (0.91 mmol/L).
Central obesity	Waist circumference >102 cm in men and >88 cm in women	Waist-to-hip ratio >0.9 or body mass index >30 kg/m ²
Target organ damage	–	Microalbuminuria: urinary albumin excretion rate >20 µg/min

Table 2

Characteristics of the Analysis Sample and the Distribution of the Study Variables among 8,099 participants
30 years old followed in NH2MS[†]

Variables	N	(%)
Age (Years)		
<65	6475	71.4
65	2591	28.6
Gender		
Male	4264	47.0
Female	4802	53.0
Race		
White	8070	89.0
Black	996	11.0
Obesity ¹	1370	15.2
Diabetes ²	821	9.1
Hypertension ³	5169	57.4
Low HDL-C ⁴	3074	41.4
Triglyceride ⁵	1647	37.2
CVD Death	854	9.4

¹Body mass index >30 (justified use—WHO guideline);

²Fasting blood sugar >110 mg/dl or had diabetes mellitus;

³Blood pressure medication or blood pressure level >130/>85;

⁴HDL cholesterol <40 mg/dl in men, <50 mg/dl in women;

⁵Triglyceride level >150 mg/dl

Prevalence of the different components of metabolic syndrome by race and gender among 8,099 participants 30 years old followed in NH2MS

Table 3

Variables	White			Black		
	Males	Females	Total	Males	Females	Total
	(N=3815)	(N=4255)	(N=8070)	(N=449)	(N=547)	(N=996)
Age >65 years	27.6 %	29.5 %	28.6	29.2 %	27.8 %	28.4
Obesity	11.3 %	16.4 %	14.0	18.6 %	31.2 %	25.5
Diabetes	8.5 %	8.8 %	8.7	12.0 %	12.6 %	12.4
Hypertension	61.0 %	51.9 %	56.2	67.0 %	66.7 %	66.8
Low HDL-C	40.8 %	44.7 %	42.9	20.1 %	34.6 %	28.3
High triglycerides	43.4 %	34.4 %	38.7	27.4 %	20.7 %	23.8

1: Body mass index >30 (justified use—WHO guideline); 2: Fasting blood sugar >110 mg/dl or had diabetes mellitus; 3: Blood pressure medication or blood pressure level >130/>85; 4: HDL cholesterol <40 mg/dl in men, <50 mg/dl in women; 5: Triglyceride level >150 mg/dl

Table 4

Components of metabolic syndrome and their associated relative risk of CVD death across racial/ethnic groups and in the overall analysis sample*

Different Components of Metabolic Syndrome	White RR (95% CI)	Black RR (95% CI)	Relative Risk (95% CI)¶
Diabetes *	3.1 (2.6–3.8)	4.0 (2.5–6.5)	3.23 (2.70–3.88)
Hypertension *	2.2 (1.9–2.6)	3.0 (1.7–5.2)	2.28 (1.94–2.67)
Obesity	1.1 (0.9–1.4)	1.3 (0.8–2.1)	1.14 (0.94–1.38)
Low HDL	1.2 (1.0–1.4)	1.2 (0.7–2.1)	1.20 (1.01–1.41)
High triglycerides *	1.6 (1.3–2.0)	2.0 (1.1–3.7)	1.63 (1.34–2.00)

CI: confidence interval; RR, relative risk;

* Statistically significant

Table 5

Dose-response relationship of the number of components of metabolic syndrome and the risk of CVD death in the overall analysis sample

Number of Components of Metabolic Syndrome	CVD Death Rate
0	4.67% (3.76–5.58)
1	8.81% (7.88–9.74)
2	11.42% (10.10–12.74)
3	15.73% (12.87–17.59)
4	15.82% (11.67–19.97)
5	18.42% (6.09–30.75)

The p value for linear trend is <0.0001